CASE REPORT

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A Crohn's patient presenting with acute hypokalemic paralysis with hypophosphatemia: Case report

Akut hipokalemik paralizi ve hipofosfatemi ile başvuran Crohn olgu sunumu

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53 years old man admitted to the emergency service with the history of muscle weakness and hypokalemic paralysis. Although the patient had no gastrointestinal symptoms at presentation, he investigated for the underlying cause and he diagnosed Crohn's disease. As far as we know this is the first case with hypokalemic paralysis caused by Crohn's disease, without gastrointestinal symptoms in the literature. Crohn's disease should be considered in the differential diagnosis for patients presenting with nutrient deficiencies or metabolic disease.

Anahtar kelimeler: *Crohn's disease, hypokalemia, paralysis*

Elliüç yaşında erkek hasta kaslarda güçsüzlük ve hipokalemik paralizi ile acil servise başvurdu. Hastanın başvuru esnasında hiçbir gastrointestinal semptomu olmamasına rağmen, yapılan araştırmalar sonucunda Crohn hastalığı tanısı aldı. Bu olgu bildiğimiz kadarı ile gastrointestinal semptomu olmadan hipokalemik paralizi tanısı alan literatürdeki ilk olgudur. Crohn hastalığı nutrisyonel eksiklikler ve metabolik hastalık ile başvuran hastaların ayırıcı tanısında düşünülmelidir.

Key words: *Crohn hastalığı, hipokalemi, paralizi*

INTRODUCTION

Acute hypokalemic paralysis is a rare condition that can be potentially life threatening but resolves with potassium infusion, if recognized early. We report the unusual case of hypokalemic and hypophosphatemic paralysis in a male patient with Crohn's disease.

CASE REPORT

53 years old male admitted to emergency department for his extreme weakness with the history of gradually increasing weakness for the past 3 months, without sensory loss. He reported weight loss of approximately 5 kgs, last month. He denied having any fever, diarrhea, chills, headache, abdominal pain, visual disturbances, paresthesias, vomiting and nausea. He reported 2-3 times defecation daily, since adolescence, without blood or mucus. He had no known allergies and medications. He had history for neither smoking nor alcohol. There was no positive family history for such disturbances.

On physical examination, the patient appeared to be uncomfortable and generally fatigued, but he was fully oriented. His vital signs; arterial blood pressure of 110/70 mmHg, respiratory rate of 16 breaths/min, and a axillary body temperature of 37.0°C. His pulse had a regular rhythm with a rate of 48 beats per minute and oxygen saturation was 97% on room air. The extraocular muscles were intact; without nystagmus. His pupils were symmetric and equally reactive to light, and the optic discs appeared normal. His abdomen was nontender, without masses, and there was no appreciable costovertebral angle tenderness. His lungs were clear to auscultation with a normal respiratory effort. The heart rate was regular but bradycardic, with normal S1 and S2 heart sounds. There was no edema in extremities. The skin was clear without any rash, petechiae, or ecchymoses. Otherwise, the neurologic examination revealed 3/5 muscle strength throughout bilateral upper and lower extremities, including deep tendon reflexes, and cerebellar tests, were all within normal range.

The patient's laboratory results are reported in Table 1. ECG showed bradycardia and significant ST depression with bifasic and negative T waves, and significant positive U waves, as well, in most derivations.

The patient hospitalized with the diagonosis of acute hypokalemic and hypophosphatemic paralysis. Because of bradycardia he was monitored. 20 mmol of potassium chloride per 100 ml of normal saline and izolyte fluid was infused hourly through a central venous line. By the day 3, serum potassium and phosphorus had improved (3.1 mmol/ l and 3.5 mg/dL) and the patient explained improvement in muscle weakness.

