CLINICAL SCIENCE OF NUTRITION

Case Report

Clin Sci Nutr 2019; 1(2): 110-2 • DOI: 10.5152/ClinSciNutr.2019.50

Zinc deficiency might occur in patients receiving parenteral nutrition

Barış Gülcü, Ersoy Taşpınar, Ersin Öztürk

Department of General Surgery, Medicana Bursa Hospital, Bursa, Turkey

ORCID ID of the author: B.G. 0000-0002-9754-8755; E.T. 0000-0002-6663-0190; E.Ö. 0000-0001-8593-5101

Cite this article as: Gülcü B, Taşpınar E, Öztürk E. Zinc deficiency might occur in patients receiving parenteral nutrition. Clin Sci Nutr 2019; 1(2): 110-2.

ABSTRACT

Zinc is an essential trace element. Many important enzymatic activities are zinc dependent. Therefore, it is not surprising that zinc deficiency is clinically expressed in rapidly growing tissues. The clinical features of zinc deficiency are not specific. The clinical manifestations of zinc deficiency include inadequate growth, hypogonadism, anorexia, alopecia, diarrhea, taste, and smell alterations, impaired wound healing, skin lesions, and diminished immune function. Acquired zinc deficiency is an uncommon entity. Zinc deficiency is defined most frequently in patients who had burns, malabsorption, severe losses from the gastrointestinal tract, and total parenteral nutrition (PN) with inadequate or no zinc in the nutrient mixture. In this case report, we present a 46-year-old female patient with altered mental status due to zinc deficiency, who was operated for ulcerative colitis, and who was fed with PN for a long time that did not contain zinc in the nutrient mixture.

Keywords: Deficiency, parenteral nutrition, zinc

Introduction

Zinc is an essential trace element that has a ubiquitous subcellular presence and is involved in catalytic, structural, and regulatory roles in the human body. It has been identified as a part of 120 different enzymes, such as carbonic anhydrase, carboxypeptidase, alkaline phosphatase (ALP), oxidoreductases, transferases, ligases, hydrolases, lyases, and isomerases (1). Endogenous stores of zinc are mobilized in the fasting state, but do not meet metabolic needs during anabolism because the net movement of zinc is into tissues, and circulating zinc is reduced. Although the syndrome of zinc deficiency cannot be identified easily, zinc deficiency does have a pronounced effect on nucleic acid metabolism, thus influencing protein and amino acid metabolism (2).

In healthy individuals, dietary zinc deficiency does not occur, mainly because zinc is widely distributed in food. Zinc deficiency has been described in a variety of patient populations including patients with advanced age, alcohol use disorder, postoperative status, burns, malabsorption syndromes, wound drainage, or gastrointestinal (GI) losses (2). Since zinc is involved in many processes, the clinical features of zinc deficiency are not specific. The clinical manifestations of zinc deficiency include inadequate growth, hypogonadism, anorexia, alopecia, diarrhea, taste, and smell alterations, impaired wound healing, and diminished immune function (1-5).

Trace element and vitamin supplements should be provided for long-term parenteral nutrition (PN). In 1976, prior to the routine inclusion of trace elements in PN, Kay et al. published one of the first case series of zinc deficiency in patients receiving PN and also noted that symptoms are responsive to the addition of zinc (3, 6). By 1979, the Nutrition Advisory Group of the American Medical Association recommended the routine addition of trace elements, such as zinc, copper, chromium, and manganese, to PN (1, 3). After the shortage of trace elements production in the USA in 2014, many diseases have been reported due to PN-dependent trace elements deficiency (3, 4).

We report a case of a woman with altered mental status due to zinc deficiency who received long-term PN and who was operated on for inflammatory bowel disease.

Corresponding Author: Barış Gülcü; drbarisgulcu@gmail.com

Submitted: 11.12.2018 **Accepted:** 24.01.2019

Case Presentation

A 46-year-old female patient who had been treated for ulcerative colitis for 13 years was admitted to our clinic for abdominal pain, severe diarrhea, and depleted health status. She had received high doses of steroids and 5-aminosalicylic acid medication. She had been hospitalized in a gastroenterology clinic elsewhere for >3 months receiving intravenous antibiotics, steroids, and PN.

