bones. They are very often ingested accidentally, without the affected person even being aware, as in our case. The most likely areas for an FB to become impacted or cause perforation are where there is physiological narrowing of the gastrointestinal tract, such as the pyloric region, duodenal curves, the angle of Treitz or ileocaecal valve.[2] The associated complications range from complete lodging in the gastric wall, which could be mimicking subepithelial tumours or pseudo-pancreatic tumours with or without biliary obstruction, to liver abscesses.[3–5]

Initial diagnosis is mainly made by imaging techniques. However, there needs to be a high diagnostic suspicion, given that the signs may be hidden and it may not always present with symptoms of persistent and progressive abdominal pain, even presenting with fever. The test of choice is CT, thanks to the different reconstructions that can be performed in different planes. In a large proportion of cases, CT can assess the complication and detect the FB as a radiopaque image in the area showing radiological signs of inflammation or complication.[5]

Treatment has classically been surgery, but an increasing number of cases are being published of endoscopic management, with this being simple, fast and without complications in the majority of cases.[2] For FB which have penetrated deeply and are not visible from the gastrointestinal lumen, there are reports of using endoscopic ultrasound techniques to help detect them and then extracting them by submucosal dissection.[3,4] In our case, the FB was embedded in the gastric wall, although ulceration was still visible proximal to where it had penetrated and we were able to grab it by its proximal tip and extract it with biopsy forceps. The patient's rapid clinical improvement after the FB was extracted was impressive, in line with other reports in the literature.[4,5]

In conclusion, when a sharp or pointed FB penetrating or perforating the gastrointestinal system is diagnosed, in view of its less aggressive nature and high likelihood of resolution, we should opt for gastrointestinal endoscopy before resorting to surgery.

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Acute infection due to *Mycobacterium marinum* in a patient with ulcerative colitis and metabolic syndrome on infliximab therapy

Infección aguda por *Mycobacterium marinum* en un paciente con colitis ulcerosa y síndrome metabólico bajo tratamiento con infliximab

Opportunistic infections by atypical mycobacteria are rare complications of anti-tumor necrosis factor (TNF)-α therapy. We report the case of a patient with ulcerative colitis and metabolic syndrome on infliximab who developed a cutaneous infection caused by *Mycobacterium marinum*.

A 54-year-old man was diagnosed with ulcerative rectosigmoiditis in 1999 and treated with topical and oral mesalazine with poor therapeutic adherence. There was a history of obesity, type 2 diabetes mellitus, depressive syndrome and chronic heavy alcohol intake of 120 g ethanol/day over the last 35 years. In November 2011, he was admitted to the hospital because of a severe flare of ulcerative colitis (UC). Contrast-enhanced ultrasound showed extension of inflammation up to the splenic flexure and severe hepatic steatosis. High-dose intravenous corticosteroids were ineffective. After screening for latent infections, an excellent clinical response was obtained with infliximab at standard induction doses. Before the second infusion dose of infliximab, the patient presented with history of fever (39 °C) and appearance of a painful indurated erythematous nodule on the back of the left hand with subsequent sporotrichoid spread to the arm (Figures 1 and 2). He was not aware of any trauma but reported to take care of a fish tank at home. A skin biopsy showed positivity for non-tuberculous mycobacteria and culture yielded growth of *M. marinum*. Treatment with ethambutol (1200 mg/day) and rifampin was prescribed, but after identification of the causative organism, rifampin was substituted by clarithromycin (500 mg twice daily), and infliximab was stopped. During the next 4 years, the clinical course was characterized by intermittent clearance and reappearance of the cutaneous infection with negative and positive cultures, thus requiring multiple antimicrobial combinations. In April 2014, quadruple treatment with clarithromycin, trimethoprim–sulfamethoxazole, ethambutol and rifampin (600 mg/day) was given. Definitive clearance of the lesions and negative cultures for *M. marinum* were achieved in