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Table 1. Patient's laboratory results

White blood cell	14410/mm^3 (5000-10.000/mm^3)
Hemoglobin	11.6 g/dL (12-16 g/ dL)
Platelet	490.000/mm^3 (150-400.000/mm^3)
Na	136 mEq/L (135-145 mEq/L)
K	1.8 mEq/L (3.5-5.5mEq/L)
Albumin	3 g/dL (3.4- 4.8 g/dL)
Calcium	8.2 mg/dL (8.7-10.7 mg/dL)
Phosphorus	0.7 mg/dL (2.7-4.5 mg/dL)
CRP	31 mg/L (<5mg/L)
Kreatinine	1.4 mg/dL (<1.2 mg/ dL)
Urea	46 mg/ dL (13-45 mg/ dL)
Serum iron	62 mcg/dL (53-167mcg/dL)
Total iron-binding capacity	213 mcg/dL (291-430mcg/dL)
Ferritin	132 ng/mL (30-400 ng/mL)
Vitamin B_{12}	143 pg/mL (197-866 pg/mL)
Vitamin D	11 mcg/L (15-60 mcg/L)
Parathormone	65 pg/ mL (15-65pg/ mL)
Folic asid	1.7 ng/mL (4.2-19.9 ng/mL)
Fibrinogen	550 mg/dl (175-400 mg/dl)
Erythrocyte sedimentation rate	46 mm/hour
Myoglobin	696 ng/dL (28-72 ng/dL)
Creatine kinase	394 U/L (< 170 U/L)
Thyroid-stimulating hormone	2.5 mlU/L (0,5-3,5 mlU/L)
Aspartate aminotransferase	14 IU/L (3-45 IU/L)
Alanine aminotransferase	10 IU/L (5-45 IU/L)

The patient was further investigated for possible underlying causes. There was no significant renal loss of potassium or alcalosis or renal function impairment. The levels of antibodies against tissue transglutaminase were normal. After stabilization, abdominal ultrasonography is performed; increased wall thickness of the terminal ileum seen. Abdominopelvic computed tomography (CT), with the suspicion of malignancy, (history of weight loss and anemia) showed no pathology except from increased wall thickness of the terminal ileum. Then, he underwent endoscopy, and colonoscopy. Gastroduodenoscopy demonstrated antral and duodenal aphtoid ulcers (Figure 1,2). Biopsy from these lesions showed mild chronic inflammation of the lamina propria, minimal villous atrophy, no increase in intraepithelial lymphocytes and no granulom, no parasites (Figure 3,4).

In colonoscopy, there were aphthous ulcers in terminal ileum and throughout the colon to the rectum with discontinuous involvement and cobble stoning (Figure 5,6). The biopsy yielded chronic inflammation of the lamina propria, rare epitheloid noncaseating granulomas, lymphoid aggregates in the submucosa, as well (Figure 7,8). Pathological specimen were stained with CD3 and CD68 was shown (Figure 9,10).

Since Crohn's disease, localized at upper gastrointestinal system and ileum and, colon, was diagnosed , medical treatment started as prednisolone 40 mg/day p.o., 5-amino Salysilate 3 gr/ day p.o., and azathioprine 100mg/day p.o. Femur bone mineral densitometry showed T score -5.0 kg/m2, added alendronate 70mg/week, calcium 1000 mg/day and vitamin D 60 IU/day, orally. Vitamin B_{12} and folic acid were also replaced.

After a 3 -month follow-up period, he remained clinically well with normal hemoglobin level and normal serum biochemistry. One year later endoscopy and colonoscopy is perfomed again all of the bowel segments were normal. The biopsy yielded low-grade chronic inflammation of the lamina propria in biopsies from the colon and ileum. Two year later the control of the femur bone mineral densitometry showed T score -3.5.

