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CLINICAL CASE

Olmesartan-Induced Enteropathy: An Unusual Cause of Villous Atrophy

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Abstract We report a case of a 63-year-old-man presenting with chronic diarrhea and weight loss while on olmesartan treatment for hypertension. Investigation showed multiple nutritional deficiencies associated with diffuse intestinal villous atrophy. Serologies for celiac disease were negative and other causes of villous atrophy were excluded. Olmesartan as a precipitant agent was suspected and withdrawn. Clinical improvement occurred in days with no need for other therapeutic measures. Follow-up at three months showed clinical remission and almost complete recovery of intestinal atrophy.

Olmesartan is an angiotensin receptor blocker commonly prescribed for the management of hypertension. Spruelike enteropathy associated with this drug is a recently described entity with few cases reported. It presents with chronic diarrhea and intestinal villous atrophy and should be included in its differential diagnosis. This case intends to alert clinicians for the possibility of this event in a patient on treatment with this drug.
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PALAVRAS-CHAVE
Atrofia;
Doenças Gastrointestinais;
Antanosistas dos Recetores da Angiotensina II/efeitos adversos

Enteropatia Induzida pelo Olmesartan: Uma Causa Incomum de Atrofia Vilositária

Resumo Apresentamos o caso de um homem de 63 anos com diarreia crónica e perda ponderal. Apresentava hipertensão arterial tratada com olmesartan. A investigação complementar mostrou múltiplos défices nutricionais associados a atrofia vilositária intestinal difusa. As serologias de doença celíaca foram negativas e outras causas de atrofia vilositária foram excluídas. Suspeitou-se do olmesartan como agente precipitante, sendo este suspenso. Observou-se melhoria clínica em dias, sem necessidade de outras medidas terapêuticas. No seguimento, aos 3 meses, constatou-se remissão clínica e recuperação quase completa da atrofia intestinal.

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O olmesartan é um bloqueador dos recetores da angiotensina, geralmente prescrito no tratamento da hipertensão. A enteropatia ''spruelike'' associada a este fármaco é uma entidade recentemente descrita, com poucos casos reportados. Manifesta-se por diarreia crónica associada a atrofia vilositária intestinal, devendo ser incluída no seu diagnóstico diferencial. Com este caso pretende-se alertar os clínicos para a possibilidade deste evento em doentes sob tratamento com este fármaco.
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1. Introduction

The most common cause of villous atrophy is celiac disease.[1,2] The villous atrophy results from injury to the small intestine and leads to loss of absorptive surface area, reduction of digestive enzymes, and consequential impaired absorption of micronutrients.[3] Negative celiac serology or nonresponse to a gluten-free diet implies a broad and challenging differential diagnosis which includes Crohn's disease, enteric infections (e.g. *Giardia lamblia*), collagenous sprue, tropical sprue, common variable immunodeficiency, autoimmune enteropathy, hematological malignancies and medication-associated enteropathy.[2] Regarding the latter, olmesartan medoxomil, an angiotensin receptor blocker for the management of hypertension, has been recently recognized as a cause of ''sprue-like enteropathy''.[4–6]

We report a case of severe enteropathy associated with olmesartan use.

2. Clinical case

A 63-year-old man was admitted to our department complaining of progressive diarrhea and significant weight loss (12 kg) for one year. He reported between 5 and 7 daily episodes of bulky, watery and nonbloody diarrhea. Over the preceding two weeks, it was associated with severe fatigue, anorexia and vomiting. He denied abdominal pain, fever or other symptoms. There was no history of recent travels or sick contacts.

Apart from arterial hypertension, treated with olmesartan and hydrochlorothiazide (20/12.5 mg) for two years, his past medical history was unremarkable.

The patient had already undergone total colonoscopy and abdominal computed tomography with no remarkable findings. A gluten and lactose-free diet were tried without improvement. He also failed initial conservative treatment with a trial of oral antibiotic for possible small bowel bacterial overgrowth.

On presentation at our department, his body mass index was 20 kg/m^2 (normal (N): 18.5–24.99 kg/m^2), close to the lower limit of normal. Muscle wasting was also seen without evident weakness. There was no peripheral edema or other relevant findings on physical examination.

Laboratory evaluation revealed: hemoglobin 11.6 g/dL (normal: 13–17) with normal mean corpuscular volume and mean corpuscular hemoglobin; serum potassium 1.8 mmol/L (N: 3.6–5.1), phosphorus 1.7 mg/dL (N: 2.3–4.7), magnesium 1.3 mg/dL (N: 3.6–5.1), corrected calcium 8.6 mg/dL (N: 8.8–10); albumin 2.9 g/dL (N: 3.4–4.8), total protein 4.9 g/dL (N: 6.2–8.5), aspartate aminotransferase 205 UI/L (N: 5–34), alanine aminotransferase 106 UI/L (N < 55) and protein C-reaction 25 mg/L (N: <5 mg/L). The prothrombine time (PT) was increased (26.6 s; N: 9.4–13) as well as activated partial thromboplastin time (aPTT) (50 s; N: 20–40).

Other laboratory work-up was unremarkable including leucogram, serum glucose, B12 vitamin, folic acid, transglutaminase antibodies, serum immunoglobulins, thyroid stimulating hormone and serology for human immunodeficiency virus. Platelets, bleeding time and fibrinogen were also normal.

Other causes for hypertransaminasemia were additionally excluded (no alcohol consumption; bilirubin, alkaline phosphatase, gamma-glutamyl transpeptidase, serum iron, ferritin, transferrin saturation, cholesterol and triglycerides were normal; hepatitis B and C serologies were negative; antinuclear antibodies and smooth muscle antibodies were negative and abdominal ultrasound excluded liver or biliary abnormalities).

Stool examination namely cultures, *Clostridium difficile* toxin assay, ova and parasites was unrevealing.

A colonoscopy was repeated and, despite all efforts, the terminal ileum could not be intubated. Colonic random biopsies excluded microscopic colitis or other abnormalities. Upper endoscopy evidenced a discrete attenuation of duodenal villous pattern without other findings (Fig. 1).

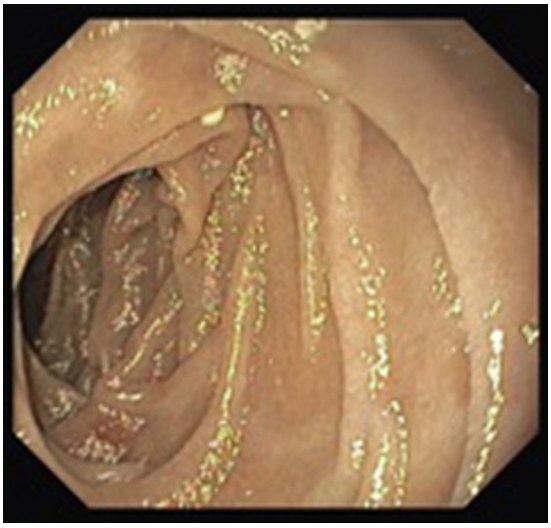

Figure 1 Initial upper endoscopy showing a discrete attenuation of villous pattern of the second portion of the duodenum.

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