apy was started. Additionally, because of food intolerance and vomiting, a palliative endoscopic stent was placed. However, the patient evolved unfavorably and died after 4 months.

Alpha-fetoprotein producing gastric cancer has been previously described in patients with chronic liver disease,[3–5] including cases associated with liver cirrhosis secondary to chronic hepatitis B[3] or unknown etiology[4] or with noncirrhotic chronic hepatitis C.[5] However, to the best of our knowledge, we describe the first case in association with liver transplantation. Even though this association could be coincidental, it is possible that post-transplant immunosuppression may have played a role in the development of malignancy. Further studies are needed to determine whether immunosuppression is effectively a risk factor for this uncommon malignancy.

In conclusion, this case demonstrates that alpha-fetoprotein producing gastric cancer must be considered in the differential diagnosis of elevated alpha-fetoprotein, particularly in the presence of associated dyspeptic symptoms. Although the incidence of this malignancy does not appear to be increased in patients with liver disease, hepatologists should be aware of its existence when following these patients due to frequent use of alpha-fetoprotein in this setting.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Wong RJ, Ahmed A, Gish RG. Elevated alpha-fetoprotein: differential diagnosis – hepatocellular carcinoma and other disorders. Clin Liver Dis. 2015;19:309–23.
2. Baek SK, Han SW, Oh DY, Im SA, Kim TY, Bang YJ. Clinicopathologic characteristics and treatment outcomes of hepatoid adenocarcinoma of the stomach, a rare but unique subtype of gastric cancer. BMC Gastroenterol. 2011;11:56.
3. Gallo P, Gentilucci UV, Taffon C, Galati G, De Vicentis A, Muda AO, et al. A challenging alfa-fetoprotein in a cirrhotic patient. Acta Gastroenterol Belg. 2014;77:66–7.
4. Umekawa Y, Watanabe M, Ikeda S, Fukumoto S, Hirakawa H, Shimada Y. Alpha-fetoprotein-producing early gastric cancer accompanying liver cirrhosis: a case report. J Gastroenterol. 1994;29:66–70.
5. Sohda T, Tomioka Y, Inomata S, Morita I, Eguchi K, Aoyagi K, et al. Alpha-fetoprotein (AFP)- and des-gamma-carboxy prothrombin (DCP)-producing adenocarcinoma of the stomach with liver metastasis in a patient with chronic hepatitis C. Intern Med. 2005;44:294–8.

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Tofacitinib for the treatment of inflammatory condition of the ileoanal pouch refractory to infliximab

Tofacitinib para el tratamiento de la afección inflamatoria del reservorio refractaria a infliximab

Up to 5%–15% of patients with ulcerative colitis (UC) require surgical treatment, with proctocolectomy with ileoanal pouch being the technique of choice. Pouchitis is its most common complication. Pouchitis includes a range of situations that remain ill-defined, ranging from classical pouchitis to Crohn's disease of the pouch.[1] The increasing incidence of this complication and the risks and loss of quality of life associated with the extraction of the pouch have increased interest in finding therapeutic alternatives in this situation.[2] We present the case of a patient with a pouch and chronic iron deficiency anaemia secondary to patchy inflammatory involvement of the pouch who received rescue treatment with tofacitinib after failure of treatment with TNF inhibitors.

This was a 48-year-old male patient with left-sided UC of 18 years of evolution requiring proctocolectomy with pouch due to refractory disease. Five years after surgery, there was onset of rectorrhagia and severe iron deficiency anaemia. Hence, a pouchoscopy revealed deep, friable ulcers in the pouch, causing stenosis of the access to afferent and efferent loops. Other causes of anaemia were ruled out (negative coeliac disease study, gastroscopy and capsule endoscopy without findings) and, given the characteristics of the lesions, it was decided to start treatment with oral budesonide with no clinical response, so combined treatment with infliximab (5 mg/kg every eight weeks) and azathioprine was started. Despite this, the patient required high-dose intravenous iron repletion and even periodic blood transfusion, with no endoscopic improvement, so infliximab was intensified to every six weeks. However, in successive endoscopies, ulcerated stenosis of the access orifice to afferent and efferent loops persisted, so local injection of infliximab and antibiotic therapy with metronidazole 500 mg/every eight hours for four weeks was administered without success. Given the patient's reluctance to have the pouch removed and the lack of therapeutic alternatives in this situation, treatment with infliximab was maintained for 10 years, with a progressive increase in the requirement for