On admission, she was offered three-stage colectomy. The first step of surgery was emergent total colectomy with end ileostomy that would be followed by proctectomy and ileal pouch-anal anastomosis (IPAA) and diverting ileostomy after 6 months and eventually ileostomy takedown.

After emergent total colectomy, she was started on oral intake and was discharged on postoperative day 12 with oral loperamide to reduce ileostomy output. During her hospital stay, she took oral and PN supplements. Her nutrition risk screening score was 6, and weight loss was >15% in the last 3 months.

After 6 days from discharge, she was admitted to the emergency clinic with high stoma output. She had signs of dehydration. Her laboratory results were as follows: blood glucose level 81 mg/dL (70-99 mg/dL), white blood cell 18,300/mm^3 (3500-10,000/mm^3), hemoglobin 9.6 g/dL (11.5-15.1 g/dL), hematocrit 33.4% (34.4%-44.2%), blood urea 96 mg/dL (17-43 mg/dL), creatinine 2.3 mg/dL (0.6-1.2 mg/dL), serum sodium 136 mmol/L (135-148 mmol/L), serum potassium 5.2 mmol/L (3.5-5.5 mmol/L), ionized calcium 4.92 mg/dL (4.64-5.28 mg/dL), serum chloride 107 mmol/L (98-106 mmol/L), aspartate aminotransferase/alanine aminotransferase within normal limits, serum albumin 2.1 g/dL (3.5-5.5 g/dL), total protein 6.8 g/dL (6.7-8.6 g/dL), ALP 27 U/l (33-96 U/L), and C-reactive protein (CRP) 17 mg/dL (0-10 mg/dL).

Rehydration and loperamide were started followed by PN with high doses of proteins (2 g/kg), vitamins, and trace elements. She experienced pneumonia that was treated with intravenous piperacillin and tazobactam. Although there was progression in clinical and nutritional status and laboratory tests, the patient's mental status was reduced. She showed agitation, dyslexia, and unresponsiveness to physical stimuli. Serum electrolyte levels were normal in this period. A neurology consultant evaluated the patient, and cranial magnetic resonance imaging was performed, revealing no pathology. Then, trace element levels were studied. Serum zinc level was below normal limits [40 µg/dL (60-120 µg/dL)], and serum copper level was within normal limits [75 µg/dL (70-140 µg/dL)].

A dose of 10 mg intravenous zinc was administered daily. After 3 days, she showed progression in her mental status, and after 8 days, she was completely normal. Serum zinc level was within normal limits [82 µg/dL (60-120) µg/dL)] after intravenous treatment. She was discharged after 12 days of intravenous zinc initiation.

Completion proctectomy with IPAA and further ileostomy takedowns were later performed uneventfully.

Discussion

Zinc deficiency is rarely seen in healthy individuals because zinc is widely distributed in food. However, prolonged use of PN and abnormal loss from the GI tract (diarrhea, fistula, stoma, or bariatric surgery) also cause zinc deficiency, such as other trace elements. Zinc deficiency may be asymptomatic or lead to lethal clinical manifestations extending to coma. Zinc, which is the most commonly used trace element after iron in the body, deficiency should not be neglected.

Long-term use of PN and GI losses, such as diarrhea, fistula, or ileostomy, may cause zinc deficiency. Zinc deficiency may be asymptomatic but may lead to mental problems and coma as well. Zinc is the second most utilized trace element after iron in the body; therefore, zinc deficiency probability should always be kept in mind.

Major national shortages of vitamin and trace element products for PN formulations have occurred in the late 1980s, the late 1990s, and finally between 2009 and 2014 (3). Several studies have been reported due to trace element deficiency during this shortage (3, 4). Although our patient manifested mental problems due to zinc deficiency, the clinical manifestations of zinc deficiency could also include erosive eczema, alopecia, cheilitis, anorexia, abdominal pain, hypogonadism, depression, and coma (1-5). Altered mental status can occur in the setting of severe deficiency and may be explained partially by hyperammonemia resulting from the impairment of the urea cycle when zinc levels are decreased. However, we did not measure blood ammonia levels.