DISCUSSION

The syndrome of hypokalemic and hypophosphoremic paralysis represents a heterogeneous group of disorders characterised clinically with acute systemic weakness and hypokalemia. Most cases are familial or primary hypokalemic periodic paralysis; sporadic cases are associated with other various conditions including barium poisoning, hyperthyroidism, renal disorders, certain endocrinopathies and gastrointestinal potassium losses (1). Gastrointestinal potassium loss is a rare cause, although reported in celiac disease (2), tropical sprue

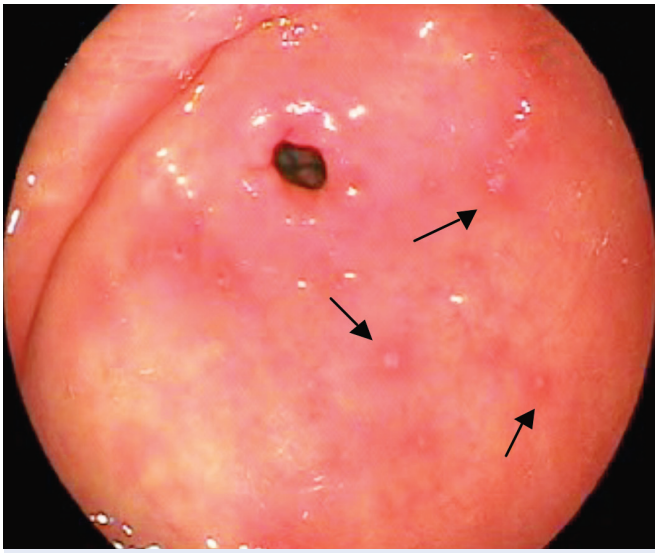

Figure 1. Antral aphtoid ulcers.

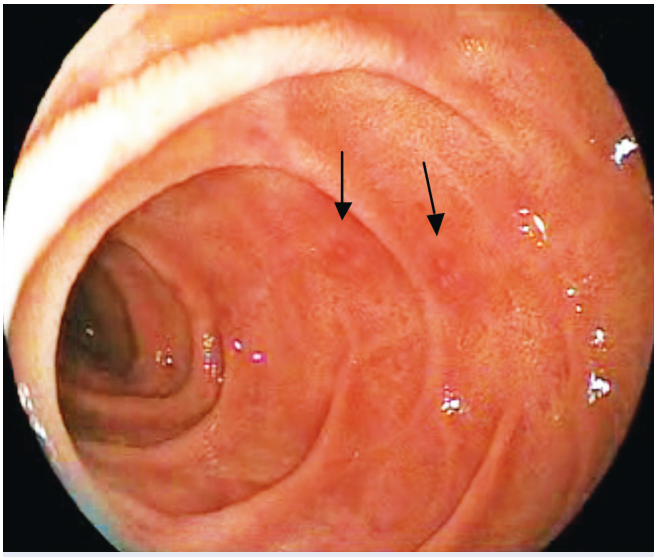

Figure 2. Duodenal aphtoid ulcers.

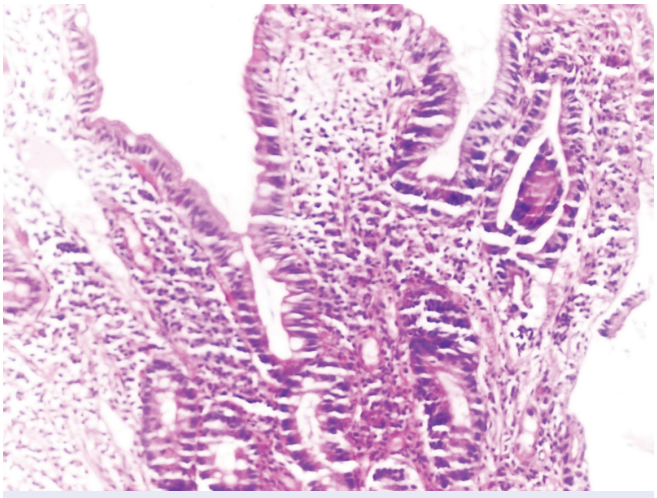

Figure 3. Villous atrophy.

