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CLINICAL SCIENCE OF NUTRITION
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Research Article
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Assessment of educational gaps and training needs regarding oral nutritional supplements among community pharmacists

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ABSTRACT

Objective: Pharmacists play a key role in managing malnutrition, yet their effectiveness depends on adequate training. This study evaluated community pharmacists' knowledge of oral nutritional supplements (ONS), focusing on administration and storage, and identified educational gaps.

Methods: A cross-sectional study was conducted using a 23-item survey to collect demographic data and assess ONS-related knowledge.

Results: Most pharmacists (78%,) reported no undergraduate or postgraduate education on ONS, and only 17% considered their knowledge sufficient. Based on 12 knowledge questions, the median score was 8.0 (IQR:3.0). Pharmacists who had received ONS education achieved higher median scores than those without training. Participants were generally more knowledgeable about storage conditions for unopened products, while significant deficiencies were observed regarding the stability and storage of opened ONS products.

Conclusion: The findings reveal substantial educational gaps and underscore the need to integrate structured nutrition education into pharmacy curricula and continuing education to improve counselling practices, patient adherence, and overall nutritional treatment outcomes.

Keywords: community pharmacist, oral nutritional supplements, malnutrition, education

Introduction

Malnutrition is a frequently observed serious health issue, and it might hinder the treatment process, causing some negative results such as prolonged recovery or increased risk of complications or infections.[1] Individuals at risk of malnutrition include patients with chronic diseases like cancer, those with neurological or psychiatric conditions, people with swallowing difficulties, and socially disadvantaged groups. Malnutrition might affect individuals at all ages; advancing age is often associated with an increased risk of malnutrition.[2] Older adults are more vulnerable to malnutrition due to reduction in food intake resulting from acute and chronic diseases, which are common among older individuals.[3] Evaluating patients' nutritional status and providing appropriate nutritional therapies is crucial for the effectiveness of the entire treatment process.[4] Patients with or at risk of

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malnutrition should be given oral nutritional supplements (ONS) when considered necessary, both during their hospital stay and after the discharge.[3] However, ONSs are frequently prescribed inappropriately or used incorrectly, and community adherence to nutritional therapy is suboptimal, partly because of insufficient coordination and communication between healthcare professionals and community pharmacists or their lack of knowledge.[5,6]

Although ONSs are commonly utilized in clinical practice, they are not regulated with the same level of care as medications. Numerous steps, like prescription, patient delivery, storage conditions, and usage instructions—many steps suffer from information gaps.[7-9] One of the often overlooked but essential factors is the storage conditions. Stability of nutrition products is critical to ensure that the products are used safely and effectively. The stability and safety of these products are guaranteed throughout their shelf life under the presence of appropriate storage conditions.[10] When stored in inappropriate conditions, not only does their nutritional value decrease, but also the risk of microbial contamination increases. Consequently, patients are unable to obtain all the necessary nutrients.[11] Pharmacists play a critical role in ensuring the proper storage of ONSs and counselling patients with accurate information. However, there is insufficient evidence regarding community pharmacists' knowledge of the storage conditions and stability of ONSs.[11,12]

Although the greater involvement of pharmacists is increasingly emphasized in the field, a limited number of studies have been conducted to evaluate community pharmacists' level of knowledge about ONS usage and storage conditions.

Main Points

- Most community pharmacists reported receiving no formal education on oral nutritional supplements (ONS).
- Educational interventions should prioritize the management and storage of opened ONS products, where the greatest knowledge gaps were identified.
- Improving nutrition education in pharmacy curricula and continuing education programs may enhance pharmacists' counselling practices.

Material and Methods

A prospective observational study of community pharmacists using an online questionnaire from December 1, 2024, to February 1, 2025. The online questionnaire was disseminated via email to the community pharmacists who were registered through a continuing education platform, and participants who did not respond, were unreachable, or had missing information were excluded.

The 23-question questionnaire collected sociodemographic data, ONS storage conditions knowledge, practical experiences, challenges, and willingness to receive ONS education. The research team developed the questionnaire items for this study, drawing on previous studies and adapting them to the specific context of this research.[2,6,10,12] The draft questionnaire was reviewed by experts (3 nurses, 2 physicians, 2 clinical pharmacists, 2 dietaticians) in the fields of clinical nutrition and pharmacy practice, and their feedback was incorporated prior to finalization. A pilot study was conducted with a small group of pharmacists who were not included in the main study, and minor revisions were made to the questionnaire based on their responses to improve clarity and comprehensibility.

The online survey was distributed to all pharmacists (n = 1,080) registered on the continuing education platform

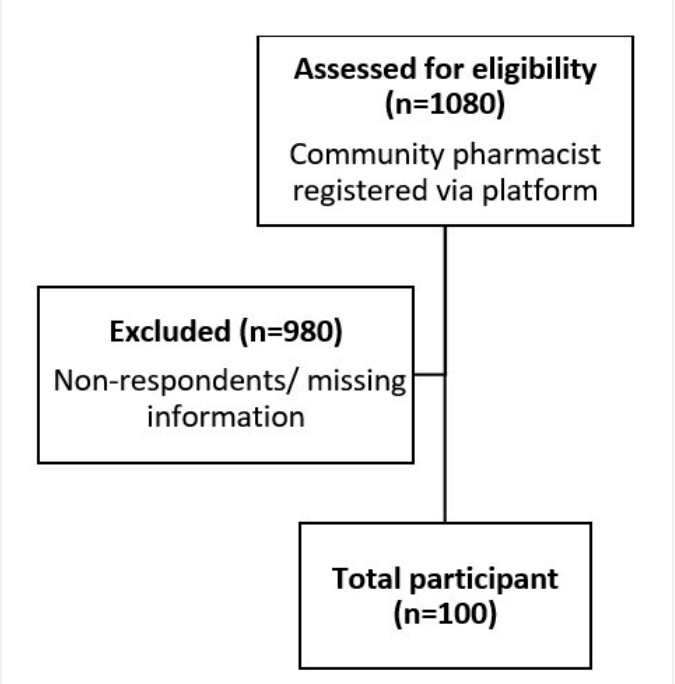

Figure 1. Participant flow chart

widely used by community pharmacists in Türkiye at the time of the study via e-mail. This online continuing education platform regularly provides pharmacy-related training, with the vast majority of its members consisting of community pharmacists. Participants who did not respond, or had missing information had exluded (n=980). A total of 100 community pharmacists (~9.3%) participated in the study, participation was entirely voluntary (Figure 1).

Statistical analyses were conducted utilizing SPSS version 27.0 (IBM Corp., Armonk, NY). The Kolmogorov-Smirnov test was employed to evaluate the normality of continuous variables, revealing a significant deviation from normal distribution ($p < 0.05$). Consequently, nonparametric tests were employed for comparisons. The Mann-Whitney U test was utilized for comparisons between two independent groups, while the Kruskal-Wallis test was employed for comparisons involving multiple groups. A p-value below 0.05 was deemed statistically significant.

Results

A total of 100 community pharmacists participated in the study. The median age was 36.5 years (min-max: 24-65), and 22% of pharmacists had a postgraduate degree (Table 1). Participants' pharmacy locations varied, with 44.0% working in local pharmacies, 30.0% in downtown settings, and 26.0% close to hospital pharmacies. However, neither knowledge scores nor survey responses showed a statistically significant difference based on pharmacy location.

While 78% (n=78) of pharmacists indicated that they had not received any education on ONSs previously, only 17% (n=17) reported they had sufficient knowledge about ONSs. Almost half of the pharmacists (53%) conveyed storing ONSs in a proper area (avoid direct sunlight, humidity, etc.). Forty-three percent of the pharmacists reported that they were storing ONS in the area left by the person who transported it from the warehouse. In other words, they stated that they did not determine a specific area.

As for ONS prescriptions, the participant pharmacists most frequently provided information to patients or caregivers regarding usage (85%), storage conditions (65%), and prescription details (52%). A majority of pharmacists (n=67,67%) reported being unaware of the disposal method for expired ONSs. It was found that the most frequently consulted sources of information about ONS storage conditions were drug databases (n=86,86%) and product labels/packaging (n=69,69%). Furthermore, the majority of pharmacists (n=94,94%) expressed an interest in receiving education on the use of ONSs. Specifically, they indicated a need for education on general information about ONSs (89%), their use in different disease stages (84%), and their storage conditions (58%). The responses provided by pharmacists regarding ONSs are shown in Table 2.

Regarding the sources used by pharmacists to obtain information about ONS storage conditions, the findings revealed that the majority of participants relied primarily on drug databases (86.0%) and product labels and package inserts (69.0%), while the use of evidence-based resources such as clinical guidelines was notably limited (16.0%). Internet resources were used by only 26.0% of participants, and seminars or professional training events were reported as an information source by merely 9.0% of respondents.

Most participants demonstrated awareness of appropriate storage requirements for unopened products, such as keeping them in a cool and dark environment (83%), away from heat and humidity (83%) and sunlight (79%), and ensuring that the cap is tightly closed (70%).

Table 1. Demographic Characteristics of Pharmacists

Category	**n**	**%**
Gender		
Female	69	69.0
Male	31	31.0
Education status		
Undergraduate	78	78.0
Postgraduate	22	22.0
Location of the pharmacy		
Local pharmacy (Far from city center)	44	44.0
Downtown pharmacy (Located in the city center, shopping mall, or street)	30	30.0
Nearby a hospital	26	26.0
	Median	**Min-Max**
Age (years)	36.5	24-65
Professional experience (years)	10.0	1-42

Table 2. Pharmacists' responses to questions regarding ONSs

Questions	n	%
Have you received any education on ONSs previously?		
Yes	22	22.0
No	78	78.0
Do you think that you have adequate information on ONSs?		
Yes	17	17.0
Partially	72	72.0
No	11	11.0
Do you have anyone around you who uses ONS?		
Yes	60	60.0
No	40	40.0
How often do you dispense ONSs at your pharmacy?		
Always	3	3.0
Frequently	26	26.0
Sometimes	44	44.0
Rarely	25	25.0
Never	2	2.0
Where do you store ONS products until you dispense them to the patients?		
I store them in a proper area.	53	53.0
I don't determine in a specific area.	43	43.0
ONS products are not sold or dispensed	4	4.0
Which information do you provide to patients and caregivers about ONSs?		
Usage	85	85.0
Side effect	20	20.0
Storage conditions	65	65.0
Expiration date	40	40.0
Payment terms	41	41.0
Prescription information	52	52.0
I do not provide any information	7	7.0
Which procedure do you use for the disposal of expired ONSs?		
Responses categorized as not disposed properly	16	16.0
Throwing it the trash	5	5.0
Pouring it into toilet/sink	5	5.0
Asking patients to dispose on their own	6	6.0
Responses categorized as disposed properly	17	17.0
Delivering it to the company	11	11.0
Delivering it to the warehouse	6	6.0

Table 2. Continued

Questions	n	%
I don't know/I haven't met.	67	67.0
Do you get feedback from patients about the use of ONS? Do you follow these patients?		
Yes	60	60.0
No	40	40.0
Which sources do you use to find information about the storage conditions of ONS products?		
Product label/packaging	69	69.0
Drug databases	86	86.0
Guidelines	16	16.0
Seminars	9	9.0
Internet resources	26	26.0
Would you like to take a course on ONS?		
Yes	94	94.0
No	6	6.0
What subjects would you like to learn from a course specifically on ONSs?		
General information	89	89.0
New ONS product	58	58.0
ONS use in different diseases	85	85.0
Storage conditions of ONS products	65	65.0
Other	5	5.0

In addition, 76% provided the correct answer for the recommended storage temperature for unopened ONSs. In contrast, only 54% and 31% of pharmacists accurately responded to the storage duration of opened products in a refrigerator or at room temperature, respectively, while knowledge regarding powdered ONS products was lower, with only 37% providing the correct response. Regarding ONS usage, correct responses were highest for not consuming the product when it is too hot (80%) or too cold (55%), not mixing it with food or medications (77%), and not diluting it (46%).

A knowledge score was calculated for each participant based on their correct responses to 12 questions assessing ONS-related knowledge. One point was awarded for each correct answer, the total score reflecting overall knowledge. Among the pharmacists, the median knowledge score was 8.0 (IQR:3.0). The knowledge score comprised a total of 12 questions: 5 questions on unopened ONSs (score range:1–5) and 7 questions on opened ONSs (score range:1–7). Despite this difference, independent analysis of the two groups demonstrated equal median and interquartile range (IQR) values [4.0 (IQR: 2.0)].

The difference in knowledge scores between the participant pharmacists with and without an educational background on ONSs was evaluated using the Mann-Whitney U test. The analysis revealed a statistically significant difference between the two groups (p=0.005), with higher median knowledge scores [Median (IQR):9.0(3.25)] for those who received ONS's education when compared to the other group [Median (IQR):8.0(3.0)].

A significant difference in knowledge scores regarding opened ONSs was observed between these two groups (p=0.001, Mann–Whitney U Test). Those with an educational background demonstrated higher median knowledge scores [Median (IQR):5.0(2.0)] compared to those who were not educated on ONSs [Median (IQR):4.0(1.0)], indicating that education was associated with better knowledge on handling opened ONSs.

Additionally, disparities in knowledge scores among the participant pharmacists who fully believed, partially believed, or did not believe they had sufficient knowledge about ONSs were evaluated using the Kruskal–Wallis test. A significant difference was observed (H=8.45, df=2, p=0.015), with median knowledge scores (IQR) of 9.0(3.5) for those who fully believed, 8.0(2.75) for those who partially believed, and 6.0 (5.0) for those who did not believe they had sufficient knowledge. Furthermore, a statistically significant difference was also found in knowledge scores regarding opened ONSs among these groups (H=12.43, df=2, p=0.02), indicating that pharmacists' self-perceived knowledge was associated with their actual knowledge.

Discussion

This study's findings indicate an important gap between the expanding clinical role of pharmacists in malnutrition management and their educational background. While the majority of participants indicated a defiency in confidence and knowledge concerning ONSs, this deficiency is not merely an individual practice gap but a systemic reflection of pharmacy curricula. Our findings correspond with prior research suggesting that healthcare professionals generally lack adequate training on nutritional products.[12,13] Nonetheless, differentiating from previous literature, our data indicates that this knowledge deficit specifically stems from the insufficient coverage of clinical nutrition and products storage conditions in undergraduate programs. A qualitative study in Portugal revealed that although all community pharmacists reported that they counsel their patients on daily intake and timing of administration, only half provided guidance on storage precautions of ONSs.[12] Consistently, although most pharmacists in this study reported to have provided instructions on ONS usage, fewer offered information regarding proper storage conditions, highlighting a persistent gap between pharmacy education and practice.

In chronic disease management, consistent monitoring and follow-up are essential for the assessment of the effectiveness and safety of pharmacotherapy and patients' adherence to treatment. The literature indicates that regular communication between pharmacists and patients along with active follow-up facilitates the identification and resolution of medication-related problems, strengthens the patient–pharmacist relationship, and enhances treatment adherence.[14] In this study, only about half of the pharmacists reported routine follow-ups with patients using ONSs, indicating that pharmacist-patient communication remains limited in the process of using nutritional products. Given the long-term and regular use required for ONSs, a systematic follow-up by pharmacists is crucial. Such a follow-up ensures accurate product use, allows assessment of the effectiveness of different ONS products for each patient separately, and enables pharmacists to provide tailored recommendations based on their clinical experience in addition to the information provided by manufacturers. Implementing structured follow-up mechanisms is likely to enhance both patient outcomes and overall efficacy of nutritional interventions.[7,12]

According to the findings of this study, 43% of pharmacists stated that they do not provide a proper area for storage of ONSs. The information given to patients or caregivers varies, just like the responses to questions regarding storage conditions. These discrepancies in knowledge and practice highlight the critical need for specialized education on ONS storage for pharmacists.[7,12] In addition, 67% of pharmacists reported that they lack information on the proper disposal of expired ONSs, indicating a need for guidance on correct recycling and waste management practices. Given the multiple sources of ONS wastage, pharmacists also play a role in educating patients about proper disposal.[7]

The participating pharmacists demonstrated higher accuracy in responding to inquiries about the storage of unopened ONSs relative to opened products, indicating greater familiarity with pharmacy-related storage practices. The lack of knowledge regarding the handling and use of opened ONS products observed in our study is particularly concerning from a clinical safety perspective. Once a nutritional product package is opened, contamination may occur through air flow, fluid drip, or consumer handling, which may introduce new pathogens or accelerate microbial growth.[15] Comprehensive guidance on appropriate storage throughout the duration of ONS use is, thus, essential to support patient adherence.[6,10]

In clinical practice, some issues in ONS prescribing may arise, potentially including treatment effectiveness and patient adherence.[3] Pharmacists play a central role in addressing these challenges by facilitating communication between patients and physicians, identifying inappropriate use, and promoting adherence. A lack of sufficient expertise or confidence among pharmacists and other healthcare professionals in diagnosing malnutrition and counseling patients

effectively can negatively impact treatment outcomes and adherence, underscoring the important role of pharmacists in managing ONS therapy.[9]

Regarding the sources used by pharmacists to obtain information about ONS storage conditions, the majority of participants relied primarily on drug databases and product labels, while clinical guidelines and peer-reviewed literature were rarely consulted. Notably, seminars were among the least frequently reported sources, likely reflecting the scarcity of structured continuing education programs on ONS-related topics. These findings highlight a critical gap in both information-seeking behavior and the availability of targeted professional development initiatives for community pharmacists.

Undergraduate pharmacy curricula often lack nutrition education, which is offered only through elective courses/extended programs at a limited number of universities.[16] This global gap in nutrition education affects pharmacy, medical, and nursing programs.[17] Undergraduate nutrition education can be advantageous for students who want to work in healthcare professions.

In our study, although pharmacists with postgraduate degrees showed a numerical trend towards higher knowledge scores, the lack of statistical significance suggests that even advanced degrees may not sufficiently cover specific ONS competencies unless focused specifically on clinical nutrition. This underscores a broader need: nutrition education must be integrated not just as theoretical knowledge but as a practical, competency-based component of the core pharmacy curriculum.

To address the identified gaps, particularly in product storage conditions and patient follow-up, pharmacy educators should consider adopting active learning strategies. ONS management should be prioritized in Continuing Professional Development programs to support the current workforce.

This study has several limitations that should be acknowledged. The relatively small sample size and the low response rate limit the generalizability of the results to the broader community pharmacist population in Turkey. This limited response may be due to variations in pharmacists' familiarity and interest, as some community pharmacists still do not have a thorough understanding of ONS. Consequently, future studies with larger and more geographically diverse samples are warranted to confirm and extend these findings.

Despite these limitations, the study offers valuable insights into pharmacists' current knowledge and practices regarding ONSs, and it provides a baseline for future research to assess the effects of targeted training and follow-up strategies.

This study highlights significant gaps in pharmacists' education and knowledge regarding ONSs, particularly concerning the storage and use of opened products compared to unopened ones. The participants' strong interest in further training—particularly on disease-specific use and storage conditions—reflects a high awareness of these deficiencies and a willingness to enhance patient care. Integrating nutrition education into pharmacy curricula may address current knowledge gaps and enable pharmacists to implement better interventions and counselling in order to optimize patient outcomes, adherence to ONS therapy, and the overall effectiveness of nutritional treatment.

Author contributions

Conception: Ö.G., N.B., K.D.; Design: Ö.G., B.K.Ç., K.D.; Data acquisition: N.B.; Data analysis: Ö.G., B.K.Ç.; Data interpretation: Ö.G., B.K.Ç.; Drafting of the manuscript: Ö.G., N.B.; Critical revision of the manuscript: B.K.Ç., K.D. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Hacettepe University Research Ethics Committee for Health Sciences (Date: 27.11.2024, Decision/Protocol No: SBA 24/1180). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data supporting the findings of this study are not publicly available due to .

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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Effects of nutrition nurse-led education on clinical nutrition readiness and knowledge gain

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ABSTRACT

Background: Malnutrition is an important clinical problem associated with increased morbidity, mortality, and prolonged hospital stay. However, nursing students' knowledge and preparedness regarding clinical nutrition are often reported to be insufficient. This study aimed to evaluate the effect of a Nutrition Nursing course, delivered under the leadership of a nutrition nurse and based on the Practice-to-Academia educational model, which aims to transfer clinical experience into academic education, on nursing students' clinical nutrition knowledge.

Methods: This quasi-experimental study was conducted with third-year nursing students enrolled in the Nutrition Nursing course in the nursing department of a university during the 2023–2024 academic year. Data were collected using a Descriptive Information Form and a 25-item Nutrition Knowledge Test. A pre-test was administered at the beginning of the course and a post-test after the 15-week training period. Data were analyzed using SPSS 27.0, and pre-test–post-test comparisons were performed using the Wilcoxon Signed-Rank Test.

Results: Following the educational program, a significant increase was observed in students' nutrition knowledge across all major domains ($p < 0.001$). The greatest improvement was identified in the domain of basic concepts of clinical nutrition therapy. Significant increases were also observed in knowledge related to malnutrition screening, clinical nutrition therapy practices, and nursing responsibilities.

Conclusion: A clinically oriented educational program led by a nutrition nurse and based on the Practice-to-Academia model significantly improves nursing students' knowledge of clinical nutrition therapy.

Keywords: clinical nutrition education, nutrition nursing, malnutrition screening, nursing students, Practice-to-Academia model

Introduction

Malnutrition is a prevalent and significant clinical problem associated with increased morbidity, mortality, and prolonged hospital stays.[1,2] European Society for Clinical Nutrition and Metabolism (ESPEN) provides guidance on clinical nutrition therapy, nutritional care processes, enteral feeding practices, and the identification and management of nutrition-related problems, thereby

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supporting healthcare professionals in delivering evidence-based nutritional care.[1]

Therefore, adequate preparedness in clinical nutrition should be considered one of the core professional competencies in nursing education.[3-5] However, existing studies indicate that nursing students' knowledge and preparedness regarding clinical nutrition practices need further development.[5-7] Furthermore, review and scoping studies emphasize that nutrition education at the undergraduate level is often limited in scope and predominantly theoretical, which may create a persistent gap between knowledge acquisition and clinical practice.[8,9]

Competence in clinical nutrition cannot be achieved through theoretical instruction alone; it requires contextual learning integrated with real clinical decision-making processes. Patricia Benner's Novice to Expert model[10] emphasizes that clinical competence develops through experiential learning, progressing from rule-based knowledge to contextual and intuitive clinical reasoning. This theoretical framework suggests that the active involvement of experienced clinicians in the educational process may facilitate students' deeper and more practice-oriented understanding of clinical knowledge.[6,11,12]

Within this context, a model led by nurses and integrating clinical experience into undergraduate education may provide an important approach to bridging the gap between theory and practice.[5] Experiential learning is particularly critical in areas such as malnutrition assessment and the appropriate management of clinical nutrition therapy, which directly influence patient outcomes.[13]Therefore, the present study aimed to evaluate the effects of a nutrition nurse–led educational model on undergraduate nursing students' readiness for clinical nutrition and their knowledge gain.

Main Points

- Education led by a nutrition nurse improved nursing students' clinical nutrition knowledge.
- The Practice-to-Academia model integrated clinical experience into nursing education.
- Knowledge levels improved in malnutrition screening and clinical nutrition therapy.

Methods

Study participants

The study population consisted of all third-year nursing students (n = 48) enrolled in the Nutrition Nursing course at Nuh Naci Yazgan University during the 2023–2024 academic year. Since all eligible and volunteer students were included in the study, a census sampling method was used. The Nutrition Nursing course is offered as an elective course within the undergraduate nursing curriculum. Students who did not participate in either the pre-test or post-test, withdrew from the study at any stage, or submitted questionnaires containing incomplete or invalid data were excluded from the analysis. Sample size was calculated using G*Power 3.1.9.7 based on the difference between two dependent means. Assuming a two-tailed test, an effect size of

$d_z = 0.50$, $\alpha = 0.05$, and a statistical power of 0.80, the minimum required sample size was determined to be 34. The study was completed with 48 participants, indicating that the sample size was adequate.

Data collection

The data collection tool was developed by two nurses with over 10 years of experience in the field of nutrition nursing and three faculty members holding PhD in nursing. During the development of the data collection tools (pre-test and post-test), expert opinions were obtained from two nutrition nurses certified by the Republic of Türkiye Ministry of Health (RTMH), each with more than 10 years of professional experience in nutrition nursing. One of these nutrition nurses also served as the course instructor and was responsible for designing the Practice-to-Academia model. The instructor holds a PhD in Internal Medicine Nursing, a Nutrition Nurse certification from the RTMH, and an ESPEN Lifelong Learning (LLL) teaching certificate. This nurse is an educator who applies the applied academy model.

Of the three faculty members involved in the development of the data collection tool, two are Associate Professors and one is an Assistant Professor; all are affiliated with the Faculty of Nursing and have over 20 years of academic experience in nursing.

The data collection tool consisted of two sections. The first section, the Descriptive Information Form, included 10 sociodemographic questions covering variables such

as age, gender, grade point average (GPA) and whether the student had previously received nutrition education.

The second section included the Nutrition Knowledge Test (Appendix 1) which consisted of 25 multiple-choice questions, each with five options and one correct answer. The test content covered topics such as basic principles of nutrition and functions of nutrients, energy balance and food groups, nutrition in special populations (e.g., infants, older adults, pregnant women), clinical nutrition (e.g., diabetes, coronary heart disease, obesity), as well as nutrition assessment and patient education. Each correct answer was scored as 1 point, and the total score ranged from 0 to 25.

The data collection process was conducted face-to-face in the classroom under the supervision of the researchers. The procedure was carried out in two stages. The pre-test was administered in the first week of the semester following the course introduction, during which both the Descriptive Information Form and the Nutrition Knowledge Test were completed. The post-test was administered after the completion of the 15-week course period, during which only the Nutrition Knowledge Test was re-administered.

Educational process

Within the undergraduate nursing curriculum, students are generally provided with nutrition education covering fundamental concepts such as nutrients, nutritional requirements, and nutrition across different life stages. However, this education is predominantly theoretical, and limited emphasis is placed on clinical nutrition practices. Prior to this study, although students had been exposed to basic nutrition content, structured and nurse-led training specifically focused on clinical nutrition therapy had not been provided.

It is stated that the instructor responsible for delivering the course actively serves as a nutrition nurse and holds a PhD in Nursing. The instructor is certified in nutrition nursing RTMH and possesses an ESPEN LLL teaching certification. Furthermore, it is emphasized that the instructor developed the Practice-to-Academia model based on academic and clinical experience in the field of nutrition nursing and made significant contributions to both its theoretical framework and practical implementation.

The educational intervention consisted of a Nutrition Nursing course conducted over a period of 15 weeks, with two hours of instruction per week, as part of the undergraduate nursing curriculum. The course content was developed in accordance with the curriculum framework of the Council of Higher Education[14] and international standards such as those proposed by the American Association of Colleges of Nursing.[15] The educational program was designed based on the Practice-to-Academia approach, aiming to integrate clinical experience into the academic learning process.

Teaching methods included lectures, clinical case analyses, case discussions, question–answer sessions, interactive discussions, and examples based on real clinical experiences. Students were encouraged to reflect on cases related to clinical nutrition management and to develop evidence-based nursing practices.

Statistical analyses

Data were analyzed using IBM SPSS Statistics version 27.0. Prior to analysis, data were checked for completeness and consistency. Categorical variables were presented as number (percentage), and continuous variables were presented as mean ± standard deviation (SD). Normality was assessed using the Shapiro–Wilk test. Since the distribution of nutrition knowledge scores did not meet normality assumptions ($p < 0.001$), non-parametric tests were applied. Pre-test and post-test scores were compared using the Wilcoxon Signed-Rank Test. Comparisons of knowledge scores across descriptive characteristics were performed using the Mann–Whitney U test (for two groups) or the Kruskal–Wallis test (for three or more groups). A p-value of < 0.001 was considered statistically significant.

Results

The descriptive characteristics of the students are presented in Table 1. The majority of participants were female 83.3%, and 66.7% were between 20 and 22 years of age. Approximately two-thirds 68.8% had no prior formal education related to nutrition, and 62.5% of the students expressed interest in receiving additional clinical nutrition training.

The comparison of Nutrition Knowledge Test scores by descriptive characteristics is presented in Table 2. A statistically significant increase was observed in overall

nutrition knowledge scores following the course ($p < 0.001$). A significant difference was found across age groups ($p = 0.041$), with greater score increases in students aged 20–22 years (+26.57) compared to those aged 18–19 years (+23.62). No significant differences were found according to gender ($p = 0.612$) or prior nutrition education ($p = 0.303$). Although not statistically significant, higher score increases were observed in students willing to receive additional training (+28.40 vs. +21.30; $p = 0.090$).

The pre-test and post-test comparisons of the Nutrition Knowledge Test domains are presented in Table 3. Across all five knowledge domains, statistically significant differences were observed between pre-test and post-test mean scores ($p < 0.001$). The largest improvement was observed in the basic concepts of clinical nutrition therapy domain ($\Delta = -0.37$), followed by clinical nutrition therapy applications ($\Delta = -0.27$) and malnutrition risk screening ($\Delta = -0.26$). The smallest improvement was observed in the complications domain ($\Delta = -0.15$)

Table 4 includes the pre-test and post-test statistics for the analysis of the Nutrition Knowledge Test subdomains. At the subdomain level, the largest changes were observed in nutrition indications ($\Delta = -0.48$), enteral nutrition products ($\Delta = -0.44$), and indicators of malnutrition ($\Delta = -0.46$) ($p < 0.001$). No statistically significant differences were found in evaluation of treatment efficacy ($\Delta = -0.04$, $p = 0.157$) or gastrointestinal complications ($p = 1.000$). Detailed results are presented in Table 4.

Table 1. Descriptive characteristics of the students (n = 48)

Variable	n	%
Age (years)		
18–19 (n=16)	16	33.3
20–22 (n=32)	32	66.7
Gender		
Female	40	83.3
Male	8	16.7
Previous Education on Nutrition		
No	33	68.8
Yes	15	31.2
Willingness to Receive Clinical Nutrition Training		
Yes	30	62.5
No	18	37.5

Values are presented as number (percentage).

Table 2. Comparison of nutrition knowledge test scores by descriptive characteristics (n = 48)

Variable	Pre-test Mean ± SD	Post-test Mean ± SD	Difference (Post – Pre)	p*
Age (years)				
18–19	43.25 ± 10.7	66.87 ± 12.1	+23.62	0.041
20–22	45.81 ± 11.9	72.38 ± 12.3	+26.57	
Gender				
Female (n=40)	44.92 ± 11.6	70.84 ± 12.2	+25.92	0.612
Male (n=8)	45.00 ± 12.4	70.00 ± 11.8	+25.00	
Previous Nutrition Education				
Yes (n=15)	46.80 ± 10.9	72.60 ± 11.8	+25.80	0.303
No (n=33)	44.00 ± 11.7	70.00 ± 12.3	+26.00	
Willingness to Receive Clinical Nutrition Training				
Yes (n=30)	44.30 ± 11.7	72.70 ± 12.1	+28.40	0.090
No (n=18)	45.10 ± 12.2	66.40 ± 12.5	+21.30	

*Comparisons between two groups were performed using the Mann–Whitney U test, and comparisons among three or more groups were performed using the Kruskal–Wallis test.

Table 3. Nutrition knowledge test domains pre-test and post-test comparisons

Category	Pre-test Mean	Post-test Mean	Difference (Δ)	p*
Basic concepts in clinical nutrition therapy	1.65	1.27	−0.37	<0.001
Malnutrition risk screening	1.55	1.29	−0.26	<0.001
Clinical nutrition therapy applications	1.54	1.27	−0.27	<0.001
Nursing care in clinical nutrition therapy	1.58	1.34	−0.24	<0.001
Complications in clinical nutrition therapy	1.48	1.32	−0.15	<0.001

* Comparisons were analyzed using the Wilcoxon Signed-Rank Test. Negative values indicate improvement in knowledge scores.

Discussion

This study demonstrated that a nutrition nursing program conducted under the leadership of a nutrition nurse significantly improved nursing students' nutrition knowledge across all major domains, including basic concepts of clinical nutrition therapy, malnutrition risk screening, clinical nutrition therapy practices, nursing responsibilities, and complication management. The magnitude and consistency of the pre-test–post-test gains observed across nearly all subcategories indicate that targeted nutrition education can effectively address the well-documented knowledge gaps among nursing students. These findings are also consistent with the Practice-to-Academia Clinical Education Model developed in this study (Figure 1). Within this model, experience and evidence-based knowledge derived from clinical practice are transferred into the academic environment through a structured educational program led by a nutrition nurse.

Previous studies have shown that blended learning approaches supported by real-life clinical scenarios significantly improve students' knowledge and nutrition counseling skills.[16] These findings are consistent with the results of the present study and support the effectiveness of structured educational programs. Moreover, integrating academic knowledge with clinical practice through academia–clinical collaboration has been reported to enhance clinical competencies and professional development.[17] In this context, the structured, case-based educational approach implemented in this study is considered to have contributed to both knowledge acquisition and the transfer of knowledge into clinical practice, in line with the Practice-to-Academia model.