The clinical features of zinc deficiency are non-specific because it is involved in so many processes. The diagnosis of zinc deficiency is difficult. Zinc deficiency for this patient was diagnosed primarily by laboratory measurement. It cannot be easily ascertained whether the patient experienced clinical sequelae of zinc deficiency (1, 4). Although there were many clinical findings (prolonged use of PN, excess loss of stoma, decrease in turgor, amenorrhea, low ALP levels, and susceptibility to infection) in our patient,

the diagnosis of zinc deficiency was delayed. Plasma zinc levels are decreased during times of stress and infection. Zinc levels can be decreased in hypoalbuminemia due to sepsis as it is bound to albumin and alpha-macroglobulins in the blood. Furthermore, zinc has been found to correlate negatively with CRP (1). In addition, the low level of ALP may be significant for zinc deficiency (3), and it may even be an alert situation for zinc deficiency.

The use of PN created a unique situation in which it was possible to feed individuals with purified diets specifically deficient in trace elements, such as zinc (2). In patients with no oral intake or PN alone, approximately 3 mg/day zinc maintained zinc balance in the absence of GI losses (1). The current American Society for Parenteral and Enteral Nutrition clinical guidelines recommended that the supplementation of PN for zinc is 2.5-5 mg/day. Excessive GI losses, sepsis, and hypercatabolic states require additional supplementation. Patients with enterocutaneous fistula and diarrhea, as well as similar to our patient with severe loss of stoma, may require 12-17 mg/L fluid. Therefore, in patients with fistula, diarrhea, and intestinal drainage, a dose of 12 mg zinc should be added for each liter of loss (1). Amino acid infusions also increase urinary zinc losses. In the kidney, zinc infusions enhance distal reabsorption of zinc, and amino acid infusion increases proximal secretion (2). In our case, zinc treatment was delayed despite long-term PN and high-volume loss from stoma, and additional amino acid infusion was observed in addition to PN. These two states further deepened zinc deficiency.

In cases of zinc deficiency, clinical improvement is quite rapid, with results seen in 2 days and resolution in 2 weeks (1). In our case, clinical improvement was started on day 3, and resolution was seen on day 8.

In conclusion, zinc deficiency should be in the differential diagnosis of patients particularly with GI losses and receiving long-term PN with unexplained mental or metabolic disorders.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – B.G., E.Ö.; Design – B.G.; Supervision – E.Ö.; Resources – B.G., E.T., E.Ö.; Materials – B.G., E.T.; Data Collection and/or Processing – E.T.; Analysis and/or Interpretation – B.G., E.Ö.; Literature Search – B.G., E.T.; Writing Manuscript – B.G., E.Ö.; Critical Review – E.Ö.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

References

1. Jin J, Mulesa L, Carrilero Rouillet M. Trace elements in parenteral nutrition: considerations for the prescribing clinician. Nutrients 2017; 9: E440. [CrossRef]
2. Jeejeebhoy K. Zinc: an essential trace element for parenteral nutrition. Gastroenterology 2009; 137: 7-12. [CrossRef]
3. Sant VR, Arnell TD, Seres DS. Zinc deficiency with dermatitis in a parenteral nutrition-dependent patient due to national shortage of trace minerals. JPEN J Parenter Enteral Nutr 2016; 40: 592-5. [CrossRef]
4. Franck A. Zinc deficiency in a parenteral nutrition-dependent patient during a parenteral trace element product shortage. JPEN J Parenter Enteral Nutr 2014; 38: 637-9. [CrossRef]
5. Jakubovic BD, Zipursky JS, Wong N, McCall M, Jakubovic HR, Chien V. Zinc deficiency presenting with necrolytic acral erythema and coma. Am J Med 2015; 128: e3-4. [CrossRef]
6. Kay RG, Tasman-Jones C, Pybus J, Whiting R, Black H. A syndrome of acute zinc deficiency during total parenteral alimentation in man. Ann Surg 1976; 183: 331-40. [CrossRef]