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CLINICAL CASE

Olmesartan-Induced Sprue Like Enteropathy

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Abstract Chronic diarrhoea is a common clinical problem in gastroenterology practice and often it is difficult to diagnose the cause. Villous atrophy is not specific and the rarer possibility of drug-induced enteritis should always be considered. Olmesartan has recently been described as a cause of drug-induced enteropathy characterized by chronic diarrhoea and varying degrees of duodenal mucosa atrophy resembling celiac disease.

We describe two cases of sprue-like enteropathy in patients treated with olmesartan for arterial hypertension several years before the onset of symptoms. Patients presented severe diarrhoea and significant weight loss, and both had histological evidence of intestinal villous atrophy. The clinical signs completely resolved after drug withdrawal.

Olmesartan-induced enteropathy is a new clinical entity that must be included in the differential diagnosis of villous atrophy with negative celiac serology. The clinical and histological alterations easily and completely resolve after drug discontinuation, restoring quality of life to patients and avoiding unnecessary investigation.
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PALAVRAS-CHAVE
Atropfia/induzida quimicamente;
Diarreia/induzida quimicamente;
Olmesartan;
Mucosa Intestinal

Enteropatia Tipo Celíaca Induzida Pelo Olmesartan

Resumo A diarreia crónica é um problema clínico comum na prática de gastroenterologia e, muitas vezes, o diagnóstico da causa é difícil. A atrofia das vilosidades intestinais não é específica e a rara possibilidade de enterite induzida por fármacos deve ser considerada. O olmesartan foi recentemente descrito como uma causa de enteropatia induzida por fármacos caracterizada por diarreia crónica e graus variáveis de atrofia da mucosa duodenal semelhante á doença celíaca.

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Os autores descrevem dois casos de enteropatia tipo celíaca em doentes com história de hipertensão arterial medicada com olmesartan vários anos antes do início dos sintomas. Ambos os doentes apresentaram diarreia grave, perda ponderal significativa e evidência histológica de atrofia vilositária. Os sinais clínicos resolveram completamente após a interrupção do fármaco.

A enteropatia induzida por olmesartan constitui uma nova entidade clínica que deve ser incluída no diagnóstico diferencial de atrofia vilositária seronegativa. As alterações clínicas e histológicas resolvem rápida e completamente após a suspensão do fármaco restaurando a qualidade de vida aos doentes e evitando, muitas vezes, investigações invasivas desnecessárias.
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1. Introduction

Intestinal villous atrophy with negative celiac serology may be a diagnostic challenge. Apart from celiac disease, a variable degree of villous atrophy can be found in other conditions as autoimmune enteropathy, common variable immune deficiency, small bowel bacterial overgrowth, parasitic infection such as giardiasis, intestinal lymphoma, human immunodeficiency virus infection-related enteropathy, Whipple's disease, and tropical sprue. Villous atrophy can also occur with prolonged use of some medications.

An association between olmesartan and enteropathy development, which is histologically indistinguishable from celiac disease, has been described recently.[1–4] Olmesartan-induced enteropathy can cause severe chronic diarrhoea with substantial weight loss, even months or years after drug initiation. The Food and Drug Administration issued a statement on olmesartan labelling in July 2013 after a case series with 22 patients was reported by Mayo Clinic.[1] The physiopathogenic mechanism of olmesartan-induced enteropathy remains unknown. One proposed mechanism is related to a cell-mediated immune response that damages the small intestinal brush border.[1–5] Additionally, a predisposition in patients with an autoimmune background has been suggested.[5,6]

We report two cases of severe olmesartan-induced enteropathy and discuss the natural history of this condition

2. Clinical cases

2.1. Case 1

A 60 year-old Caucasian male with arterial hypertension, treated with olmesartan and hydrochlorothiazide (20 + 12.5 mg/day) for the last 4 years, was admitted to our hospital with a 3-month clinical history of abdominal pain, diarrhoea and marked weight loss (16% body weight). He reported between 5 and 7 daily episodes of watery and non-bloody diarrhoea. He denied any other symptoms suggestive of local or systemic infections, recent travel, consumption of contaminated food or water, animal contact, or changes in diet or medications within the past few years.

Because the patient was dehydrated and had tachycardia with orthostatic hypotension, antihypertensive drugs were discontinued. Abdominal palpation was diffusely painful but there were no masses or organ enlargement. On physical examination, there was also diffuse skin thickening without sclerodactyly or telangiectasias.

Blood tests showed hypokalaemia (serum potassium 2.0 mmol/L; N: 3.6–5.1), hypophosphataemia (phosphorus 1.8 mg/dL, N: 2.3–4.7), and hypoalbuminaemia (albumin 2.8 g/dL; N: 3.4–4.8). The patient's leucocyte count, C-reactive protein level, and liver chemistries were all normal. The remaining initial workup including immunoglobulin levels, serum thyroid stimulating hormone, stool cultures, Clostridium difficile toxin assay, stool ova and parasites, and stool osmolality and electrolytes was unremarkable. Conventional serologic tests (tissue transglutaminase, endomysial, and antigliadin antibodies) and a lack of clinical response to a gluten-free diet ruled out celiac disease.

An abdominal CT scan showed diffuse small bowel wall thickening with surrounding mesenteric adenopathy (Fig. 1). Pancreatic abnormalities or potential malignancies were excluded. Colonoscopy and colonic biopsies were normal, and there was no evidence of microscopic colitis or inflammatory bowel disease. Immunological assays were positive for antinuclear antibody (titre, 1:1280) and Scl70 suggesting a diagnosis of systemic sclerosis that was confirmed by a skin biopsy.

During hospitalization, the patient had progressive clinical and analytical improvements and was discharged with an indication for clinical and analytical reassessment. A month later, diarrhoea and weight loss recurred. Oral ciprofloxacin for possible small bowel bacterial overgrowth secondary to small intestinal dysmotility of systemic sclerosis was initiated, but he did not show any clinical improvement. An upper gastrointestinal endoscopy was performed and duodenal biopsies revealed moderate villous blunting and intraepithelial lymphocyte infiltration (Fig. 2). Because of clinical improvement during hospitalization (without antihypertensive drug) and intensification of complaints with the re-introduction of his usual medication, olmesartan-induced enteropathy was suspected. The drug was discontinued. Within 3 months, the patient gained weight and showed resolution of analytical changes.

2.2. Case 2

A 62 year-old Caucasian male with arterial hypertension, who was administered olmesartan (20 mg/day) for 5 years previously, was referred for a gastroenterology and internal

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