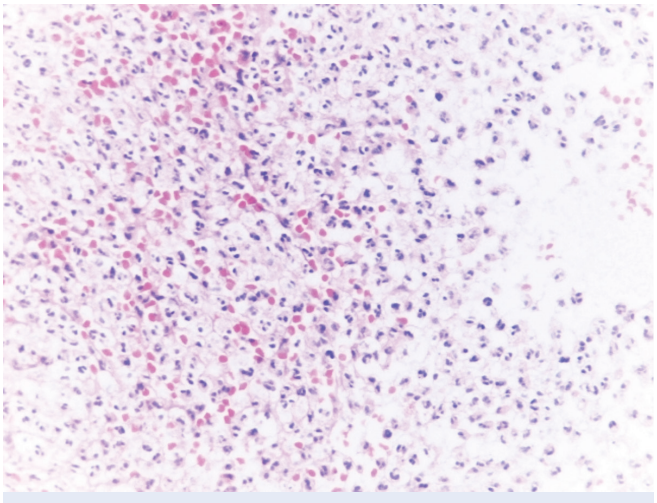

Figure 4. Chronic inflammation of the lamina propria.

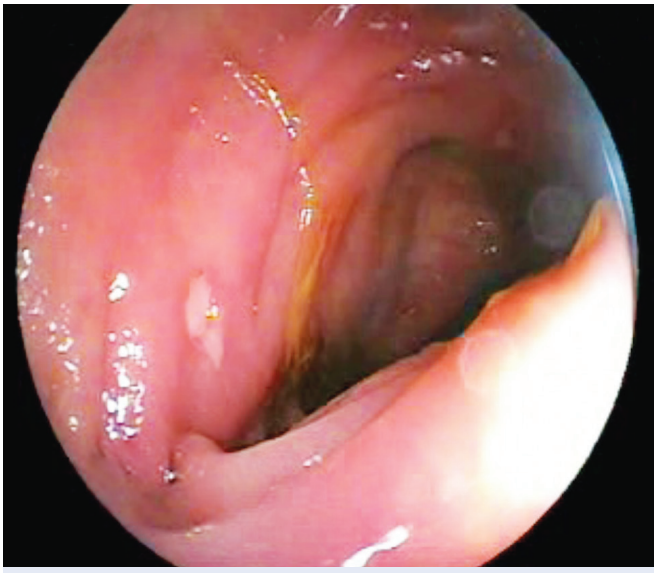

Figure 5. Aphthous ulcers in terminal ileum.

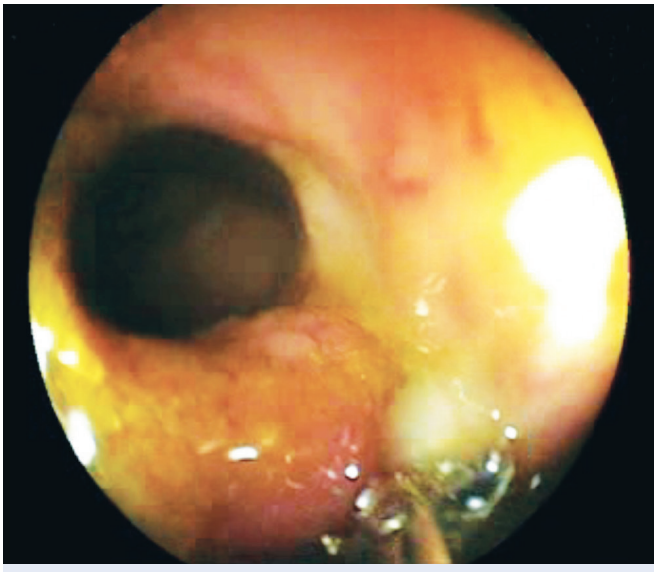

Figure 6. The swollen, ulcerated area in terminal ileum.