Consistent with previous studies, these findings confirm that nursing students often begin undergraduate programs with limited knowledge of nutrition and benefit substantially from structured educational interventions. Systematic and scoping reviews have shown that nursing students generally have insufficient baseline knowledge in both basic nutrition and clinical nutrition.[5,8] Similarly, the substantial knowledge gains observed after the course in this study support the view that nutrition education should be considered a core professional competency rather than a supplementary topic within nursing curricula.[6,14,15]

Significant improvements were also observed in knowledge related to malnutrition screening.[16] In particular, notable gains were found in students' understanding of indicators of malnutrition, screening tools, and the purposes of screening.[17] These findings are highly consistent with Bauer et al.[18], who reported that malnutrition knowledge among nursing students is generally at a moderate level and emphasized the importance of active teaching methods. Early identification of malnutrition is one of the fundamental responsibilities of nurses within the clinical care process. Therefore, adequate knowledge of screening tools is critical for the implementation of evidence-based nutritional care. Similarly, Ibrahim and Aldawsari[19] emphasized that strong nutrition knowledge forms the foundation for nurses' ability to apply evidence-based screening practices. The findings of the present study suggest that focused educational interventions can significantly strengthen students' ability to recognize nutritional risk.[20]

Significant improvements were also observed in knowledge related to nursing responsibilities in clinical nutrition therapy. Increases in students' knowledge regarding patient monitoring, documentation, sterility, assessment of patient tolerance, and interdisciplinary communication indicate that the educational program enhanced awareness of clinical responsibilities. These findings are consistent with studies showing that structured nutrition education improves both knowledge and practice-related behaviors.[21] In addition, Dogan

et al.[8] emphasized that addressing nutrition within both academic and clinical learning environments is essential to prepare nursing students for real-world clinical care settings.

Table 4. Pre-test and post-test statistics for nutrition knowledge test subdomains analysis. (n = 48)

Questions	Pre-test Mean ± SD	Post-test Mean ± SD	Difference (Post – Pre)	p*
Basic Concepts in clinical nutrition therapy				
Nutrition organizations	1.62 ± 0.49	1.19 ± 0.39	−0.44	<0.001
Nutrition indications	1.83 ± 0.38	1.35 ± 0.48	−0.48	<0.001
Enteral nutrition products	1.83 ± 0.38	1.40 ± 0.49	−0.44	<0.001
Use of parenteral nutrition products	1.27 ± 0.45	1.15 ± 0.36	−0.12	=0.014
Principles of parenteral nutrition	1.52 ± 0.50	1.15 ± 0.36	−0.38	<0.001
Malnutrition risk screening				
Definition of malnutrition	1.42 ± 0.50	1.25 ± 0.44	−0.17	0.021
Purpose of screening	1.48 ± 0.50	1.29 ± 0.46	−0.19	0.007
Nutritional screening tools	1.67 ± 0.48	1.33 ± 0.48	−0.33	<0.001
Indicators of malnutrition	1.85 ± 0.36	1.40 ± 0.49	−0.46	<0.001
Timing of screening	1.33 ± 0.48	1.19 ± 0.39	−0.15	0.020
Clinical nutrition therapy applications				
Nutritional requirements and calculations	1.62 ± 0.49	1.44 ± 0.50	−0.19	0.007
Preparation and administration of nutrition solutions	1.58 ± 0.50	1.29 ± 0.46	−0.29	0.001
Evaluation of treatment efficacy	1.10 ± 0.31	1.06 ± 0.24	−0.04	0.157
Indications for enteral/parenteral nutrition	1.67 ± 0.48	1.27 ± 0.45	−0.40	<0.001
Routes of clinical nutrition therapy	1.70 ± 0.46	1.27 ± 0.45	−0.43	<0.001
Nursing care in clinical nutrition therapy				
Basic nursing responsibilities in clinical nutrition therapy	1.79 ± 0.41	1.42 ± 0.50	−0.38	<0.001
Communication and collaboration in nutrition care	1.52 ± 0.50	1.40 ± 0.49	−0.12	0.014
Monitoring and documentation of clinical nutrition therapy	1.60 ± 0.49	1.33 ± 0.48	−0.27	0.001
Maintaining sterility and line care	1.50 ± 0.51	1.29 ± 0.46	−0.21	0.002
Assessment of patient tolerance to clinical nutrition therapy	1.46 ± 0.50	1.25 ± 0.44	−0.21	0.008
Complications in clinical nutrition therapy				
Prevention of complications	1.56 ± 0.50	1.40 ± 0.49	−0.17	0.011
Early detection of complications	1.54 ± 0.50	1.40 ± 0.49	−0.15	0.008
Gastrointestinal complications	1.15 ± 0.36	1.15 ± 0.36	0.00	1.000
Metabolic and mechanical complications	1.69 ± 0.47	1.40 ± 0.49	−0.29	<0.001
Nursing responsibilities in complication management	1.44 ± 0.50	1.29 ± 0.46	−0.15	0.008

* Comparisons were analyzed using the Wilcoxon Signed-Rank Test. Negative values indicate improvement in knowledge scores.

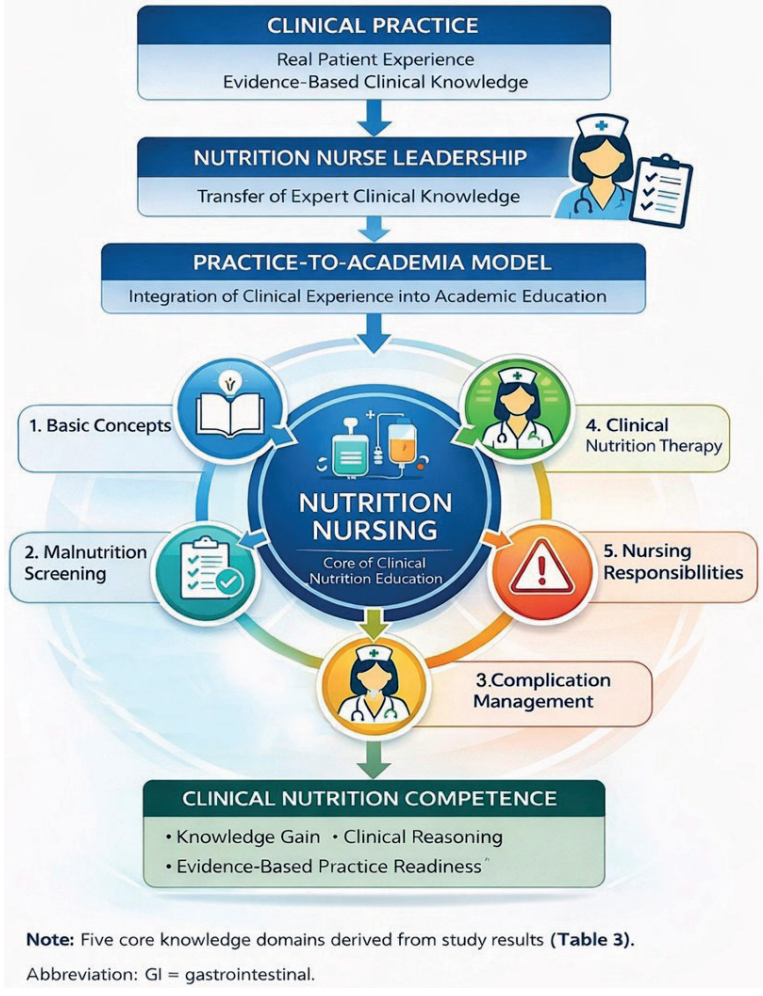

Figure 1. Practice-to-Academia Clinical Education Model led by a nutrition nurse for clinical nutrition therapy

One of the key strengths of this study is that the educational program was conducted by a nutrition nurse with clinical experience. Nutrition nurses play an active role in the assessment of malnutrition risk, planning of clinical nutrition therapy and patient monitoring. Therefore, transferring clinically grounded knowledge into the educational process may significantly contribute to the development of students' professional competencies in nutrition care. In this context, education programs led by nutrition nurses may represent an effective approach to improving nursing students' knowledge and clinical readiness in clinical nutrition.

Furthermore, this study is important as it represents the first empirical evaluation in Türkiye of an undergraduate nutrition nursing course led by a nutrition nurse. Considering that nutrition topics are often addressed only to a limited extent within nursing education in Türkiye, evaluating structured educational models of this kind may contribute significantly to both the development of educational programs and the strengthening of clinical nutrition care.

This study has several limitations. The fact that the research was conducted at a single center limits its generalizability. Furthermore, evaluating only short-term effects restricts the ability to draw conclusions about long-term outcomes. A limitation of these services is the exclusion of questions related to clinical treatment and drug administration, which are frequently encountered in the clinical settings of the researchers.

Conclusion

This study demonstrated that an educational program led by a nutrition nurse and integrating clinical experience into academic education significantly improves nursing students' knowledge of clinical nutrition therapy. The findings indicate that educational approaches focused on key areas of clinical care, particularly malnutrition screening and clinical nutrition therapy, can effectively enhance nursing students' competencies in clinical nutrition therapy.

Ethical statement

The study was approved by the Scientific Research and Publication Ethics Committee of Nuh Naci Yazgan University (Approval No: 2024/002-05, Approval date: 12.02.2024). All participants were informed about the study and provided voluntary consent prior to participation.

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Author contributions

Conception: H.K., Ö.C.; Design: H.K.; Data acquisition: H.K.; Data analysis: H.K., G.S., Z.K.; Data interpretation: H.K.; Drafting of the manuscript: H.K., Ö.C.; Critical revision of the manuscript: H.K., Ö.C. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Scientific Research and Publication Ethics Committee of Nuh Naci Yazgan University (Date: February 12, 2024, Decision/Protocol No: 2024/002-05). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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Association of HbA1c levels with anthropometric measures and body composition: Emphasis on waist-to-hip ratio as a marker of glycemic impairment

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ABSTRACT

Objective: This study aimed to evaluate the relationships between glycated hemoglobin (HbA1c) levels and anthropometric measurements (body mass index (BMI), waist circumference (WC), and waist-to-hip ratio (WHR) as well as body composition parameters.

Method: A total of 145 individuals (control, n=49; prediabetes, n=48; type 2 diabetes, n=48) were included in this cross-sectional study. HbA1c levels, BMI, WC, and WHR were measured in the participants; body composition was assessed using bioelectrical impedance analysis (BIA). Appropriate parametric and non-parametric tests were used for between-group comparisons, and relationships among variables were examined using correlation analyses. Analyses adjusted for age, gender, use of antidiabetic medications, and regular physical activity status were performed using generalized linear models (GLM).

Results: BMI, WC, and WHR values were found to be significantly lower in the control group compared to the prediabetes (preDM) and type 2 diabetes (T2DM) groups ($p<0.001$). No significant difference was observed between the preDM and T2DM groups regarding these parameters. Body fat percentage and fat mass were found to be higher in the preDM and T2DM groups compared to the control group ($p<0.001$). HbA1c levels showed a positive correlation with BMI, WC, WHR, body fat percentage, and fat mass ($p<0.05$). In analyses adjusted for gender and age, WC and WHR were found to be independently associated with HbA1c, with the strongest association observed for WHR (β: 4.301; $p=0.006$).

Conclusion: In this study, abdominal obesity indicators, particularly WC and WHR, showed stronger associations with HbA1c levels compared to BMI. These findings suggest that markers of abdominal obesity may be associated with glycemic status. However, due to the cross-sectional study design, these associations cannot be interpreted as causal.

Keywords: HbA1c, waist circumference, waist-to-hip ratio, type 2 diabetes, prediabetes

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Introduction

Diabetes mellitus (DM) has become a major public health problem due to its increasing prevalence and the serious complications it causes.[1] Type 2 diabetes mellitus (T2DM) accounts for the majority of diabetes cases and is closely associated with obesity. In particular, abdominal fat accumulation and increased body fat percentage play a significant role in insulin resistance and impaired glycemic control.[2,3]

Although body mass index (BMI) is widely used in the assessment of obesity, it has limitations as it does not reflect body fat distribution.[4] In contrast, waist circumference (WC) and waist-to-hip ratio (WHR) are indicators that better reflect abdominal adiposity and may show stronger associations with diabetes risk.[5,6]

Bioelectrical impedance analysis (BIA) is a commonly used non-invasive method for assessing body composition and allows a more detailed evaluation of parameters such as fat mass and fat-free mass.[7] It is known that changes in body composition affect glucose metabolism and insulin sensitivity.[8,9]

HbA1c is a reliable biomarker reflecting average blood glucose levels over the past 2–3 months and is widely used for long-term glycemic control assessment.[10] However, the strength and relative importance of the relationships between HbA1c and various obesity indicators and body composition parameters are not yet fully clear.

The aim of this study is to comparatively evaluate the relationships between HbA1c levels and anthropometric measurements (BMI, WC, and WHR) as well as body composition parameters determined by BIA in healthy individuals and those diagnosed with preDM and T2DM, and to elucidate the role of abdominal obesity indicators in particular. However, studies that evaluate these parameters comparatively within the same population and in conjunction with multivariate analyses are limited.

Main Points

- Indicators reflecting abdominal obesity (WC and WHR) show stronger associations with HbA1c levels compared to BMI.
- These findings suggest that considering WC and, particularly, WHR measurements may be beneficial in assessing glycemic status.
- The observed associations between HbA1c, anthropometric measurements, and body composition parameters in the prediabetes stage support the notion that metabolic impairment begins early.

Materials and Methods

Study population and design

This study was designed as a cross-sectional study. A total of 145 individuals aged between 19 and 65 years who presented to the Internal Medicine Outpatient Clinic, Ankara Bilkent City Hospital between November 2024 and June 2025 were consecutively included, provided that biochemical blood test results obtained within the previous 48 hours were available. The sample size of the study was determined prior to data collection using an a priori power analysis via G*Power software (version 3.1.9.7). Assuming an effect size of f = 0.40, a significance level of $\alpha = 0.05$, and a statistical power of 95% ($1-\beta = 0.95$), the minimum required sample size was calculated as 102 participants. A total of 145 participants were included in the study.

Participants were categorized into three groups according to their clinical diagnoses:

1. Control group: individuals with HbA1c < 5.7% and no comorbidities,
2. PreDM group: individuals with fasting plasma glucose (PG) 100–125 mg/dL and a 2-hour PG result of 140–199 mg/dL on an oral glucose tolerance test (OGTT) (75 g glucose) and/or HbA1c 5.7–6.4%,
3. T2DM group; individuals meeting any of the following criteria: fasting PG ≥126 mg/dL after at least 8 hours of fasting, or 2-hour PG ≥200 mg/dL on an OGTT (75 g glucose), or random PG ≥200 mg/dL, or HbA1c ≥6.5%, in the presence of typical DM symptoms.[2]

Individuals under 19 years of age and over 65 years of age, those unable to provide informed consent, insulin users, those with type 1 DM, gestational DM, malignancy, severe systemic disease (heart failure, liver or kidney disease, or lung disease, etc.), those with a history of psychiatric or neurological disorders, individuals with severe acute or chronic infectious diseases, and pregnant or breastfeeding women were excluded from the study.

Ethical approval for the study was obtained from the Ankara Bilkent City Hospital Scientific and Ethical Review

Board for Medical Research (TABED) on October 2, 2024, under number TABED 2 – 24 – 553.

Data collection

Data were collected using a face-to-face questionnaire. Body weight, height, waist circumference, and hip circumference were measured using standard methods, and BMI was calculated. Body weight and composition (body fat percentage, muscle mass) were measured using a BIA device. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest using a measuring tape, while hip circumference was measured at the widest part of the hips.[11]WHR was calculated as waist circumference divided by hip circumference and was analyzed as a continuous variable in the statistical analyses.

Biochemical parameters were obtained from routine laboratory analyses ordered by a physician at the hospital and analyzed at the Medical Biochemistry Laboratory of Ankara Bilkent City Hospital.

Physical activity status was assessed in accordance with the international guidelines recommending at least 150 minutes of moderate-intensity exercise per week for individuals with diabetes. Participants were questioned about their regular exercise habits, including the type (e.g., walking, running, swimming, pilates), duration, and weekly frequency of the activities. Participants who met the 150-minute threshold were categorized as 'regularly exercising', while those who did not were categorized as 'not regularly exercising'.[12]

Statistical analysis

The normality of the data obtained in the study was assessed using the Kolmogorov-Smirnov test. Continuous variables are presented as mean ± standard deviation (mean ± SD) or median (interquartile range [IQR]), as appropriate.

For comparisons between groups, one-way analysis of variance (ANOVA), the Kruskal-Wallis test, and the Mann-Whitney U test were used as appropriate. Bonferroni-adjusted post-hoc tests were applied for significant results. Categorical variables were analyzed using the chi-square test.

The relationship between HbA1c groups and anthropometric measurements and body composition parameters was examined using Spearman's correlation analysis. Analyses adjusted for gender, regular physical activity status, and antidiabetic medication use were performed using one-way analysis of covariance (ANCOVA). The independent relationships between HbA1c levels and the variables were determined using a general linear model (GLM) adjusted for gender and age. Because anthropometric variables were expressed on different numerical scales, interpretation of β coefficients was performed cautiously and focused primarily on the direction and statistical significance of associations rather than direct comparison of coefficient magnitudes. To minimize potential multicollinearity, anthropometric and body composition variables were evaluated in separate regression models. Multicollinearity was assessed using variance inflation factor (VIF), and all observed values remained within acceptable limits.

Statistical significance was accepted as $p<0.05$.[13]

The analyses were conducted using three different approaches.

1. control, preDM, and T2DM groups formed based on clinical diagnosis,
2. groups based on HbA1c levels (<5.7; 5.7–6.4; ≥6.5) to evaluate relationships independent of diagnosis,
3. correlation and covariance analyses based on HbA1c values.

In HbA1c-based analyses, the use of antidiabetic medications and regular physical activity were taken into account. The statistical tests used for each analysis are specified below the relevant tables.

Results

Demographic characteristics and HbA1c levels

A total of 145 participants (control: n=49, preDM: n=48, T2DM: n=48) were included in the study. A significant difference in age was observed between the groups ($p=0.015$); the control group was found to be younger than the preDM and T2DM groups ($p<0.05$), while no difference was observed between the preDM and T2DM groups ($p>0.05$). No difference was found in gender distribution among the groups ($p=0.081$). HbA1c levels showed significant differences among the groups ($p<0.001$), and all groups were found to be significantly different from one another ($p<0.05$).

The demographic characteristics and HbA1c levels are presented in Table 1.

Comparison of anthropometric measurements across groups

BMI, WC, and WHR values showed significant differences across groups (p<0.001 for all results). These parameters were found to be lower in the control group compared to the preDM and T2DM groups; however, no significant difference was observed between the preDM and T2DM groups.

In the analysis by gender, no significant effect was observed on BMI (p>0.05), while WC and WHR values were found to be higher in males (p<0.001).

The results of the anthropometric measurements are presented in Table 2.

Distribution of body composition parameters across groups

Body fat percentage and fat mass showed significant differences across groups (p<0.001), and these values were lower in the control group. No significant difference was observed between the PreDM and T2DM groups. No significant difference was observed between the groups in terms of FFM (p=0.09). When evaluated by gender, body fat percentage and fat mass were higher in females, while FFM was higher in males (p<0.05).

The results are presented in Table 3.

Table 1. Demographic characteristics and HbA1c levels

Variable	Control (n= 49)	PreDM (n= 48)	T2DM (n= 48)	p-value
Age (years)	41 (36-46)a	46 (41-52)b	44 (40-49)b	0.015
HbA1c (%)	5.3 (5.1-5.5)a	5.9 (5.8-6.1)b	7.6 (6.4-8.9)c	<0.001
Gender (M/F)	17/32	19/29	27/21	0.081

Data are given as median (IQR). Kruskal-Wallis test with Bonferroni post-hoc analysis was used. Different superscripts (a, b, c) indicate significant differences between groups. p<0.05 was considered statistically significant.
HbA1c: glycated hemoglobin; DM: diabetes mellitus; T2DM: type 2 diabetes mellitus; M: male; F: female.

Table 2. Distribution of anthropometric measurements across groups

Variable	Control (n= 49)	PreDM (n= 48)	T2DM (n= 48)	p-value	Gender Effect
BMI	26.7 ± 4.7a	32.3 ± 5.8b	31.9 ± 4.4 b	<0.001	Not Significant
WC	92.4 ± 13.6a	103.6 ± 12.1b	106.1 ± 9.9b	<0.001	Male> Female*
WHR	0.87 ± 0.1a	0.94 ± 0.09b	0.96 ± 0.06b	<0.001	Male> Female*

Data are given as mean ± SD. ANOVA was applied for comparisons. Different superscripts (a, b) indicate significant differences between groups.
p<0.05 was considered statistically significant. *p<0.001
BMI: body mass index; WC: waist circumference; WHR: waist-to-hip ratio; DM: diabetes mellitus; T2DM: type 2 diabetes mellitus.

Table 3. Distribution of body composition parameters across group

Variable	Control (n= 49)	PreDM (n= 48)	T2DM (n= 48)	p-value	Gender Effect
Body fat (%)	27.0 (22.5-31.5)a	36.7 (29.0-44.2)b	32.0 (26.5-37.5)b	<0.001	Female> Male*
Fat mass (kg)	21.0 (15.5-26.5)a	29.7 (20.3-39.0)b	28.6 (22.8-34.5)b	<0.001	Female> Male*
FFM	51.7 (42.7-60.7)	53.4 (44.7-61.0)	58.7 (49.4-68.0)	0.09	Male> Female*

Data are given as median (IQR). Kruskal-Wallis test with Bonferroni post-hoc analysis was used. Gender differences were analyzed using the Mann-Whitney U test. Different superscripts (a, b) indicate significant differences between groups. p<0.05 was considered statistically significant. * p<0.05
DM: diabetes mellitus; T2DM: type 2 diabetes mellitus; FFM: fat-free mass.

The association between HbA1c groups and anthropometric measurements and body composition parameters

Moderate positive correlations were found between HbA1c groups and anthropometric measurements (BMI: r=0.39; WC: r=0.42; WHR: r=0.37; p<0.001 for all). These findings indicate a association between increases in anthropometric measurements and higher glycemic levels. The strongest association among anthropometric parameters was observed with WC.

Weak-to-moderate positive associations were found between body composition parameters and HbA1c groups (body fat percentage r=0.22, fat mass r=0.31, and FFM r=0.20). This suggests that abdominal fat indicators may be more strongly associated with glycemic status compared to total body fat indicators.

After adjusting for antidiabetic medication use and regular physical activity (ANCOVA), differences between HbA1c groups remained significant for BMI, WC, WHR, body fat percentage, and fat mass (p<0.001 for all results), while no significant difference was found for FFM (p=0.202).

The results are presented in Table 4.

Association between HbA1c levels and anthropometric measurements and body composition parameters

In a general linear model (GLM) analysis adjusted for age and gender, it was found that WC and WHR were independently associated with HbA1c. WC showed a positive and independent association with HbA1c; each 1 cm increase in WC was associated with a 0.028-unit increase in HbA1c levels (β: 0.028; p=0.012). The strongest association was observed with WHR (β=4.301; p=0.006). While the association between BMI and HbA1c was borderline (p=0.065), no significant association was found with FFM (p=0.648). These findings suggest that measurements reflecting abdominal obesity may be more closely associated with glycemic status compared to general obesity and fat-free mass.

The results are presented in Table 5.

Table 4. Association between HbA1c groups and anthropometric measurements and body composition parameters

Variable	Spearman's r	p-value (Correlation)	p-value (ANCOVA)
BMI	0.39	<0.001	<0.001
WC	0.42	<0.001	<0.001
WHR	0.367	<0.001	<0.001
Body fat (%)	0.224	0.007	<0.001
Fat mass (kg)	0.306	<0.001	<0.001
FFM	0.201	0.015	0.202

Spearman's correlation analysis was used. Group comparisons were performed using ANCOVA adjusted for medication use and regular physical activity. p<0.05 was considered statistically significant.
BMI: body mass index; WC: waist circumference; WHR: waist-to-hip ratio; FFM: fat-free mass.

Table 5. Association between HbA1c values and anthropometric measurements and body composition parameters

Variable	β (Estimate)	95% Confidence Interval	p-value
BMI	0.044	-0.094	0.065
WC	0.028	0.006-0.049	0.012
WHR	4.301	1.276-7.326	0.006
FFM	0.009	-0.079	0.648

Data are given as unstandardized β coefficients with 95% confidence intervals derived from age and gender- adjusted general linear models. p<0.05 was considered statistically significant.
BMI: body mass index; WC: waist circumference; WHR: waist-to-hip ratio; FFM: fat-free mass.

Discussion

In this study, the relationships between HbA1c and anthropometric measurements and body composition parameters were evaluated in the control, preDM, and T2DM groups. The findings suggest that impaired glucose metabolism may be closely associated with abdominal adiposity, particularly from the early stages onward.

When anthropometric measurements were examined, BMI, WC, and WHR were lower in the control group, while similar values were observed in the preDM and T2DM groups. This pattern indicates that obesity—particularly abdominal adiposity—may be linked to glycemic impairment from early stages. Previous studies have also demonstrated that abdominal fat accumulation is strongly associated with insulin resistance and glucose metabolism.[14-16]

Although BMI is known to be associated with glycemic control,[17,18] the stronger associations observed for WC and WHR in this study are noteworthy. This finding supports the notion that fat distribution, rather than total body weight, may be a more important determinant of glycemic status. Consistent with our results, several studies have reported that WC and WHR are more strongly associated with HbA1c levels than BMI.[19,20] These findings are consistent with studies indicating that abdominal fat accumulation may be associated with an increased risk of diabetes even in individuals with normal BMI values.[3]

When body composition parameters were evaluated, it was observed that body fat percentage and mass were increased in the preDM and T2DM groups compared to the control group; however, no significant difference was found between these two groups. This suggests that an increase in body fat may occur in the early stages of dysglycemia. The literature also reports that increased body fat is associated with metabolic disorders and that higher fat levels have been observed in individuals with diabetes.[15,21,22]

The fact that no significant difference was observed between groups in terms of FFM suggests that body fat distribution may be a more decisive factor in the association with HbA1c compared to muscle mass. However, some studies have also reported that low muscle mass is associated with an increased risk of diabetes.[23,24] This highlights the complex and multifactorial nature of body composition in relation to glycemic regulation.

Correlation analyses revealed significant positive associations between HbA1c groups and anthropometric parameters as well as body composition parameters. The strongest correlations were observed with WC, BMI, and WHR, suggesting that measurements reflecting abdominal adiposity may be more strongly associated with glycemic status. Similar associations have previously been demonstrated for WC,[25] BMI,[17]and WHR.[26] In contrast, the associations with body fat percentage and body fat mass were weaker. One study reported that although fat mass is strongly associated with T2DM risk—particularly in women and younger individuals—it is not a better predictor than WC.[27]Additionally, another study found that short-term changes in fat mass do not significantly affect HbA1c levels in patients with diabetes.[28]

The fact that the results remained statistically significant even after adjusting for regular physical activity and the use of antidiabetic medications indicates that the observed associations are relatively independent of these factors. However, variables such as duration of medication use and treatment adherence, along with the cross-sectional study design, represent key limitations of this study. Furthermore, although age and gender were included as adjustment variables in the generalized linear models, residual confounding related to age differences between the study groups cannot be completely excluded.

One of the key contributions of this study is the combined evaluation of anthropometric and body composition parameters across different glycemic states. In multivariate analyses, WC and, particularly, WHR were found to be more strongly associated with HbA1c compared to BMI and FFM. Because WHR is expressed on a narrower numerical scale than BMI and WC, the magnitude of the β coefficient should be interpreted cautiously and not considered a direct measure of effect size. The observed relationship between increases in WC and HbA1c levels supports the potential importance of abdominal obesity indicators. The fact that BMI was only marginally associated may be explained by its limitations in reflecting fat distribution. In addition, no evidence of substantial multicollinearity was observed in the regression analyses, supporting the stability of the reported associations.

Overall, the findings indicate that parameters reflecting abdominal fat distribution are more closely associated with glycemic status. However, due to the cross-sectional design of the study, these associations should

not be interpreted causally. From a clinical perspective, incorporating WC and, in particular, WHR measurements into routine evaluations may contribute to a more accurate assessment of metabolic risk.

Conclusion

In this study, the relationships between HbA1c levels and anthropometric as well as body composition parameters were evaluated, and indicators reflecting abdominal obesity (WC and WHR) were found to have stronger associations with HbA1c compared to BMI. These findings suggest that considering abdominal obesity indicators may be beneficial in the assessment of glycemic status. However, due to the cross-sectional design, causal inferences cannot be made. Further large-scale and prospective studies are needed to better elucidate these relationships.

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Author contributions

Conception and design: G.Ç.A., A.D.; Data acquisition: G.Ç.A., B.E., E.S.Ş.; Data analysis: G.Ç.A.; Data interpretation: G.Ç.A., A.D., E.F.O.; Drafting of the manuscript: G.Ç.A., A.D., B.E., E.F.O., E.S.Ş.; Critical revision of the manuscript: G.Ç.A., A.D. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Ankara Bilkent City Hospital Scientific and Ethical Review Board for Medical Research (TABED) (Date: 2 October, 2024, Decision/Protocol No: TABED 2 – 24 – 553). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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Longitudinal assessment of malnutrition by GLIM criteria and NRS-2002 screening in head and neck cancer patients undergoing radiotherapy or chemoradiotherapy: a prospective observational study

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ABSTRACT

Objective: This study aimed to examine the concordance between Global Leadership Initiative on Malnutrition (GLIM) criteria and Nutrition Risk Screening-2002 (NRS-2002) screening scores, and to describe longitudinal changes in anthropometric parameters and C3-derived skeletal muscle metrics, in patients with head and neck cancer (HNC) undergoing radiotherapy (RT) or chemoradiotherapy (CRT).

Methods: A total of 32 HNC patients undergoing RT or CRT were prospectively enrolled and assessed at baseline (Day 1) and at Week 5 of treatment, with all patients receiving standardized oral nutritional supplementation and weekly clinical monitoring throughout the study period. Anthropometric measurements, skeletal muscle mass (SMM) at the third cervical vertebra (C3), and nutritional status using the NRS-2002 and GLIM criteria were assessed at baseline (Day 1) and at the end of the fifth week of treatment.

Results: From the first day to the fifth week of anticancer treatment, the percentage of patients at risk of malnutrition (NRS-2002 scores ≥ 3) increased significantly from 40.6% to 71.9% (P = .002), while the prevalence of malnutrition based on GLIM criteria increased from 46.9% to 71.9% (P = .005). Despite nutritional support, significant reductions in body weight, BMI, and C3 cross-sectional muscle area all P < .05 were observed. NRS-2002 scores correlated significantly with GLIM classifications at baseline and at Week 5 (all P < .005). No significant associations were observed between C3-derived skeletal muscle parameters and either assessment tool.

Conclusions: Despite standardized nutritional supplementation and weekly clinical monitoring, significant declines in body weight, BMI, and C3 cross-sectional muscle area were observed during the five-week treatment period. The GLIM criteria identified a progressive increase in malnutrition prevalence, particularly in the severe (Stage 2) category. NRS-2002 scores showed stronger associations with anthropometric parameters than with SMM, suggesting potential limitations of SMM assessments in this patient population. These findings underscore the need for individualized, proactive nutritional management in HNC patients undergoing RT or CRT. The utility of C3-based skeletal muscle assessment as a monitoring tool during active treatment requires further validation in larger prospective studies.

Keywords: head and neck neoplasm, malnutrition, nutritional assessment, radiotherapy

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Introduction

Head and neck cancer (HNC) is one of the cancer types with the highest prevalence of malnutrition, due to the compromising impact of lifestyle habits adopted prior to diagnosis as well as the location of the tumor on food intake, the hypercatabolic state induced by the malignancy itself and the toxicities of radiotherapy (RT) or chemoradiotherapy (CRT).[1,2]

Approximately 70% of weight loss in HNC patients involves the decrease in lean body mass (LBM), with skeletal muscle mass (SMM) being its primary component.[1,3] Reduced SMM is increasingly recognized as a critical factor contributing to poor short- and long-term outcomes in oncology, including diminished treatment tolerance, increased toxicity, and worse survival.[4,5] In clinical practice, the gold standard for SMM assessment is based on cross-sectional imaging at the third lumbar vertebra (L3); however, this is not routinely available in HNC patients.[1,3-6] Instead, SMM assessment at the third cervical vertebra (C3), which is typically included in HNC imaging protocols, has been validated as a reliable and cost-effective alternative, showing a strong correlation with L3 measurements.[7,8] Despite this, the diagnostic sensitivity of C3-based SMM assessments in identifying malnutrition remains limited, particularly in sarcopenic or nutritionally compromised patients.

