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Case Report

Abdominal Pain Crisis in a Young Patient with B12 Deficiency: A Case Report

Neda Nozari[1], Akram Pourshams[1],Parisa Divsalar[2]

[1] Digestive Disease Research Center, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran
[2] The Priory Hospital Cheadle Royal, Cheadle, Cheshire, Manchester, UK

ABSTRACT

Vitamin B12 deficiency is an unusual disease in younger individuals.This case report has demonstrated how a patient with vitamin B12 deficiency can present with unusual symptoms.Two episodes of severe abdominal pain were identified in a 35-year-old man in the emergency ward. During both episodessurgical consultations were requested. A gastroduodenoscopy revealed macroscopic atrophic gastritis and pathology reports showed mucosal atrophy in the gastric corpus with evidence of intestinal metaplasia. Laboratory data showed a low level of vitamin B12 in the patient's serum. In rare cases, vitamin B12 deficiency can present with severe abdominal pain associated with abdominal rigidity.

Keywords: Vitamin B12 deficiency; Atrophic gastritis; Abdominal pain

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INTRODUCTION

Vitamin B12 deficiency is an uncommon and unusual disease among younger populations (1-3). The median age at diagnosis is 60 years due to slow disease progression (1, 2).Women are affected more than men(1). Vitamin B12 deficiency may occur due to the malabsorption syndrome.

Vitamin B12 deficiency can affect major organs that have rapid cell turnover such as the bone marrow and gastrointestinal tract(1,4). The onset of vitamin B12 deficiency can be silent, insidious and vague. Patients often adapt to their symptoms due to slow disease progression(5). Rare manifestations of vitamin B12 deficiency have been reported in one review of a case study (89 cases). Manifestations included neurologic pain, as well as jerking and involuntary movements in the abdominal muscles(6).

The current case report shows how a vitamin B12 deficiency can present with unusual symptoms.

Corresponding author:
Neda Nozari, MD
Digestive Disease Research Center, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran
Tel: +98 02182415000
Fax:+98 21 82415400
E-mail: nozari_neda@yahoo.com
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CASE REPORT

A 35-year-old man who resided in Ardebil, Iran with a history of aninitial episode of severe abdominal pain three weeks prior presented to our emergency room with complaints of a second episode of severe acute abdominal pain upon awakening. He woke up with the pain four hours prior to his arrival. The onset of pain was in the epigastric region and then became diffuse. The pain was exacerbated by movement. He denied nausea, vomiting, fever, chills or rigor. Pain was not related to eating. The patient had suffered

from chronic mild abdominal pain (almost constant) for six months which was occasionally exacerbated while eating. His appetite was poor but there was no significant weight loss. Pain gradually progressed during the previous six months. There was no history of any vegetarian diet, neurologic symptoms (ataxia, paresthesis, etc.), diarrhea or malabsorption. During the physical examination he appeared uncomfortable and had diffuse tenderness with guarding and rigidity. Normal bowel sounds were auscultated throughout the abdomen. Laboratory studies showed the following: hemoglobin(3.9g/dlit), hematocrit(10), WBC (1100/mm^3), and platelets (40000/mm^3). Indices showed MCV of 100fL, MCH of 39pg/cell, and RDW of 16. The reticulocyte count was 2% after correction due to anemia and his LDH level was 396U/L.Liver function tests reported total bilirubin (2.8mg/dlit), direct bilirubin (0.4mg/dlit), AST (38U/L), and ALT (29U/L).Kidney function, amylase, and lipase tests were normal. The plain abdominal radiograph was normal. An abdominal ultrasound showed no evidence of any inflammatory changes. After 24 hours, there was a reduction in his abdominal painwithout any specific treatment. More laboratory tests were requested. The stool specimen was negative for ova or parasites and he had a normal urinalysis. HBS antigen, HCV antibody, and HIV tests were negative. The G6PD level was sufficient. Celiac serology was negative. Hyper-segmented polymorphonuclear leukocytes were visualized in the peripheral blood smear; his B12 level was 197 pg/ml (normal: 200-970) and serum folate was 15 pg/ml (normal range). An upper gastrointestinal endoscopy showed atrophic gastritis that was macroscopic in appearance which was confirmed by gastric biopsy. The biopsy showed mucosal atrophy in the gastric corpus with evidence of intestinal metaplasia, but not reported *H. Pylori* .Bone marrow biopsy showed megaloblastic anemia with CD33 as 15%-20% of immature myeloid series and CD34 as 5% of marrow nucleated cells. Magnetic resonance enterography imaging was normal (motility and mucosal appearance). Abdominal vessel Doppler sonography was normal (excluded vessel thrombosis). CD55 and CD59 were negative for paroxysmal nocturnal hemoglobinuria. A genetic test was performed to exclude familial Mediterranean fever. His lead level was in the normal range. MRI of the cervical and thoracic area showed no lesions in spinal cord.