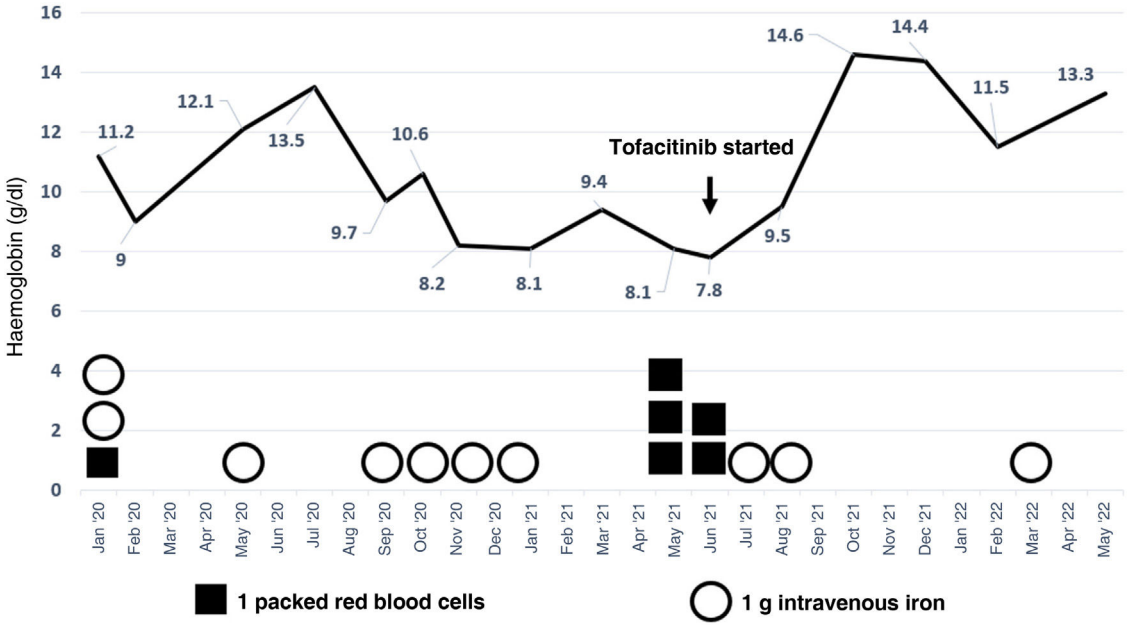

Figure 1 Evolution of haemoglobin values (g/dl) in the last two years (January 2020–May-2022).

intravenous iron therapy and blood transfusions in the last two years (Fig. 1).

After another pouchoscopy in which no changes were observed, the anaemia was exacerbated with haemoglobin of 7 g/dl. Resection of the pouch and end ileostomy were considered, but given the patient's reluctance to undergo further surgery, rescue treatment with tofacitinib was agreed on, following the guidelines approved for UC (10 mg every 12 h for eight weeks, followed by 5 mg/12 h), with the patient presenting with frank clinical improvement (only some isolated rectorrhagia). At five months after the start of treatment, the anaemia resolved for the first time in the last 15 months (Fig. 1). At one year of follow-up, only 1 g of intravenous iron was required without transfusion support, although there was no endoscopic improvement.

Pouchitis occurs in up to 30% of patients with pouches. The therapeutic requirements of pouchitis are high, with up to 80% of patients requiring biologics and 15%–30%, surgery. However, TNF inhibitors have been shown to be effective in only half of patients with pouchitis and this, together with the desire of most patients to avoid having to have the pouch removed, requires a search for effective therapeutic alternatives. Although we do not have controlled clinical trials comparing the efficacy of tofacitinib (JAK1 and JAK3 inhibitor, approved for UC without response to conventional therapy) with other drugs, a recent meta-analysis equates it in efficacy to other biologic therapies in the treatment of UC. Additionally, high response rates to tofacitinib have been reported in patients with UC refractory to biologic treatment. In this scenario, the lack of controlled clinical trials in treating pouchitis has traditionally demanded the use of drugs that have been effective in ulcerative colitis, so the use of tofacitinib seems reasonable. To date, there are only nine cases of chronic refractory pouchitis treated with tofacitinib in the literature.[3–5]

References

1. Akiyama S, Ollech JE, Rai V, Glick LR, Yi Y, Traboulsi C, et al. Endoscopic Phenotype of the J Pouch in Patients With Inflammatory Bowel Disease: A New Classification for Pouch Outcomes. Clin Gastroenterol Hepatol. 2022;20:293–302.e9, http://dx.doi.org/10.1016/j.cgh.2021.02.010.
2. Shen B, Kochhar GS, Rubin DT, Kane SV, Navaneethan U, Bernstein CN, et al. Treatment of pouchitis, Crohn's disease, cuffitis, and other inflammatory disorders of the pouch: consensus guidelines from the International Ileal Pouch Consortium. Lancet Gastroenterol Hepatol. 2022;7:69–95, http://dx.doi.org/10.1016/S2468-1253(21)00214-4.
3. Dalal RS, Bains K, Marcus J, McClure EL, Allegretti JR. Tofacitinib for the Treatment of Pouch-Related Disorders: A Case Series. Inflamm Bowel Dis. 2022:izac147, http://dx.doi.org/10.1093/ibd/izac147. Epub ahead of print. PMID: 35792486.
4. Bauer CM, Barnes EL, Herfarth HH. Tofacitinib in the Treatment of Crohn's-Like Disease of the Pouch. Am J Gastroenterol. 2020;115:2116–7, http://dx.doi.org/10.14309/ajg.0000000000000801.
5. Okano S, Yoshimura N, Sako M, Takazoe M. A case of refractory chronic pouchitis successfully treated with tofacitinib. Clin J Gastroenterol. 2020;13:560–3, http://dx.doi.org/10.1007/s12328-020-01108-5.

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