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SCIENTIFIC LETTERS

Silodosin: An overlooked cause of drug-induced diarrhea☆

Silodosina: causa desapercibida de diarrea farmacológica

Drug-induced diarrhoea is the second most common cause of acute diarrhoea after infection, but is very seldom taken into account. It should also be remembered that it is not only caused by typical drugs, such as antibiotics or metformin.[1]

We present the case of an 80-year-old man with a significant history of ischaemic heart disease, colonic diverticulosis and benign prostatic hyperplasia (BPH), who underwent surgery in February 2015, where he received 1 cycle of antibiotic therapy for a urinary tract infection. He had been on standard omeprazole and carvedilol treatment for years at stable doses. Silodosin had been prescribed upon discharge from the urology department; during admission he had received neither non-steroidal anti-inflammatory drugs, a new cycle of antibiotics nor other diarrhoea-inducing drugs. He was admitted to hospital approximately 3 weeks after discharge from urology for symptoms of diarrhoea of around 20 low-volume watery stools per day with no mucous, pus or blood, and no rectal tenesmus or urgency. Symptoms did not worsen with food intake, nor were they accompanied by abdominal pain or fever. The patient denied travel abroad that month or other epidemiological history of interest. He had never presented any similar gastrointestinal episode that would suggest a chronic origin or recurrent symptoms. He also reported weight loss of 4 kg since onset.

On arrival at the emergency department, laboratory tests indicated prerenal acute kidney failure, for which he received intravenous fluids. In view of a suspected infectious aetiology, empirical antibiotic therapy was initiated with ciprofloxacin and metronidazole. *Clostridium difficile* toxin testing was negative, and faecal cultures were inconclusive. Blood samples were taken for serology; all tests for diarrhoea (including antibodies for coeliac disease, thyroid hormones) were negative. Complete colonoscopy was performed—with excellent preparation—with the sole finding of diverticulosis. He had not had any bowel movements since the day following admission, so as he remained asymptomatic, he was discharged. The patient was readmitted the following week for new symptoms of diarrhoea of 20 stools per day with the same characteristics as in the previous admission. Again, bowel movements ceased the day after admission. Given the chronology, and the fact that the symptoms were self-limiting during admission, differential diagnosis was considered with drug-induced diarrhoea. The patient's history was taken again, in which he reported that he had recently commenced silodosin, a medication that had not been administered during the previous admission due to lack of availability. Nevertheless, the colonoscopy was repeated to take biopsies in order to rule out microscopic colitis, but revealed no histological alterations.

Given the suspicion of severe diarrhoea secondary to silodosin, treatment was discontinued, and the patient's symptoms ceased completely.

Silodosin is an alpha-1a adrenergic receptor antagonist that causes smooth muscle relaxation in the lower urinary tract, thereby decreasing bladder outlet resistance without affecting detrusor smooth muscle contractility. It is used mainly for BPH, which is why our patient was taking it. In the Summary of Product Characteristics, diarrhoea is described as a common side effect (10%),[2] but in clinical trials it appears in only 4% of patients and is not described as a serious adverse event.[3,4]

Drug-induced diarrhoea occasionally goes unnoticed, and must be considered specifically for correct diagnosis. Nevertheless, differential diagnosis should always be made with other causes of diarrhoea, and faecal, blood, serological and endoscopic tests should be carried out to make a diagnosis of exclusion. Treatment consists of discontinuation of the causal drug. Diarrhoea can be one of the most common adverse effects of pharmacological treatments, but is usually mild. It should not be forgotten that, occasionally, it can cause serious symptoms that require hospital admission, as our case shows.[5,6]

As medicine advances, we will increasingly find ourselves dealing with polymedicated patients. This case underlines the importance of a complete anamnesis, always including the patient's overall treatment so that the drug origin does not go unnoticed, since patients can present with severe diarrhoea. This is yet another example of how an accurate, detailed, and carefully-taken medical history is the key not only to reaching the diagnosis of drug-induced diarrhoea,

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but to be able to withdraw the causal agent and achieve healing.

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Conflict of interests

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Lower gastrointestinal bleeding as a presentation of miliary tuberculosis

Hemorragia digestiva baja como presentación de una tuberculosis miliar

A 44-year-old male, native of Bolivia but living in Spain for the last 10 years without recent travels to his country, and with no history of interest, attended the emergency room complaining of rectal bleeding with hemodynamic instability. The patient also addressed a three-month period of diffuse abdominal pain (predominantly in left flank), dysmotility, dysthermia, night sweats and weight loss. Physical examination, including neurological examination, was unremarkable except for malnutrition and pain in the left flank's deep palpation without detecting masses, organomegaly or ascites.

Tests revealed 7.6 g/dL hemoglobin levels with ferropenia profile, 135 mg/dL CRP, 60% Quick I. and severe malnutrition. The rest of the parameters, including WBC count, electrocardiogram and chest radiograph resulted without alterations. Colonoscopy was performed without finding mucosal lesions, though with hematic content in terminal ileum. Gastroscopy was normal. CT scan showed jejunal thickening with plenty intraluminal hematic content without actually observing bleeding points. In addition, a thickening of the omentum, multiple peritoneal implants, ascites, and lymphadenopathy were demonstrated. Double contrast CT and gastrointestinal transit were performed without new discoveries (Fig. 1).

On admission the blood culture, HIV and Mantoux tests were negative. Early laparoscopy was performed, obtaining images that suggested miliary TB vs. peritoneal carcinomatosis (Fig. 2).

Histology showed abundant caseating granulomas. The Ziehl Neelsen stain and PCR for M. Tuberculosis in peritoneal fluid were negative. Due to these findings the patient was isolated until the negative result of sputum smear microscopy arrived. Pleural effusion was found in a second radiography with negative RCP for Mycobacteria.

Treatment was initiated with rifampicin, isoniazid, pyrazinamide and ethambutol in weight-adjusted doses, with resolution of symptoms. As an adverse effect the patient presented transient elevation of transaminases. After 8 weeks, sputum results arrived: both solid and liquid medium cultures were positive for Mycobacterium Tuberculosis sensitive to all TB drugs. Peritoneal fluid and urine cultures in both solid and liquid medium were negative. We had no peritoneum sample for culture. Currently our patient is progressing well with a 5 kg weight gain and continues with rifampicin and isoniazid, intending to maintain the treatment for at least 12 months.

In this case gastrointestinal bleeding related to jejunal affectation and probably secondary to peritoneal extension, allowed the diagnosis of miliary tuberculosis.

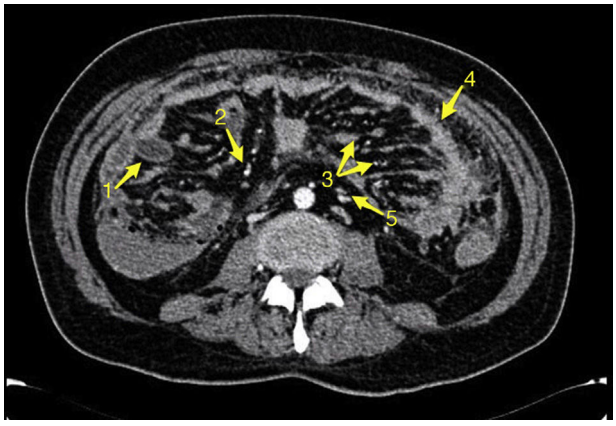

Figure 1 CT showing (1) blood content in jejunum, (2) multiple peritoneal implants, (3) omental cake, (4) thickening of the omentum, (5) lymphadenopathy.