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LETTERS TO THE EDITOR

Hypertrophic pyloric stenosis in adults☆

Estenosis hipertrófica pilórica del adulto

To the Editor,

We present the case of a 72-year-old woman, allergic to iodine contrast, with a history of type 2 diabetes mellitus, hypercholesterolaemia, goitre and hypothyroidism due to Hashimoto thyroiditis. She had been admitted on multiple occasions since 1997 for acute gastric dilatation secondary to pyloric stenosis, which was of unknown aetiology due to the patient's refusal to undergo diagnostic tests. She had severe ischaemic colitis, with secondary sigmoid perforation in 2006, requiring surgical resection and permanent colostomy in the left iliac fossa.

She was admitted to our department for sudden onset abdominal pain, accompanied by vomiting and absence of flatulence. On physical examination, she presented severe abdominal distension, tympanism and findings of peritoneal irritation. Blood tests found compensated metabolic acidosis, with normal full blood count, coagulation, C-reactive protein and basic biochemistry. Abdominal X-ray revealed severe gastric dilatation (Fig. 1), so an urgent abdominal computed tomography (CT) scan was requested (Fig. 2A and B), in which gastric distension secondary to complete pyloric stenosis and pneumoperitoneum was observed, with no clear perforation site.

The patient underwent surgical treatment with total gastrectomy and bowel transit reconstruction by Roux-en-Y oesophagojejunostomy. We were unable to use the laparoscopic approach given the patient's severe gastric distension.

A stomach measuring 30 cm at the longest axis was observed in the surgical specimen, with a thickened pylorus (Fig. 3); histopathology showed muscle fibres in bundles with an irregular arrangement and hypertrophy. No perforation site was found in the specimen, attributing the pneumoperitoneum to diffusion of air secondary to the severe gastric dilatation.

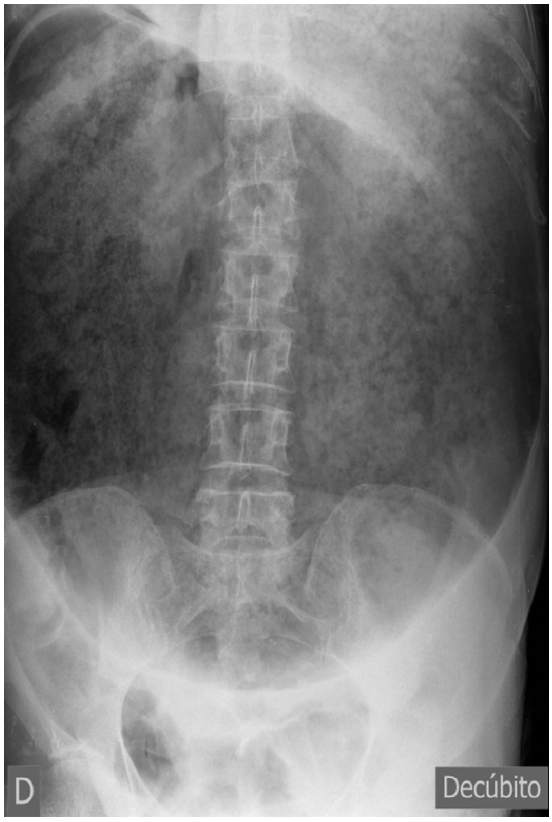

Figure 1 Severe gastric dilatation on conventional abdominal X-ray.

Hypertrophic pyloric stenosis in adults is a rare entity, with a wide range of symptoms (although it can be asymptomatic), producing dyspepsia, vomiting or even occlusive symptoms with pneumoperitoneum.[1] Symptoms can appear between the ages of 14 and 85, with an average age of 50 years; it is more common in men, with an incidence of 3:1.[2,3]

The aetiology remains uncertain, but several hypotheses have been proposed, namely[1,2,4]:

1. *Primary causes*: No apparent lesion identified:

(a) Neuromuscular incoordination due to changes in the Auerbach plexus or vagal hyperactivity.
(b) Protracted pylorospasm.