Main Points

- Malnutrition progressed significantly during treatment, with GLIM-defined malnutrition prevalence increasing from 46.9% at baseline to 71.9% by Week 5 despite nutritional supplementation.
- The proportion of severely malnourished patients (GLIM Stage 2) tripled during the five-week treatment period.
- NRS-2002 scores increased in parallel with GLIM staging and were consistently associated with anthropometric deterioration, supporting their complementary use for early malnutrition identification and monitoring in routine clinical practice.
- Significant reductions in body weight, BMI, and C3 cross-sectional muscle area were observed during the five-week treatment period, confirming measurable anthropometric and regional muscle deterioration in this patient population.
- C3-based skeletal muscle parameters were not significantly associated with GLIM staging or NRS-2002 scores, and their role as a monitoring tool during active treatment requires further validation.

Recently, Global Leadership Initiative on Malnutrition proposed global consensus-based universal criteria (GLIM criteria) for diagnosis and severity grading of malnutrition in adults in different clinical settings after individuals were determined to be at risk of malnutrition according to a validated nutritional screening tool.[9] The diagnosis of malnutrition is based on any combination of at least one phenotypic criterion (nonvolitional weight loss, low body mass index [BMI] or reduced muscle mass) and one etiologic criterion (reduced food intake or disease burden / inflammation), while the severity classification (stage 1 / moderate or stage 2 / severe) is based on the degree of loss for the phenotypic criteria.[9] Notably, given that they are currently developed as a consensus-based framework, the validity and reliability of these operational criteria need to be tested across different clinical populations.[10,11] However, to date, only a limited number of studies have investigated the prevalence of malnutrition according to GLIM specifically in the HNC setting.[10,12,13]

This study aimed to examine the concordance between GLIM staging and NRS-2002 scores prospectively and describe longitudinal changes in anthropometric parameters and C3-derived skeletal muscle metrics over the first five weeks of RT or CRT, in a real-world outpatient HNC cohort under standardized oral nutritional supplementation and weekly clinical monitoring.

To our knowledge, this represents one of the few prospective studies to simultaneously apply all three assessment modalities — GLIM criteria, NRS-2002 screening, and C3-based skeletal muscle metrics — within the same HNC cohort, thereby enabling a direct comparison of their concordance and methodological limitations under routine clinical conditions.

Methods

Study population

A total of 32 patients with HNC (median age 60 years, range 18-85 years, 81.3% were men) who received RT or CRT with concomitant nutritional support were included in this prospective longitudinal study conducted at a tertiary care radiation oncology clinic between April 2020 and April 2021. Patients were eligible if they were 18 years or older and provided written informed consent

after a detailed explanation of the study protocol. Exclusion criteria included prior anticancer treatment, secondary malignancies, or inability to complete the planned treatment protocol. Of the 58 patients initially screened, 26 were excluded due to prior anticancer treatment (n = 12), secondary malignancy (n = 6), inability to complete the planned treatment course (n = 5), or refusal to provide informed consent (n = 3). All 32 enrolled patients completed both assessment time points (Day 1 and Week 5) with no loss to follow-up. All patients received volumetric modulated arc therapy (VMAT) with standard fractionation (1.8–2 Gy per fraction). Patients undergoing CRT received concurrent weekly platinum-based chemotherapy according to institutional protocols; cetuximab-based regimens were not used in this cohort.

Written informed consent was obtained from each subject following a detailed explanation of the objectives and protocol of the study, which was conducted in accordance with the ethical principles stated in the 'Declaration of Helsinki' and approved by the institutional ethics committee. This study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Assessments

Data on patient demographics (age, sex), comorbid diseases, smoking status, tumor characteristics (TNM stage, location) and treatment modality (definitive/adjuvant RT, CRT) were recorded at baseline. Data on anthropometrics (weight [kg], BMI [kg/m^2], mid-arm circumference [cm], calf circumference [cm]), SMM at the level of the third cervical vertebra (C3) including paravertebral cross-sectional muscle area (CSMA, cm^2) and skeletal muscle index (cm^2 / m^2), and nutritional status assessment via NRS-2002 and GLIM criteria were recorded on the first day and 5th week of anti-cancer treatment.

Study parameters

First-day vs. fifth-week data on anthropometrics, SMM and nutritional status (NRS-2002, GLIM) were compared. The correlation of NRS-2002 scores with anthropometrics and SMM as well as the change in NRS-2002 scores, anthropometrics and SMM according to the first day and fifth week GLIM classification groups were also evaluated. The characteristics of the patient and treatment were also evaluated according to anthropometric parameters, SMM, and nutritional status.

SMM assessment

Skeletal muscle mass was assessed by measuring cross-sectional muscle area at the C3 vertebral level on planning computed tomography (CT) scans obtained at baseline (Day 1) and at Week 5. All scans were acquired with a 2.5-mm slice thickness using the institutional radiotherapy simulation protocol.

Muscle contours were manually delineated at the C3 level according to previously validated anatomical landmarks and methodology.[8] Skeletal muscle tissue was segmented using Hounsfield unit thresholds of −29 to +150 HU and cross-sectional muscle area was calculated using the volume analysis tool of the treatment planning system (Eclipse v15.1, Varian Medical Systems).

CSMA was defined as the sum of the bilateral paravertebral muscles (PVM); the sternocleidomastoid muscles (SCM) were excluded from all measurements owing to the risk of contour inaccuracy in the presence of cervical lymph node involvement. The C3 skeletal muscle index (SMI) was calculated by dividing total CSMA by the square of the patient's height (cm^2/m^2).

Measurements were independently performed by two trained observers using a standardized segmentation protocol, both blinded to each other's measurements and to the patients' nutritional assessment results. To evaluate reproducibility, a randomly selected subset comprising 25% of the scans was reanalyzed. Intra- and interobserver agreement were assessed using a two-way random effects intraclass correlation coefficient (ICC) model with absolute agreement. The analysis demonstrated good reproducibility, with intraobserver ICC values of 0.86 (95% CI: 0.80–0.93) and interobserver ICC values of 0.82 (95% CI: 0.75–0.90).

Nutritional status assessment

Nutritional status assessment was based on NRS-2002 and GLIM criteria. Patients with NRS 2002 scores ≥ 3 were considered at risk of malnutrition that required nutritional intervention. Based on GLIM criteria, patients were classified as stage 1 (moderate malnutrition; weight loss: 5–10% within the past 6 months, or 10–20% beyond 6 months; low BMI: < 20 if < 70 years, < 22 if ≥ 70 years, reduced muscle mass: mild to moderate deficit) and stage 2 (severe malnutrition; weight loss: >10% within the past 6 months, or >20% beyond 6 months; low BMI: < 18.5 if <70 years, < 20 if ≥ 70 years; reduced muscle mass: severe deficit) [9].

For the etiologic criterion, all patients were classified as meeting the criterion of 'disease burden/inflammation' on the basis of a confirmed active malignancy requiring anticancer treatment, consistent with the GLIM framework's guidance that cancer constitutes a chronic disease with systemic inflammation.

Nutritional intervention and monitoring

All patients received standardized nutritional counseling and oral nutritional supplements (ONS) starting at treatment initiation. Standard, commercially available balanced ONS formulations routinely used in clinical practice were prescribed (approximately 1.2–1.5 kcal/mL; 200–220 mL per serving), without disease-specific or protein-enriched formulations.

The initial prescription consisted of 2–3 bottles per day, with dose adjustments based on weekly body weight trends, treatment tolerance, and patient-reported intake.

Nutritional monitoring was performed weekly through structured face-to-face physician-led interviews (approximately 10 minutes each) during RT/CRT. Each interview included a symptom-directed assessment (dysphagia, odynophagia, mucositis, nausea, and taste alterations), a 24-hour dietary recall, and a pragmatic adherence evaluation based on patient-reported supplement consumption. Bottle counts were used to corroborate self-reported adherence where available.

Adherence to ONS was pragmatically categorized as acceptable when patients reported consuming approximately ≥75% of the prescribed amount on most days of the week. Given the pragmatic outpatient design, total caloric and protein intake from all dietary sources was not formally quantified.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics version 23.0 (IBM Corp., Armonk, NY). Descriptive statistics were presented as means ± standard deviation (SD), medians (min-max), or percentages. The Shapiro-Wilk test was used to assess normality. Parametric data were analyzed using paired t-tests and one-way analysis of variance (ANOVA) with post hoc Tukey HSD tests, while non-parametric data were analyzed using the Mann-Whitney U test and Kruskal-Wallis test. Categorical variables were compared using the chi-square test, with the McNemar-Bowker test applied for time-based comparisons. Pearson's correlation coefficients were calculated for relationships between quantitative variables. $P < .05$ was considered statistically significant.

Results

Baseline characteristics

The study population included 32 HNC patients with a median age of 60 years (range: 18–85 years), of whom 81.3% were male. Hypertension and chronic obstructive pulmonary disease (COPD) were the most common comorbidities (18.8% each), and 37.5% of patients were active smokers. Most tumors were located in the larynx (56.3%) or oral cavity (21.9%), and the predominant histological subtype was squamous cell carcinoma (93.8%). Overall, 65.6% of patients had locally advanced diseases (T3-T4). A total of 68.8% of patients received CRT, while 31.3% underwent RT alone.

All patients were managed exclusively in an outpatient setting throughout the five-week treatment period. No patient required hospitalization, nasogastric tube feeding, percutaneous endoscopic gastrostomy (PEG), or parenteral nutritional support during the study. No grade 3 acute toxicity occurred that necessitated escalation of nutritional support or treatment interruptions longer than 2 days. Among the 32 enrolled patients, 27 (84.3%) demonstrated acceptable adherence to the prescribed ONS regimen (≥75% of the prescribed daily dose on most monitored days) based on weekly interview-based assessment. The remaining five patients (15.6%) reported suboptimal adherence, primarily attributable to nausea (n = 3), odynophagia (n = 1), and dysgeusia (n = 1).

Further baseline characteristics are detailed in Table 1.

Changes in anthropometrics and SMM during treatment

Between Day 1 and Week 5 of treatment, there were significant reductions in body weight (mean±SD: 69.38 ± 15.43 kg vs. 65.98 ± 16.08 kg, $P = .001$), BMI (23.83 ± 4.74 vs. 22.92 ± 4.68 kg/m², $P < .001$), and calf circumference (36.39 ± 3.12 cm vs. 35.66 ± 2.88 cm, $P < .001$) (Table 2). The percentage weight loss averaged 5.08% (mean ± SD: 5.08 ± 9.21%) across the study population, with significant differences observed between GLIM stages ($P = .004$).

Table 1. Baseline characteristics

Patient characteristics		
Age (year)	Mean (SD)	57.8 (15.1)
	Median (min-max)	60 (18-85)
Age group, n (%)	≤65 year	21 (65.6)
	>65 year	11 (34.4)
Sex, n (%)	Male	26 (81.3)
	Female	6 (18.8)
Comorbidity, n (%)	Hypertension	6 (18.8)
	Diabetes	4 (12.5)
	COPD	6 (18.8)
	Other	6 (18.8)
Active smoking, n (%)		12 (37.5)
Tumor characteristics, n (%)		
Histological subtype	Squamous cell carcinoma	30 (93.8)
	Undifferentiated	1 (3.1)
	Adeno-squamous	1 (3.1)
TNM Stage	0	1 (3.1)
	Stage I	4 (12.5)
	Stage II	6 (18.8)
	Stage III	5 (15.6)
	Stage IV	16 (50.0)
Treatment characteristics, n (%)		
Type of treatment	CRT	22 (68.8)
	RT	10 (31.3)
RT approach	Definitive	18 (56.3)
	Adjuvant	14 (43.8)
RT field	Nasopharynx	4 (12.5)
	Oropharynx	1 (3.1)
	Hypopharynx	1 (3.1)
	Larynx	18 (56.3)
	Oral cavity	7 (21.9)
	Unknown	1 (3.1)
RT Dosage	≤50 Gy	1 (3.1)
	>50 Gy	31 (96.9)

COPD: chronic obstructive pulmonary disease; RT: Radiotherapy; CRT: Chemoradiotherapy

There was also a significant decrease in cross-sectional muscle area (CSMA) at the C3 vertebra level (4.76 ± 1.39 cm^2 vs. 4.46 ± 1.21 cm^2, P = .038), while changes in skeletal muscle index (SMI) were not statistically significant (P = .058). (Table 2)

Nutritional status at baseline and week 5

The prevalence of malnutrition based on GLIM criteria increased significantly from 46.9% at baseline to 71.9% by Week 5 (P = .005). Of these, the proportion of patients classified as severely malnourished (Stage 2) increased from 9.4% to 28.1%. Similarly, the percentage of patients at risk of malnutrition based on NRS-2002 scores (≥ 3) increased from 40.6% at baseline to 71.9% at Week 5 (P = .002). (Table 2)

Correlation of NRS-2002 scores with anthropometrics and SMM

Anthropometric parameters (body weight, BMI and mid-arm circumference) were correlated negatively with first day and 5th week NRS-2002 scores (r: ranged from -0.387 for mid-arm circumference to -0.609 for BMI; P ranged from .03 to < .001) and positively with first day and 5th week CSMA (r: ranged from 0.598 for mid-arm circumference to 0.699 for body weight; P < .001) and skeletal muscle index (r: ranged from 0.538 for mid-arm circumference to 0.648 for BMI; P ranged from .004 to < .001) measured at the level of C3. (Table 3)

No significant correlations were observed between NRS-2002 scores and C3-derived skeletal muscle parameters at either time point (all P > .05; Table 3).

Correlation of GLIM categories with anthropometrics and SMM

Staging based on GLIM criteria was negatively correlated significantly with both BMI (r = -0.391, P = .027) and midarm circumference (r = -0.356, P = .045) on the first day, while only with BMI (r = -0.435, P = .013) in the fifth week. By Week 5, only BMI retained statistical significance with GLIM classification. No significant correlation was observed between GLIM stages and SMM parameters. (Table 3).

Table 2. First day vs. 5th week data on anthropometrics, SMM and nutritional status

		First day		5th week		P value[1]
		Mean±SD	Median(min-max)	Mean±SD	Median(min-max)	
Anthropometrics						
Weight (kg)		69.38 ± 15.43	67.00 (45.00 - 95.00)	65.98 ± 16.08	65.20 (27.00 - 91.00)	.001
BMI (kg/m^2)		23.83 ± 4.74	23.65 (13.90 - 30.70)	22.92 ± 4.68	21.85 (14.00 - 31.20)	< .001
Mid arm circumference (cm)		29.78 ± 3.73	30.50 (21.00 - 35.50)	29.42 ± 3.80	30.00 (19.50 - 35.00)	.15
Calf circumference (cm)		36.39 ± 3.12	36.25 (31.00 - 43.00)	35.66 ± 2.88	36.25 (30.00 - 42.00)	< .001
SMM (C3)						
CSMA (cm^2)		4.76 ± 1.39	4.50 (2.30 - 7.40)	4.46 ± 1.21	4.45 (2.20 - 6.50)	.04
SMI (cm^2/m^2)		1.64 ± 0.46	1.60 (0.77 - 2.61)	1.54 ± 0.44	1.47 (0.80 - 2.48)	.06
Nutritional status						
NRS-2002 assessment						
Total score		2.31 ± 1.28	2.00 (1.00 - 5.00)	3.19 ± 1.31	3.00 (1.00 - 5.00)	< .001
Risk of malnutrition (≥3), n (%)		13 (40.6)		23 (71.9)		.002[2]
GLIM classification, n (%)						
Non-malnourished		17 (53.1)		9 (28.1)		.005[3]
Malnourished	Total	15 (46.9)		23 (71.9)		
	Stage 1	12 (37.5)		14 (43.8)		
	Stage 2	3 (9.4)		9 (28.1)		

BMI: Body mass index; SMM: Skeletal muscle mass; CSMA: Cross-sectional muscle area; SMI: Skeletal muscle index, NRS: Nutritional Risk Screening, GLIM: Global Leadership Initiative on Malnutrition

[1]Paired samples t test, [2]McNemar test, [3]McNemar-Bowker test

Change over time in nutritional status by GLIM assessment

When stratified by GLIM classification, 50% of patients who were moderately malnourished (Stage 1) at baseline progressed to Stage 2 by Week 5. Among the non-malnourished at baseline, 41.2% developed malnutrition (Stage 1 or 2) during treatment. (Table 4)

NRS-2002 scores, anthropometrics and SMM according to GLIM groups

Stage 1 and stage 2 vs. non-malnourished GLIM groups on the first day had a significantly higher first day (3.17 ± 1.03 and 4.00 ± 1.00 vs. 1.41 ± 0.62, P < .001) and 5th week (4.00 ± 0.74 and 4.67 ± 0.58 vs. 2.35 ± 1.12, P = .002) NRS-2002 scores.

Stage 1 and stage 2 vs. non-malnourished GLIM groups on the 5th week also had significantly higher first day (2.50 ± 1.23 and 3.00 ± 1.41 vs. 1.33 ± 0.50, P = .012) and 5th week (3.50 ± 0.94 and 4.22 ± 0.67 vs. 1.67 ± 0.87, P < .001) NRS-2002 scores. (Table 5)

The 5th week vs. the first day NRS-2002 scores were significantly higher in non-malnourished (2.35 ± 1.12 vs. 1.41 ± 0.62, P = .001) and stage 1 malnourished (4.00 ± 0.74 vs. 3.17 ± 1.03, P = .005) patients based on the first day GLIM assessment, and in stage 1 (3.50 ± 0.94 vs. 2.50 ± 1.23, P < .001) and stage 2 (4.22 ± 0.67 vs. 3.00 ± 1.41, P = .005) malnourished patients based on the 5th week GLIM assessment. (Table 5)

SMM parameters did not differ significantly across GLIM groups at either assessment point (all P > .05; Table 5). Given the small number of patients within each GLIM subgroup, these comparisons should be considered exploratory.

Table 3. Correlation of NRS-2002 scores with anthropometrics and skeletal muscle assessment

		NRS-2002 score		GLIM-stage		C3 CSMA		C3 SMI	
		First day	5th week	First day	5th week	First day	5th week	First day	5th week
C3 CSMA	r	-0.060	-0.225	-0.099	-0.182	-	-	0.952	0.945
	p	.75	.22	.59	.33	-	-	**< .001**	**< .001**
C3 SMI	r	-0.087	-0.285	-0.185	-0.210	0.952	0.945	-	-
	p	.64	.11	.31	.25	**<.001**	**<.001**	-	-
Body weight	r	-0.527	-0.430	-0.252	-0.26	0.649	0.689	0.564	0.542
	p	.002	.01	.16	.15	**< .001**	**< .001**	**.001**	**.001**
Body mass index	r	-0.545	-0.609	-0.391	-0.435	0.622	0.674	0.630	0.648
	p	.001	< .001	.03	.01	**<.001**	**<.001**	**<.001**	**< .001**
Midarm circumference	r	-0.454	-0.387	-0.356	-0.19	0.661	0.598	0.635	0.538
	p	.009	.03	.05	.30	**< .001**	**< .001**	**< .001**	**.001**
Calf circumference	r	-0.335	-0.257	-0.109	-0.024	0.385	0.283	0.278	0.136
	p	.06	.16	.55	.90	.03	.12	.12	.46

CSMA: Cross-sectional muscle area; SMI: Skeletal muscle index
Pearson correlation analysis; r: correlation coefficient

Table 4. Comparison of the first day versus the fifth week of nutritional status based on GLIM assessment

	GLIM-5th week			P value
	Non-malnourished (n=9)	Stage 1 (n=14)	Stage 2 (n=9)	
GLIM-first day, n(%)				
Non-malnourished (n=17)	9 (100.0)	7 (50.0)	1 (11.1)	.005
Stage 1 (n=12)	0 (0.0)	7 (50.0)	5 (55.6)	
Stage 2 (n=3)	0 (0.0)	0 (0.0)	3 (33.3)	

McNemar-Bowker test

Patients classified as not malnourished on the first day and fifth week of GLIM evaluation had significantly higher BMI compared to stage 1 (25.80 ± 3.78 vs. 21.46 ± 4.87 kg/m2, P = .037) and stage 1 and stage 2 patients (26.79 ± 4.09 vs. 21.39 ± 4.29 and 21.44 ± 3.86, P = .009), respectively. (Table 5)

Characteristics of the patient and treatment according to study parameters

Females vs. males (median -1.50 vs. -1.00, P = .03) and patients who received CRT vs. RT (median -1.0 vs. 0.0, P = .030) had more marked deterioration in NRS-2002 scores, while definitive vs. adjuvant RT (72.2 vs. 28.6%, P = .02) was applied more frequently in non-malnourished patients based on first day GLIM (Table 6).

Age, smoking status and tumor stage had no significant influence on anthropometrics, SMM and nutritional status parameters (Table 6).

Discussion

Although several studies have investigated GLIM criteria in HNC patients[10,12,13], the present study adds to this literature by simultaneously incorporating longitudinal C3-based skeletal muscle assessment alongside both GLIM staging and NRS-2002 monitoring within a prospective real-world outpatient cohort. This integrated,

Table 5. NRS-2002 scores, anthropometrics and SMM according to the first day and fifth week GLIM classification

	GLIM- first day				GLIM-5th week			
	Non-malnourished	Malnourished			Non-malnourished	Malnourished		
		Stage 1	Stage 2	p value[2]		Stage 1	Stage 2	P value[2]
NRS-2002 scores, mean±SD								
First day	1.41 ± 0.62	3.17 ± 1.03	4.00 ± 1.00	<.001	1.33 ± 0.50	2.50 ± 1.23	3.00 ± 1.41	.01
5th week	2.35 ± 1.12	4.00 ± 0.74	4.67 ± 0.58	.002	1.67 ± 0.87	3.50 ± 0.94	4.22 ± 0.67	< .001
p value[1]	.001	.005	.18		.08	<.001	.005	
Difference	-0.94 ± 0.90	-0.83 ± 0.83	-0.67 ± 0.58	.86	-0.33 ± 0.50	-1.00 ± 0.78	-1.22 ± 0.97	.053
Anthropometrics, mean±SD								
Body weight (kg)	73.82 ± 13.64	63.50 ± 16.35	67.67 ± 18.72	.21	75.80 ± 13.29	60.41 ± 16.74	64.83 ± 14.35	.08
BMI (kg/m^2)	25.80 ± 3.78	21.46 ± 4.87	22.17 ± 5.73	.04	26.79 ± 4.09	21.39 ± 4.29	21.44 ± 3.86	.009
Calf circumference (cm)	36.82 ± 2.84	35.75 ± 3.03	36.50 ± 5.63	.67	36.51 ± 2.55	34.68 ± 2.68	36.33 ± 3.32	.24
Mid-arm circumference (cm)	31.32 ± 3.05	27.71 ± 3.93	29.33 ± 3.21	.030	31.33 ± 3.16	28.18 ± 4.20	29.44 ± 3.24	.15
SMM, mean±SD								
C3 CSMA (cm^2)	4.94 ± 1.33	4.48 ± 1.54	4.83 ± 1.40	.69	4.81 ± 1.14	4.37 ± 1.19	4.23 ± 1.37	.58
C3 SMI (cm^2/m^2)	1.73 ± 0.45	1.51 ± 0.49	1.59 ± 0.43	.47	1.71 ± 0.44	1.52 ± 0.46	1.39 ± 0.39	.31

BMI: Body mass index; SMM: Skeletal muscle mass; CSMA: Cross-sectional muscle area; SMI: Skeletal muscle index
[1]Paired samples t test, [2]One way ANOVA

Table 6. Characteristics of the patient and treatment according to anthropometrics, SMM, and nutritional status

	Difference (first day score - 5th week score), mean±SD				GLIM-first day, n(%)			GLIM-5th week, n(%)		
	BMI (kg/m²)	C3 CSMA (cm²)	C3 SMI (cm²/m²)	NRS-2002 score	Non-malnourished	Stage 1	Stage 2	Non-malnourished	Stage 1	Stage 2
Sex										
Female	1.40 ± 0.87	0.25 ± 0.18	0.08 (0.03 - 0.18)	-1.50 (-2.00 -1.00)	3 (50.0)	3 (50.0)	0 (0.0)	1 (16.7)	2 (33.3)	3 (50.0)
Male	0.80 ± 1.29	0.32 ± 0.88	0.10 (-0.95 - 0.66)	-1.00 (-3.00 - 0.00)	14 (53.8)	9 (34.6)	3 (11.5)	8 (30.8)	12 (46.2)	6 (23.1)
p value	.29[1]	.86[1]	.96[2]	.03[2]	.60[3]			.41[3]		
Age group										
<65 year	1.15 ± 1.28	0.31 ± 0.50	0.11 ± 0.18	-1.00 (-3.00 - 0.00)	12 (57.1)	7 (33.3)	2 (9.5)	6 (28.6)	8 (38.1)	7 (33.3)
> 65 year	0.45 ± 1.03	0.29 ± 1.20	0.08 ± 0.43	0.00 (-2.00 - 0.00)	5 (45.5)	5 (45.5)	1 (9.1)	3 (27.3)	6 (54.5)	2 (18.2)
p value	.12[1]	.96[1]	.87[1]	.11[2]	.79[3]			.60[3]		
Smoking										
No	0.91 ± 1.12	0.28 ± 0.94	0.05 (-0.95 - 0.66)	-1.00 (-2.00 - 0.00)	10 (50)	8 (40)	2 (10)	5 (25)	9 (45)	6 (30)
Yes	0.92 ± 1.45	0.34 ± 0.50	0.13 (-0.17 - 0.55)	-0.50 (-3.00 - 0.00)	7 (58.3)	4 (33.3)	1 (8.3)	4 (33.3)	5 (41.7)	3 (25)
p value	.98[1]	.84[1]	.86[2]	.35[2]	.90[3]			.87[3]		
Disease stage										
Early stage (stage I-II)	0.68 ± 0.91	0.20 (-0.40 - 1.50)	0.07 (-0.16 - 0.55)	-0.50 (-2.00 - 0.00)	6 (60)	4 (40)	0 (0)	4 (40)	4 (40)	2 (20)
Advanced (stage III-IV)	0.95 ± 1.36	0.40 (-2.50 - 2.10)	0.13 (-0.95 - 0.66)	-1.00 (-3.00 - 0.00)	10 (47.6)	8 (38.1)	3 (14.3)	5 (23.8)	9 (42.9)	7 (33.3)
p value	.58[1]	.70[2]	.75[2]	.24[2]	.44[3]			.60[3]		
RT approach										
Definitive	0.79 ± 1.46	0.20 (-2.50 - 1.50)	0.07 (-0.95 - 0.55)	-0.50 (-3.00 - 0.00)	13 (72.2)	5 (27.8)	0 (0)	7 (38.9)	7 (38.9)	4 (22.2)
Adjuvant	1.06 ± 0.89	0.35 (-0.60 - 2.10)	0.12 (-0.23 - 0.66)	-1.00 (-2.00 - 0.00)	4 (28.6)	7 (50.0)	3 (21.4)	2 (14.3)	7 (50)	5 (35.7)
p value	.56[1]	.78[2]	.70[2]	.50[2]	.02[3]			.30[3]		
Treatment										
CRT	1.20 ± 1.37	0.25 (-2.50 - 2.10)	0.08 (-0.95 - 0.66)	-1.00 (-3.00 - 0.00)	11 (50)	8 (36.4)	3 (13.6)	4 (18.2)	10 (45.5)	8 (36.4)
RT	0.26 ± 0.41	0.25 (-0.40 - 1.50)	0.09 (-0.16 - 0.55)	0.00 (-1.00 - 0.00)	6 (60)	4 (40)	0 (0)	5 (50)	4 (40)	1 (10)
p value	.006[1]	.54[2]	.58[2]	.03[2]	.47[3]			.12[3]		

BMI: Body mass index; CSMA: Cross-sectional muscle area; SMI: Skeletal muscle index; RT: radiotherapy; CRT: Chemoradiotherapy
[1]Independent samples t test, [2]Mann-Whitney U test, [3]χ^2 test

multimodal approach enables a direct comparison of assessment tools and muscle metrics under routine clinical conditions, which has not been systematically reported in previous studies focusing primarily on malnutrition prevalence.

Malnutrition remains a significant concern in HNC patients due to the combined effects of tumor biology, treatment-related toxicities, and decreased oral intake.[14-16] The baseline malnutrition prevalence observed in this study (46.9%) is consistent with prior research, reflecting the inherent nutritional vulnerability of this patient population.[10,15,17] By Week 5, prevalence increased to 71.9%, consistent with evidence that malnutrition frequently worsens during anticancer treatment owing to the cumulative mucosal and systemic toxicities of RT or CRT, including mucositis, dysphagia, and anorexia.[12,13]

NRS-2002 scores correlated significantly with GLIM-based malnutrition stages at baseline, and this concordance was maintained through Week 5 of treatment. These findings support the feasibility of applying GLIM criteria to prospectively identify patients at high nutritional risk prior to anticancer treatment, including those with severe malnutrition (Stage 2) who are at higher risk of postoperative complications and 90-day all-cause mortality.[9,18,19]

In addition, NRS-2002 scores were significantly associated with body weight, BMI and mid-arm circumference, whereas GLIM-defined malnutrition was significantly correlated with BMI alone at both time points. This pattern likely reflects the dominant role of low BMI as the most frequently met GLIM phenotypic criterion in this cohort, given that the majority of patients had BMI values near or below the GLIM threshold (<20 kg/m² for patients under 70 years). Weight loss and reduced muscle mass criteria, while clinically present, may have been less consistently met or may not have reached phenotypic threshold severity in all patients at baseline.[20,21]Similarly, Steer et al. reported that GLIM-defined malnutrition in HNC patients was significantly associated with BMI alone.[10]

The lack of significant association between GLIM classification and C3-derived muscle parameters may reflect the short observation period, limited sample size, and methodological challenges of detecting early muscle changes during active RT/CRT rather than a true lack of biological relationship.

In the present cohort, despite standardized ONS prescription aligned with ESPEN recommendations and weekly clinical monitoring, significant declines in body weight, BMI, and C3 CSMA were observed throughout the five-week treatment period. However, as total dietary intake from all sources was not formally quantified, the degree to which individual patients met recommended energy and protein targets cannot be confirmed; the observed nutritional deterioration should therefore be interpreted in the context of prescribed but incompletely verified nutritional support. These findings highlight the limitations of standard ONS-based interventions in HNC patients, particularly those with severe baseline nutritional deficits. Prior studies have reported similarly mixed results regarding the efficacy of ONS alone in maintaining nutritional status during RT or CRT, and nutritional strategies incorporating proactive measures — such as prophylactic enteral feeding or intensive individualized dietary counseling — may better address the complex needs of this population.[22-26]

CRT was associated with greater declines in BMI and nutritional status compared with RT alone, reflecting the heightened toxicity of combined treatments. This is consistent with prior evidence identifying CRT as a major risk factor for nutritional deterioration, owing to its exacerbation of mucositis, dysphagia, and gastrointestinal symptoms.[12,27-30] Early and intensive nutritional intervention is therefore essential to minimize treatment interruptions and improve clinical outcomes in these patients.[12,22,29]

C3 cross-sectional muscle area (CSMA) has been proposed as a practical alternative to L3-based skeletal muscle assessment in HNC patients, given its routine availability on planning CT imaging.[7,8] In this study, C3 CSMA declined significantly during treatment, whereas changes in the skeletal muscle index (SMI) did not reach statistical significance, and neither parameter was significantly correlated with GLIM staging or NRS-2002 scores. The lack of significant association between GLIM classification and C3-derived muscle parameters may reflect the short observation period, limited sample size, and methodological challenges inherent to detecting early muscle changes during active RT/CRT, rather than a true absence of biological relationship. Similar constraints have been reported in prior studies evaluating C3-based assessments, particularly regarding sensitivity to small but clinically meaningful muscle losses.[31]

A further consideration is the potential influence of local radiotherapy-related tissue changes on C3 muscle

measurements. In the acute treatment phase, soft tissue edema and early lymphatic alterations within the irradiated neck region could theoretically affect cross-sectional area estimates. However, clinically relevant RT-induced skeletal muscle atrophy and volumetric changes are generally reported months to years after treatment completion rather than within the first weeks of therapy.[32,33] For this reason, the Week 5 assessment time point was intentionally selected to capture early treatment-related nutritional changes while minimizing exposure to long-term structural RT effects. Nevertheless, the possibility that local treatment-related tissue changes may partially confound C3 CSMA measurements cannot be entirely excluded and should be considered when interpreting these results.

By incorporating percentage weight loss and skeletal muscle metrics (CSMA and SMI) into the assessment, this study adds to the growing evidence supporting GLIM as a robust tool for malnutrition diagnosis and staging. The findings further suggest that GLIM can identify patients at higher risk of adverse outcomes, such as those with severe malnutrition (Stage 2), a subgroup often overlooked in routine clinical care.

This study has several limitations. First, the relatively small sample size (n = 32), heterogeneous distribution of tumor subsites (larynx 56.3%, oral cavity 21.9%, other 21.8%), range of treatment indications (definitive vs. adjuvant RT/CRT), and single-center design limit the generalizability of these findings. Additionally, the predominantly male cohort (81.3%) may reduce applicability to female HNC patients, in whom body composition thresholds for muscle depletion may differ. Second, total energy and protein intake from all dietary sources were not formally quantified. Although weekly structured interviews included a 24-hour dietary recall and pragmatic adherence assessment, the accuracy of patient-reported supplement consumption is subject to recall bias. Third, although C3-based measurements were performed using a standardized protocol with high reproducibility, local RT-related tissue changes may partially influence neck-level muscle estimates. Fourth, the absence of a control group precludes definitive conclusions regarding the effectiveness of nutritional support in mitigating malnutrition progression. Finally, given the small number of patients within each GLIM subgroup, subgroup-level findings should be interpreted as exploratory.