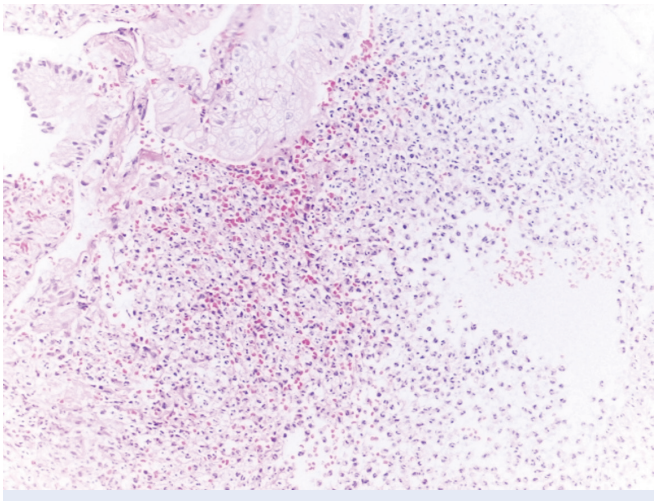

Figure 7. Chronic inflammation in the lamina propria.

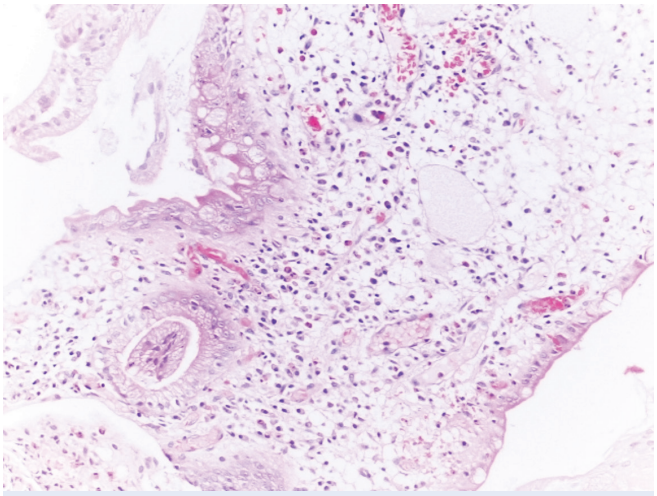

Figure 8. Noncaseating granulomas and lymphoid aggregates.

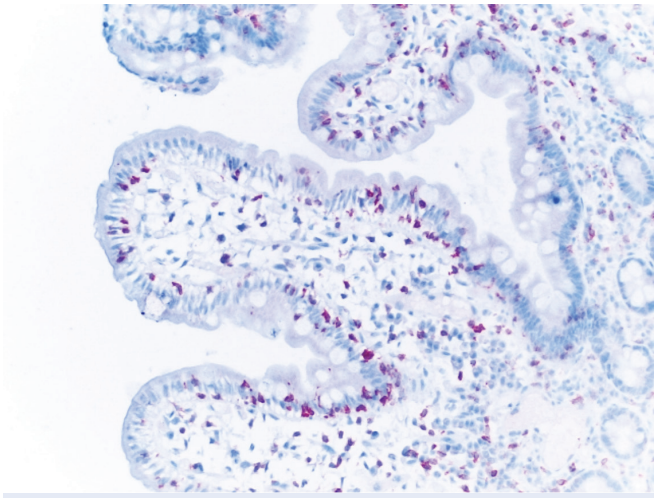

Figure 9. CD 3, T lymphocyte surface marker, stomach.

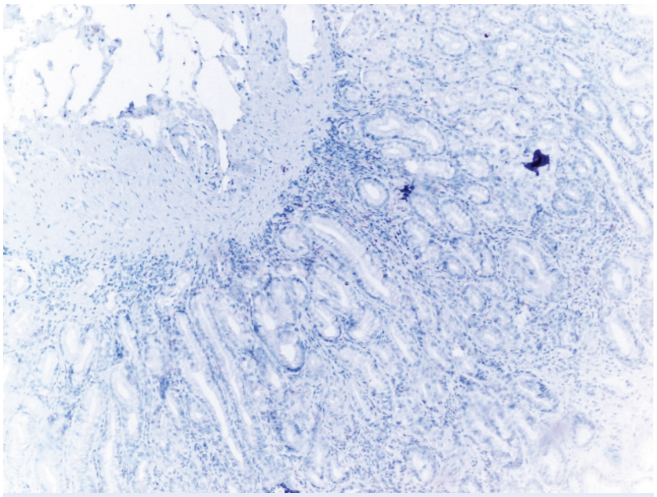

Figure 10. CD 68, macrophage surface marker, colon.

