

Recurrent tetany in a male patient with short bowel syndrome and Crohn's disease

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In the course of Crohn's disease (CD), several clinical symptoms may occur, primarily associated with gastrointestinal problems. However, CD may also cause other important health disturbances, such as atypical metabolic disturbances or electrolyte imbalance.

A 48-year-old male with CD (diagnosed September 2022) and reactive arthritis was admitted to the Internal Medicine Department. The patient had a history of surgically treated gastric ulcer perforation complicated by diffuse peritonitis, peritoneal adhesions, and iatrogenic small intestine perforation, which required ileostomy formation (August 2022).

In October 2022 the patient was admitted because of tetany with motor aphasia, binocular diplopia, weakness of the lower extremities, and with tongue and upper limb stiffening. Additionally, the patient reported pain located in the left elbow joint and the left knee joint.

On admission, a physical examination revealed a postoperative surgical wound in the middle epigastrium, with small amounts of serous and cloudy liquid and ileostomy in the right middle abdomen.

A preliminary diagnosis initially indicated stroke as a possible cause of the symptoms; however, computed tomography (CT) of the head excluded this. CT enterography revealed inflammatory lesions in the small and large intestine distal to the stoma, with the presence of adhesions and interloop fistulas, a small amount of fluid in the abdominal, pelvic, and pleural cavities, hepatosplenomegaly, and enlarged external iliac (18 mm) and left periaortic (15 mm) lymph nodes.

Laboratory blood tests showed severe hypocalcaemia (5.35 mg/dl [reference: 8.6–10.00]), low ionised calcium concentration (0.67 mmol/l [reference: 1.15–1.35]), hypomagnesaemia (0.55 mg/dl [reference:

1.6–2.6]), hyperphosphataemia (5.17 mg/dl [reference: 2.5–4.5]), hypoproteinaemia (5.52 g/dl [reference 6.6–8.3]), hypoalbuminaemia (2.8 g/dl [reference 3.5–5.2]), high C-reactive protein (111 mg/l [reference: < 5.0]), high D-dimers (1772 ng/ml [reference: < 500]), high parathyroid hormone (PTH) (92.48 pg/ml [reference: 15.00–65.00]), low red blood cell count (RBC) ($3.08 \times 10^6/\mu\text{l}$ [reference: $4.2\text{--}5.7 \times 10^6/\mu\text{l}$]), low haemoglobin (10.4 g/dl [reference: 13.5–16.5]), low folic acid (3.83 ng/ml [reference: 3.89–26.8]), and low 25-hydroxy vitamin D₃ (VitD3) (17.00 ng/ml [reference: 30.00–50.00]) concentrations. The kidney function was preserved with creatinine concentration of 0.83 mg/dl [reference: 0.67–1.17] and eGFR > 60 ml/min/1.73 m².

The diagnosis was malabsorption due to CD and short bowel syndrome. The treatment involved sulphasalazine and corticosteroids, as well as empiric antibiotic therapy and supplementation of electrolytes, which resulted in improved joint mobility, lowered inflammatory markers, and normalized electrolytic profile. The patient was discharged and advised to take prednisone (5 mg/day for 5 days, then 2 mg/day for 7 days), sulphasalazine (1000 mg/day), azathioprine (200 mg/day), esomeprazole, calcium, cholecalciferol, magnesium, potassium, iron, vitamin C, and folic acid. During the subsequent 6 weeks, the patient presented to the emergency department (ED) on 2 separate occasions, with joint cramps and similar symptoms to those at the first admission. In both cases laboratory test results showed hypocalcaemia, hypomagnesaemia, and hyperphosphataemia; the symptoms subsided after electrolyte deficiency treatment. One and a half weeks after the last discharge the patient presented to his GP clinic for intravenous supplemen-

tation of calcium and magnesium because of muscle cramps persisting for several days.

At the end of January 2023, the patient was admitted to the Internal Medicine Department for gastroscopy and colonoscopy, necessary for qualification to biological therapy of CD.

Malabsorption in patients with CD may not seem surprising. Several case reports also mention hypomagnesaemia in 13–88% of patients with intestinal bowel disease [1]. However, the mechanism responsible for the electrolyte imbalance may be more complex than just impaired absorption, and may include secondary hyperparathyroidism and complex interactions between hormones (i.e. PTH and vitamin D₃) and electrolytes (magnesium, calcium, phosphates). Other authors have also described cases of tetany in patients with CD [2–8]. However, there was always a triggering or exacerbating factor, i.e. a specific IV drug [7] or pregnancy [8], and the electrolyte imbalance was quite typical, i.e. hypocalcaemia, hypomagnesaemia [8], elevated PTH [3, 5], and additionally lowered vitamin D₃ concentrations and hypophosphataemia [6, 7]. Interestingly, the patient first presented with simultaneous hyperphosphataemia, hypocalcaemia, hypomagnesaemia, high PTH, and low vitamin D concentrations. One and a half months later his vitamin D and PTH levels were normal, but the electrolyte profile demonstrated hypocalcaemia, hypomagnesaemia, and hyperphosphataemia. The potential explanations, also mentioned in similar case reports, include malabsorption of calcium and magnesium, caused by reduced intestine length because of many fistulas, as well as exacerbation of hypocalcaemia by hypomagnesaemia, due to reduced PTH secretion [9]. In the case of the described patient, the latter mechanism should not have applied because his PTH levels were either high or within the normal values with accompanying hypomagnesaemia. However, none of these mechanisms explain the hyperphosphataemia. Hypomagnesaemia may also cause end-organ resistance to PTH [9], which is a likely cause of the electrolyte imbalances in our patient, including hyperphosphataemia. This may be indicative that PTH secretion was preserved, while only the end-organ response was impaired, as described by another case report [3]. Hyperphosphataemia could also have been the result of an inflammatory process, either in the digestive tract or in the peritoneum. Taking everything into account, secondary hyperparathyroidism in our patient could be a result of 4 coexisting factors, causing hypocalcaemia: malabsorption, vitamin D₃ deficiency, the resistance of organs to PTH, and calcium being bonded with the excess of phosphates.

Concluding, this case report describes a patient with CD and ileostomy, with recurrent tetany and atypical

phosphate imbalance. This case report highlights various metabolic symptoms that may be present in patients with CD.

Conflict of interest

The authors declare no conflict of interest.

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