Future studies with larger cohorts and more robust methodologies are needed to validate these findings and explore the utility of GLIM criteria in predicting long-term clinical outcomes.

In conclusion, our findings revealed deterioration in anthropometrics, C3 CSMA, and NRS-2002 scores along with no change or even deterioration in baseline malnourished status according to GLIM in most HNC patients under anticancer treatment. Although all patients received standardized ONS prescription and weekly monitoring, progression of malnutrition was still observed during treatment. This likely reflects the complex multifactorial nature of nutritional decline in HNC, where treatment-related symptoms, inflammation, and catabolic stress may overcome nutritional interventions, particularly when nutritional intake cannot be strictly quantified in routine outpatient settings. NRS-2002 scores were significantly correlated with GLIM-based stages at baseline and the relationship between the two tests also maintained its significance over time, whereas neither GLIM stages nor NRS-2002 scores were associated with SMM parameters (C3 CSMA and skeletal mass index) within the short observation period. These findings suggest that anthropometric and clinical screening tools remain central in early nutritional monitoring, while the role of C3-based measurements requires further validation in larger studies with longer follow-up.

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Author contributions

Conception and design: M.A.; Data acquisition: M.A., C.G.; Data analysis: M.A., C.G.; Data interpretation: M.A., C.G.; Drafting of the manuscript: M.A.; Critical revision of the manuscript: M.A., C.G. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Gazi University Ethics Committee (Date: March 16, 2020, Decision/Protocol No: 452). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable requests subject to institutional regulations and applicable ethical/privacy restrictions.

Conflict of interest

The authors declare the following potential conflict of interest: M.A. reports a relationship with Turkish Society for Radiation Oncology (TROD) that includes: unpaid Board Member.

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Generative AI statement

The authors declare that during the preparation of this study, the following AI-assisted technology was used: ChatGPT-5, OpenAI on 18/12/2025. Extent of Use: AI-assisted technology was used only for language editing, grammar and clarity improvement, and proofreading. It was not used for study conception or design, data collection, data extraction, statistical analysis, data interpretation, figure generation, or creation of scientific conclusions. All AI-assisted suggestions were critically reviewed and edited by the authors, who take full responsibility for the integrity, accuracy, originality, and final content of the manuscript. The authors confirm that they have critically reviewed and edited any AI-generated content and take full responsibility for the integrity, accuracy, and originality of the publication. The authors certify that the original human contribution is maintained and that AI-assisted tools are not listed or cited as authors.

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Oral health and its relationship with frailty, sarcopenia risk and nutritional status in middle-aged and older adults undergoing dental treatment

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ABSTRACT

Objective: Middle-aged and older adults with poor oral health may be at higher risk of being frail during dental treatment. This study aimed to investigate the associations between oral health status and frailty, as well as their relationships with malnutrition and sarcopenia risk, in middle-aged and older adults undergoing dental treatment.

Methods: This cross-sectional study included adults aged ≥55 years receiving dental care for restorative or prosthetic treatment. Nutritional status, sarcopenia risk, and frailty were assessed using the Mini Nutritional Assessment, the SARC-F questionnaire, and the FRAIL scale, respectively. Oral health status was assessed using the Geriatric Oral Health Assessment Index (GOHAI).

Results: A total of 90 adults were included in the study, of whom 58 (64.4%) were classified as pre-frail/frail. Participants in the pre-frail/frail group were significantly older and showed higher rates of malnutrition risk/malnutrition and sarcopenia risk compared with those with non-frailty. The pre-frail/frail group had lower GOHAI scores, greater tooth loss, different dental treatment profiles, and more frequent weight loss after dental treatment. GOHAI scores were associated with frailty, sarcopenia risk and nutritional status.

Conclusion: Poor oral health was associated with higher frailty, malnutrition and sarcopenia risk among middle-aged and older adults receiving dental care. These findings underscore the importance of incorporating comprehensive nutritional assessments into dental treatment to identify older adults and support multidisciplinary care approaches.

Keywords: frailty, oral health, sarcopenia, malnutrition, geriatric dentistry

Introduction

Oral health is a fundamental component of overall health and functional well-being in older adults.[1] Its importance has become increasingly evident as the global prevalence of oral diseases continues to rise. Oral diseases represent some of the most common non-communicable diseases worldwide and include both inflammatory and non-inflammatory conditions such as dental caries, periodontal disease, tooth loss, oral cancer, xerostomia, and dysphagia, all of which disproportionately affect older populations.[2] Age-related physiological changes, the cumulative burden of chronic diseases, and declining oral hygiene practices further increase older adults' susceptibility to dental problems.[3,4]

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Malnutrition, sarcopenia, and frailty, which are nutrition related disorders highly prevalent among community-dwelling older adults and share common etiological factors, including chronic inflammation, inadequate food intake, reduced physical activity, age-related hormonal changes, and multiple sociodemographic, behavioral, and disease-related factors.[5-7] These conditions are associated with adverse health outcomes, such as functional decline, increased fall risk, reduced quality of life, hospitalization, and mortality.[8] Importantly, suboptimal oral health, including functional problems such as broken or missing teeth or ill-fitting dentures, can compromise nutritional intake by limiting food choices and impairing chewing and swallowing, thereby contributing to deteriorations in nutritional status among older adults.[9] A recent meta-analysis demonstrated that in older adults, fewer teeth, chewing or swallowing difficulties, dry mouth, and the absence of removable dentures were significantly associated with malnutrition, highlighting the close interplay between oral health and nutritional status in aging populations.[10] Emerging evidence indicates that nutritional status is closely associated with both dental status and frailty, highlighting that maintaining oral health alongside adequate nutrition is a key strategy to prevent or delay frailty in older adults.[11]

Current evidence supports a significant relationship between oral health and frailty. Poorer self-rated oral health is consistently associated with higher frailty in community-dwelling middle-aged and older adults.[12,13] Furthermore, recent systematic reviews indicate that a broad spectrum of oral health indicators, including dental-related measures, oral hygiene practices, oral functional parameters, and comprehensive oral health assessment scores are significantly associated with frailty in older adults.[14] Despite increasing recognition of these associations, studies specifically examining the interrelationships between oral health, frailty, sarcopenia, and nutritional status in older adults undergoing dental treatment remain limited. Given the high demand for dental treatments in aging populations, clarifying the relationship between oral health, nutritional assessment and functional status is essential for integrated dental care. Specifically, clarifying the impact of treatment-induced alterations in oral function on nutritional disorders is of importance in older adults, a population inherently vulnerable to malnutrition, sarcopenia, and frailty. Therefore, this study aimed to examine the associations between oral health status and frailty, as well as its relationships with malnutrition and sarcopenia risk, in middle-aged and older adults undergoing dental treatment.

Main Points

- Nutrition related disorders are a common problem during dental treatment.
- Perceived poor oral health is associated with higher frailty, sarcopenia risk, and malnutrition in middle-aged and older patients undergoing dental treatment.
- Frailty is associated with treatment parameters such as number of missing teeth, number of fillings, and type of treatment.

Materials and Methods

Study design and population

This cross-sectional study, conducted between July 2025 and October 2025, involved individuals aged 55 years and older who consecutively applied to the dental clinic. The exclusion criteria were severe cognitive impairment preventing reliable responses, neurological disease (e.g., recent stroke, parkinson disease), current chemotherapy/radiotherapy or immunosuppression and terminal-stage cancer, active oral disease or recent oral/maxillofacial surgery that prevents oral examination and hearing or speech disorders. The study was approved by the Ethics Committee of Sivas Cumhuriyet University. All participants were informed about the study, and each provided written voluntary consent in accordance with the principles of the Declaration of Helsinki.

Measurements

The sociodemographic and clinical variables recorded included age, sex, marital status, smoking status, the presence of chronic disease, and regular medication use. Oral health related variables comprised the type of dental treatment, treatment initiation date, and treatment duration. Participants were also asked about weight loss prior to dental treatment, pain or discomfort while eating due to dental problems, changes in eating habits after treatment, and changes in chewing function following treatment. Further clinical variables included the number of missing teeth, number of dental fillings, and the presence of a teeth-clenching habit. The patients' data

was obtained through the administration of a structured questionnaire and from their medical records.

Oral health assessment

The Geriatric Oral Health Assessment Index (GOHAI) was used to determine the oral health status of older adults.[15]The scale consists of 12 items and assesses oral health problems across three domains: physical function, psychosocial function, and pain or discomfort. Each item is scored on a 5-point Likert-type scale, yielding a total score ranging from 12 to 60, with higher scores indicating better oral health. Based on cut-off values used in previous studies, patients with GOHAI scores of 12–50 and 51–56 were classified as having low and moderate perceived oral health, respectively, whereas those with scores ≥ 57 were considered to have high perceived oral health.[16,17] The Turkish version of GOHAI is valid and reliable.[18]

Frailty assessment

Frailty was evaluated using the FRAIL scale, which consists of five components: fatigue, resistance, ambulation, illnesses, and weight loss. The total score ranges from 0 to 5, with higher scores indicating greater frailty. According to standard cut-off points, a score of 0 indicates robustness, scores of 1–2 indicate pre-frailty, and scores >2 indicate frailty.[19] The Turkish version of the FRAIL scale has been validated and shown to be reliable in older adults.[20] In the present study, participants were categorized into two groups: normal and pre-frailty/frailty.

Nutritional assessment

Anthropometric measurements were performed, including weight and height, from which body mass index (BMI, kg/m^2) was calculated. Weight was measured to the nearest 0.1 kg and height to the nearest 0.1 cm using a standardized stadiometer, with participants wearing light clothing and no shoes. Calf circumference was measured at the widest point of the calf. Nutritional status was assessed using the Mini Nutritional Assessment (MNA), a reliable tool widely used to evaluate the nutritional status of older adults.[21] According to the total MNA score, a score of ≥24 indicates "normal nutritional status," scores between 17 and 23.5 indicate "risk of malnutrition," and a score of <17 indicates "malnutrition".

Sarcopenia risk assessment

Sarcopenia risk was assessed using the SARC-F (The Simple Questionnaire for Rapidly Diagnose of Sarcopenia), which consists of five parameters evaluating the following components: strength, assistance in walking, rising from a chair, climbing stairs, and falls. The total score ranges from 0 to 10, and a score of ≥4 indicates an increased risk of sarcopenia.[22]

Statistical analysis

Statistical analyses were performed using SPSS (version 23, IBM). Normality of the variables was assessed using Kolmogorov–Smirnov test. Non-normally distributed variables were summarized using medians (Q1–Q3), normally distributed variables were presented as means ± standard deviations, and categorical variables were reported as frequencies and percentages. Comparisons between the normal and pre-frail/frailty groups were performed using the independent samples t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Associations between oral health status and nutritional and functional outcomes were assessed using Spearman's rank correlation analysis, with correlation coefficients (r) and p values reported. A p-value of < 0.05 was considered statistically significant.

Results

Baseline characteristics and nutritional status of the participants are presented in Table 1. The study included a total of 90 adults, of whom 58 (64.4%) were classified as pre-frail/frail. The mean age of the participants was 59.6 ± 4.1 years, and individuals in the pre-frail/frail group were significantly older than those in the normal group (60.7 ± 4.3 vs. 57.6 ± 3.0 years, $p = 0.001$). There was no significant difference between the groups in sex, marital status, smoking habits and anthropometric measurements ($p > 0.05$). Malnutrition status and sarcopenia risk were significantly associated with pre-frailty/frailty, with higher proportions observed in the pre-frail/frail group compared with the normal group ($p < 0.01$).

Table 2 presents oral health characteristics according to frailty status. The pre-frail/frail group had significantly lower median GOHAI scores than the normal group ($p = 0.011$), whereas GOHAI categories did not differ

Table 1. Sociodemographic, clinical, and nutritional characteristics of participants according to frailty status

	Normal	Pre-frail/frailty	p	Total
Age, year, mean±SD	57.6±3.0	60.7±4.3	0.001	59.6±4.1
Sex, n (%)				
Female	12 (37.5%)	32 (55.2%)	0.108	44 (48.9%)
Male	20 (62.5%)	26 (44.8%)		46 (51.1%)
Education				
Primary/secondary school	-	12 (20.7%)	0.007	12 (13.3%)
High School/university	32 (100%)	46 (79.3%)		78 (86.7%)
Marital status, n (%)				
Single/widowed/divorced	7 (21.9%)	12 (20.7%)	0.895	19 (21.1%)
Married	25 (78.1%)	46 (79.3%)		71 (78.9%)
Smoking, n (%)				
Yes	19 (59.4%)	28 (48.3%)	0.313	47 (52.2%)
No	13 (40.6%)	30 (51.7%)		43 (47.8%)
Chronic illness, n (%)				
Yes	15 (46.9%)	42 (72.4%)	0.016	57 (63.3%)
No	17 (53.1%)	16 (27.6%)		33 (36.7%)
BMI, kg/m^2, mean±SD	25.3±3.0	25.6±4.1	0.708	25.5±3.7
BMI classification, kg/m^2				
18.5-24.9	14 (43.8%)	25 (43.1%)		39 (43.3%)
25-29.9	16 (50.0%)	26 (44.8%)	0.664	42 (46.7%)
≥30	2 (6.2%)	7 (12.1%)		9 (10.0%)
Calf circumference, mean±SD	36.5±3.2	35.6±4.2	0.328	35.9±3.8
Malnutrition status, n (%)				
Normal nutrition	32 (100%)	39 (67.2%)	<0.001	71 (78.9%)
Malnutrition risk	-	14 (24.2%)		14 (15.5%)
Malnutrition	-	5 (8.6%)		5 (5.6%)
Sarcopenia risk, n (%)				
Normal	32 (100%)	43 (74.1%)	0.002	75 (83.3%)
Sarcopenia risk	-	15 (25.9%)		15 (16.7%)

significantly between groups ($p > 0.05$). There was a significant association between frailty status and dental treatment types, the number of missing teeth, and the number of filled teeth ($p < 0.05$). Weight loss after initiation of dental treatment was more frequently reported in the pre-frail/frail group ($p = 0.035$), while oral pain during eating did not differ between groups ($p > 0.05$).

As shown in Table 3, there was a negative correlation between GOHAI and FRAIL, SARC-F scores, and a positive correlation with MNA scores ($p < 0.01$). There is a positive correlation between FRAIL and SARC-F scores, and a negative correlation between FRAIL and MNA scores ($p < 0.001$).

Table 2. Oral health status, dental treatment characteristics, and eating-related outcomes according to frailty status

	Normal	Pre-frail/frailty	p	Total
GOHAI, median (Q1-Q3)	49.5 (42-53)	42.5 (32.8-51)	0.011	44.5 (34-52)
GOHAI classification, n (%)				
Low oral health	18 (56.2%)	42 (72.4%)	0.119	60 (66.7%)
Moderate oral health	14 (43.8%)	16 (27.6%)		30 (33.3%)
Type of dental treatment, n (%)				
Implant	15 (46.9%)	35 (60.3%)	0.002	50 (55.6%)
Endodontic and restorative treatment	17 (53.1%)	13 (22.4%)		30 (33.3%)
Dental prosthesis	-	10 (17.2%)		10 (11.1%)
Number of filled teeth, mean±SD	3.8±2.0	2.7±2.1	0.010	3.1±2.1
Number of missing teeth, mean±SD	4.8±3.6	10.6±8.4	<0.001	8.6±7.6
Weight loss after initiation of dental treatment, n (%)				
Yes	4 (12.5%)	19 (32.8%)	0.035	23 (25.6%)
No	28 (87.5%)	39 (67.2%)		67 (74.4%)
Oral pain or discomfort while eating, n (%)				
Yes	30 (93.8%)	511 (87.9%)	0.381	81 (90%)
No	2 (6.2%)	7 (12.1%)		9 (100%)
Improved eating habits after treatment, n (%)				
Yes	9 (28.1%)	33 (56.9%)	0.009	42 (46.7%)
No	23 (71.9%)	25 (43.1%)		48 (53.3%)

Table 3. Relation between oral health scale and nutritional disorders score

	FRAIL		MNA		SARC-F	
	r	p	r	p	r	p
GOHAI	-0.399	<0.001	0.590	<0.001	-0.322	0.002
FRAIL	-	-	-0.598	<0.001	0.721	<0.001
MNA	-0.598	<0.001	-	-	-0.564	<0.001
SARC-F	0.721	<0.001	-0.564	<0.001	-	-

Discussion

This study demonstrated an association between oral health status, as assessed by the GOHAI, and frailty, sarcopenia risk, and nutritional status among middle aged and older adults receiving dental treatment. In the present study population, participants in the pre-frail/frail group had lower GOHAI scores compared with non-frail participants, indicating poorer perceived oral health. Moreover, GOHAI scores were negatively correlated with FRAIL and SARC-F scores and positively correlated with MNA scores, suggesting that declines in oral health are associated with increased frailty and sarcopenia risk as well as poor nutritional status.

In this study, the prevalence of sarcopenia risk and malnutrition status among participants is consistent with previous systematic reviews and epidemiological studies conducted on community-dwelling older adults.[23-25] In contrast, a relatively high prevalence of pre-frailty/frailty (64.4%) was observed in the present study. A large meta-analysis including over 40,000 community-dwelling

adults aged ≥60 years reported a wide prevalence range of frailty (8–80%), with a pooled prevalence of 42%, highlighting that frailty is common but heterogeneous across populations.[26]The higher prevalence observed in the present study may be partially explained by the inclusion of individuals receiving dental treatment, who may represent a more vulnerable subgroup with underlying functional limitations and health problems.

Oral health status in the present study was assessed using the GOHAI, a widely validated instrument that evaluates physical and psychosocial functioning, pain, and discomfort as core components of oral health related quality of life, which are closely associated with dietary intake and functional capacity in both older and adult populations.[27,28] In the current study, GOHAI scores were negatively correlated with FRAIL and SARC-F scores and positively correlated with MNA scores, indicating that poor perceived oral health is associated with higher frailty and sarcopenia risk as well as poor nutritional status. Our results are consistent with previous studies reporting an association between poor self-rated oral health, as measured by GOHAI, and pre-frailty/frailty status[29], as well as findings from hospitalized older adults showing that poor self-reported oral health is independently associated with higher frailty scores, even after adjusting for nutritional status and comorbidities.[17] Likewise, oral health status has been widely reported to be associated with nutrition related disorders, including malnutrition, sarcopenia, and frailty, particularly among community-dwelling older adults.[30-32] Oral diseases may contribute to these conditions through multiple pathways such as pain, tooth loss, impaired mastication, infection, and chronic inflammation, all of which can lead to reduced dietary intake and exacerbate age-related pathophysiological changes.[33,34]

In recent years, oral frailty has received increasing attention and has been defined as an age-related decline in oral structure and function.[35,36] Previous studies have shown that oral frailty is associated with nutritional status and muscle function.[37,38] Although oral frailty was not specifically assessed in this study, the evaluation of overall oral health using GOHAI provides partial insight into oral status, encompassing some physical, functional, and psychosocial aspects. Further studies using specific oral frailty indices are needed to clarify these relationships.

In the present study, weight loss following the initiation of dental treatment was more frequently found among participants in the pre-frail/frail group. Although dental treatment is generally expected to improve oral function over time, short-term factors such as postoperative discomfort, adaptation to dental prostheses, and functional limitations associated with prosthetic rehabilitation may adversely affect dietary nutrient intake.[39,40] It should be noted that the weight loss data were obtained based on patient self-report, which may introduce bias. Despite this situation, these findings highlight the importance of integrating nutritional assessment and close monitoring into dental care, particularly for older adults with pre-existing frailty or sarcopenia risk.

A large population-based longitudinal study demonstrated that tooth loss was associated with a more rapid progression of frailty over time.[41] Similarly, previous studies have shown that progressively severe tooth loss, edentulism, and periodontal disease are associated with a higher risk of developing frailty, whereas a greater number of natural teeth is associated with a lower frailty risk.[42,43] These findings suggest that impaired oral health and functional limitations observed during dental treatment may be closely linked to frailty-related outcomes and could contribute to the accelerated frailty progression reported in longitudinal studies. This need is further supported by qualitative evidence indicating that, although general dental practitioners recognize the importance of providing dietary advice, its routine implementation in primary dental care is constrained by unclear professional roles, time pressures, and inadequate remuneration.[44] In this context, greater involvement of dietitians may help support individualized medical nutrition therapy for dental patients. Furthermore, systematic reviews suggest that combining dietary interventions with dental care can improve both masticatory function and nutritional outcomes.[45] These findings underscore the importance of addressing both oral function and nutrition concurrently to optimize health outcomes in older adults receiving dental treatment.

There are some limitations in the study. First, this study employed a cross-sectional design and recruited patients from a single dental clinic; therefore, causal inferences cannot be drawn. Second, this study enrolled individuals aged 55 and above, while most previous studies on oral health and frailty have focused on older adults, although some included participants starting from 40 years of age[13,46], which may affect the generalizability of the results. Third, sarcopenia risk was assessed using the SARC-F questionnaire, without direct measurements of muscle mass or function. Another limitation of this study is that oral health status was assessed using the GOHAI,

which is primarily based on self-reported symptoms and may not fully reflect objective clinical evaluations conducted by dental professionals. Therefore, there is potential reporting bias. Fourth, weight loss data during dental treatment were also based on patient self-report and were not documented in medical records, making them inherently subjective and limiting their reliability. Also, the relatively small sample size and the limited number of participants in certain subgroups restricted the ability to perform multivariate regression analyses. It is important to note that the absence of multivariate regression analysis is a significant limitation because it restricts consideration of potential confounding factors. Finally, due to the relatively small sample size and the cross-sectional nature of the study, the interpretation of the associations between oral health status, frailty, sarcopenia risk, and nutritional status during dental treatment should be made with caution.

In conclusion, oral health status had a significant association with frailty, nutritional status, and sarcopenia risk in older adults receiving dental treatment, with poorer perceived oral health linked to higher frailty and sarcopenia risk as well as nutritional impairment. Integrating routine nutritional screening and frailty assessment into dental care settings may enable dietitians to identify older adults at increased risk and facilitate early, multidisciplinary interventions. Future longitudinal and multicenter studies are warranted to clarify causal relationships, enhance the generalizability of findings, and determine whether targeted oral and nutritional interventions reduce the progression of frailty and improve overall health outcomes.

Author contributions

Conception and design: M.K., E.B.T.; Data acquisition: E.B.T.; Data analysis: M.K.; Data interpretation: M.K.; Drafting of the manuscript: M.K.; Critical revision of the manuscript: M.K. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Sivas Cumhuriyet University Health Sciences Research Ethics Committee (Date: July 10, 2025 (July 10, 2025), Decision/Protocol No: 2025-07/46). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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Knowledge levels of nursing students regarding the nutritional care of surgical patients: a descriptive study

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ABSTRACT

Objective: This study aimed to assess the knowledge levels of nursing undergraduate students regarding the nutritional care of surgical patients.

Methods: The descriptive study included 240 second-, third-, and fourth-year nursing students at a university in Türkiye's capital during the 2024–2025 academic year. Students were eligible if they were enrolled in these years of the nursing program, volunteered to participate, and had Internet access. The Student Demographic Information Form and the Questionnaire for Nutritional Care of Surgical Patients were used to collect the research data. Kruskal-Wallis H test was used to data analysis.

Result: The median age of the students was 22 (min-max: 19-26) years. Most (84.6 %) nursing students were female, and second-year students (47.5 %). The evaluation of students' knowledge levels by grade showed that second-year students had the highest scores (35.26 ± 10.73), while fourth-year students had the lowest (32.52 ± 7.84). The median scores were 36, 32, 32, and 36 for second-year, third-year, fourth-year, and total groups, respectively. The overall mean for all students was 34.73±10.50. No statistically significant difference was found in knowledge levels between grades (p=0.065).

Conclusion: Nursing students demonstrated only moderate knowledge of nutritional care for surgical patients, with notable gaps in key areas, including malnutrition, timing of postoperative nutrition, and enteral–parenteral nutrition principles. The findings underscore the need to integrate a dedicated course on nutritional care, encompassing both theoretical and practical components, into the curriculum.

Keywords: knowledge, nursing students, nutritional care, surgical patients

Introduction

Malnutrition is defined as a condition arising from insufficient intake or absorption of nutrients, resulting in altered body composition (reduced fat-free mass) and body cell mass, which leads to lower physical and mental function and impaired clinical outcomes from disease.[1,2] In surgical patients, prolonged preoperative fasting, the metabolic and endocrine response to surgical trauma, surgical complications, and patients' inability to express hunger and inadequate nutrient intake are risk factors for malnutrition. [3,4] It is reported that approximately 24-65% of patients who undergo surgery are at risk of malnutrition.[3]Nutritional therapy aims to prevent existing and potential malnutrition, strengthen the immune system, accelerate wound healing, minimize catabolic effects, and reduce mortality, complications, and

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infection rates associated with malnutrition in patients hospitalized in surgical clinics.[5,6]

Nurses play a significant role and bear considerable responsibility in nutritional care.[7,8] It plays an essential role in early identification of patients at risk of malnutrition, and in implementing nutritional interventions.[1] However, it has been reported that nurses' education and knowledge of nutritional care are insufficient, potentially limiting their ability to identify and manage malnutrition in patients.[9,10]

The literature reveals that nurses in internal and surgical intensive care units lack adequate expertise and fail to properly assess the quality of the nutritional therapy administered to patients in their units [11-16], it also reveals that their awareness and adherence to some evidence-based practice recommendations for nutritional care are inadequate [17,18], and that their knowledge levels regarding nutritional care need to be supported through theoretical and practical training.[19,20]

This necessitates giving special importance to equipping nursing students with knowledge and skills in nutritional management throughout their education. Studies have reported that the knowledge levels of actively practicing nurses regarding nutritional care are insufficient, underscoring the need to determine the knowledge levels of nursing students who have not yet graduated in this area. To the best of our knowledge, no study in the literature has evaluated nursing students' knowledge of nutritional care for surgical patients. This study aimed to assess the knowledge levels of nursing undergraduate students regarding the nutritional care of surgical patients. It is predicted that the study results will enable the integration of lessons on the nutritional care of surgical patients into the nursing education curriculum, thereby guiding it.

Main Points

- Nursing students demonstrated moderate knowledge of nutritional care of surgical patients.
- Knowledge levels peaked in the second year, when Surgical Nursing lessons were taught, but declined thereafter.
- The findings show that undergraduate nursing programs need to strengthen their nutrition instruction.
- A nutrition course, including both theoretical and practical components, should be added to the nursing curriculum.

The research questions to be answered are as follows:

1. What is the level of knowledge among nursing students regarding the nutritional care of surgical patients?
2. Is there a difference in the knowledge level of nursing students regarding the nutritional care of surgical patients based on class variable?

Material and Methods

Study design

This study is descriptive. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines were used in study reporting.

Settings and participants

The study population included second, third-, and fourth-year students of a nursing faculty in the capital of Türkiye's during the academic year 2024–2025. In their first year, students take a course on "nutritional requirements" as part of their "Fundamentals of Nursing" curriculum, which is approximately eight hours long; this course covers enteral and parenteral nutrition types and care. In the second year, nutrition is not directly included in the "Surgical Nursing" curriculum; it is briefly touched upon within course, and the nutritional recommendations of the Enhanced Recovery After Surgery (ERAS) protocol for the are included in this course. The inclusion criteria were (a) a student enrolled in the second, third, or fourth year of the undergraduate nursing program in the academic year 2024–2025, (b) volunteering to participate in the research, and (c) having Internet access. The study sample included 240 students, determined using a sample calculation formula with a known universe (95% confidence interval, 5% error rate).

Instruments

The Student Demographic Information Form and the Questionnaire for Nutritional Care of Surgical Patients were used to collect research data.

Student Demographic Information Form: It consists of three questions: age, gender, and grade level.

Questionnaire for Nutritional Care of Surgical Patients: It was developed by researchers through a literature review [1,3,6,21-23] to assess nursing students' knowledge of nutritional care for surgical patients. The questionnaire consists of 15 questions with "true," "false," and "don't know" options. The form was sent to 8 experts, including academics and clinical nurses, for evaluation of content validity. Expert opinions were analyzed using the Lawshe technique [24]. Experts were asked to evaluate each item as "not appropriate," "appropriate but requires modification," or "appropriate," and to provide reasons for not finding the items appropriate and suggestions for modification. According to Lawshe's critical values , the minimum Content Validity Ratio (CVR) for an element to be considered valid by a panel of 8 experts was set at 0.75.[24] Accordingly, elements deemed "essential" by at least seven experts were considered suitable for content validity. The questionnaire was finalized after revisions based on expert recommendations. The minimum score for the questionnaire is 0, and the maximum is 60.

Data collection

After obtaining approval from the ethics committee and written permission from the faculty, the completed forms were collected between June and November 2025 by sending the 'Google Forms' questionnaire link to the students through WhatsApp and e-mail. Participants could view the research questions only after reading the informed consent form. They took approximately 20 minutes to complete the questionnaire.

Data analysis

The IBM SPSS 26 (Statistical Package for the Social Sciences 26 for Windows) was used to analyze the data. Descriptive statistics were presented as median (minimum–maximum) and mean ± standard deviation (X ± SD) for numerical variables, and as number (n) and percentage (%) for categorical variables. The Kolmogorov-Smirnov (K-S) and Shapiro-Wilks tests were used to determine whether the data were normally distributed. Because the normality assumption was not met, the nonparametric Kruskal-Wallis H test was used to compare total scores across grade levels. The significance level was accepted as .05 in all analyses.

Ethical considerations

Written permission was obtained from the university ethics committee (No.: E-51986023-605-00004199531), and institutional approval was obtained from the Faculty of Nursing Dean's Office. The recommendations of the Association of Internet Researchers' Ethics Study Committee were followed for Internet research. The informed consent form was the first page of the questionnaire. Each participant accepted the following statement: 'I have read the information, and I agree to participate in the research voluntarily' and agreed to voluntary participation by clicking the consent button. Participants who did not click the consent button in the survey could not reach the questions page. Participants were assured of their right to refuse to participate in the study and the confidentiality of the information they provided. By signing the voluntary consent form, nursing students were assured of anonymity, the freedom to withdraw from the study at any time, and that their data would be used only in the academic field. Additionally, the researchers did not declare any ethical conflicts or financial benefits.

Results

The median age of the students was 22 (min-max: 19-26) years. Most (84.6 %) nursing students were female, and second-year students (47.5 %). Table 1 shows demographic data for students by current year of study.

When students' knowledge levels were evaluated by grade in the study, it was determined that second-year students had the highest score (35.26±10.73), while

Table 1. Nursing students' demographic data according to year of study

	Second year (n=114- %47.5)	Third year (n=103- %42.9)	Fourth year (n=23- %9.6)	All participants (n=240- %100)
Gender n (%)				
Female	97 (85.1)	89 (86.4)	17 (73.9)	203 (84.6)
Male	17 (14.9)	14 (13.6)	6 (26.1)	37 (15.4)
Median age (min-max)	22 (19-26)	22 (20-26)	23 (22-26)	22 (19-26)

fourth-year students had the lowest (32.52±7.84). The overall mean for all participants was 34.73±10.50. The median scores were 36, 32, 32, and 36 for second-year, third-year, fourth-year, and total groups, respectively. No statistically significant difference was found in knowledge levels between grades (p=0.065) (Table 2).

When the knowledge levels of 240 students regarding nutrition practices were evaluated, it was determined that, overall, high accuracy rates were achieved on a significant portion of the questions. Still, there were substantial knowledge deficiencies in some basic concepts and clinical practice areas. Correct answer rates for the questions range from 20.8% to 94.2%. It was observed that the response rates to questions such as the definition of malnutrition (question 2), the time to start postoperative feeding (question 7), and the types of enteral and parenteral nutrition (questions 9 and 10) ranged from 20.8% to 40%. Nearly half of the students answered these questions incorrectly. On the other hand, the statement that nutritional assessment should be performed when patients are admitted to the clinic had the highest percentage of correct answers (94.2%) (Table 3).