(3), short bowel syndrome (4), and acute gastroenteritis (5). Underlying causes are anorexia, active inflammation, bile salt and fat malabsorption, decreased of absorption surface, rapid gastrointestinal transit, lack of fat-soluble vitamins (6-7).

There has been reported only one case with hypokalemic paralysis caused by intestinal Crohn's disease in the literature (8). We know our patient is the first report of hypokalemic paralysis caused by involvement in upper gastrointestinal system and ileocolic Crohn's disease, as far. Interestingly, the patient had no gastrointestinal symptoms at presentation, despite evidence of malabsorption.

Malnutrition is an important complication and frequently observed in patients with Crohn's Disease (CD), usually associated with nutritional deficiencies, especially vitamins (both water- and fat-soluble) and essential trace elements. It is often due to the disease activity; poor oral intake or restrictive diets, bacterial overgrowth, bile salt malabsorption, extensive small-bowel disease, fructose malabsorption, inflammation, lactose malabsorption, multiple resections, short-bowel syndrome and surgery (9).

Anemia and osteoporosis are the most frequent complications generated by nutritional deficiencies in inflammatory bowel diseases (IBD). Osteoporosis is caused by low intakes of calcium and vitaminD; anemia, may be associated with chronic iron loss, long lasting inflammation and nutritional deficiencies suh as folate and vitamin B_{12} (10,13).

The cause of IBD iron deficiency anemia (IDA) is multifactorial and due to a combination of iron deficiency and chronic gastrointestinal blood loss and, in some cases, self-imposed dietary restrictions. A study showed that vitamins of A, C, D, E, B_{12}, folate and level of calcium, iron and potassium were low in blood with Crohn's disease (11). Gastrointestinal bleeding is the most common etiology for iron-deficiency anemia in IBD. Although patients with ileal CD may be at particular risk for vitamin B_{12} deficiency, the unaffected small bowel may adapt and increase its ability to absorb vitamin B_{12} efficiently. Distal ileal resection or active disease in this area, ie. the last 60 cm of ileum where B_{12} is absorbed, resuled in B_{12} deficiency, as well. Vitamin B_{12} should be given parenterally, in such cases, due to lack of functional absorptive area in the terminal ileum. Beside these, the diarrhea, high-output stoma or fistulae, short-bowel syndrome, malabsorption and vomiting result in excessive amount electrolyte losses such as potassium, sodium, magnesium, selenium and zinc (12). However, hypophosphatemia may occur due to both vitamin D deficiency and decreased intestinal absorption. Hypophosphatemia is commonly missed due to nonspecific signs and symptoms, but it causes considerable morbidity and even mortality. Phosphate is abundantly present in many foods. Isolated dietary phosphate deficiency is uncommon, and the

deficiency usually occurs with generalized malnutrition. Intestinal phosphate absorption is up-regulated by 1,25(OH) vitD3. Generalized muscle weakness is the most common symptom of hypophosphatemia; weakness and fatigue are related with acquired hypophosphatemia. Rhabdomyolysis is one of the most important clinical consequences in hypophosphatemia (14).

Calorie-protein malnutrition may lead in the humoral and cellular immunodeficiency. Its effects on the intestine: decreasing the efficiency of the mucosal barrier, leading alteration in the mucosa-associated lymphoid tissue functions and finally a greater risk of infection by bacterial translocation. Moreover, hypoplasia of the intestinal villi perpetuates malabsorption and increases the risk of infections (15).

Our case had paralysis due to hypokalemiae and hypophosphatemia associated with malabsorbtion. Patients with Crohn's disease may present with the malabsorbtion symptoms, solely. Therefore, one must consider IBD, espcially Crohn disease, in a patient, even if with no gastrointestinal symptom.

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