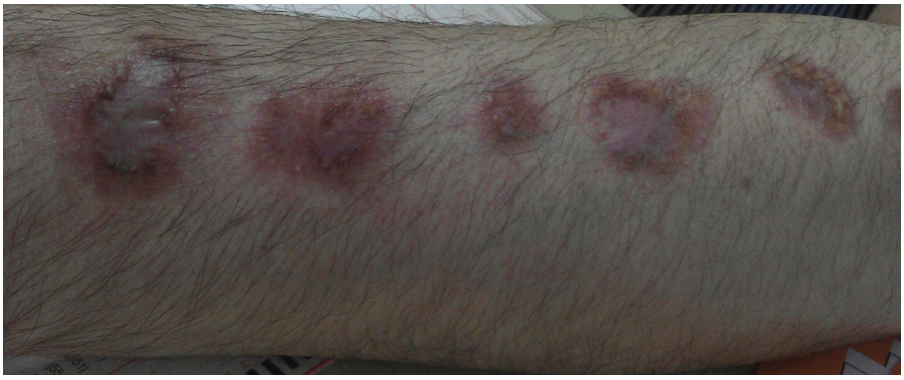

Figure 1 Erythematous painful indurated nodule on the back of the left hand, with sporotrichoid spread to the arm.

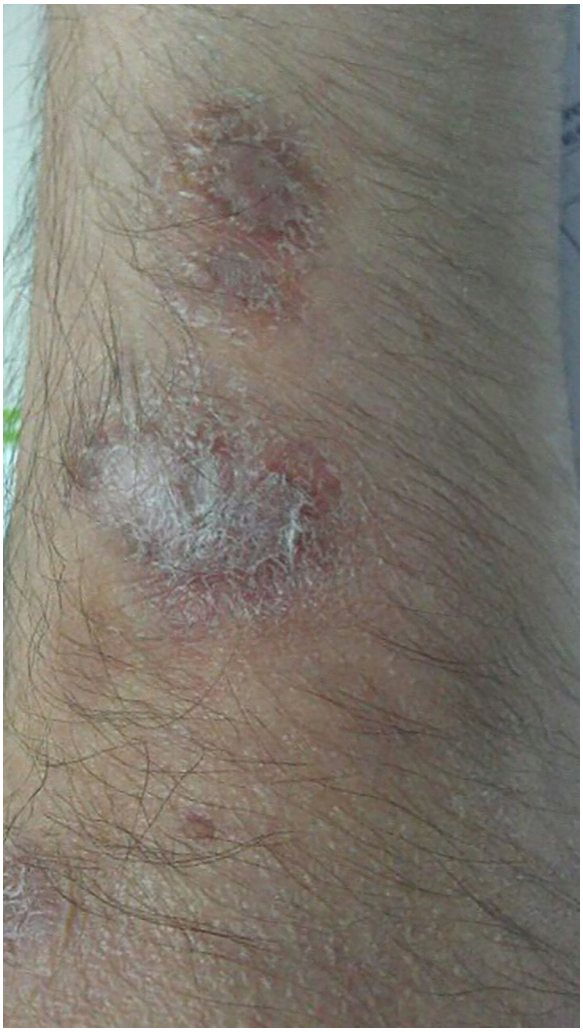

Figure 2 Residual scar after antibiotic treatment.

February 2016. During this 4-year period, remission of the UC was maintained with mesalazine, although in May 2016, the patient presented a mild flare, which was successfully treated with beclomethasone dipropionate. Treatment with azathioprine was started. At present, the UC is in remission, and there are no signs of cutaneous infection.

M. marinum has been rarely described as the causative organism of non-tuberculous cutaneous granulomas in patients Crohn's disease treated with anti-TNF-α drugs,[1–4] with only a single previous case of a patient with UC treated with infliximab.[5] Therefore, with the patient here described there are apparently only two case reports of *M. marinum* cutaneous infections in infliximab-treated patients with UC. Most *M. marinum* infections in infliximab-treated patients stem from aquarium exposure, cleaning fish tanks,[1–4] although Fallon et al.[2] reported an infection in the leg after swimming on holiday in the Canary Islands. Definitive diagnosis, however, is established by identification of the microorganism in cultures of skin biopsies, which is positive in 70–80% of cases. In our patient, cultures grew *M. marinum* but PRC amplification and DNA sequencing of the hsp65 gene fragment was not performed.