Table 2. Scores knowledge levels of nursing students according to year of study

Academic year	n	Knowledge level	
		mean±SD (median)	min-max (Q1-Q3)
Second year	114	35.26±10.73 (36)	0-60 (32-40)
Third year	103	34.64±10.81 (32)	8-60 (28-40)
Fourth year	23	32.52±7.84 (32)	24-60 (28-36)
Total	240	34.73±10.5 (36)	0-60 (28-40)
p*	.065		
H*	5.468		

SD, standard deviation; min, minimum; max, maximum; Q1, First Quartile; Q3, Third Quartile
*Kruskal Wallis Test
Grouping Variable: academic year

Discussion

This study aimed to determine nursing students' knowledge levels regarding the nutritional care of surgical patients during their undergraduate education. The study found that students' overall knowledge of nutritional care for surgical patients was generally moderate. It was determined that second-year students had the highest knowledge scores, but there was a downward trend in moderate knowledge levels as they progressed toward their fourth year. Although the Surgical Nursing course is taught in the second year and there is no independent topic specifically on nutritional care within the curriculum, the limited treatment of nutritional care in content such as ERAS protocols can be considered an influential factor in this outcome. The fact that students do not take an additional undergraduate course in this field until the fourth grade is also considered another factor supporting the decrease in knowledge levels. However, the absence of a statistically significant difference between grade levels is noteworthy. This situation can be attributed to students' continuous participation in clinical practice until graduation and their continued experience with the nutritional requirements of surgical patients across different clinical settings. In the systematic review by Doménech Briz et al., it was found that nurses' knowledge of nutritional assessment, a fundamental component of nutritional care, was insufficient.[20]In a study conducted in Italy, the attitudes of 245 nursing students towards nutritional care were evaluated, and it was found that 54% of first-year students, 98.5% of second-year students, and 100% of third-year students received training in nutrition and nutritional care. The majority of students in all classes had a neutral attitude towards nutritional care [25], in a study conducted by Buxton et al. (2013) with 166 3rd- and 4th-year nursing students, it was determined that 3.6% had good nutritional knowledge, 62.7% were sufficient, and 33.7% were inadequate.[26]

The findings of this study indicate that students have significant knowledge gaps in some basic information related to nutritional care. The fact that the accuracy rates for questions specifically regarding the definition of malnutrition, the timing of starting nutrition in the postoperative period, and the characteristics of enteral and parenteral nutrition ranged from 20.8% to 40% indicates that students have knowledge gaps in these areas.

Particularly within ERAS protocols, there is strong evidence that initiating oral fluid intake early postoperatively (e.g., at the second hour) is safe and

Table 3. The percentage of correct answers for each question

No	Questions	The correct answer to the question	Number of participants choosing the correct answer n (n=240) n (%)
1.	A nutritional assessment should be performed upon the patient's initial admission to the clinic.	T	226 (94.2)
2.	A body mass index below 20.5 kg/m2 is defined as malnutrition.	F	96 (40)
3.	Patients at serious risk of malnutrition should receive nutritional therapy 7-14 days before surgery.	T	204 (85)
4.	Parenteral nutrition can be administered in situations where oral/enteral nutrition is not feasible.	T	218 (90.8)
5.	Patients undergoing surgery who are not at risk of aspiration can drink clear liquids up to two hours before anesthesia.	T	164 (68.3)
6.	Patients undergoing surgery who are not at risk of aspiration can eat solid food up to six hours before anesthesia.	T	167 (69.6)
7.	Patients can be given liquid food in the second postoperative hour without waiting for auscultation of bowel sounds.	T	50 (20.8)
8.	Patients can be given solid foods without waiting for bowel sounds to be auscultated after the eighth postoperative hour.	F	145 (60.4)
9.	Enteral nutrition can be administered through a peripheral or central venous catheter.	F	104 (43.3)
10.	Feeding via gastrostomy is a type of parenteral nutrition.	F	119 (49.6)
11.	Enteral nutrition can be provided while the patient is in the supine position.	F	162 (67.5)
12.	Enteral nutrition products should not hang in the feeding bag for more than 8 hours.	T	184 (76.7)
13.	Multiple medications can be administered at the same time through an enteral feeding tube.	F	141 (58.8)
14.	Infusion sets used in parenteral nutrition should be changed every 24 hours.	T	182 (75.8)
15.	Parenteral nutrition bags should be protected from direct sunlight.	T	228 (95)

T; True; F;False

promotes healing.[22] However, the low rate of correct answers in our findings, such as those related to this type of question, suggests that students may have been influenced by more traditional clinical practices rather than current evidence-based approaches. Indeed, even if students have learned current guidelines theoretically during their training, they may still observe traditional approaches widely practiced in clinical area, such as waiting for bowel sounds. This situation can lead to a mismatch between students' knowledge and their perception of practice, and this may be reflected in our results.

The inability to accurately define malnutrition makes it challenging to identify at-risk patients early on and can negatively impact the creation of appropriate nutritional treatment plans.[1] In a study by Yfanti et al., the nutritional knowledge of 506 nursing students was evaluated, and it was found that they had insufficient knowledge of assessing body mass index.[27] In a cross-sectional study by Chonnail et al., it was found that nursing students had low knowledge of nutritional care for cancer patients.[28] This study also indicates a high level of misinformation regarding postoperative nutrition and that clinical practices crucial for preventing complications and supporting the healing process have not been sufficiently internalized. Misconceptions about enteral and parenteral nutrition indicate a potential lack of comprehensive knowledge among students about the fundamental principles, practices methods, and

indications of nutritional therapy. On the other hand, the statement that nutritional assessment should be performed upon patients' admission to the clinic has a high accuracy rate of 94.2%, indicating that students have a strong awareness of the clinical importance of nutritional assessment. This result suggests that a general awareness has formed at the theoretical level, but it is not sufficiently supported by knowledge of the details of clinical processes. Strengthening hospital collaborations and enabling students to participate in clinical practices in hospital clinical nutrition units can significantly contribute to increasing their knowledge and practical skills. Accordingly, incorporating more structured clinical practices into the program and encouraging closer collaboration with hospital staff can help reduce the gap between theory and practice and improve educational outcomes.[8,9,15,26]In this context, the findings clearly demonstrate the need to review the current curriculum and provide a more comprehensive, in-depth, and structured education in clinical nutrition. Additionally, adding a course on clinical nutrition to the curriculum is considered requirement for enhancing students' professional competence and preparing them more effectively for clinical practice.[29] Increasing opportunities for practice-based learning is crucial for addressing knowledge gaps. The use of educational modules enriched with case studies, in particular, can improve students' ability to apply theoretical knowledge to real-life scenarios. Furthermore, integrating interactive teaching methods that incorporate clinical decision-making processes related to nutritional therapy into the curriculum will strengthen students' critical thinking and problem-solving skills. Such approaches support not only rote memorization but also the effective application of knowledge.[26] Student-centered and self-directed learning approaches have become more prevalent in higher education in recent years, replacing teacher-centered instruction. Students must assume more responsibility for locating, obtaining, and critically analyzing instructional resources due to the decline in traditional lecture hours. This change is especially significant in the context of nursing education since evidence-based practice relies on the capacity to independently find and use current information.[30] In nursing education programs, there are courses on nutritional care based on international regulations. In the United States, nutritional knowledge level is among the topics assessed in the National Council Licensure Examination (NCLEX), the nursing licensing exam.[31] In Europe, Regulation 2005/36/EC emphasizes that nutritional care is included in the theoretical part of nursing education, within the basic sciences. [29] However, current research shows that nurses' knowledge of nutrition is often inadequate. [32,33] Furthermore, some studies indicate that nurses do not make sufficient effort to update their nutritional knowledge after graduation.[34] Macaninch and Martyn et al. delivered British Association for Parenteral and Enteral Nutrition (BAPEN's) clinical nutrition training modules to nursing students online [35], while Xia et al. aimed to increase knowledge levels by providing clinical nutrition training via e-learning [36] . In Buxton & Davies' study, it was suggested that nursing students need more education in nutritional care, and that professionals planning the university's nursing curriculum should develop it with these results in mind.[26]

Limitations

The unequal distribution of participants across grade levels is one of the study's primary limitations; in particular, the very low presence of fourth-year students may have decreased the statistical power of between-group comparisons. It is therefore advised that more balanced samples be used in future research.

Conclusion

This study showed that nursing students have moderate overall knowledge of nutritional care for surgical patients, with significant deficiencies in critical areas, including the definition of malnutrition, postoperative nutritional timing, and the principles of enteral and parenteral nutrition. Although second-year students scored higher, the general decline in later years and the lack of significant differences across grades suggest deficiencies in the curriculum. These findings highlight the need to improve curriculum for nursing students so they can provide effective and evidence-based nutritional care. In this context, integrating a structured nutritional care course into the curriculum is recommended. Furthermore, strengthening interdisciplinary collaboration with the nutrition team and increasing students' clinical practice experience in the field of nutrition are also important.

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Author contributions

Conception: R.E.S., Z.Ö.; Design: R.E.S., Z.Ö., D.S.; Data acquisition: Z.Ö., D.S.; Data analysis: R.E.S.; Data interpretation: R.E.S.; Drafting of the manuscript: R.E.S., Z.Ö., D.S.; Critical revision of the manuscript: R.E.S. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Hacettepe University Social Sciences and Humanities Researches Ethics Board (Date: May 6, 2025, Decision/Protocol No: No.: E-51986023-605-00004199531). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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The relationship between nutritional risk and clinical outcomes in medical patients transferred from internal medicine wards to the intensive care unit

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ABSTRACT

Objective: Malnutrition is common in hospitalized medical patients but is frequently underrecognized and undertreated. This study aimed to determine the prevalence of nutritional risk at intensive care unit (ICU) admission among patients transferred from internal medicine wards of a university hospital, to evaluate nutritional interventions during hospitalization in the wards, and to investigate the association between nutritional risk and ICU outcomes.

Methods: This retrospective observational study included adult patients transferred from internal medicine wards to a tertiary ICU between November 2022 and June 2025. Nutritional risk was assessed within 24 hours of ICU admission using the modified Nutrition Risk in the Critically Ill (mNUTRIC) score. Patients were categorized as high (mNUTRIC ≥5) or low (mNUTRIC <5) risk of malnutrition. Clinical characteristics, nutritional interventions before ICU admission, and ICU outcomes were compared between groups. Multivariable logistic regression analysis was performed to identify independent predictors of ICU mortality.

Results: Among 340 patients, 205(60.3%) had high malnutrition risk at ICU admission. High-risk patients were older and had significantly higher APACHE II and SOFA scores. Nutritional support during hospitalization in the ward was initiated in fewer than half of patients with high malnutrition risk (46.3%). Compared with low-risk patients, those with high malnutrition risk more frequently required invasive mechanical ventilation (63.2% vs 31.9%, p<0.001), developed ICU-acquired infections (33.7% vs 20.9%, p=0.011), and experienced higher ICU mortality (56.1% vs 25.9%, p<0.001). In multivariable analysis, high nutritional risk independently predicted ICU mortality (OR 2.85, 95%CI: 1.64–4.98, p<0.001), together with RIFLE Failure stage (OR 2.36, 95% CI: 1.23–4.53, p=0.010), ICU admission due to sepsis (OR 1.84, 95% CI: 1.07–3.17, p=0.028), and hypoalbuminemia (OR 2.36, 95% CI: 1.40–3.99, p=0.001).

Conclusion: These findings highlight nutritional risk as a key determinant of outcomes in patients transferred from internal medicine wards to the ICU and underscore the importance of early nutritional assessment in wards.

Keywords: malnutrition, nutritional risk, intensive care unit, mnutric, critical illness, clinical outcomes

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Introduction

Malnutrition is a common but frequently underrecognized condition among hospitalized medical patients and is associated with adverse clinical outcomes, including infections, prolonged hospital stay, functional decline, and increased mortality.[1-3] Malnutrition is a multifactorial condition resulting from the combined effects of inadequate nutritional intake, disease burden, and systemic inflammation, rather than a simple deficiency of calories or nutrients.[4] In this context, international frameworks have proposed a structured approach to malnutrition management, beginning with nutritional risk screening followed by diagnostic assessment using phenotypic and etiologic criteria.[4]

Data from Türkiye and other European countries demonstrate that nutritional risk is highly prevalent in internal medicine wards, particularly among older adults and patients with multiple chronic comorbidities.[5,6] Large-scale hospital studies have reported malnutrition risk rates ranging from 20% to over 50% in medical inpatients, depending on patient characteristics and the screening tool used.[5,6] Despite this high prevalence, several studies have consistently shown that a substantial proportion of patients identified as being at malnutrition risk do not receive timely or adequate nutritional support during hospitalization.[5-7]

Critically ill patients represent a subgroup in whom the clinical consequences of malnutrition are further affected by hypermetabolism, catabolic stress, and organ dysfunction.[8] The European Society for Clinical Nutrition and Metabolism (ESPEN) emphasizes the importance of early nutritional risk assessment and individualized nutritional therapy in the intensive care unit (ICU), as cumulative energy and protein deficits may negatively affect outcomes.[8] In critically ill patients, nutritional risk screening is essential to identify individuals most likely to benefit from targeted nutritional interventions. Among available tools, the modified Nutrition Risk in the Critically Ill (mNUTRIC) score has been widely validated across diverse ICU populations.[9-11]

Main Points

- High nutritional risk is common among patients transferred from internal medicine wards to the ICU.
- Nutritional support during ward hospitalization is initiated in fewer than half of high-risk patients.
- High mNUTRIC score independently predicts ICU mortality.
- The ward-to-ICU transition represents a potential window for early nutritional intervention.

While the impact of malnutrition in hospitalized patients and ICU populations has been well described, less attention has been paid to patients transferred from internal medicine wards to the ICU. This transition period may represent a missed opportunity for early nutritional screening and intervention. Randomized controlled trials in medical inpatients have demonstrated that individualized nutritional support initiated during ward hospitalization can improve clinically relevant outcomes, including survival.[7,12] However, data specifically addressing malnutrition risk at ICU admission and the adequacy of nutritional interventions before ICU transfer in internal medicine patients remain limited. Therefore, this study aimed to evaluate the prevalence of malnutrition risk at ICU admission among patients transferred from internal medicine wards, to assess whether nutritional support was initiated during ward hospitalization, and to investigate the association between malnutrition risk and ICU outcomes.

Material and Methods

Study design and setting

This retrospective observational study was conducted in a 9-bed tertiary-level internal medicine ICU of Gazi University Hospital, Ankara, Türkiye, between November 1, 2022, and June 30, 2025. The ICU primarily admits adult patients transferred from internal medicine wards, including general internal medicine, nephrology, medical oncology, endocrinology, geriatrics, hematology, gastroenterology, and rheumatology services. The study was designed and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[13]

Ethical approval

The study protocol was approved by the Gazi University Clinical Research Ethics Committee (approval date: September 23, 2025; approval number: 2025-1599). Due to the study's retrospective nature, informed consent was waived. The study was conducted in accordance with the Declaration of Helsinki.

Study population

Adult patients (≥18 years) transferred from internal medicine wards to the ICU during the study period were eligible for inclusion. Patients were excluded if they were admitted from non–internal medicine units, had an ICU length of stay shorter than 24 hours, had repeated ICU admissions during the same hospitalization, or were classified as terminal at the time of ICU admission.

Data collection

Demographic characteristics, comorbid conditions, source ward, ICU admission diagnoses, and clinical data were obtained retrospectively from electronic medical records. Disease severity at ICU admission was assessed using the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and organ dysfunction was evaluated using the Sequential Organ Failure Assessment (SOFA) score.[14,15] Comorbidity burden was assessed using the Charlson Comorbidity Index.[16] Performance status prior to ICU admission was evaluated using the Eastern Cooperative Oncology Group (ECOG) performance scale.[17] Acute kidney injury was classified according to the Risk, Injury, Failure, Loss, and End-stage kidney disease (RIFLE) criteria.[18] Laboratory data obtained at ICU admission included hemoglobin, white blood cell count, platelet count, blood urea nitrogen, creatinine, serum albumin, electrolytes, liver function tests, C-reactive protein, and procalcitonin.

Assessment of nutritional risk

Nutritional risk was assessed within the first 24 hours of ICU admission using the modified Nutrition Risk in the Critically Ill (mNUTRIC) score, which includes age, APACHE II score, SOFA score, number of comorbidities, and the number of days from hospital admission to ICU admission.[9-11] Patients were classified as having high nutritional risk (mNUTRIC ≥5) or low nutritional risk (mNUTRIC <5).

Nutritional support before ICU admission

Information regarding consultation with the nutrition support unit and initiation of nutritional support during ward hospitalization before ICU transfer was recorded. Nutritional support was defined as the initiation of enteral and/or parenteral nutrition documented in the medical records. The adequacy of caloric or protein delivery could not be evaluated due to the retrospective design.

Outcomes

The primary outcome was ICU mortality. Secondary outcomes included the need for invasive mechanical ventilation, ICU-acquired infections, ICU length of stay, and duration of hospitalization prior to ICU admission.

Statistical analysis

Continuous variables were assessed for normality and expressed as median and interquartile range (25th–75th percentile). Comparisons between groups were performed using the Mann–Whitney U test. Categorical variables were expressed as numbers and percentages and compared using the chi-square test or Fisher's exact test, as appropriate. Variables with a P value <0.05 in univariable analyses and clinically relevant variables were entered into a multivariable logistic regression model to identify independent predictors of ICU mortality. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs). Statistical analyses were performed using IBM SPSS Statistics (IBM Corp, Armonk, NY, USA). A two-sided P value <0.05 was considered statistically significant.

Results

A total of 340 patients transferred from internal medicine wards to the ICU were included in the analysis, of whom 205 (60.3%) were classified as having high nutritional risk (mNUTRIC ≥5) (Table 1). Despite the high burden of nutritional risk at the time of ICU transfer, nutritional support before ICU admission was initiated only in 46.3% of the patients with high malnutrition risk. Moreover, only 28% of the patients received nutritional support team consultation before ICU transfer, which was not significantly higher among those at risk (31.7% vs 23.7%, p=0.110) (Table 1).

Demographic characteristics, source wards, nutritional interventions during ward stay, and comorbidities by nutritional risk status are presented in Table 1. ICU admission characteristics, including performance status, RIFLE classification, reasons for ICU admission, organ dysfunction scores, laboratory findings, and clinical outcomes according to nutritional risk status, are summarized in Table 2. At ICU admission, patients with high nutritional risk had greater disease severity and organ dysfunction, as reflected by higher APACHE II and SOFA scores, lower GCS scores, and higher Charlson Comorbidity Index values (p<0.05) (Table 2). Sepsis was

Table 1. Demographic characteristics, source wards, nutritional interventions during ward stay, and comorbidities of patients according to malnutrition risk

Variable	All Patients (n=340)	High Malnutrition Risk (n=205)	Low Malnutrition Risk (n=135)	P value
Admission Data				
Age, median (IQR)	73 (64–81)	77 (70–83)	66 (55–74)	**<0.001**
Female sex, n (%)	143 (42.1)	82 (40.0)	61 (45.2)	0.343
Height (cm), median (IQR)	165 (160–173)	167 (160–174)	165 (160–173)	0.823
Weight (kg), median (IQR)	70 (60–76)	70 (60–75.5)	70 (60–77)	0.783
BMI (kg/m^2),median(IQR)	24.2 (22–27)	24.2 (22.1–27)	24 (22–27)	0.936
Source Ward, n (%)				
Medical Oncology	117 (34.4)	66 (32.2)	51 (37.8)	0.289
Nephrology	75 (22.1)	51 (24.9)	24 (17.8)	0.122
Geriatrics	60 (17.6)	44 (21.5)	16 (11.5)	0.015
Gastroenterology	38 (11.2)	18 (8.8)	20 (14.8)	0.084
General Internal Medicine	28 (8.2)	15 (7.3)	13 (9.6)	0.448
Endocrinology	6 (1.8)	3 (1.5)	3 (2.2)	0.603
Hematology	8 (2.4)	5 (2.4)	3 (2.2)	0.897
Rheumatology	8 (2.4)	3 (1.5)	5 (3.7)	0.182
Nutritional Support During Ward Stay				
NST consultation, n (%)	97 (28.5)	65 (31.7)	32 (23.7)	0.110
Nutritional support before ICU, n (%)	134 (39.4)	95 (46.3)	39 (28.9)	**<0.001**
Comorbidities, n (%)				
Hypertension	201 (59.3)	143 (69.8)	58 (43.3)	**<0.001**
Chronic kidney disease	118 (34.7)	85 (41.5)	33 (24.4)	**<0.001**
Diabetes mellitus	115 (33.8)	69 (33.7)	46 (34.1)	0.937
Malignancy (solid or hematologic)	126 (37.0)	72 (35.1)	54 (40.0)	0.623
Coronary artery disease/Heart failure	165 (48.7)	113 (55.1)	52 (38.8)	0.002
COPD/Asthma	107 (31.5)	59 (28.8)	48 (35.6)	0.188
Cerebrovascular disease	72 (21.2)	52 (25.4)	20 (14.8)	0.013

Values are presented as median (interquartile range, IQR) or number (%), as appropriate. High malnutrition risk was defined as an mNUTRIC score ≥5.
BMI; Body mass index, ICU; Intensive Care Unit, COPD; Chronic obstructive pulmonary disease, n; Number, NST; Nutritional Support Team.

the most common reason for ICU admission and was more frequent among patients with high nutritional risk ($p<0.05$).

High nutritional risk was associated with worse ICU outcomes. Patients in the high-risk group required invasive mechanical ventilation more frequently, developed ICU-acquired infections more often, and had higher ICU mortality than those in the low-risk group ($p<0.05$) (Table 2). Length of hospital stay before ICU admission was longer in patients with high nutritional risk, whereas ICU length of stay and the use of enteral or parenteral nutrition in the ICU did not differ significantly between groups. Laboratory findings at ICU admission showed higher blood urea nitrogen, creatinine, aspartate aminotransferase, and procalcitonin levels, together with lower serum albumin concentrations and more frequent hypoalbuminemia in the high-risk group ($p<0.05$) (Table 2).

Table 2. ICU admission characteristics, laboratory findings, and clinical outcomes according to malnutrition risk

Variable	All Patients (n=340)	High Malnutrition Risk (n=205)	Low Malnutrition Risk (n=135)	P value
Organ Dysfunction and Prognostic Scores at ICU Admission, median (IQR)				
APACHE II score	23 (16–31)	27 (22–34)	17 (13–20)	**<0.001**
SOFA score	6 (4–9)	8 (5–11)	4 (3–7)	**<0.001**
GCS score	14 (9–15)	12 (7–15)	15 (13–15)	**<0.001**
Charlson comorbidity index	6 (5–8)	6 (5–8)	5 (3–7)	**<0.001**
Reasons for ICU Admission, n (%)				
Sepsis	221 (65.2)	149 (72.7)	72 (53.7)	**<0.001**
Renal	122 (35.9)	80 (39.0)	42 (31.1)	0.137
Pulmonary	203 (59.7)	123 (60.0)	80 (59.3)	0.892
Cardiac	38 (11.2)	29 (14.1)	9 (6.7)	0.022
Gastrointestinal	52 (15.3)	24 (11.7)	28 (20.7)	**0.018**
Hepatobiliary	29 (8.5)	15 (7.3)	14 (10.4)	0.324
Neurologic	41 (12.1)	24 (11.7)	17 (12.6)	0.806
Rheumatologic	2 (0.6)	1 (0.5)	1 (0.7)	0.765
Metabolic/endocrine	12 (3.5)	5 (2.4)	7 (5.2)	0.179
Performance Status (ECOG), n (%)				
ECOG 1	55 (16.2)	23 (11.2)	32 (23.7)	0.002
ECOG 2	86 (25.3)	47 (22.9)	39 (28.9)	0.216
ECOG 3	106 (31.2)	76 (37.1)	30 (22.2)	0.003
ECOG 4	72 (21.2)	56 (27.3)	16 (11.9)	<0.001
RIFLE Classification, n (%)				
Risk	96 (28.2)	55 (26.8)	41 (30.4)	0.478
Injury	41 (12.1)	25 (12.2)	16 (11.9)	0.924
Failure	64 (18.8)	42 (20.5)	22 (16.3)	0.04
Loss	18 (5.3)	14 (6.8)	4 (3.0)	0.119
End-stage	30 (8.8)	26 (12.7)	4 (3.0)	**0.001**
Mechanical Ventilation Support in ICU, n (%)				
Invasive	172 (50.7)	129 (63.2)	43 (31.9)	<0.001
Non-invasive	77 (22.8)	46 (22.8)	31 (23.0)	0.967
ICU-acquired infection, n (%)	96 (28.6)	68 (33.7)	28 (20.9)	**0.011**

Values are presented as median (interquartile range, IQR) or number (%), as appropriate. ICU mortality was defined as death during the ICU stay. Hypoalbuminemia: Albumin < 3g/dL

APACHE II; Acute Physiology and Chronic Health Evaluation II, SOFA; Sequential Organ Failure Assessment, GCS; Glasgow Coma Scale, ECOG; Eastern Cooperative Oncology Group performance status, RIFLE; Risk, Injury, Failure, Loss, and End-stage kidney disease classification, IMV; Invasive mechanical ventilation, NIMV; Non-invasive mechanical ventilation, WBC; White blood cell count, PLT; Platelet count, BUN; Blood urea nitrogen, ALT; Alanine aminotransferase, AST; Aspartate aminotransferase, ALP; Alkaline phosphatase, GGT; Gamma-glutamyl transferase, CRP; C-reactive protein, n; Number

Table 2. Continued

Variable	All Patients (n=340)	High Malnutrition Risk (n=205)	Low Malnutrition Risk (n=135)	P value
Nutritional Support in ICU, n (%)				
Enteral	205 (60.7)	130 (63.7)	75 (56.0)	0.153
Parenteral	90 (26.7)	56 (27.7)	34 (25.2)	0.606
Laboratory Values at ICU Admission, median (IQR)				
Hemoglobin (g/dL)	9.6 (8.3–11.5)	9.3 (8.2–11.0)	10.0 (8.3–12.0)	0.073
WBC (×10^3/μL)	11.3 (7.3–15.7)	11.6 (7.7–16.4)	10.8 (6.7–14.2)	0.113
Platelets (×10^3/μL)	176 (97–261)	169 (100–242)	184 (95–281)	0.441
BUN (mg/dL)	41.5 (26–64)	47 (32–65)	33 (19–57)	**<0.001**
Creatinine (mg/dL)	1.5 (0.8–2.6)	1.8 (1.0–3.1)	1.2 (0.7–2.1)	**<0.001**
Albumin (g/dL)	2.9 (2.5–3.3)	2.8 (2.4–3.1)	3.0 (2.6–3.5)	**0.001**
Hypoalbuminemia, n (%)	211(62%)	141(69%)	70(52%)	**<0.001**
Sodium (mEq/L)	138 (134–142)	137 (134–141)	138 (133–142)	0.835
Potassium (mEq/L)	4.1 (3.6–4.6)	4.1 (3.6–4.7)	4.1 (3.8–4.6)	0.262
ALT (U/L)	27 (14–54)	29 (15–61)	25 (14–42)	0.070
AST (U/L)	37 (24–76)	41 (25–88)	33 (21–64)	**0.014**
ALP (U/L)	98 (69–168)	99 (70–176)	97 (67–157)	0.557
GGT (U/L)	48 (24–100)	49 (24–100)	47 (23–102)	0.596
CRP (mg/L)	91 (35–164)	91 (41–160)	87 (27–170)	0.633
Procalcitonin (ng/mL)	0.9 (0.2–3.7)	1.3 (0.3–4.7)	0.5 (0.1–2.4)	**<0.001**
Length of stay before ICU (days)	4 (1–10)	5 (2–16)	3 (1–9)	**<0.001**
Length of ICU stay (days)	6 (3–12)	7 (4–13)	5 (3–10)	0.209
ICU mortality, n (%)	150 (44.1)	115 (56.1)	35 (25.9)	**<0.001**

Values are presented as median (interquartile range, IQR) or number (%), as appropriate. ICU mortality was defined as death during the ICU stay.
Hypoalbuminemia: Albumin < 3g/dL
APACHE II; Acute Physiology and Chronic Health Evaluation II, SOFA; Sequential Organ Failure Assessment, GCS; Glasgow Coma Scale, ECOG; Eastern Cooperative Oncology Group performance status, RIFLE; Risk, Injury, Failure, Loss, and End-stage kidney disease classification, IMV; Invasive mechanical ventilation, NIMV; Non-invasive mechanical ventilation, WBC; White blood cell count, PLT; Platelet count, BUN; Blood urea nitrogen, ALT; Alanine aminotransferase, AST; Aspartate aminotransferase, ALP; Alkaline phosphatase, GGT; Gamma-glutamyl transferase, CRP; C-reactive protein, n; Number

Table 3. Independent Predictors of ICU mortality identified by multivariable logistic regression analysis

Variable	Odds Ratio (OR)	95% Confidence Interval (CI)	P value
RIFLE Failure stage	2.36	1.23–4.53	**0.010**
ICU admission due to sepsis	1.84	1.07–3.17	**0.028**
High malnutrition risk (mNUTRIC ≥5)	2.85	1.64–4.98	**<0.001**
Hypoalbuminemia (albumin <3 g/dL)	2.36	1.40–3.99	**0.001**

mNUTRIC; Modified Nutrition Risk in the Critically Ill Score, RIFLE; Risk, Injury, Failure, Loss, and End-stage kidney disease classification, ICU; Intensive Care Unit.

In multivariable logistic regression analysis, high nutritional risk was independently associated with ICU mortality (OR: 2.85, 95% CI: 1.64–4.98, p < 0.001). Additional independent predictors of ICU mortality included RIFLE Failure stage (OR:2.36, 95%CI:1.23–4.53, p=0.010), ICU admission due to sepsis (OR:1.84, 95%CI:1.07–3.17, p=0.028), and hypoalbuminemia (albumin <3 g/dL) (OR:2.36, 95%CI:1.40–3.99, p=0.001) (Table 3).

Discussion

In this retrospective cohort of patients transferred from internal medicine wards to the ICU, nearly two-thirds presented with high nutritional risk at ICU admission. High nutritional risk was associated with greater disease severity, increased need for invasive mechanical ventilation, higher rates of ICU-acquired infections, and higher ICU mortality. Most importantly, our data reveal a clinically critical gap between nutritional vulnerability and clinical action during ward hospitalization; despite the high burden of nutritional risk at the time of ICU transfer, nutritional support before ICU admission was initiated in fewer than half of patients with high malnutrition risk, and nutrition support unit consultation was not consistently higher among those at risk.

This gap between nutritional risk and nutritional intervention is the key finding of the present study, because it identifies a potentially modifiable challenge of critical illness. Previous data have consistently shown that a substantial proportion of hospitalized patients at high malnutrition risk do not receive timely nutritional support, even when reduced intake is evident.[5-7] Randomized evidence in medical inpatients further supports addressing this gap, demonstrating that protocol-guided individualized nutritional support can improve outcomes, including survival.[7] Our study extends these observations by focusing on a vulnerable subgroup, patients deteriorating on internal medicine wards and subsequently requiring ICU admission, and by linking inadequate pre-ICU nutritional intervention to worse ICU outcomes. Together, these findings suggest that the ward to ICU transition represents a missed opportunity for earlier nutritional screening and timely intervention in a population that experiences poor ICU outcomes.

A high mNUTRIC score was not only a marker of worse baseline status but also remained an independent predictor of ICU mortality in the multivariable analysis. This finding is clinically meaningful because mNUTRIC was originally designed to identify ICU patients most likely to experience harm from underfeeding and who might benefit from targeted nutritional strategies, rather than categorizing malnutrition severity.[9-11] In our cohort, consisting of patients deteriorating on internal medicine wards, the mNUTRIC score showed a multidimensional risk profile reflecting age, comorbidity burden, acute illness severity, and pre-ICU hospital exposure, beyond what is explained by conventional severity scores alone. Methodologically, because mNUTRIC incorporates acute severity variables and time from hospital admission to ICU, its independent association with mortality should be interpreted as reflecting cumulative depletion of physiologic reserve rather than an isolated evaluation of nutritional risk in this transitional population.

A longer duration of ward hospitalization prior to ICU transfer among patients with high malnutrition risk represents another important finding of the present study. This observation suggests that nutritional vulnerability may progress during the ward phase of hospitalization and may indicate prolonged exposure to reduced nutritional intake or metabolic stress prior to ICU admission. Importantly, these findings identify the ward hospitalization period as a potential therapeutic window during which early nutritional assessment and targeted support may modify subsequent ICU outcomes, whereas interventions initiated only after ICU admission may represent a delayed response to a process that has already progressed during the pre-ICU period.

Beyond malnutrition risk, sepsis-related ICU admission, RIFLE Failure stage, and hypoalbuminemia remained independent predictors of mortality, underscoring the combined impact of systemic inflammation, acute organ dysfunction, and impaired metabolic reserve on outcomes.[12,18,19] Patients with high nutritional risk were more frequently admitted with sepsis and had higher procalcitonin levels, indicating coexisting systemic inflammation and nutritional vulnerability at the time of clinical deterioration in this patient population.[12] Advanced renal dysfunction may further amplify this vulnerability through metabolic instability, accumulation of uremic toxins, and impaired amino acid utilization, contributing to adverse outcomes. Hypoalbuminemia, although not a specific marker of malnutrition, reflects the integrated effects of inflammation, capillary leak, hepatic synthesis, and nutritional reserve and consistently correlates with adverse outcomes.[19,20] Together, these factors define a clinical profile characterized by systemic inflammatory stress, impaired organ reserve, and reduced metabolic resilience.