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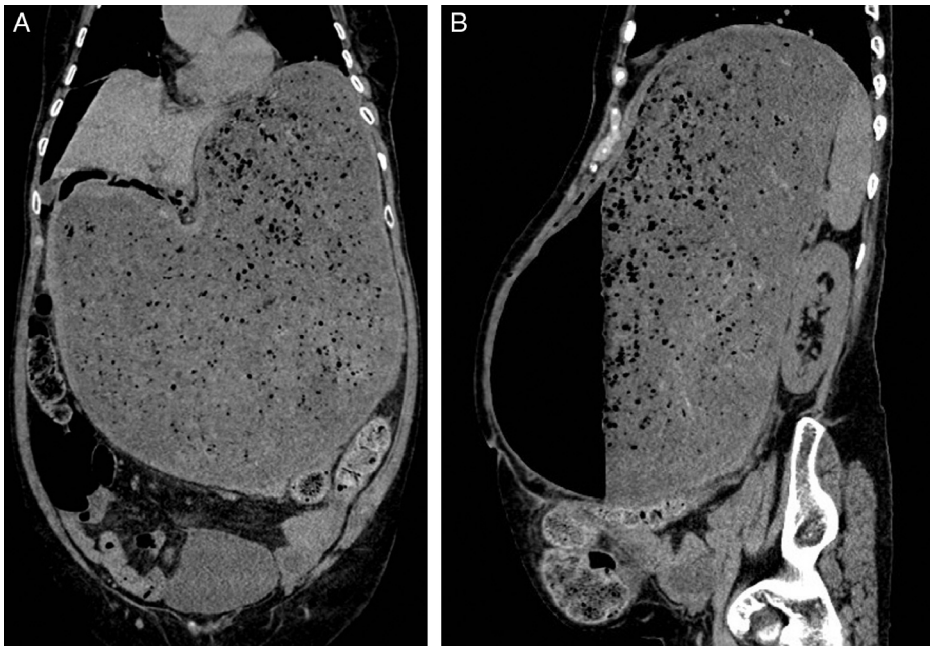

Figure 2 Abdominal computed tomography images. (A) Coronal slice; (B) Sagittal slice: stomach very distended, containing food and air, with no passage of these to the duodenum observed, in relation to known pyloric stenosis. Pneumoperitoneum in upper abdomen and small amount of perigastric free fluid.

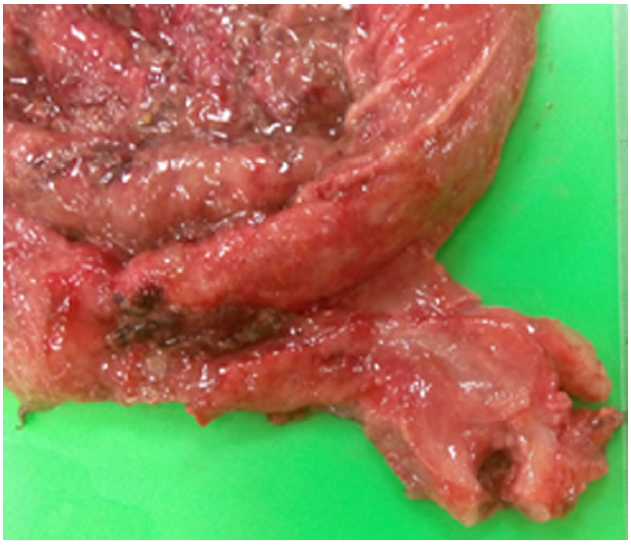

Figure 3 Hypertrophy at pyloric level in transverse slice of surgical specimen.

(c) Persistence of childhood stenosis. Most authors favour this theory, in which the disease remains latent and is reactivated by secondary factors such as inflammation, oedema, and spasm. Pyloric stenosis in adults and children presents similar anatomical and histopathological abnormalities, which supports this hypothesis.

2. *Secondary causes*: Associated with gastric, pyloric or duodenal lesion, such as gastritis, peptic lesions or tumours.

Clinical symptoms are variable and rarely diagnostic, with predominantly abdominal pain, persistent vomiting, early satiety, anorexia, dyspepsia and weight loss.[2,3] Diagnosis is mainly radiological (identifying a pyloric canal greater than 1 cm) and endoscopic (where a narrow pylorus with smooth border is observed), although diagnostic confirmation is histological.

Asymptomatic patients or those with few symptoms do not require treatment, while patients with clinical evidence require surgery. Among the various surgical techniques used, pylorectomy with minimal gastrectomy seems to obtain better outcomes. Endoscopic treatment by means of pyloric dilation has also been described, but has a high rate of recurrence and should be used only in selected cases (patients with high surgical risk or who refuse surgery).[2–4]

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Ana Belén Fernández Laso[a,*], Idoia Ganchegui Aguirre[a], Amaia Campos Ruiz[a], Leire Urtasun Arlegui[a], Irati Gorroño Zamalloa[a], Candido Martinez Blazquez[b], Maria Elena Carreter De Granda[c], Maria Begoña Atares Pueyo[d], Aitor Orive Calzada[a]

[a] *Servicio de Aparato Digestivo, Hospital Universitario de Araba, Vitoria-Gasteiz, Araba, Spain*
[b] *Servicio de Cirugía General, Hospital Universitario de Araba, Vitoria-Gasteiz, Araba, Spain*
[c] *Servicio de Radiodiagnóstico, Hospital Universitario de Araba, Vitoria-Gasteiz, Araba, Spain*
[d] *Servicio de Anatomía Patológica, Hospital Universitario de Araba, Vitoria-Gasteiz, Araba, Spain*

* Corresponding author.
E-mail address: anabelen.fernandez.laso@gmail.com (A.B. Fernández Laso).