The rapid onset of infection, only 10 days after starting treatment with infliximab and the torpid clinical course, with intermittent phases of clearance and reappearance of lesions despite early and aggressive therapy, may be explained by associated obesity, metabolic syndrome, and chronic liver disease present in our patient combined with anti-TNF-α therapy.

Optimal treatment for skin lesions has not yet been established nor the duration of treatment. In our patient, different antimicrobial combinations were administered but clearance of lesions was finally achieved with clarithromycin, trimethoprim-sulfamethoxazole, ethambutol and rifampin quadruple regimen. It seems advisable to prolong treatment at least between 2 and 6 months after resolution of the lesions. Given the protracted clinical course, antimicrobial treatment was exceptionally maintained for 48 months. The decision to discontinue treatment with infliximab should be individualized but in our case biological therapy was withdrawn.

Infection caused by *M. marinum* in infliximab-treated patients with inflammatory bowel disease should be suspected in the presence of skin lesions with a sporotrichoid pattern of spread, especially when minor trauma and history of injury from fish fins or exposure to contaminated water such as home fish tanks are recalled by the patients.

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Azathioprine-induced acute submandibular sialadenitis in a patient with Crohn's disease☆

Submaxilitis aguda secundaria a la toma de azatioprina en paciente con enfermedad de Crohn

Use of immunosuppressive therapy with thiopurines is very common in inflammatory bowel disease.[1,2] We present the case of a submandibular sialadenitis in a patient with Crohn's disease (CD) related to the use of azathioprine (AZA) and mercaptopurine (MP). As this is the first time such a side effect has been reported, we believe it is very important for it to be published.

This was a case of a 39-year-old man with CD diagnosed in September 2016; A2 L3 B1 according to the Montreal classification. In February 2017, as the patient met the criteria for steroid dependence, treatment was started with AZA at a dose of 2.5 mg/kg. Two weeks later he went to the emergency department complaining of general malaise and severe submandibular pain. Physical examination detected bilateral inflammation of the submandibular glands and blood analysis showed leucocytosis and hugely elevated c-reactive protein (CRP) at 179 mg/l (normal values: <5). The patient was prescribed empirical antibiotic therapy with amoxicillin/clavulanic acid (875/125 mg/8 h) and prednisone (50 mg/day followed by descending regimen for 10 days), the azathioprine was discontinued, and his condition completely improved.

At follow-up, two months later, he was asymptomatic from the ear, nose and throat (ENT) point of view, with no pain and no signs of inflammation in the salivary glands on physical examination. However, he had begun to suffer once again from diarrhoea and right iliac fossa pain, and blood tests showed altered acute phase reactants. It was therefore decided, in the absence of reports of any other cases of sialadenitis with azathioprine, to start treatment with mercaptopurine as an immunomodulator. After the first dose of 50 mg of MP, prescribed along with oral budesonide, the patient again developed pain and inflammation in both submandibular glands, with no pyrexia or other signs of infection. Ultrasound showed a bilateral increase in the size of the submandibular glands, with no lithiasis, cysts or other abnormalities (Fig. 1). Serology for mumps virus, cytomegalovirus and Epstein-Barr

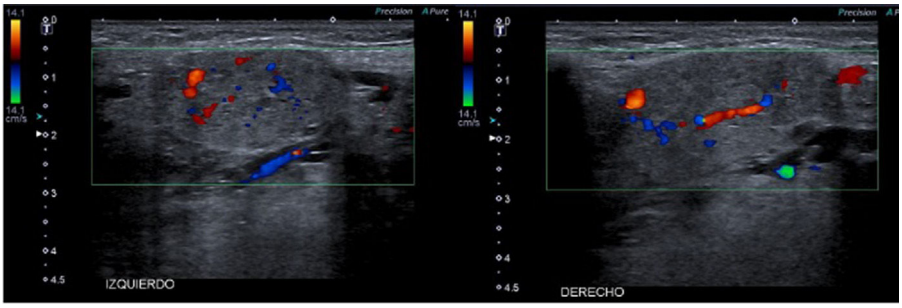

Figure 1 Ultrasound of the right and left submandibular glands. Increase in size with heterogeneity of the parenchyma and increased vascularisation. No images of lithiasis or dilation of excretory ducts. No peri- or intraglandular collections.

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