After a comprehensive evaluation, his treatment consisted of daily interamuscular administration of vitamin B12 (1mg) for one week followed by 1mg weekly administration for four weeks and 1mg monthly. The patient was prescribeddaily folic acid (5mg) and ferrous sulphate (325mg) due to his severe anemia. He did not show any symptoms during three months of follow up. The anemia improved and his abdominal pain did not recur. Final laboratory tests showed the following: WBC (6500/mm3), hemoglobin (13.3mg/dlit), MCV (85fL), platelets (280,000/mm3), AST (24U/L), ALT (20U/L), ALP (138µ/L), total bilirubin (1.4mg/dlit), direct bilirubin (0.8mg/dlit), folate(25pg/ml), and vitamin B12 (1081pg/ml).

DISCUSSION

Megaloblastic anemiais associated with hypersegmented polymorphonuclear leukocytes,low serum B12 levels(1,7), and an abnormal shilling test in laboratory studies(7).Multiple causes include autoimmunity, absolute intrinsic factor (IF) deficiency and dysfunctional IF(1,8).

Gastric biopsy can confirm chronic atrophic gastritis (type A). Peripheral blood smears show macrocytosis with hypersegmented polymorphonuclear leukocytes, anemia, leukopenia, and thrombocytopenia or pancytopenia. Bone marrow examination will show megaloblastic changes (giant metamyelocytes)(1).

Peripheral neuropathy (paresthesis and numbness) (1,2,9) and lesions in the posterior and lateral columns of the spinal cord(subacute combined degeneration)(1,9) and in the cerebrum can occur in vitamin B12 deficiency. Progression from demyelination to axonal degeneration may cause serious complications which may not be reversed after vitamin B12 replacement therapy.Spinal cord injuries are a combination of posterior column signs (loss of vibration and position sense, and sensory ataxia with a positive Romberg's sign) and lateral column signs(limb weakness, spasticity, and extensor plantar responses)(1).We were surprised this case did not haveevidence of neurologic signs and symptoms when he presented with two episodes of abdominal pain crisis that we attributed to spinal cord pathology. MRI of the cervical and thoracic areas did not show evidence of lesion in the spinal cord.

MRI can show involvement of the cervical spinal cord in most patients but pathology initially presents in the thoracic cord. MRI confirms involvement of multiple segments in the cord. Cord pathology can improve with treatment if cord atrophy is not visualized on MRI(10). One review case study from the UShas reported rare manifestations of vitamin B12 deficiency that included neurologic pain, jerking and involuntary movements in the abdominal muscles(6). This case report introduced

another unusual manifestation of vitamin B12 deficiency in one young patient.

Although vitamin B12 deficiency is a rare disease among younger individuals, clinicians should obtain vitamin B12 levels in cases of macrocytic anemia. Vitamin B12 deficiency in patients rarely can present with severe abdominal pain associated with abdominal rigidity which is likely due to spinal cord pathology.

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