Another finding of the study was that patients with high malnutrition risk had substantially worse functional status at ICU admission, with higher proportions of ECOG 3–4, reflecting advanced dependency and poor performance status before critical illness. Consistent with this profile, patients with high malnutrition risk required invasive mechanical ventilation more frequently and experienced higher rates of ICU-acquired infections, reflecting impaired physiologic reserve and host defense as reported in previous literature.[21,22] High malnutrition risk was also associated with longer hospital stay before ICU transfer, suggesting a period of progressive deterioration and cumulative nutritional deficit on the wards.[5,7] In contrast, ICU length of stay did not differ significantly between groups despite higher mortality among high-risk patients, suggesting that the observed mortality difference may have been related to earlier deaths during the ICU course rather than longer ICU stays. These findings may reflect progressive deterioration during ward hospitalization rather than events occurring solely after ICU admission.

This study has several limitations. First, its retrospective single-center design limits generalizability. Second, detailed data on actual caloric and protein delivery, the timing of nutritional initiation, and achievement of nutritional targets were not available in ICU nutrition, precluding evaluation of nutritional adequacy and dose-response relationships. Third, functional and long-term outcomes after ICU discharge were not assessed. Finally, ward nutritional support was evaluated as a binary variable, with no detailed information on adequacy, duration, or achieved energy or protein targets; therefore, the findings should be interpreted as reflecting the presence of a nutritional intervention rather than the precise amount of nutritional delivery. Despite these limitations, the study provides clinically relevant insights into nutritional risk and care process gaps in a vulnerable transitional population.

Conclusion

In conclusion, high nutritional risk is common among patients transferred from internal medicine wards to the ICU and is independently associated with increased mortality. Despite this, nutritional interventions during ward hospitalization remain insufficient. These findings indicate that systematic nutritional screening, combined with early, individualized nutritional support on the wards, may represent a critical, modifiable opportunity to improve outcomes for patients at risk of critical illness.

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Author contributions

Conception: K.İ., N.B.D.; Design: K.İ., G.A., A.O.T., S.Ç., A.Y., D.Y.A., E.T.M., B.K.; Data acquisition: K.İ., N.B.D., G.A., A.O.T., S.Ç., A.Y., D.Y.A., E.T.M., B.K., M.T.; Data analysis: K.İ.; Data interpretation: K.İ.; Drafting of the manuscript: K.İ.; Critical revision of the manuscript: G.A., M.T.; All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Gazi University Clinical Research Ethics Committee (Date: Sep 23,2025, Decision/Protocol No: 2025-1599).

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that during the preparation of this work, the following AI-assisted technology was used: Name and Manufacturer of the Tool / Service: ChatGPT, OpenAI; Grammarly, Grammarly Inc. Date(s) of Use: During manuscript preparation and revision process.

Reason and Extent of Use: ChatGPT and Grammarly were used solely for language editing, grammar checking, sentence-level clarity, and improving the readability of the manuscript.

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Evaluation of the dietary quality of selected public, university, and private hospital menus

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ABSTRACT

Objective: The aim of this study is to examine the dietary quality of public, university, and private hospital menus.

Methods: In this study, the meal menus for October 2025 and April 2026 were analyzed for hospitals in the provinces of Ankara and Konya (one public, one university, and one private hospital from each province). The nutrient adequacy ratio (NAR) and mean adequacy ratio (MAR) were used to assess diet quality. Additionally, the nutrient content of the menus was analyzed and compared with the recommendations of the Türkiye Dietary Guidelines-2022 (TDG-2022).

Results: There was no significant difference in MAR values between public hospitals (72.5-79.1%) and university hospitals (71.4-78.1%), while the MAR value for private hospital menus (76.3-82%) was significantly higher than the other two hospitals ($p=0.001$, $\eta^2 \leq 0.05$). The NAR values for calcium, magnesium (for men only), potassium (for men only), and folate in public and university hospital menus were below 66%. The NAR values for calcium (for women aged 51-70) and magnesium (for men aged 31-70) in the private hospital menu were below 66%. The fiber, vitamin B2, vitamin B6, calcium, potassium, magnesium content, and energy percentage from polyunsaturated fatty acids of all hospital menus were found to be below the TDG-2022 recommendations, while the energy ratio from total fat and saturated fat and the sodium content were found to be above the TDG-2022 recommendations. Furthermore, the vitamin C content of public and university hospital menus was below the TDG-2022 recommendations, while the cholesterol content of public and private hospital menus was above the TDG-2022 recommendation.

Conclusion: The fiber, vitamin B2, vitamin B6, calcium, potassium, and magnesium content of all hospital menus examined does not meet the TDG-2022 recommendations. The menus need to be improved in terms of the nutrients found to be lacking, while saturated fat and sodium content should be reduced. Based on MAR scores, the menus of public and university hospitals need to be improved in terms of the examined nutrients, taking into account the nutritional needs of men.

Keywords: nutritional assessment, hospital care, calcium, malnutrition, menu planning

Introduction

Nutrition plays a central role in maintaining and improving health. Inadequate and unbalanced nutrition is associated with an increase in the prevalence of noncommunicable diseases and healthcare expenditures, as well as reduced quality of life.[1] Malnutrition refers to deficiencies or excesses in nutrient intake, imbalances in essential nutrients, or impaired nutrient utilization.[2] Increased

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nutrient requirements associated with acute or chronic illness, and disease-related malnutrition resulting from symptoms affecting nutrition, represent one of the most common conditions in the hospital setting.[3] Since hospital menus are the sole source of nutrition for hospitalized patients, the dietary quality of these menus plays a major role in supporting healthy nutrition and improving the patient's nutritional status and health.[4] Delayed wound healing, increased risk of complications and infection, prolonged hospital stay, higher readmission and mortality rates are some of the poor health outcomes associated with hospital malnutrition.[5] A systematic review reported that malnutrition affects 20-50% of hospitalized patients and that meal dissatisfaction is among the frequently reported causes of malnutrition.[6] A study conducted with 200 patients hospitalized in the internal medicine department of a hospital in Türkiye reported that 75% of patients ate less than half of the hospital meals and that the incompatibility of the meals on the menu in terms of content was an effective factor in this situation.[7]

Although hospital food services are often less appreciated than other clinical care services, they are an integral part of treatment.[8] Patients report that the service they remember most after discharge is nutrition services.[7] Furthermore, since healthcare institutions are expected to be advocates for a health-promoting lifestyle, the menus offered in such settings have the potential to influence the public's perception of what constitutes healthy eating.[1] Research examining the dietary quality of hospital menus is needed to ensure patients receive optimal nutrition.[3] Such studies can identify interventions that could improve the health and well-being of patients receiving nutrition services and potentially lead to policy changes to ensure that healthy foods predominate in hospital menus.[9] The aim of this study is to evaluate the dietary quality of menus for patients on a normal diet in public, university, and private hospitals using the nutrient adequacy ratio (NAR) and mean adequacy ratio (MAR).

Main Points

- The fiber, B2, B6 vitamin, calcium, potassium, magnesium content, and percentage of energy from polyunsaturated fatty acids in all hospital menus were below the recommendations of TDG-2022.
- It was found that the total fat and saturated fat energy percentage of all hospital menus, as well as their sodium content, exceeded the recommendations of TDG-2022.
- Based on mean adequacy ratio scores, public and university hospitals need to improved in terms of the examined nutrients, taking into account the nutritional needs of men.

Methods

Hospital selection and data collection

In this study, the meal menus for October 2025 and April 2026 were analyzed for hospitals in the provinces of Ankara and Konya (one public, one university, and one private hospital from each province). The menus are consist of three meals: breakfast, lunch and dinner. Hospitals were selected using a web-based randomization program (random.org). The public and university hospitals was selected from among hospitals with monthly three-meal menu available on their website. Since the private hospitals menus were not available on their websites, the institution authorities were contacted. All hospital menus were prepared by institutional dietitians. Hospital menus do not consist of menus that repeat on a weekly or biweekly cycle. A menu analysis was conducted for 122 days (31 days in October and 30 days in April, with two hospitals in each group) for hospitals in each category (public, university, and private).

Analysis and evaluation of menus

The information regarding the ingredients and portion sizes of the dishes on the menus is based on the Standard Recipes for Turkish Cuisine[10] and the Bed-based Treatment Institutions Operating Regulations.[11] A total of 150 grams of white bread per day, 50 grams per meal, has been added to the menus for all hospitals. This practice has been implemented to avoid bias. This is because while some hospital menus include bread with meals, others do not specify it on the menu. In hospitals, patients are usually given a roll with their meals, and each roll weighs 50 grams. However, when foods such as cake, simit, pogaca, pastry, lahmacun, and meat bread are included in a meal, 50 grams of white bread has not been added to that meal. The energy and nutrient content of the menus was analyzed using the Nutrition Information System software.[12] The nutrient content of the menus was then compared with the recommendations of the Türkiye Dietary Guideline-2022 (TDG-2022).[13]

Mean adequacy ratio (MAR) and nutrient adequacy ratio (NAR) calculation

NAR and MAR scores were used to assess diet quality. NAR scores were calculated using the following formula, based on Dietary Reference Intake (DRI) levels categorized by age and gender:[14]

NAR = Daily amount of the nutrient analyzed in the menu x 100 / DRI level of the nutrient

NAR values range from 0 to 100. A score closer to 100 indicates diets more consistent with nutrient intake references. To mitigate the effects of high consumption, the calculated NAR values have been truncated at 100.[15] In this study, NAR scores were calculated for ten nutrients (calcium, magnesium, potassium, vitamins A, E, C, B1, B2, B9, and B12). As in previous studies, a NAR score of <66% for a nutrient was considered an inadequate intake.[16,17]

The MAR score was obtained by taking the average of the NAR scores calculated for the ten nutrients. As in previous studies, a MAR score of <75% was considered indicative of overall micronutrient intake inadequacy.[16,18] NAR and MAR scores were calculated separately for women and men aged 19-70 years.

Statistical analysis

The normal distribution of the data was tested using the Shapiro-Wilk test and skewness-kurtosis values. The homogeneity of variances was assessed using the Levene test. Quantitative data with normal distribution are presented as mean ($\bar{x}$) ± standard deviation (SD), while quantitative data without normal distribution are presented as median (interquartile range). In addition %95 confidence intervals (%95 CI) for the means have been calculated. One-way ANOVA and Kruskal-Wallis test, whichever was appropriate, were used for inter-hospital comparisons of the nutritional content of menus. The post-hoc Tukey test or Tamhane's T2 test was used to identify the group causing the difference. To assess the magnitude of differences between groups, the effect size measure eta square (η^2) was calculated. The classifications proposed by Cohen were used to interpret the effect sizes ($\eta^2 \approx 0.01$: small effect; $\eta^2 \approx 0.06$: moderate effect; $\eta^2 \approx 0.14$: large effect).[19] The unit of analysis is the number of days. As previously explained, a 122-day menu analysis was conducted for each hospital group (public, university, private). A two-tailed $p<0.05$ value was considered statistically significant for all statistical analyses. All statistical analyses were performed using the SPSS 26.0 Windows (SPSS Inc.; Chicago, USA) software.

Results

In this study, which analyzed 122 daily menus from each hospital group (public, university, and private), the energy and nutrient contents of the menus are presented in Table 1. There was no significant difference in the average daily energy content of public (1833.12 kcal) and university (1866.08 kcal) hospital menus, while the energy content of private hospital menus (1984.91 kcal) was significantly higher than the other two hospitals ($p<0.001$, $\eta^2=0.09$). The average daily carbohydrate and fat amount in the menus of public and university hospitals was significantly lower than that in the menus of private hospitals ($p<0.001$, $\eta^2=0.09$ and $p=0.003$, $\eta^2=0.03$ respectively), while no significant difference was observed between the three hospitals in terms of the percentage of energy derived from carbohydrates and sucrose in the menus ($p=0.077$ and $p=0.434$ respectively). The fiber content in public and university hospital menus was similar (18.93 and 18.20 grams, respectively) and was found to be significantly lower than that in private hospital menus (21.14 grams) ($p<0.001$, $\eta^2=0.05$). No significant differences were observed among the hospital groups in terms of the cholesterol content of the menus, the percentage of energy derived from fat, or the percentages of energy derived from monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs) ($p>0.05$). The saturated fat content of public and university hospital menus was found to be similar and significantly lower than that of private hospital menus ($p<0.001$, $\eta^2=0.06$). While no significant difference was observed between the groups in terms of the average protein content of the menus ($p=0.171$), the proportion of energy derived from protein in private hospital menus (14.57%) was found to be significantly lower than that in public and university hospital menus (16.22% and 16.40%, respectively) ($p<0.001$, $\eta^2=0.07$).

The levels of certain micronutrients were similar in public and university hospital menus but were found to be significantly lower in private hospital menus (e.g., vitamin E, B9, vitamin C, calcium, potassium, and magnesium). The differences between groups showed a moderate effect size for potassium ($\eta^2=0.07$) and a small effect size for other nutrients (η^2 values ranging from 0.03 to 0.05). The zinc and vitamin B12 content in the menus was similar in the public and private hospital groups but was found to be significantly lower in the university hospital group

Table 1. Energy and nutrient content of hospital menus

Energy and nutrients	**Public**		**University**		**Private**		**p value1**	**Eta Squared (η2)**
Number of hospitals	**n=2**		**n=2**		**n=2**			
Total number of menu days	**n=122**		**n=122**		**n=122**			
	x̄ ± SD or M (IQR)	**%95 CI**	**x̄ ± SD or M (IQR)**	**%95 CI**	**x̄ ± SD or M (IQR)**	**%95 CI**		
Energy (kcal)	1833.12± 178.37[a]	1801.1-1865.1	1866.08± 209.12[a]	1828.6-1903.6	1984.91± 237.12[b]	1942.4-2027.4	**<0.001**	0.09
Carbohydrate (gr)	204.59± 28.31[a]	199.5-209.6	205.33± 36.92[a]	198.7-211.9	226.27± 33.17[b]	220.3-232.2	**<0.001**	0.09
Carbohydrate (%E)	45.75± 5.21	44.8-46.7	45.31± 5.48	44.3-46.3	46.86± 5.77	45.8-47.9	0.077	0.01
Fat (gr)	78.80± 16.04[a]	75.9-81.7	80.64± 15.86[a]	77.8-83.5	86.30± 20.32[b]	82.7-89.9	0.003	0.03
Fat (%E)	38.01± 5.50	37-39	38.24± 5.15	37.3-39.2	38.55± 6.92	37.3-39.8	0.773	0.001
Protein (gr)	72.18± 12.64	69.9-74.4	74.28± 13.38	71.9-76.7	70.78± 17.43	67.7-73.9	0.171	0.01
Protein (%E)	16.22± 3.09[a]	15.7-16.8	16.40± 2.89[a]	15.9-16.9	14.57± 3.22[b]	14-15.2	**<0.001**	0.07
Fiber (gr)	18.93± 4.68[a]	18.1-19.8	18.20± 4.56[a]	17.4-19.0	21.14± 7.03[b]	19.9-22.4	**<0.001**	0.05
Fiber (gr)/ 1000 kcal	10.34± 2.38[a,b]	9.9-10.8	9.81± 2.41[a]	9.4-10.3	10.69± 3.43[b]	10.1-11.3	**0.049**	0.02
Sucrose (%E)	5.69± 3.64	5-6.4	5.93± 4.74	5.1-6.8	6.35± 3.57	5.7-7	0.434	0.01
SFA (%E)	15.15± 2.91[a]	14.6-15.7	15.57± 2.88[a]	15.1-16.1	16.99± 3.93[b]	16.3-17.7	**<0.001**	0.06
MUFA (%E)	13.51± 2.73	13-14	13.60± 2.40	13.2-14	13.17± 2.76	12.7-13.7	0.398	0.01
PUFA (%E)	6.11± 2.16	5.7-6.5	5.94± 1.86	5.6-6.3	5.82± 2.23	5.4-6.2	0.552	0.003
Cholesterol (mg)	306± 124.33	283.7-328.3	287.27± 114.19	266.8-307.7	315.71± 157.60	287.5-344	0.240	0.01
Vitamin A (µg)	906 (640.85)	992.1-1225.1	810.8 (556.55)	932.3-1356.7	1209.95 (781.03)	1157-1561.5	0.116*	0.01
Vitamin E (mg)	10.66± 4.67[a]	9.8-11.5	9.56± 4.88[a]	8.7-10.4	12.50± 7.39[b]	11.2-13.8	**<0.001**	0.04
Vitamin B_1 (mg)	0.77± 0.15[a]	0.74-0.80	0.80± 0.21[a,b]	0.76-0.84	0.84± 0.21[b]	0.8-0.88	**0.028**	0.02
Vitamini B_2 (mg)	1.17± 0.24	1.13-1.22	1.22± 0.32	1.17-1.28	1.25± 0.36	1.19-1.32	0.141	0.01
Niacin equivalent (mg)	27.06± 7.07[a,b]	25.8-28.3	28.80± 6.68[a]	27.6-30	25.48± 8.85[b]	23.9-27.1	**0.003**	0.03
Vitamin B_6 (mg)	1.19± 0.25[a,b]	1.14-1.24	1.14± 0.23[a]	1.1-1.19	1.24± 0.35[b]	1.18-1.3	**0.035**	0.02
Vitamin B_9 (µg)	250.85± 64.44[a]	239.3-262.4	253.21± 95.89[a]	236-270.4	288.94± 93.28[b]	272.2-305.7	**0.001**	0.04
Vitamin B_{12} (µg)	4 (2.72)[a]	3.8-4.4	5 (3.4)[b]	5-6.4	3.75 (3.1)[a]	3.3-4.7	**<0.001***	0.05

CI: Confidence Interval, E: energy, IQR: Interquartile Range, M: Median, MUFA: monounsaturated fatty acids, PUFA: polyunsaturated fatty acids, SFA: saturated fatty acids 1ANOVA (post hoc Tukey test) or *Kruskal Wallis test (post hoc Tamhane's T2 test), whichever is appropriate, was used.
Different lowercase superscripts on the same row indicate a statistically significant difference between groups

Table 1. Continued

Energy and nutrients	Public		University		Private		p value1	Eta Squared (η2)
Number of hospitals	n=2		n=2		n=2			
Total number of menu days	n=122		n=122		n=122			
	x̄ ± SD or M (IQR)	%95 CI	x̄ ± SD or M (IQR)	%95 CI	x̄ ± SD or M (IQR)	%95 CI		
Vitamin C (mg)	78.64± 33.81[a]	72.5-84.7	77.99± 40.37[a]	70.7-85.2	101.73± 60.80[b]	90.8-112.6	**<0.001**	0.05
Calcium (mg)	620.34± 156.56[a]	592.3-648.4	613.97±163.28[a]	584.7-643.2	712.66± 236.61[b]	670.3-755	**<0.001**	0.05
Magnesium (mg)	247.28± 44.15[a]	239.4-255.2	247.96± 42.14[a]	240.4-255.5	267.03± 75.11[b]	253.6-280.5	**0.008**	0.03
Sodium (mg)	3449.49± 551.59[a]	3350.6-3548.4	3972.10± 837.01[b]	3822.1-4122.1	4289.63± 1259.92[c]	4063.8-4515.5	**<0.001**	0.12
Potassium (mg)	2104.29± 341.76[a]	2043-2165.5	2101.53± 352.51[a]	2038.3-2164.7	2359.73± 586.85[b]	2254.5-2464.9	**<0.001**	0.07
Iron (mg)	9.66± 1.94	9.3-10	9.89± 1.98	9.5-10.2	10.42± 3.58	9.8-11.1	0.070	0.02
Zinc (mg)	10.57± 2.53[a]	10.1-11	11.87± 2.54[b]	11.4-12.3	10.44± 3.01[a]	9.9-10.9	**<0.001**	0.05

CI: Confidence Interval, E: energy, IQR: Interquartile Range, M: Median, MUFA: monounsaturated fatty acids, PUFA: polyunsaturated fatty acids, SFA: saturated fatty acids 1ANOVA (post hoc Tukey test) or *Kruskal Wallis test (post hoc Tamhane's T2 test), whichever is appropriate, was used.
Different lowercase superscripts on the same row indicate a statistically significant difference between groups

(p<0.001, η^2=0.05). There are no significant differences in vitamin A, vitamin B2, and iron content among the groups (p>0.05).

The sodium content of the menus was 3449.49 mg in the public hospital group, 3972.1 mg in the university hospital group, and 4289.63 mg in the private hospital group; there was a statistically significant difference among all groups (p<0.001, η^2=0.12).

When compared with the TDG-2022 recommendations, the proportion of energy derived from carbohydrates and protein in the menus was consistent with the recommendations across all groups, whereas the proportion of energy derived from fat exceeded the recommended range (20–35%). The total fiber content of the menus and the fiber content per 1000 kcal were below the TDG-2022 recommendations (at least 25 grams and 14 g/1000 kcal, respectively) in all groups. It was found that the percentage of energy derived from saturated fat in the menus of all groups exceeded the TDG-2022 recommendations (<10%). While the percentage of energy derived from MUFAs was consistent with the TDG-2022 recommendations across all groups, the percentage of energy derived from PUFAs was below the TDG-2022 recommendations (7–10%). The cholesterol content of public and private hospital menus (306 and 315 mg, respectively) exceeds the TDG-2022 recommendations (<300 mg). In all groups, the vitamin B2, vitamin B6, calcium, magnesium, and potassium content of the menus was below the TDG-2022 recommendations, while the sodium content exceeded the recommended 2000 mg. The vitamin C content of public and university hospital menus falls below the TDG-2022 recommendations. The vitamin A, vitamin B1, vitamin B9, niacin equivalent, and iron content of the menus meet the TDG-2022 recommendations for all groups.

Data on NAR and MAR scores used to evaluate the dietary quality of hospital menus are presented in Table 2. Calcium, magnesium (excluding those calculated for women), potassium and folate NAR scores were similar in public and university hospital menus but significantly lower than in private hospital menus (p<0.05). This significant difference corresponds to a small to moderate effect size (η^2 values ranging from 0.02 to 0.07). The NAR score calculated for vitamin B12 was similar in the public and university hospital groups but significantly higher than that of the private hospital group (p<0.001, η^2=0.06). The NAR scores for calcium, magnesium (calculated only for men), potassium (calculated only for men), and folate

Table 2. NAR and MAR scores for hospital menus by age and gender groups

Variables	**Public**		**University**		**Private**		**p value[1]**	**Eta Squared (η^2)**
Number of hospitals	**n=2**		**n=2**		**n=2**			
Total number of menu days	**n=122**		**n=122**		**n=122**			
NAR Calcium (%)	**$\bar{x}$ ± SD**	**%95 CI**	**$\bar{x}$ ± SD**	**%95 CI**	**$\bar{x}$ ± SD**	**%95 CI**		
Men aged 19-70, Women aged 19-50	61.9± 15.2[a]	59.1-64.6	61.4± 16.3[a]	58.5-64.3	69.3± 18.7[b]	65.9-72.7	**<0.001**	0.05
Women aged 51-70	51.7± 13.0[a]	49.4-54	51.2± 13.6[a]	48.7-53.6	58.7± 17.5[b]	55.5-61.9	**<0.001**	0.05
NAR Magnesium (%)								
Men aged 19-30	61.8± 11.0[a]	59.8-63.7	61.9± 10.5[a]	60.1-63.8	66.0± 16.8[b]	63-69.1	**0.018**	0.02
Men aged 31-70	58.9± 10.5[a]	56.9-60.7	59.0± 10.0[a]	57.2-60.8	63.1± 16.4[b]	60.1-66	**0.013**	0.02
Women aged 19-30	78.9± 12.5	76.7-81.2	79.4± 12.4	77.2-81.6	81.0± 15.4	78.3-83.8	0.442	0.004
Women aged 31-70	76.8± 12.6	74.5-79	77.1± 12.4	74.9-79	79.2± 15.9	76.4-82.1	0.308	0.01
NAR Potassium (%)								
Men aged 19-70	61.8± 10.0[a]	60.1-63.7	61.8± 10.3[a]	59.9-63.6	68.8± 15.5[b]	66.1-71.6	**<0.001**	0.07
Women aged 19-70	80.5± 12.1[a]	78.3-82.7	80.2± 12.2[a]	77.9-82.3	85.4± 13.9[b]	82.9-87.8	**0.002**	0.03
NAR Vitamin A (%)								
Men aged 19-70	87.9± 16.1[a,b]	85.1-90.8	84.8± 16.9[a]	81.7-87.8	91.7± 14.8[b]	89.1-94.4	**0.003**	0.03
Women aged 19-70	95.4± 10.1	93.6-97.2	94.0± 11.3	91.9-96.1	96.7± 9.2	95.1-98.3	0.125	0.01
NAR Vitamin E (%)								
Men and women aged 19-70	67.0± 23.3[a,b]	62.9-71.2	60.6± 24.5[a]	56.2-65	70.5± 24.5[b]	66.1-74.8	**0.005**	0.03
NAR Vitamin C (%)								
Men aged 19-70	77.4± 23.3[a,b]	73.2-81.6	73.5± 25.3[a]	68.9-78.1	82.5± 22.6[b]	78.4-86.5	**0.014**	0.02
Women aged 19-70	84.8± 21.1[a,b]	81-88.6	80.6± 23.2[a]	76.5-84.7	88.2± 19.6[b]	84.6-91.6	**0.023**	0.02
NAR Vitamin B_1 (%)								
Men aged 19-70	64.5± 12.8[a]	62.2-66.7	66.1± 15.3[a,b]	63.4-68.9	69.4± 16.4[b]	66.5-72.3	0.033	0.02
Women aged 19-70	70.0± 13.2[a]	67.7-72.4	71.6± 15.6[a,b]	68.8-74.4	74.8± 16.4[b]	71.9-77.8	0.041	0.02

MAR: Mean Adequacy Ratio, NAR: Nutrient Adequacy Ratio

[1]ANOVA and the Tukey post hoc test were used.

Different lowercase superscripts on the same row indicate a statistically significant difference between groups.

Table 2. Continued

Variables	**Public**		**University**		**Private**		**p value**[1]	**Eta Squared (η^2)**
Number of hospitals	**n=2**		**n=2**		**n=2**			
Total number of menu days	**n=122**		**n=122**		**n=122**			
NAR Calcium (%)	**$\bar{x}$ ± SD**	**%95 CI**	**$\bar{x}$ ± SD**	**%95 CI**	**$\bar{x}$ ± SD**	**%95 CI**		
NAR Vitamin B_2 (%)								
Men aged 19-70	86.3± 13.5	83.9-88.8	86.8± 14.2	84.3-89.4	88.1± 14.5	85.6-90.7	0.603	0.003
Women aged 19-70	94.1± 9.7	92.4-95.8	93.7± 9.7	91.9-95.4	94.5± 10.6	92.6-96.4	0.814	0.001
NAR Folate (%)								
Men and women aged 19-70	62.5± 15.9[a]	59.7-65.3	61.6± 17.7[a]	58.4-64.7	70.2± 18.7[b]	66.9-73.6	**<0.001**	0.05
NAR Vitamin B_{12} (%)								
Men and women aged 19-70	96.2± 10.7[a]	94.3-98.2	97.9± 8.7[a]	96.4-99.5	89.5± 19.9[b]	85.9-93.1	**<0.001**	0.06
MAR (%)								
Men aged 19-30	72.8± 7.8[a]	71.4-74.1	71.7± 8.6[a]	70.1-73.2	76.6± 10.5[b]	74.7-78.5	**<0.001**	0.05
Men aged 31-70	72.5± 7.8[a]	71.1-73.9	71.4± 8.6[a]	69.8-72.9	76.3± 10.5[b]	74.5-78.2	**<0.001**	0.05
Women aged 19-30	79.1± 7.3[a]	77.8-80.4	78.1± 8.2[a]	76.6-79.5	82.0± 9.5[b]	80.3-83.7	**0.001**	0.04
Women aged 31-50	78.9± 7.3[a]	77.6-80.2	77.9± 8.2[a]	76.4-79.3	81.8± 9.5[b]	80.1-83.5	**0.001**	0.04
Women aged 51-70	77.9± 7.2[a]	76.6-79.2	76.9± 8.0[a]	75.4-78.3	80.8± 9.3[b]	79.1-82.4	**0.001**	0.04

MAR: Mean Adequacy Ratio, NAR: Nutrient Adequacy Ratio

[1]ANOVA and the Tukey post hoc test were used.

Different lowercase superscripts on the same row indicate a statistically significant difference between groups.

in public and university hospital menus were found to be below 66%. In addition, the NAR score for vitamin B1 (calculated for men only) in public hospital menus and the NAR score for vitamin E in university hospital menus were found to be below 66%. The NAR scores for calcium (calculated for women aged 51–70) and magnesium (calculated for men aged 31–70) in private hospital menus were found to be below 66%.

There was no significant difference in the MAR score calculated for all age and gender groups examined between public hospitals (72.5-79.1%) and university hospitals (71.4-78.1%), while the MAR score for private hospitals (76.3-82.0%) was significantly higher than the other two hospitals ($p<0.001$, η^2 values ranging from 0.04 to 0.05). The MAR scores calculated for men based on public and university hospital menus were found to be below 75% (indicating an overall deficiency in micronutrient intake).

Discussion

Hospital menus are a key communication tool between hospitals and inpatients and can also support disease-specific nutrition education.[20] Ensuring that menus meet energy and nutrient requirements is essential for recovery and the prevention of adverse outcomes.[5] In this study, menus from public, university, and private hospitals consistently failed to meet TDG-2022 recommendations for fiber, vitamin B2, vitamin B6, calcium, potassium, magnesium, and PUFAs energy contribution, while exceeding recommended limits for sodium, total fat, and saturated fat. This pattern indicates a systematic imbalance in menu planning, characterized by energy-dense but nutrient-poor food choices. Our findings have important implications for improving healthcare practices and policies in the areas of identifying and managing malnutrition in hospital settings.

The coexistence of high sodium and fat levels with low fiber and micronutrient content suggests a reliance on processed foods and insufficient inclusion of plant-based food groups and dairy. Our findings are similar to the results of studies conducted in hospital settings in Türkiye and other countries.[1,3,21] A study examining five hospital menus from four different regions of Türkiye found that all hospital menus contained higher levels of energy, protein, fat, saturated fat, and sodium than recommended by TDG-2022, while fiber content was below TDG-2022 recommendations.[21] A study conducted in Spain found that menus offered to patients were deficient in vitamin E, magnesium, calcium, potassium, and zinc.[3] High sodium, low dietary fiber, and low potassium intake are well-known risk factors for noncommunicable diseases such as hypertension and cardiovascular disease.[22]

Nutrient adequacy analyses further reinforce these concerns. MAR scores indicated that menus in public and university hospitals require improvement, while NAR results highlighted insufficient intake of key micronutrients such as calcium, magnesium, potassium, vitamin E, vitamin B1, and folate. In a study examining the menus of four public hospitals in Ankara, although overall adequacy scores were reported to be higher, the NAR scores calculated for calcium, magnesium, and dietary fiber were found to be lower than those of other nutrients.[8] The consistent identification of low calcium, magnesium, fiber, and high fat, saturated fat and sodium levels across studies suggests a persistent structural issue in menu composition rather than isolated deviations.[1,8,22]

In a study examining menus at a hospital in Greece during different seasons, it was found that the dairy content of the menus did not meet European guidelines.[23] In a study examining the menus of a hospital in Portugal, it was found that the menus evaluated during the study period did not comply with nutritional recommendations in terms of cooking techniques, vegetable, fruit, and fish content.[4]

In our study, the observed nutrient profile may be explained by menu composition, particularly the higher proportion of red meat and processed foods and the limited inclusion of vegetables, fruits, legumes, and dairy products. Processed foods have low nutrient density and contain higher levels of saturated fat, sugar, and sodium. An analysis of hospital menu in Greece found that the median energy value from ultra-processed foods was 25.2%, and that the energy provided by minimally processed foods was positively associated with the menu's energy, protein, zinc, selenium, iron, and vitamin B12 content.[24] Ultra-processed food consumption has been linked to an increased risk of cardiovascular disease and cancer, making it crucial to limit such foods in hospital menus for the protection and promotion of health.[25]

International experiences demonstrate that implementing nutritional standards in hospital food services can significantly improve menu quality.[22,24,26] However, compliance with such standards remains inconsistent. A systematic review of five observational studies involving

thirty hospitals reported that approximately half of the hospitals did not comply with nutritional standards.[9]

This study contributes to the literature by comparing different hospital types and evaluating nutrient adequacy across demographic groups. However, the findings should be interpreted in light of several limitations. The first limitation is that the study only examined the nutritional profiles of menus over a total of two months at hospitals in the provinces of Ankara and Konya. Each hospital group (public, university, and private) is represented by only two hospitals. Although the unit of analysis was defined as the daily menu, the evaluated menus were derived from a limited number of hospitals. Therefore, the observations may not be fully independent, which may introduce a potential risk of pseudoreplication and reduce the generalizability of the findings. In addition, multiple statistical comparisons were conducted across numerous nutrients and dietary quality indicators, which may increase the risk of type I error inflation despite the application of appropriate statistical procedures. Seasonal variations and geographic region are important factors influencing menu planning. In our study, we analyzed the autumn (one-month) and spring (one-month) menus of hospitals in the Central Anatolia Region (with limited representativeness). This does not constitute a sufficient source of information about the nutrient profile of menus in hospitals across Türkiye. The second limitation concerns the fact that the consumption status of the examined menus was not questioned. The analysis of the nutritional values of meals served to hospitalized patients is an indicator of dietary intake when the entire meal is consumed. In this study, only the menus served by the hospital kitchen were evaluated, and the consumption records of these foods by patients were not examined. Since our study lacks patient-level dietary intake data, the use of NAR and MAR to assess dietary quality may overestimate nutrient adequacy. In addition, 50 grams of roll bread was included in the analysis for each meal, but actual consumption could not be verified. Another limitation of the study is its focus on normal diet menus. In reality, hospitals offer a wide variety of diet menus tailored to patients' specific needs, such as low-sodium, diabetic, and low-fat diets. Furthermore, since patients' energy and nutrient requirements may vary depending on their medical conditions, the use of standard DRI values in menu evaluations may lead to inaccurate assessments. Future research should incorporate actual intake data and clinical outcomes, such as length of hospital stay and malnutrition incidence, to better understand the impact of menu quality on patient health.