Intestinal and peritoneal tuberculosis in a non-immunocompromised patient☆

Tuberculosis intestinal y peritoneal en paciente no inmunodeprimido

To the Editor,

Intestinal involvement in tuberculosis is rare, but when it occurs, the ileocaecal area is most often affected.[1] The incidence of tuberculosis is increasing, however, as a result of HIV, chronic renal disease, immunosuppressive therapy and immigration.[1,2] It is the great imitator, and is a diagnostic challenge.[1]

We present the case of a 67-year-old Spanish woman with a history of hypothyroidism and chronic constipation, who had been admitted for abdominal pain three times in the previous 2 months. She reported a 5-day history of postprandial abdominal pain, vomiting and constipation. The patient was afebrile, with unquantified weight loss.

On examination, her abdomen was slightly distended, tympanitic, with generalised pain, no signs of peritoneal irritation, no masses or organomegaly and diminished bowel sounds.

In the complementary examinations, blood tests were normal, while an abdominal X-ray showed dilatation of small bowel loops, with air-fluid levels. Abdominal scan revealed dilatation of the terminal jejunum and ileum with caecal wall thickening, and a change in calibre at the level of the terminal ileum, with small lymphadenopathies in the adjacent fat and root of the mesentery. Colonoscopy showed an oedematous and deformed ileocaecal valve with minute fibrinous ulcers, and erythematous mucosa and small aphthoid lesions in the last few centimetres of the terminal ileum, from which biopsies were taken.

Due to the patient's clinical deterioration, an exploratory laparotomy was performed, which found multiple peritoneal implants with a miliary appearance, with segmental thickening of the ileum (Fig. 1). The histological result of the ileal biopsies was chronic necrotising granulomatous ileitis, and peritoneal implants and mesenteric lymphadenopathy of necrotising granulomas with caseous necrosis (Fig. 2). Ziehl–Neelsen staining and culture were negative, and a polymerase chain reaction (PCR) test was positive for *M. tuberculosis*. The Mantoux test was positive, even though the patient had been BCG vaccinated in childhood, but the interferon-gamma release assay was also positive, suggesting latent tuberculosis. Chest X-ray was normal and the HIV and hepatotropic virus studies were negative.

The patient started tuberculostatic treatment with quadruple therapy, with gradual improvement.

Diagnosing intestinal tuberculosis in a immunocompetent patient such as ours is challenging, and even more so when there is no evidence of lung disease. The lungs are involved in only 20% of cases of intestinal tuberculosis.[1] Peritoneal tuberculosis accounts for between 1%-3% of all cases of tuberculous disease.[3]

In our case, the disease commenced with sub-occlusive symptoms; these should be considered in the differen-

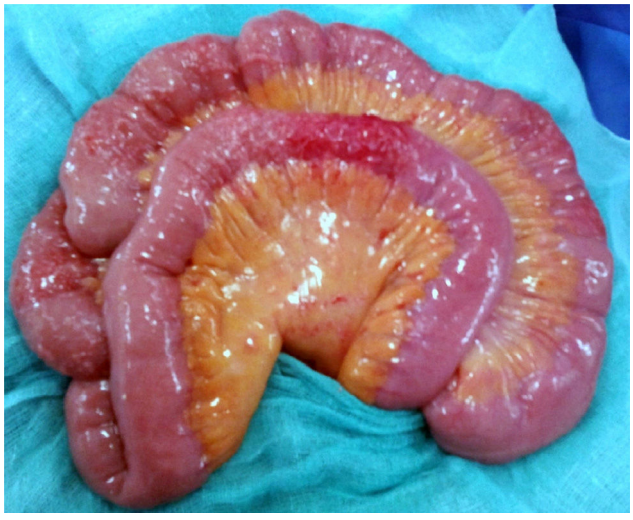

Figure 1 Image of the ileal loops, after exploratory laparotomy: multiple miliary implants in the ileal wall and meso-ileum.

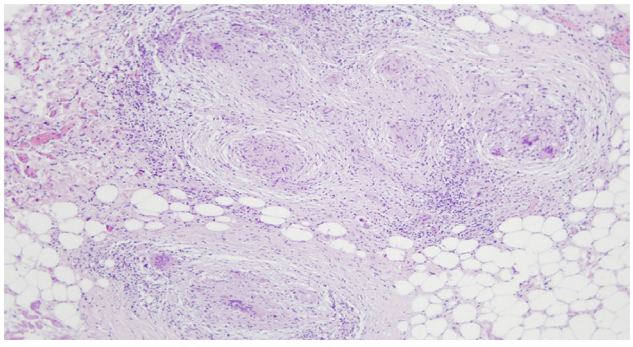

Figure 2 Necrotising granulomas, with central caseous necrosis surrounded by multinucleated giant cells.

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