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SCIENTIFIC LETTER

A challenging alpha-fetoprotein after liver transplantation

Un diagnóstico desafiante de alfafetoproteína elevada en paciente trasplantado hepático

To the Editor,

The most common causes for elevation of serum alpha-fetoprotein are hepatocellular carcinoma and germ cell tumors.[1] Less commonly, it may be associated with alpha-fetoprotein producing gastric cancer, a rare subtype of gastric adenocarcinoma (2–15%) that is usually diagnosed in advanced stage and typically associated with poor prognosis and higher rates of lymphatic and venous invasion, lymph node metastasis and liver metastasis than conventional gastric cancer. Accordingly, it is demonstrated that increased alpha-fetoprotein is associated with overall poorer prognosis in gastric adenocarcinoma.[2]

We report the case of a 39-year-old female with previous medical history of liver transplantation for alcoholic liver cirrhosis performed 4 years earlier, and abstinent since then, who was referred to our hospital for investigation of a progressive rise of serum alpha-fetoprotein (peak: 1100 mg/dL). Since liver transplantation, she had been under immunosuppression with tacrolimus without any signs of graft rejection. She referred asthenia, post-prandial fullness, nausea and vomiting. Abdominal computed tomography scan revealed gastric parietal thickening suggestive of malignancy (Fig. 1A) associated with peri-gastric lymphadenopathy and two hypodense liver nodules with 10 and 32 mm, both located in segment II and demonstrating peripheral hyperenhancement during arterial phase without evident washout during portal phase, suggestive of liver metastasis. Liver allograft did not display signs of cirrhotic transformation. Therefore, she undergone upper digestive endoscopy that showed a large ulcerated polypoid lesion involving the smaller curvature along body and antrum (Fig. 1B). Biopsies of the lesion were performed and histopathological examination revealed adenocarcinoma (Fig. 1C), with strong immunohistochemical positivity for alpha-fetoprotein (Fig. 1D), consistent with alpha-fetoprotein producing gastric cancer (stage IV). After multidisciplinary discussion, palliative chemother-

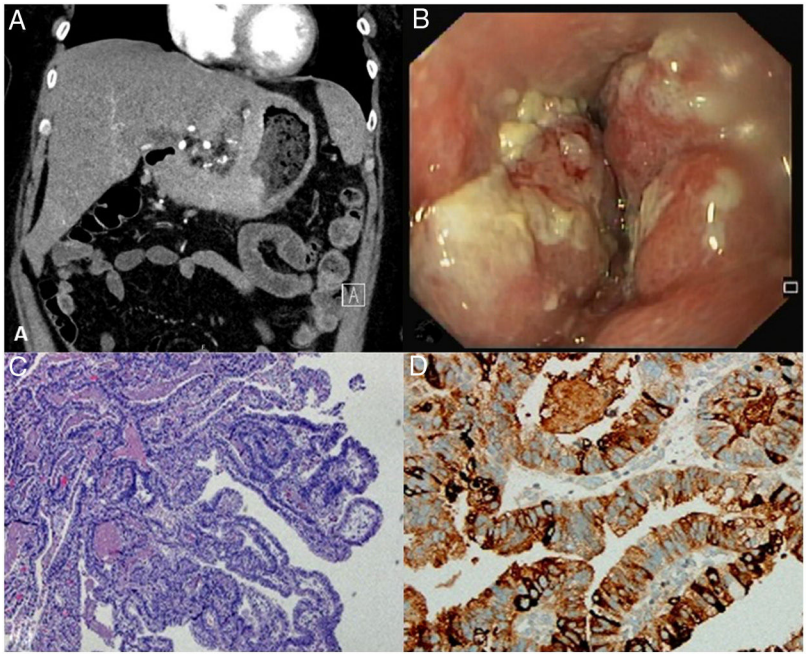

Figure 1 (A) Abdominal computed tomography scan revealed gastric parietal thickening and a luminal protrusion suggestive of malignancy. (B) Esophagogastroduodenoscopy revealed a large protruding ulcerated lesion extending along the smaller curvature of antrum and body. (C) Histopathological examination revealed a solid neoplasia forming glandular structures consistent with adenocarcinoma. (D) Immunohistochemistry showed strong positivity for alpha-fetoprotein in neoplastic cells.

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.

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

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

Hasta un 5-15% de los pacientes con colitis ulcerosa (CU) requieren tratamiento quirúrgico, siendo la proctocolectomía con reservorio ileoanal la técnica de elección. La afección inflamatoria del reservorio (AIR) es su complicación más frecuente. La AIR incluye un abanico de situaciones aún mal definidas que incluyen desde la reservoritis clásica a la enfermedad de Crohn del reservorio[1]. La incidencia creciente de esta complicación y los riesgos y pérdida de calidad de vida asociados a la extracción del reservorio han incrementado el interés por hallar alternativas terapéuticas en esta situación[2]. Presentamos el caso de un paciente portador de reservorio con anemia ferropénica crónica secundaria a afectación inflamatoria parcheada del reservorio en el que se realizó tratamiento de rescate con tofacitinib tras fracaso al tratamiento con anti-TNF.

Varón de 48 años con CU izquierda de 18 años de evolución que requirió proctocolectomía con reservorio por enfermedad refractaria. A los 5 años de la cirugía inicia rectorragias y anemia ferropénica grave por lo que se realiza una reservorioscopia que evidencia úlceras profundas y friables en el reservorio que subestenosan el acceso a asas aferente y eferente. Se descartaron otras causas de anemia (estudio de enfermedad celiaca negativo, gastroscopia y cápsula endoscópica sin hallazgos) y, dadas las características de las lesiones, se decidió iniciar tratamiento con budesonida oral sin obtenerse respuesta clínica, por lo que se inició tratamiento combinado con infliximab (5 mg/kg cada 8 semanas) y azatioprina. A pesar de ello, el paciente presentaba elevados requerimientos de hierro intravenoso e incluso transfusión sanguínea periódicos, sin evidenciarse mejoría endoscópica, por lo que se intensificó infliximab a cada 6 semanas. Sin embargo, en las sucesivas endoscopias persistía la estenosis ulcerada del orificio de acceso a asas aferente y eferente, realizándose inyección local de infliximab y antibioterapia con metronidazol 500 mg/cada 8 h durante 4 semanas, sin éxito. Dada la reticencia del paciente a la extracción del reservorio y la ausencia de alternativas terapéuticas en esta situación, se mantuvo el tratamiento con infliximab durante 10 años, con progresivo aumento de los requerimientos de ferroterapia intravenosa y trasfusiones sanguíneas en los dos últimos años (fig. 1).