Conclusion

Hospital meals are an important part of institutional care and nutritional support for inpatients, so their nutrient content should be reviewed at regular intervals. Our study found that none of the hospital menus examined met the fiber, vitamin B2, vitamin B6, calcium, potassium, magnesium, and PUFAs content recommendations of TDG-2022. Menus should be improved to address identified nutrient deficiencies, and the levels of energy from fat, energy from saturated fat, and sodium content that exceed recommended limits should be reduced. In addition, hospital menus need to be developed with due consideration for the micronutrient requirements of specific age and gender groups (particularly men and women aged 51–70). It is thought that reducing the frequency of red meat and increasing the frequency of vegetables, fruits, and dairy products in menus would be a beneficial approach. However, it should be noted that our study has a limited sample size and includes only a two-month analysis of menus; therefore, our findings may not be applicable to all hospital menus.

In conclusion, this study advances the existing body of knowledge by providing a comprehensive comparison of menus in public, university, and private hospitals in Türkiye in terms of their nutrient content. The findings highlight an urgent need to standardize hospital food services nationwide in strict accordance with the TDG-2022 guidelines. From a practical and policy perspective, our results offer valuable evidence for Turkish healthcare policymakers and administrators.

Ethical approval

Ethical approval for this study was obtained from the Non-Interventional Clinical Research Ethics Committee of Alanya Alaaddin Keykubat University (Approval No. E-25767966-050.04-325976).

Author contributions

Conception and design: A.K., A.E.B.; Data acquisition: A.K.; Data analysis: A.K.; Data interpretation: A.K.; Drafting of the manuscript: A.K., A.E.B.; Critical revision of the manuscript: A.K., A.E.B. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

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Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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Evaluation of the relationship between mini nutritional assessment-short form and prognostic nutritional index in individuals aged 80 years and older

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ABSTRACT

Objective: This study investigated the relationship between the Mini Nutritional Assessment-Short Form and the Prognostic Nutritional Index in clinically stable, community-dwelling individuals aged 80 years and older, while examining associations with chronic diseases and demographic characteristics.

Materials and Methods: The study was conducted with 167 elderly individuals registered at the Konya City Hospital Healthy Living Outpatient Clinic. Participants' demographic data, caregiver status, and chronic comorbidities were retrieved from patient medical records. Nutritional status was assessed using the Mini Nutritional Assessment-Short Form. The Prognostic Nutritional Index was calculated using serum albumin levels and total lymphocyte counts recorded in the hospital database within the preceding three weeks. Data analysis was performed using SPSS version 27.0. For variables with non-normal distribution, the Mann–Whitney U test and Spearman's correlation coefficient were employed.

Results: The median age was 85 years, with a high prevalence of multiple chronic conditions. No significant differences in Prognostic Nutritional Index values were found across Mini Nutritional Assessment categories ($p > 0.05$). The correlation between the Mini Nutritional Assessment and the Prognostic Nutritional Index was weak and not statistically significant ($r = 0.088$; $p = 0.260$). Furthermore, neither assessment showed significant associations with age, sex, or the total number of chronic diseases ($p > 0.05$).

Conclusion: Findings suggest that the Mini Nutritional Assessment and the Prognostic Nutritional Index reflect different dimensions of nutritional status in the very old and do not necessarily yield parallel results. In this age group, functional assessment tools may provide more discriminative information than biochemical parameters. Larger-scale studies are warranted to clarify the potential complementary use of these two methods in geriatric nutritional evaluation.

Keywords: nutritional assessment, nutritional status, geriatric population, aged, 80 and over

Introduction

Malnutrition plays a decisive role in determining morbidity and mortality in the advanced age group, as it substantially affects both physical and functional capacity. Age-related factors, such as sarcopenia, increased burden of chronic diseases, polypharmacy, loss of appetite, and decline in functional performance, significantly elevate the risk of malnutrition among older adults.[1] Considering that malnutrition serves as a precursor to numerous

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pathological conditions, either through direct or indirect pathways, its early detection and clinical management are paramount for optimizing patient outcomes in healthcare settings.[2] Previous studies have reported the prevalence of malnutrition in this population ranging between 15% and 29%.[3-5] Therefore, early identification of nutritional status in elderly individuals is crucial for preventing adverse clinical outcomes and for planning timely and appropriate interventions.

The Mini Nutritional Assessment–Short Form (MNA-SF) is one of the most widely used screening tools for detecting malnutrition in older populations, and its validity and reliability have been confirmed in numerous studies.[6-8] Incorporating multidimensional parameters such as weight loss, appetite, mobility, neuropsychiatric status, and body mass index, it provides a comprehensive evaluation of nutritional status in elderly individuals.

In contrast, the Prognostic Nutritional Index (PNI) is a simple yet effective immuno-nutritional marker based on serum albumin levels and total lymphocyte count. Initially introduced by Onodera and colleagues to predict surgical mortality, PNI has increasingly been utilized in geriatric populations in recent years.[9,10] While serum albumin is associated with chronic inflammation, liver function, and protein–energy malnutrition, lymphocyte count reflects immune response capacity. Accordingly, PNI has been shown to correlate with morbidity, risk of infection, frequency of hospitalization, and overall life expectancy.[9,11,12]

Although MNA-SF and PNI assess different physiological domains, both have been reported to be useful in identifying nutritional risk in older adults and to be associated with clinical outcomes. However, studies directly comparing MNA-SF and PNI are limited, and the nature of their relationship within the same population remains unclear. It has been suggested that MNA-SF offers a broader evaluation due to its focus on functional and behavioral components, whereas PNI primarily reflects biochemical and immunological status.[13] Evaluating these two methods together may therefore provide a more integrated perspective on nutritional status in elderly individuals.

Main Points

- Lack of Association: In clinically stable octogenarians, MNA-SF scores were not significantly correlated with PNI values or risk categories.
- Distinct Dimensions: Functional screening (MNA-SF) and immuno-nutritional markers (PNI) assess different physiological dimensions and may not yield parallel results in very old adults.
- Methodological Impact: High functional stability and ceiling effects in community-dwelling elders can limit the discriminatory capacity of standard PNI cutoffs.
- Multidimensional Need: Geriatric nutritional evaluation in octogenarians requires a comprehensive strategy combining both functional and population-specific biochemical indicators.

The present study aimed to examine MNA-SF and PNI values in individuals aged 80 years and older and to assess the relationship between these two nutritional measures. Additionally, the associations between nutritional status, chronic disease burden, and demographic variables were investigated to provide evidence regarding the use of practical and reliable nutritional screening tools in older adults. As the elderly population continues to grow, comparing nutritional assessment instruments and clarifying their role in clinical practice is increasingly important for the development of healthy aging policies.

Materials and Methods

This study is a retrospective, cross-sectional, and descriptive research conducted on clinically stable individuals aged 80 years and older, registered at the Konya City Hospital Healthy Living Outpatient Clinic, who exhibited no signs of active acute illness. Data were obtained through a retrospective review of records from scheduled home visits. Ethical approval for the study was granted by the KTO Faculty of Medicine Ethics Committee on January 29, 2026 (Decision No: 2026/34). A total of 167 elderly individuals, who were visited at their residences and whose information was recorded in patient files between August 1, 2024, and July 31, 2025, were included in the study. These participants represented a community-dwelling population evaluated during routine geriatric home visits, and their clinical stability was characterized by the absence of acute hospital-level complaints at the time of evaluation. Exclusion criteria consisted of severe cognitive impairment, active infection, and known malignancy. In addition, individuals were excluded if they had incomplete biochemical data, laboratory measurements obtained outside the 3-week period preceding the home visit, or incomplete clinical records.

A total of approximately 170 individuals were excluded, primarily due to missing or incomplete laboratory data. Due to the retrospective nature of the study and reliance on pre-existing hospital records, the exact number of excluded participants per category could not be systematically retrieved. Furthermore, history of systemic corticosteroid exposure could not be systematically screened or verified from the available retrospective records. Severe cognitive impairment was defined as a Mini-Mental State Examination (MMSE) score of <10, based on previously recorded assessments in patient medical records. Individuals with MMSE scores at or below this threshold were excluded from the study. Active infection was assessed clinically and supported by laboratory markers, including C-reactive protein (CRP), to exclude acute inflammatory conditions. Patients with known malignancy were excluded due to their potential impact on serum albumin levels and lymphocyte counts, which could confound PNI values.

Participants' demographic characteristics (age, sex), caregiver status (self-care, spouse, child/grandchild, professional caregiver), and the presence of chronic diseases (diabetes mellitus, hypertension, hyperlipidemia, coronary artery disease, dementia, thyroid disease, osteoporosis/osteoarthritis, asthma-COPD, chronic kidney disease, and cerebrovascular accident) were retrieved from patient records, along with Mini Nutritional Assessment (MNA-SF) scores obtained during the visits. Recent weight loss was not treated as an independent exclusion criterion, as it was already systematically evaluated and scored as a core component within the MNA-SF framework. Comorbidities were evaluated as the presence or absence of predefined chronic conditions and as the total number of comorbid diseases. No weighted comorbidity index or disease severity scoring system was applied due to the retrospective design of the study. For the biochemical data required to calculate the Prognostic Nutritional Index (PNI), serum albumin and lymphocyte counts performed within the three weeks preceding the visit date were screened via the hospital's electronic medical record system. Individuals whose laboratory tests exceeded this timeframe or were missing were excluded from the study. No new blood samples were collected within the scope of this research.

Nutritional assessment

Mini nutritional assessment (MNA-SF)

Nutritional status was evaluated using the Mini Nutritional Assessment–Short Form (MNA-SF). The MNA-SF consists of six components: appetite loss, weight loss, mobility, neuropsychiatric status, acute disease/stress, and body mass index or calf circumference.

Total scores range from 0 to 14 and were interpreted as follows:

- 12–14 points: Normal nutritional status
- 8–11 points: At risk of malnutrition
- 0–7 points: Malnutrition.[6]

Prognostic nutritional index (PNI)

PNI was calculated using serum albumin levels (g/L) and total lymphocyte count (10^3/μL) according to the following formula:

PNI = Albumin (g/L) + 5 × Lymphocyte count (10^3/μL)

Based on international literature:

- PNI ≥ 50: Normal nutritional status
- PNI < 50: Risk of malnutrition.[9]

The study was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical analyses were performed using IBM Statistical Package for the Social Sciences (SPSS) for Windows, version 27.0. Descriptive statistics were presented as frequency (n) and percentage (%) for categorical variables; mean ± standard error for normally distributed continuous variables; and median with interquartile range (25th–75th percentile) for non-normally distributed variables. Normality was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. The Chi-square test was used to compare categorical variables. For continuous variables that were not normally distributed, the Mann–Whitney U test was applied. Correlations between numerical variables were analyzed using Spearman's correlation test. A p-value < 0.05 was considered statistically significant.

Table 1. Demographic and clinical characteristics of the study population

Age (years)	85 (83-88)	
Median (IQR)		
	%	**n**
Sex		
Female	67.1	112
Male	32.9	55
Caregiver status		
Self	15.0	25
Spouse	14.4	24
Child/grandchild	70.0	117
Professional caregiver	0.6	1
Chronic diseases*		
Diabetes mellitus	31.7	53
Hypertension	68.3	114
Hyperlipidemia	11.4	19
Coronary artery disease	28.1	47
Dementia	18.0	30
Thyroid disorders	3.6	6
Osteoporosis/osteoarthritis	9.0	15
Asthma–COPD	26.9	45
Chronic kidney disease	2.4	4
Cerebrovascular disease	5.4	9
MNA-SF score	12 (10-14)	
Median (IQR)		
PNI score	47.61 ± 0.48	
Mean± SE		

MNA-SF: Mini Nutritional Assessment; PNI: Prognostic Nutritional Index; COPD: Chronic Obstructive Pulmonary Disease
*More than one chronic condition could be selected
IQR: Interquartile range; SE: Standard error

Table 2. Comparison of PNI values according to MNA-SF groups

	MNA-SF ≥12	**MNA-SF <12**	**p**
PNI	47.20 (43.45-51.20)	48.30 (43.20-52.10)	0.979

MNA-SF: Mini Nutritional Assessment; PNI: Prognostic Nutritional Index

Table 3. Comparison of MNA-SF values according to PNI groups

	PNI ≥50	**PNI <50**	**p**
MNA-SF	12 (10-14)	12 (10-13)	0.658

MNA-SF: Mini Nutritional Assessment; PNI: Prognostic Nutritional Index

Table 4. Spearman correlations between MNA-SF, PNI, Age, and number of chronic diseases

	r	**p**
MNA-SF-PNI	0.088	0.260
MNA-SF-age	0.035	0.655
PNI-age	-0.036	0.643
MNA-SF- chronic diseases	-0.081	0.297
PNI- chronic diseases	-0.067	0.387

MNA-SF: Mini Nutritional Assessment; PNI: Prognostic Nutritional Index

Results

The median age of the participants was 85 years (interquartile range: 83–88). Of the study population, 67.1% were female. Hypertension was the most frequently observed chronic condition (68.3%). According to the MNA-SF classification, the majority of participants were within the normal nutritional range (Table 1).

PNI values were similar between individuals with normal nutritional status and those at risk of malnutrition according to the MNA-SF classification (p = 0.979) (Table 2).

MNA-SF scores were comparable between participants with low PNI (<50) and those with normal PNI (≥50), and no statistically significant difference was observed between the two groups (p = 0.658) (Table 3).

No significant correlations were found between MNA-SF and PNI, age, or the number of chronic diseases (r = 0.088, p = 0.260; r = 0.035, p = 0.655; r = –0.081, p = 0.297, respectively).

Similarly, no correlation was observed between PNI and age (r = –0.036, p = 0.643). In addition, there was no statistically significant association between PNI and the number of chronic diseases (r = –0.067, p = 0.387) (Table 4).

Table 5. Comparison of PNI levels according to the presence of comorbid diseases

Diseases	With disease – median (IQR) PNI	Without disease – median (IQR) PNI	p
Diabetes mellitus	48.20 (41.52-53.20)	47.52 (44.06-50.82)	0.662
Hypertension	47.30 (43.00- 52.10)	48.50 (43.75-51.75)	0.703
CAD	47.00 (43.00-50.50)	48.20 (43.50-52.25)	0.344
Dementia	46.70 (41.20-49.20)	48.25 (43.80-52.25)	0.084
Asthma–COPD	48.50 (45.00-50.70)	47.30 (43.00-52.10)	0.653

PNI: Prognostic Nutritional Index. IQR: Interquartile range CAD: Coronary Artery Disease COPD: Chronic Obstructive Pulmonary Disease

To further explore PNI as a continuous variable, a binary logistic regression analysis was conducted, treating continuous PNI as an independent predictor and the presence of malnutrition risk (MNA-SF < 12) as the dependent outcome. The analysis revealed that continuous PNI values were not significantly associated with the odds of being at risk for malnutrition (Odds Ratio [OR] = 1.002, 95% Confidence Interval [CI] = 0.941–1.066, p = 0.957).

No statistically significant differences were observed in PNI levels according to the presence of diabetes mellitus, hypertension, coronary artery disease (CAD), dementia, or asthma/COPD (p > 0.05) (Table 5).

Discussion

This study was conducted to explore the relationship between the Mini Nutritional Assessment (MNA-SF) and the Prognostic Nutritional Index (PNI) in individuals aged 80 years and older. Although the study population consisted of community-dwelling individuals followed in a Healthy Aging program, this should not be interpreted as a completely healthy cohort. A considerable proportion of participants had multiple chronic diseases, reflecting a multimorbid elderly population. However, as these individuals were clinically stable and not experiencing acute illness, variability in biochemical and functional parameters may have been relatively limited. This context may have contributed to the lack of significant associations observed. Given the lack of statistically significant associations and the low post-hoc statistical power observed in this cohort, any definitive conclusion regarding whether the MNA-SF and PNI reflect entirely different dimensions of nutritional status remains strictly hypothetical. While it is theoretically plausible that function-based tools and biochemical markers assess distinct physiological pathways, the observed lack of correlation cannot be confidently attributed to a true physiological divergence. Instead, it must be carefully weighed against the high probability of a Type II error due to sample size limitations. Therefore, our current data do not allow for a clear differentiation between a true absence of clinical overlap and an underpowered statistical artifact. An important characteristic of the study population was that all participants were clinically stable, community-dwelling individuals without active medical complaints. This context is particularly relevant, as biochemical indicators may not reflect acute physiological stress in such a stable population.

The absence of a statistically significant association between MNA-SF categories and PNI values is in line with previous studies reporting that functional nutritional measures and immunonutritional biomarkers do not always demonstrate parallel trends in elderly populations. Gong et al. demonstrated that functional measures such as MNA-SF and immunonutritional indicators like PNI do not necessarily correlate in orthopedic and neurosurgical patients.[14] Similarly, Ayraler et al. reported no significant relationship between PNI and nutritional status in their cohort.[10] These findings are consistent with the results of the present study.

Several contextual factors may help explain these observations. All participants were registered at a Healthy Aging Clinic and were clinically stable without acute complaints, which may have limited variability in both MNA-SF and PNI values. In addition, home-based assessments may have contributed to relatively preserved nutritional status, potentially reflecting adequate social support and family-based care. Furthermore, PNI was calculated using laboratory data obtained within a three-week window, reducing the likelihood of acute physiological fluctuations influencing biochemical parameters. Taken together, these factors may partly account for the observed lack of statistically detectable association in this cohort.

Although serum albumin has long been considered a marker of malnutrition, studies indicate that in older adults, it is also influenced by chronic inflammation, hydration status, liver function, and overall comorbidity burden.[15,16] Because our participants were clinically stable and not experiencing active disease exacerbations, large fluctuations in PNI values would not be anticipated. Furthermore, the prognostic value of PNI has been shown to be more pronounced in populations with malignancy, advanced organ failure, significant inflammatory burden, or acute catabolic states.[17-20] In such high-risk groups, PNI may more sensitively reflect disease progression and nutritional deterioration. In contrast, the largely stable and community-based nature of our cohort may have attenuated the discriminatory capacity of PNI. These findings suggest that while PNI may serve as a valuable monitoring tool in high-risk or progressive clinical conditions, function-based instruments, such as MNA-SF, may offer greater explanatory value in stable, community-dwelling elderly populations.

Similarly, lymphocyte count—another component of the PNI formula—is influenced by various non-nutritional factors in older adults, including chronic inflammation, infections, medications (e.g., corticosteroids), and immunosenescence associated with aging. Consequently, the lymphocyte multiplier included in the PNI calculation may not adequately capture nutritional status when considered in isolation in very old individuals.

The minimal and statistically non-significant correlation between MNA-SF and PNI may also be attributable to the functional independence of many participants and the strong family support structures observed within this population. Home-based evaluations indicated that participants generally maintained regular meal patterns, received social support, and had their care needs adequately addressed. The literature consistently emphasizes that living at home and receiving family support are associated with better nutritional outcomes in older adults.[21,22] This social context may have influenced overall nutritional status and contributed to reduced variability in both MNA-SF and PNI values.

The lack of significant associations between MNA-SF or PNI and age, sex, or total chronic disease burden further highlights the heterogeneity of the very old population. Among individuals aged 80 years and above, biological age and physiological reserve may vary substantially; therefore, the number of chronic conditions alone may not reliably determine nutritional risk.

This study has several limitations. Its single-center design and relatively modest sample size may limit the generalizability of the findings. A post-hoc power analysis indicated low statistical power (0.204), suggesting an increased risk of type II error. Therefore, the lack of statistically significant associations between MNA-SF and PNI should be interpreted with caution, as it may reflect limited statistical power rather than absence of a true relationship.

The PNI cut-off value (≥ 50), originally derived from surgical and oncological populations, may not be fully applicable or sufficiently discriminatory in very old, community-dwelling individuals. In the present cohort, the mean PNI value (47.61 ±0.48) indicates that a substantial proportion of stable participants fell below this surgical threshold without exhibiting functional malnutrition. To avoid the loss of statistical power associated with artificial categorization, we evaluated PNI as a continuous measure through Spearman's correlation analysis; however, no significant linear association was detected. This lack of significance strongly reinforces that standard tumor-associated or acute-care cut-offs suffer from a floor effect in stable geriatric screening setups. Population-specific, age-adjusted cut-off values are therefore urgently warranted in geriatric medicine to accurately capture subclinical nutritional deficits. Furthermore, the distributional characteristics of the MNA-SF within this cohort present an inherent statistical constraint that helps interpret the negative findings. The MNA-SF scores exhibited a notable ceiling effect, characterized by a high median score of 12 and a tight interquartile range (IQR 10–14), positioning a large proportion of the participants near the maximum possible score of 14. This skewed distribution severely limited the variance and statistical variability of the nutritional status data. Mathematically, a restricted variance and a prominent ceiling effect significantly diminish the statistical power of correlation coefficients, rendering the detection of potential linear relationships with the PNI highly improbable. Consequently, the observed lack of significant association must be interpreted not only as a potential clinical divergence but also as a statistical artifact resulting from restricted variability and a ceiling effect within this stable, community-dwelling geriatric sample.

Although MNA-SF was assessed at the time of home visits, serum albumin values used for PNI calculation were obtained from the 3-week period preceding the assessment. Given the retrospective design, this

approach was adopted to maximize data availability and to approximate biochemical status close to the time of functional evaluation. Considering the approximately 20-day half-life of albumin, this interval is unlikely to introduce major bias; however, a degree of temporal mismatch cannot be completely excluded.

Although C-reactive protein (CRP) values were available within the same time window, they were not systematically included in the analysis as an inflammatory covariate. Therefore, residual confounding due to subclinical inflammation may have influenced albumin and lymphocyte-related parameters, potentially affecting PNI interpretation. Future prospective studies incorporating structured, continuous inflammatory biomarkers like CRP as direct covariates are needed to better isolate the independent nutritional predictive value of the PNI.

Comorbidities were assessed based on presence or total number, without evaluation of severity or use of validated indices such as the Charlson Comorbidity Index. Given the heterogeneity of health status in individuals aged ≥80 years, this approach may not fully capture comorbidity burden and its relationship with nutritional status.

The use of the Mini Nutritional Assessment–Short Form (MNA-SF) instead of the full MNA-SF may have reduced sensitivity in relatively well-functioning older adults, potentially resulting in a ceiling effect and limiting variability in nutritional classification.

The reported prevalences of certain chronic comorbidities—such as chronic kidney disease (2.4%), thyroid disorders (3.6%), and osteoporosis/osteoarthritis (9.0%)—appear lower than standard geriatric epidemiological expectations for a cohort with a median age of 85 years. This discrepancy stems from the retrospective nature of our study; the comorbidity data were strictly gathered from hospital electronic health records and the national 'e-Nabız' system based on documented ICD-10 codes. In general health registry systems, secondary, stable, or subclinical chronic conditions are prone to under-documentation or under-coding during routine institutional encounters. Consequently, these rates reflect documented diagnostic codes rather than proactive, systematic clinical screening, which should be considered a form of documentation bias inherent to retrospective designs.

A substantial number of individuals (approximately 170) were excluded from the study primarily due to missing electronic records, incomplete laboratory parameters, or unresolvable data gaps. Because the exact baseline clinical characteristics and demographic distribution of these excluded individuals could not be retrieved due to the retrospective archive framework, a potential risk of significant selection bias must be acknowledged. It is highly plausible that these excluded patients represented a frailer, older, or more nutritionally vulnerable subgroup who may have been less capable of undergoing comprehensive screening or who suffered from severe acute comorbidities that led to incomplete documentation. Consequently, our final cohort might represent a relatively healthier or more stable survival sample, which could potentially limit the generalizability of our findings to the entire geriatric or malnourished population. This limitation underscores the need for future prospective, multi-center studies with comprehensive, consecutive patient enrollment to mitigate selection bias.

Strengths of the study include data collection through home visits, which reduces hospital-based selection bias, and the exclusive focus on individuals aged 80 years and older, a population that remains underrepresented in nutritional research. Overall, these findings suggest that MNA-SF and PNI reflect different dimensions of nutritional status in older adults, highlighting the multidimensional nature of geriatric nutritional assessment.

Conclusion

In conclusion, this study demonstrates no statistically significant correlation between MNA-SF and PNI categories or continuous scores in clinically stable individuals aged 80 years and older. However, due to the low post-hoc statistical power of our sample, it remains statistically uncertain whether this lack of association reflects a genuine conceptual independence between functional and immunonutritional dimensions or a limitation in sample size. Consequently, no definitive assertions can be made regarding the interchangeable or complementary nature of these tools based on our data alone. To resolve this ambiguity, adequately powered, large-scale prospective trials are required to rigorously evaluate the clinical interplay between functional screening tools and biochemical indices in geriatric populations.

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Author contributions

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Ethical approval

This study was approved by the Ethics Committee of the KTO Faculty of Medicine (Date: January 29, 2026, Decision/Protocol No: 2026/34). Informed consent was obtained from all participants involved in this study.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that during the preparation of this study, the following AI-assisted technology was used: Gemini on 06/05/2026. Extent of Use: During the preparation of this work, the authors used Gemini for language support and editorial editing. The authors reviewed and edited the output as needed and take full responsibility for the content of the published article.. The authors confirm that they have critically reviewed and edited any AI-generated content and take full responsibility for the integrity, accuracy, and originality of the publication. The authors certify that the original human contribution is maintained and that AI-assisted tools are not listed or cited as authors.

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The adipokine functions of nesfatin-1: Its effects on adipose tissue physiology and metabolic diseases

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ABSTRACT

Nesfatin-1, a pleiotropic peptide derived from the nucleobindin-2 precursor, exhibits widespread distribution in the central nervous system and peripheral tissues. It plays a critical role in regulating energy homeostasis. Its high concentration in the appetite control centers of the hypothalamus indicates that Nesfatin-1 is a potent anorexigenic peptide, which suppresses food intake through melanocortin-dependent but leptin-independent mechanisms. Its capacity to augment insulin secretion and regulate glucose metabolism by activating L-type calcium channels in pancreatic β-cells renders Nesfatin-1 a promising therapeutic target, particularly for type 2 diabetes. Nesfatin-1 has also been shown to be active in hypothalamic and limbic circuits associated with stress, anxiety, and behavioral responses. Levels of Nesfatin-1 exhibit fluctuations in response to acute and chronic stress conditions. Nesfatin-1 has been reported to increase arterial pressure via central oxytocinergic and melanocortinergic pathways in the cardiovascular system. At the peripheral level, it produces vasoconstrictive effects via eNOS inhibition. Although Nesfatin-1 has been demonstrated to possess anti-inflammatory and anti-apoptotic properties in experimental models, the findings from clinical studies are heterogeneous, and its potential as a biomarker remains uncertain. In summary, Nesfatin-1 is a significant molecule that has the potential to contribute to the development of novel treatment strategies for obesity, diabetes, neuropsychiatric disorders, and inflammatory processes. However, it is imperative to define its mechanisms with precision.

Keywords: hypothalamus, nesfatin-1, energy homeostasis, adipokines, obesity

Introduction

The intricate interplay among energy homeostasis, appetite regulation, glucose metabolism, and neuroendocrine signaling plays a pivotal role in maintaining physiological balance and in the pathogenesis of metabolic disorders such as obesity, type 2 diabetes mellitus, and metabolic syndrome. Nesfatin-1, a member of the anorexigenic peptide family, has emerged as a key regulatory molecule involved in these complex processes. Following its initial identification in hypothalamic nuclei by Oh-I et al. in 2006, nesfatin-1 rapidly gained attention as a critical factor in metabolic and neuroendocrine research, paving the way for extensive experimental and clinical investigations.[1,2]

Nesfatin-1 is an 82–amino acid peptide generated through the proteolytic processing of Nucleobindin-2 (NUCB2), a preproprotein composed of a 396–amino acid core region and a 24–amino acid signal peptide, resulting in a total length of 420 amino acids. During post-translational modification, NUCB2 is cleaved by peptidases into three biologically relevant fragments: nesfatin-1 (residues 1–82), nesfatin-2 (residues 85–163), and nesfatin-3 (residues 166–396). Among these cleavage products,

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nesfatin-1 has been identified as the primary biologically active fragment, exerting significant effects on energy balance, appetite suppression, and glucose metabolism.[2] Furthermore, nesfatin-1, derived from the NUCB2 gene, is synthesized not only within the central nervous system but also in several peripheral tissues, including the gastric mucosa, adipose tissue, pancreas, and testes, indicating its pleiotropic physiological functions.[3]

The high density of nesfatin-1 expression in hypothalamic nuclei involved in appetite regulation, such as the arcuate nucleus (ARC), paraventricular nucleus (PVN), supraoptic nucleus (SON), and lateral hypothalamus, underscores its central role in food intake control and energy homeostasis. Notably, hypothalamic nesfatin-1 expression exhibits sensitivity to feeding and fasting states, suggesting a close association between nesfatin-1 signaling and metabolic status. In addition, nesfatin-1 concentrations in the gastric oxyntic mucosa are reported to be approximately 20-fold higher than those in the brain, emphasizing the importance of peripheral nesfatin-1 as a contributor to systemic energy regulation.[4]

Recent studies have revealed that nesfatin-1 exerts a broader spectrum of biological effects than previously anticipated. Beyond its anorexigenic action, nesfatin-1 expression has been documented across multiple organ systems, implicating the peptide in diverse physiological and pathological conditions. As summarized in Figure 1, alterations in nesfatin-1 levels have been associated with a wide range of gastrointestinal and inflammatory disorders, including gastric ulcer, gastric cancer, colitis, necrotizing enterocolitis, pancreatitis, cholecystitis, and obstructive jaundice. At the cellular level, nesfatin-1 demonstrates cytoprotective properties, mediated through the inhibition of proinflammatory cytokine release, regulation of oxidative stress balance, and suppression of apoptosis. These mechanisms highlight nesfatin-1 as a potential therapeutic target for modulating inflammation, reducing tissue injury, and limiting disease progression.[5]

This review systematically and comprehensively addresses the biosynthesis and tissue distribution of nesfatin-1, its central and peripheral actions, and its involvement in metabolic and neuroendocrine regulation. Additionally, current clinical findings, controversial issues within the existing literature, and future research directions are critically discussed. In this context, nesfatin-1 is regarded as a pivotal neuroendocrine mediator of energy balance and is considered a promising candidate for the development of novel therapeutic strategies targeting metabolic and inflammatory diseases.

Main Points

- Nesfatin-1 functions as a potent anorexigenic adipokine that plays a central role in energy homeostasis and modulates appetite regulation through leptin-independent pathways.
- Within adipose tissue, nesfatin-1 promotes lipolysis and reduces lipid accumulation, highlighting its importance in the pathophysiology of obesity and metabolic syndrome.
- Nesfatin-1 influences insulin secretion and glucose metabolism, suggesting its potential as a therapeutic target in type 2 diabetes and related metabolic disorders.
- By modulating cardiovascular function—including central regulation of blood pressure and peripheral vascular tone—nesfatin-1 may contribute to the development of obesity-related hypertension.
- The diverse metabolic and neuroendocrine effects of nesfatin-1 position it as a promising biomarker and future therapeutic candidate for obesity, diabetes, and stress-associated metabolic dysfunctions.

General Information

Nesfatin-1 and the structures that secret

In 2006, Oh-1 and colleagues discovered nesfatin-1, a hormone secreted from the hypothalamic nucleus that regulates appetite. In this study, it was reported that food intake was inhibited by nesfatin-1 even in obese mice lacking the leptin gene.[2] Consequently, the potential of nesfatin-1 has generated optimism for the treatment of obese individuals with leptin gene mutations. The release of nesfatin-1 is known to decrease during periods of hunger, suggesting a regulatory role in energy balance.[6]

Nesfatin-1 and its precursor peptide have been reported to be widely and abundantly distributed throughout the central nervous system, indicating a multifunctional neuroregulatory role. The presence of this peptide has been clearly demonstrated in key hypothalamic nuclei critically involved in energy balance, stress response, and neuroendocrine regulation, including the arcuate nucleus (ARC), paraventricular nucleus (PVN), supraoptic nucleus (SON), and lateral hypothalamus, as well as in the pituitary gland, highlighting its potential influence on hypothalamic–pituitary axis activity. Furthermore,

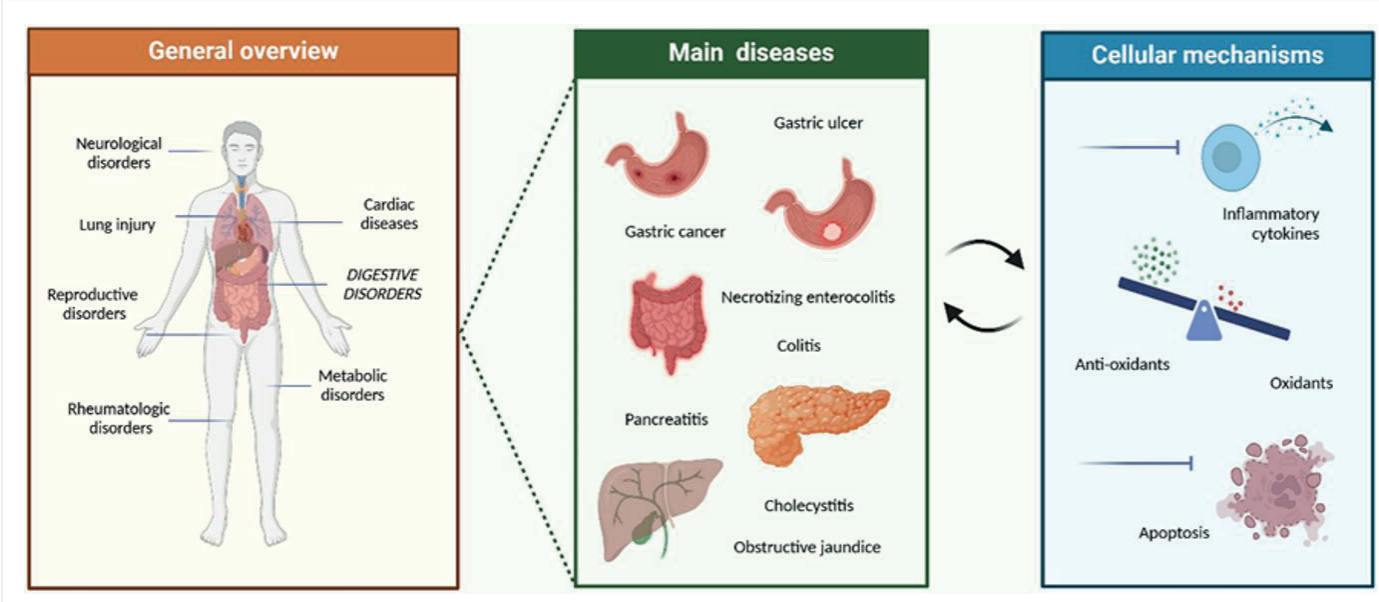

Figure 1. Schematic summary of the systemic effects and key cellular mechanisms of Nesfatin-1[5]

nesfatin-1 expression has been identified in the nucleus tractus solitarius (NTS), a major integrative center within the brainstem responsible for autonomic and visceral signal processing, in addition to multiple nuclei of the forebrain, midbrain, and the central amygdaloid complex, suggesting its involvement in emotional regulation, stress-related behaviors, and autonomic control.[4]

In addition, accumulating evidence indicates that nesfatin-1 is expressed in extra-hypothalamic and lower brain regions, including the cerebellum and ventrolateral medulla, as well as in the preganglionic neurons of the sympathetic spinal cord at the thoracolumbar level and the parasympathetic spinal cord at the sacral level in experimental rat models.[7] This widespread neuroanatomical distribution strongly supports the notion that nesfatin-1 participates not only in metabolic regulation but also in autonomic nervous system modulation and integrative neurophysiological processes.[7]

A multitude of studies have reported the secretion of nesfatin-1 by the central nervous system (CNS) as well as by peripheral tissues, including but not limited to adipose tissue, gastric mucosa, endocrine pancreatic beta cells, and testes.[4] The level of this substance in the gastric oxyntic mucosa was found to be 20 times higher than in the brain (Figure 2).[8]

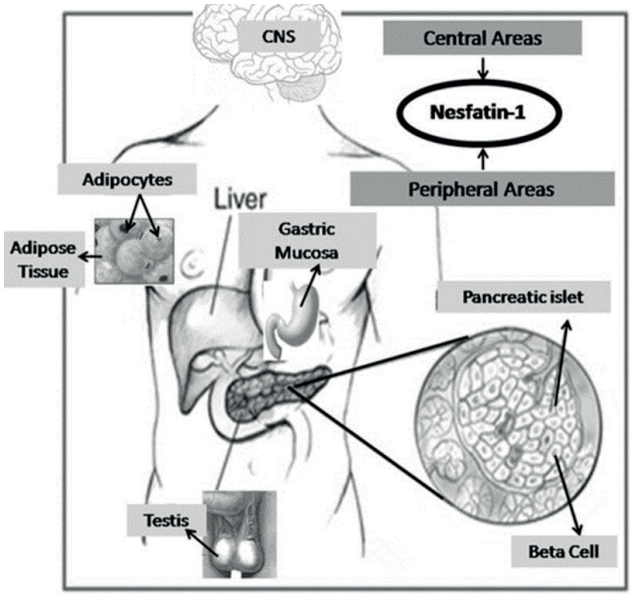

Figure 2. Structures secreting nesfatin-1[17]

The Effects of Nesfatin-1

Effect on food intake

Nesfatin-1, whose mRNA and protein expression levels are markedly elevated in key central regions such as the hypothalamic nuclei and brainstem, has been closely linked to the regulation of metabolic processes and the suppression of food intake.[9] The presence of nesfatin-1 in multiple brain regions involved in feeding behavior and emotional regulation, including the limbic system, hypothalamus, pons, and medullary nuclei, further supports its regulatory role in appetite control and energy balance.

The influence of energy status alterations associated with acute or chronic malnutrition on nesfatin-1 mRNA and protein expression levels in the hypothalamic paraventricular nucleus (PVN) and supraoptic nucleus (SON) has been well documented, highlighting its sensitivity to nutritional cues. Recent findings have demonstrated a positive correlation between increased levels of anorexigenic factors, such as cholecystokinin and melanocyte-stimulating hormone, and a subsequent upregulation of NUCB2 mRNA expression.[10] This upregulation has been shown to activate nesfatin-1–immunopositive neurons within both the hypothalamus and brainstem, indicating a coordinated neuroendocrine response to satiety signals.

Notably, nesfatin-1 has been reported to modulate both food and water intake through a leptin-independent but melanocortin-dependent signaling pathway, suggesting that it operates via distinct neuroendocrine circuits separate from classical leptin-mediated mechanisms.[11]

In dose-dependent experimental studies conducted in male Wistar rats, intracerebroventricular administration of nesfatin-1 into the third ventricle resulted in a significant reduction in both food intake and body weight.[7] These findings have been consistently replicated across different laboratories and species, including mice, rats, and goldfish, underscoring the robust and evolutionarily conserved anorexigenic effect of nesfatin-1.

Low-dose nesfatin-1 administration (5–20 pmol) into the lateral, third, and fourth ventricles, as well as the cisterna magna and specific hypothalamic regions such as the paraventricular nucleus (PVN), lateral hypothalamic area (LHA), and dorsal vagal complex (DVC), has been shown to induce pronounced anorexigenic effects. These low-dose applications were found to suppress food intake for prolonged durations (6–48 hours), particularly during the dark phase, when feeding activity is normally increased. In contrast, higher-dose injections (25–80 pmol) not only enhanced the anorexigenic response but also elicited anxiety-like behaviors, indicating a dose-dependent divergence in central effects.

Microinjection studies have further demonstrated that the paraventricular nucleus (PVN) serves as the primary hypothalamic site mediating the anorexigenic action of nesfatin-1, highlighting its central role in appetite suppression.[2,7] Additionally, pharmacological blockade of endogenous nesfatin-1 signaling using neutralizing antibodies or antisense oligonucleotides has been shown to increase food intake and promote weight gain, providing functional evidence for its physiological relevance.

Consistent with these observations, NUCB2 mRNA and protein levels in the PVN and SON regions have been reported to decrease during fasting or starvation and to return to baseline levels upon refeeding, further supporting the role of nesfatin-1 as a nutritional status–responsive regulator of energy homeostasis.[12,13]

Nesfatin-1, a hormone whose mRNA and protein levels are elevated in regions such as the hypothalamic nucleus and brain stem, has been linked to the regulation of metabolism and the suppression of food intake.[9] The presence of Nesfatin-1 in regions such as the limbic system, hypothalamus, pons, and medullary nucleus suggests that it plays a regulatory role in relation to food intake. The impact of energy shifts associated with acute or chronic malnutrition on Nesfatin-1 mRNA and protein levels in the hypothalamic PVN and SON is well-documented. Recent findings have indicated a correlation between the increase in certain substances that inhibit food consumption—such as cholecystokinin and melanocyte-stimulating hormone—and the subsequent increase in NUCB2 mRNA expression levels.[10] This increase, in turn, has been observed to lead to the activation of nesfatin-1-immunopositive neurons located within the hypothalamus and brain stem. Nesfatin-1 has been found to affect water and food intake via a leptin-independent, melanocortin-dependent system.[11]

In dose-dependent experiments conducted on male Wistar rats, it was found that intracerebroventricular injections into the third ventricle significantly reduced food intake and body weight.[7] This finding has been replicated and confirmed by different research groups in various animal models, including mice, rats, and

goldfish. The administration of low-dose injections (5–20 pmol) into the lateral, third, and fourth ventricles, the cisterna magna, and the hypothalamic nuclei PVN, lateral hypothalamic area (LHA), and dorsal vagal complex (DVC) regions has been reported to produce anorexigenic effects. Low-dose applications have been shown to reduce food intake for an extended period (6–48 hours) during the dark phase, while high-dose injections (25–80 pmol) have been observed to induce anxiety-like behaviors in addition to the anorexigenic effect. The results of microinjection studies have demonstrated that the paraventricular nucleus (PVN) is the primary hypothalamic center responsible for the anorexigenic effect of nesfatin-1.[2,7] In addition, the obstruction of endogenous nesfatin-1 signaling through the utilization of antibodies or antisense oligonucleotides has been observed to result in augmented feeding and weight gain. It has been reported that levels of NUCB2 mRNA and protein decrease in the PVN and SON regions during starvation and return to normal levels upon refeeding.[12,13]

In addition to its effects on the central nervous system, it has been demonstrated that nesfatin-1 also regulates food intake at the peripheral level. Nesfatin-1 has been detected in rodents and human plasma, and its potential sources include subcutaneous adipose tissue, endocrine cells of the gastric mucosa, intestines, and pancreatic β-cells. Plasma levels have been documented to decline during periods of fasting and return to normal following refeeding.[14] The continuous peripheral infusion led to a reduction in food intake, while the high-dose intraperitoneal administration suppressed feeding during the dark phase.[15] The durability of this anorexigenic effect in obese db/db mice, which carry a leptin receptor mutation, suggests that nesfatin-1 operates through a leptin-independent mechanism. A study of structural fragments revealed that the anorexigenic effect of nesfatin-1 originates from the central portion of the protein, specifically the amino acid sequence spanning residues 24–53.[4,16] In contrast, the N- and C-terminal fragments were found to be ineffective. Following peripheral administration, nesfatin-1 was reported to cross the blood-brain barrier and directly affect feeding centers. Additionally, it was reported to exert anorexigenic effects via indirect mechanisms through activation of vagal afferents and stimulation of POMC and CART neurons in the nucleus tractus solitarius (NTS) region.[7,17]

In summary, nesfatin-1 is a potent and multifunctional anorexigenic peptide that suppresses food intake through both central and peripheral regulatory mechanisms. These mechanisms include leptin-independent yet melanocortin-dependent signaling pathways, indicating that nesfatin-1 operates via distinct neuroendocrine circuits beyond classical satiety hormones. Its marked sensitivity to the hunger–satiety cycle further underscores its critical role in the fine-tuned regulation of energy homeostasis, positioning nesfatin-1 as a promising therapeutic target for the treatment of obesity and related metabolic disorders.

Recent experimental studies have demonstrated that nesfatin-1 modulates the activity of gastric satiety-sensitive neurons and regulates gastric motility via melanocortin signaling within the central nucleus of the amygdala, highlighting a functional link between emotional processing centers and gastrointestinal control. Furthermore, nesfatin-1 has been characterized as an inhibitory neurotransmitter that influences gastric motor function through its actions on key hypothalamic regions, including the paraventricular nucleus (PVN) and the lateral hypothalamic area (LHA).

Despite these advances, the precise neuroanatomical pathways, receptor mechanisms, and context-dependent effects through which nesfatin-1 regulates feeding behavior remain incompletely understood. Therefore, further comprehensive experimental and translational studies are required to fully elucidate the role of nesfatin-1 in appetite control, gut–brain communication, and long-term energy balance regulation.[7]

Effect on the nervous system

Evidence has been presented demonstrating the presence of neurons expressing NUCB2, the precursor peptide of Nesfatin-1, in multiple regions of the brain. A subset of NUCB2-positive neurons situated within the parvocellular region of the paraventricular hypothalamic nucleus (PVH) have been observed to express the melanocortin 4 receptor (MC4R).[18] These neurons have been shown to receive signals from α-melanocyte-stimulating hormone (α-MSH) and agouti-related protein (AgRP) fibers. It has been documented that α-MSH has the capacity to enhance the synthesis and secretion of nesfatin-1. A particular study revealed that NUCB2-expressing neurons in the PVH (pPVH) induce anorexigenic effects through the secretion of nesfatin-1.[19] However, the precise mechanism of action of nesfatin-1 remains to be elucidated.[15]

The central release of nesfatin-1 is subject to fluctuations in response to alterations in metabolic status. A multitude

of studies have demonstrated that following a 24-hour fasting period, there is a decrease in the transcription and translation levels of nesfatin-1 in both the paraventricular hypothalamic nucleus (PVH) and the supraoptic nucleus (SON) regions. In the SON region, a notable increase in nesfatin-1 synthesis and release is observed following refeeding after a 24-hour period of fasting. Peripheral injection of α-melanocyte-stimulating hormone (α-MSH) and serotonin 5-HT receptor antagonists, which have anorexigenic effects, has been demonstrated to increase hypothalamic nesfatin-1 release in rodent models. The administration of ghrelin, an orexigenic hormone, at orexigenic doses has been demonstrated to increase the activity of nesfatin-1-producing neurons located in the arcuate nucleus (ARC). Furthermore, peripheral injection of cholecystokinin (CCK), a satiety peptide, has been shown to increase the activity of neurons responsible for nesfatin-1 release in both the parvocellular region of the paraventricular nucleus (pPVN) and the nucleus tractus solitarius (NTS) regions. These findings suggest that nesfatin-1 interacts with various feeding peptides involved in satiety signaling.[14] Conversely, the sensation of hunger is believed to be modulated by peripheral metabolic signaling molecules, including leptin and ghrelin. The effects of ghrelin on the ARC have been shown to result in a decrease in the activity of proopiomelanocortin (POMC) neurons and an increase in the activity of agouti-related protein (AgRP) neurons.[15] In the 2006 study, the author and colleagues proposed that hunger reduces the activity of NUCB2-expressing neurons located in the paraventricular hypothalamic nucleus (PVH) via melanocortin signaling pathways.[2]

Its effect on stress and anxiety

It is widely believed that nesfatin-1 plays a regulatory role in stress-related responses physiological and may contribute to the development of stress-induced anorexia, an effect that is thought to arise from the activation of NUCB2-secreting neurons located in the posterior paraventricular hypothalamic nucleus (pPVH).[9] This region is known to be critically involved in integrating neuroendocrine and autonomic responses to stress, supporting the proposed role of nesfatin-1 in stress adaptation.

Magnocellular neurons, which are prominently distributed in the supraoptic nucleus (SON) and posterior pituitary projections, are primarily responsible for the regulation of water and electrolyte balance through the secretion of neurohormones such as vasopressin and oxytocin. It is therefore plausible that alterations in water balance may indirectly influence the regulatory functions of NUCB2-expressing neurons within this region, suggesting a potential interaction between fluid homeostasis and nesfatin-1–mediated stress responses.

Unexpectedly, experimental evidence indicates that classical metabolic regulators such as fasting and melanocortin signaling do not significantly affect NUCB2-expressing neurons located in the medial paraventricular hypothalamus (mPVH), SON, arcuate nucleus (ARH), lateral hypothalamus/intercalated zone (LHA/ZI), and nucleus tractus solitarius (NTS).[19] This lack of responsiveness to well-established metabolic cues highlights a significant gap in current knowledge and suggests that the precise physiological function of NUCB2 neurons in these regions remains poorly understood, warranting further targeted investigation.

In acute stress situations, an increase in nesfatin-1 levels is observed in the central nervous system; however, this increase is not reflected in plasma concentrations. However, recent studies have demonstrated that chronic stress exposure can increase plasma levels of nesfatin-1.[20,21]

A multitude of studies have demonstrated that nesfatin-1 may exert specific effects on anxiety. Recent observations have indicated that the administration of nesfatin-1 to the central nervous system results in an augmentation of anxiety-like behaviors. Furthermore, the study examined the relationship between variations in serum nesfatin-1 levels and alterations in eating behaviors observed in depressed individuals. In this context, one study reported that serum nesfatin-1 levels in individuals diagnosed with depression were significantly higher than in the healthy control group.[22] These findings suggest that nesfatin-1 may potentially play a role in regulating anxiety-like behaviors.[23,24]

Effect on the endocrine system

The influence of substances secreted in brain regions associated with nesfatin-1 on nutritional behavior, neuroendocrine regulation, autonomic control, visceral functions, sleep, mood, and pain is a subject of considerable interest. Consequently, it is plausible that nesfatin-1 may be associated with behaviors that extend beyond the realm of nutritional behavior. The significant presence of nesfatin-1 in endocrine tissues suggests a potential role in hormone secretion. Its presence in the stomach suggests a potential yet to be elucidated effect on food intake. Additionally, the presence of nesfatin-1

in the pancreas suggests a potential role in insulin- and glucagon-mediated glucose metabolism. The presence of nesfatin-1 in peripheral tissues corroborates its critical function in regulating energy homeostasis, neuroendocrine functions, and hypothalamic anorexic signaling. Additionally, the detection of nesfatin-1 in plasma is widely accepted as substantiating evidence for its hormonal activity.[25,26]

Subsequent to the central injection of Nesfatin-1, there was an increase in circulating LH and FSH hormone levels in adolescent female rats, both in the ad libitum fed and fasted groups. However, this effect was not observed in adult female rats. These findings suggest a potential role for Nesfatin in the developmental process of adolescence.[27]

A study was conducted to examine the effects of nesfatin-1 on testosterone release. The results demonstrated that central administration of this peptide caused significant increases in plasma follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone levels in rats. However, these increases did not result in a significant change in plasma gonadotropin-releasing hormone (GnRH) levels compared to the control group. These findings demonstrated for the first time that central injection of nesfatin-1 can regulate FSH, LH, and testosterone levels in males via the hypothalamic-pituitary-gonadal axis (HPGA).[28]

In accordance with the potential effects of Nesfatin-1 on the endocrine system, it has been proposed that increases or decreases in serum levels could be evaluated as biomarkers for certain diseases. In order to provide support for this hypothesis, an examination of serum nesfatin-1 levels was conducted across various patient groups. However, two of these studies reported conflicting results: One study suggested that low nesfatin-1 levels may play a role in the pathogenesis of polycystic ovary syndrome (PCOS),[29] while the other reported that high nesfatin-1 levels may be associated with PCOS.[30] The observed discrepancy between these results is hypothesized to stem from differences in study methodology, sample characteristics, experimental conditions, and genetic differences. Consequently, the identification of potential polymorphic regions in the nesfatin-1 gene may facilitate a more comprehensive understanding of this peptide's role in the development of various diseases.

Effect on diabetes mellitus

A comprehensive evaluation of the relevant literature has reported the protective and glucose-lowering effects of nesfatin-1 under hyperglycemic conditions. Intravenous administration of nesfatin-1 has been shown to induce a marked reduction in plasma glucose levels in hyperglycemic db/db mice, a widely accepted experimental model of type 2 diabetes mellitus.[31] The available data indicate that this hypoglycemic effect is primarily mediated through peripheral mechanisms and displays time-dependent and dose-dependent characteristics, as well as a partial association with circulating insulin levels.

Conversely, the absence of a comparable glucose-lowering effect of nesfatin-1 in a streptozotocin (STZ)-induced diabetes model, which is characterized by severe pancreatic β-cell damage, suggests that this mechanism may not rely exclusively on direct insulin secretion. These findings point to the involvement of insulin-independent or insulin-sensitizing pathways, although the precise molecular mechanisms underlying this effect remain incompletely elucidated.

In the same experimental framework, intracerebroventricular administration of nesfatin-1 in db/db mice resulted in a significant suppression of food intake without producing a corresponding reduction in blood glucose levels, indicating that the antihyperglycemic effect of nesfatin-1 is independent of its central anorexigenic action.[32]

Collectively, the appetite-suppressing (anorexigenic) and glucose-lowering (antihyperglycemic) properties of nesfatin-1 highlight its dual regulatory role in energy intake and glucose metabolism, respectively.[33] These observations strongly suggest that nesfatin-1 exerts a multifaceted influence on the maintenance of metabolic homeostasis. Accordingly, nesfatin-1 has been proposed as a potential therapeutic target in the management of metabolic disorders, particularly type 2 diabetes mellitus and obesity.

Moreover, experimental evidence suggests that nesfatin-1 may act as an antidiabetic agent by promoting insulin release and by enhancing insulin sensitivity, potentially through the facilitation of free fatty acid utilization and modulation of insulin resistance pathways.[34,35] Nevertheless, despite these promising

findings, the exact physiological and pathophysiological mechanisms through which nesfatin-1 regulates glucose homeostasis and energy metabolism remain to be fully clarified, underscoring the need for further mechanistic and translational studies.[35]

Effect on the immune system

A study demonstrated that nesfatin-1 exerts significant anti-apoptotic and anti-inflammatory effects in rats with a subarachnoid hemorrhage (SAH) model. In this experimental model, increases in tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6) levels were observed in brain tissue, while a significant decrease in antioxidant enzyme levels was detected.[36] Recent studies have indicated a potential correlation between decreased serum nesfatin-1 levels and obstructive sleep apnea syndrome (OSAS), suggesting a possible anti-inflammatory response as a contributing factor.[37] In addition, the results of a study conducted on individuals with emphysema-type chronic obstructive pulmonary disease (COPD) suggest that nesfatin-1 may serve as a novel biomarker associated with systemic inflammation.[38] This finding indicates that nesfatin-1 may potentially function as an inflammatory factor in this particular patient population. While these findings highlight the role of nesfatin-1 in regulating inflammatory responses, further research is needed to fully elucidate this mechanism.

Effect on the cardiovascular system

A multitude of studies have underscored the pivotal function of nesfatin-1 in modulating cardiovascular stress responses through the central nervous system. Specifically, it has been demonstrated that the intracerebrospinal injection of this peptide results in a significant increase in arterial blood pressure.[9,39] In experimental models employing melanocortin and oxytocin receptor antagonists, it has been observed that both the appetite-suppressing effects and the blood pressure-raising effects of nesfatin-1 are eliminated. Recent findings have revealed that nesfatin-1, which is localized in the paraventricular nucleus (PVN) in conjunction with oxytocin, has been shown to stimulate oxytocin release through the neuronal depolarization mechanism. In addition, it has been reported that nesfatin-1 can activate melanocortin signaling pathways via oxytocin. In light of these findings, it has been proposed that the hypertensive effects of nesfatin-1 may be associated with the central oxytocinergic system or the melanocortin pathway. However, the question of whether these two systems are activated simultaneously or sequentially, and which molecular mediators are involved in this process, remains unresolved.[14,25]

Intravenous administration of nesfatin-1 has been demonstrated to significantly inhibit nitric oxide (NO) production, thereby inducing a vasoconstrictive response and consequently leading to an increase in systemic arterial blood pressure.[40] This effect underscores the role of nesfatin-1 as an active modulator of vascular tone and cardiovascular regulation.

In a particular experimental study, peripheral administration of nesfatin-1 was shown to result in a marked reduction in endothelial nitric oxide synthase (eNOS) expression, an effect that was especially pronounced in rats subjected to chronic stress exposure.[5] The stress-dependent enhancement of this response suggests a potential interaction between nesfatin-1 signaling and stress-related neurovascular pathways. Collectively, these findings indicate that the hypertensive effect of peripheral nesfatin-1 is mediated, at least in part, through the suppression of the eNOS–NO signaling pathway, ultimately promoting vasoconstriction as the primary underlying mechanism.[41,42] This mechanism positions nesfatin-1 as a potential link between metabolic regulation, stress physiology, and cardiovascular dysfunction.

Intravenous administration of nesfatin-1 has been demonstrated to inhibit nitric oxide (NO) production, thereby inducing a vasoconstrictive effect and, consequently, an increase in systemic blood pressure.[40] In a particular study, the administration of peripheral nesfatin-1 was found to result in a significant reduction in endothelial nitric oxide synthase (eNOS) levels, particularly in rats exposed to chronic stress.[5] These findings suggest that the effect of peripheral nesfatin-1 on blood pressure occurs via eNOS and NO and likely manifests through the vasoconstriction mechanism.[41,42]

Effect on l-type calcium channels

A study employing primary hypothalamic neuronal cell cultures in rats has demonstrated that nesfatin-1 exhibits pharmacological characteristics consistent with G protein–coupled receptor (GPCR)–mediated signaling pathways. Specifically, the findings revealed that nesfatin-1 induces a significant increase in intracellular calcium (Ca^{2+}) concentration, indicating the activation of calcium-dependent signaling mechanisms. Moreover, the observation that selective inhibition of L-, P-,

and Q-type voltage-gated calcium channel subunits markedly attenuated the nesfatin-1–induced calcium response provides strong evidence that these calcium channel subtypes are directly or indirectly activated by nesfatin-1. These results further support the hypothesis that nesfatin-1 exerts its biological effects through GPCR-linked calcium signaling cascades.

In addition, this study demonstrated the neuronal expression of nesfatin-1 in both the hypothalamus and brainstem, highlighting its relevance in central neuroendocrine and autonomic regulation.[43] Furthermore, complementary investigations using hypothalamic neural cell culture models have consistently shown that nesfatin-1 facilitates intracellular calcium influx via GPCR-dependent mechanisms, reinforcing the concept that calcium signaling constitutes a key downstream pathway in nesfatin-1–mediated neuronal effects.[44,45]

A separate study indicated that nesfatin-1 enhances insulin secretion by activating L-type calcium channels in pancreatic islet beta cells. The increase in intracellular calcium is not dependent on protein kinase A and phospholipase A2. Recent findings have indicated a correlation between the increase in intracellular calcium levels and glucose levels. Specifically, following a meal, an increase in plasma glucose levels has been observed to result in elevated glucose-stimulated intracellular calcium levels in pancreatic islet beta cells, thereby inducing increased insulin secretion.[46]

In a separate study, it was discovered that long-term administration of nesfatin-1 to the peripheral region can increase the expression level of the α1c subunit protein in cardiac L-type calcium channels, particularly under chronic stress conditions. It has been hypothesized that the effect of nesfatin-1 on the heart may result in substantial damage to cardiomyocytes.[47]

Result

Nesfatin-1 is a pleiotropic and highly conserved peptide that exerts multifaceted regulatory effects on energy homeostasis, appetite regulation, glucose metabolism, neuroendocrine control, cardiovascular responses, inflammatory pathways, and stress-related physiological mechanisms. This biologically active molecule is derived from the post-translational cleavage of the NUCB2 precursor protein and is widely expressed in both central nervous system structures and diverse peripheral tissues, underscoring its systemic regulatory capacity.

Nesfatin-1 plays a crucial role as a satiety signal and contributes to the integrated regulation of whole-body physiological functions. Its prominent localization in key hypothalamic appetite- and energy-regulating centers, including the arcuate nucleus (ARC), paraventricular nucleus (PVN), supraoptic nucleus (SON), and lateral hypothalamic area (LHA), provides compelling evidence for its central involvement in the neuroendocrine control of energy balance and feeding behavior.

In this context, nesfatin-1 is regarded as a pivotal mediator in the maintenance of metabolic homeostasis, functioning at the intersection of central and peripheral signaling pathways. Moreover, its interaction with stress-responsive neuroendocrine circuits highlights its role in coordinating adaptive physiological responses to environmental and metabolic stressors, further supporting its relevance in health and disease.

The extant literature suggests that nesfatin-1 exerts potent anorexigenic effects at both central and peripheral levels through mechanisms that are both leptin-independent and melanocortin-dependent. This property facilitates the evaluation of nesfatin-1 as a potential therapeutic target, particularly in the context of obesity management, where leptin resistance is frequently observed. Moreover, the finding that nesfatin-1 increases insulin secretion and modulates glucose metabolism by activating L-type calcium channels in pancreatic β-cells is noteworthy for the treatment of type 2 diabetes. However, the literature contains conflicting findings regarding the insulin-dependence of its effects on glycemic regulation, indicating that the underlying mechanism of this molecule has not yet been fully elucidated.

The observed activity of Nesfatin-1 in neural circuits associated with stress, anxiety, and behavioral responses suggests the potential for this peptide to be linked to neuropsychiatric processes. The increases observed at the central level under acute stress conditions and elevated plasma concentrations under chronic stress conditions suggest that nesfatin-1 plays a regulatory role in stress adaptation mechanisms. However, the observation that nesfatin-1 administration can induce anxiety-like behaviors underscores the necessity for prudence in interpreting the neuropsychiatric effects of this molecule and the imperative for more exhaustive investigation of its underlying mechanisms.

Research conducted on the cardiovascular system has demonstrated that nesfatin-1 has the capacity to elevate arterial blood pressure through both central and peripheral

mechanisms. This hypertensive effect, reported to be associated with oxytocinergic and melanocortinergic pathways, is considered to potentially pose a limitation, particularly for individuals at cardiovascular risk. Another mechanism that should be carefully considered in clinical practice is vasoconstrictive effects arising from peripheral inhibition of endothelial nitric oxide synthase (eNOS) and decreased nitric oxide production. These findings underscore the importance of conducting a comprehensive investigation into the effects of nesfatin-1 on cardiovascular homeostasis, taking into account both its therapeutic potential and its potential risks.

In experimental studies conducted within the context of the immune system, nesfatin-1 has been demonstrated to exert notable anti-inflammatory and anti-apoptotic effects, supporting the hypothesis that this peptide possesses significant cytoprotective properties. These experimental findings suggest that nesfatin-1 may play a regulatory role in immune responses and inflammation-associated tissue injury.

However, results obtained from human studies have been highly heterogeneous and, in some cases, contradictory, indicating that the clinical applicability of nesfatin-1 as a reliable inflammation biomarker has not yet been clearly established. Moreover, inconsistent and disease-specific alterations in circulating nesfatin-1 levels reported in conditions such as epilepsy, polycystic ovary syndrome, and various metabolic disorders further limit its current reliability as a clinical biomarker.

Consequently, there is a clear need for advanced, standardized, and large-scale clinical investigations employing uniform measurement techniques, well-defined patient populations, and longitudinal study designs to elucidate the precise role of nesfatin-1 in immunological and metabolic processes and to determine its potential utility as a clinically meaningful biomarker.

In summary, nesfatin-1 is a neuropeptide that exerts a multifaceted role in physiological processes, demonstrating a broad spectrum of effects and high therapeutic potential. However, the fact that most of the extant studies are based on experimental animal models, while human data are limited, conflicting, and methodologically heterogeneous, necessitates careful evaluation of inferences regarding clinical applications. It is imperative that future research prioritize elucidating the detailed molecular mechanisms of action of nesfatin-1, characterizing its receptor-level interactions, determining dose-response relationships, and assessing long-term clinical safety.

In summary, nesfatin-1 is a key integrative neuroendocrine molecule involved in the coordinated regulation of energy balance, glucose metabolism, appetite control, and neuroendocrine signaling pathways. Accumulating experimental and clinical evidence suggests that nesfatin-1 holds significant promise as a biomarker and potential therapeutic target in a broad spectrum of clinical conditions, including obesity, type 2 diabetes mellitus, neuropsychiatric disorders, cardiovascular dysfunctions, and inflammatory diseases.

Despite these promising findings, the precise mechanisms of action, receptor pathways, and tissue-specific effects of nesfatin-1 remain incompletely understood. Therefore, the successful translation of its biological potential into clinical practice requires large-scale, well-designed, and long-term human studies, with particular emphasis on standardized measurement methods, population heterogeneity, and longitudinal outcome assessments. Clarifying these aspects will be essential for determining the clinical utility of nesfatin-1–based diagnostic and therapeutic approaches.

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Author contribution

The authors declare contribution to the paper as follows: Study conception and design: İDP; data collection: İDP; analysis and interpretation of results: İDP, AD; draft manuscript preparation: İDP. All authors reviewed the results and approved the final version of the article.

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Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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