etiologies (e.g., opportunistic infections); however, one must bear in mind that the risk of perforation has been reported to be as high as 20%.[1] Our case underlines some important clinical aspects and therapeutic quandaries when facing these situations, first, the importance of offending drug withdrawal and second, optimal steroid dose administration as recommended by the manufacturer, as well as a slow steroid tapering in order to avoid an early and, sometimes, steroid-refractory recurrence.[7,8] In cases of steroid-refractory colitis, infliximab therapy (5 mg/kg, usually a single dose), as a second-line treatment, should be considered.[9,10]

Financial support

None.

Ethical approval

Informed consent was obtained from the patient.

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Upper gastrointestinal bleeding due to an uncommon lymphoproliferative process☆

Hemorragia digestiva alta secundaria a un proceso linfoproliferativo infrecuente

Upper gastrointestinal bleeding is due to gastric tumours in 3% of cases,[1] most commonly gastric adenocarcinomas, which occur much more frequently than other types of tumours such as lymphomas. Whereas gastric lymphomas are generally B-cell tumours, mainly mucosa-associated lymphoid tissue (MALT) and large B-cell lymphomas, T-cell lymphomas are exceptional in this location.[2]

We present the case of an 86-year-old man who was admitted to our department for a 15-day-history of melaena, with no haemodynamic compromise. His personal history included ischaemic heart disease (acute myocardial infarction), and he was on dual antiplatelet therapy with acetyl salicylic acid and clopidogrel. He was also taking 40 mg of omeprazole daily as gastroprotective treatment. Physical examination was unremarkable, while laboratory tests showed moderate iron deficiency anaemia, with a haemoglobin result of 10.2 g/dL.

Gastroscopy detected a friable, ulcerated lesion with a submucosal appearance, 5 cm in diameter, located on the greater curvature of the lower gastric body. Biopsies pointed to a suspicion of gastric neoplasm. A chest-abdomen-pelvis computed tomography (CT) scan showed a tumour measuring 5.7 cm × 15 cm on the anterior wall of the gastric antrum. Lymphadenopathies detected in the greater omentum and aortopulmonary window were considered pathological due to their radiological characteristics, and because of an uptake similar to the primary mass revealed on subsequent scintigraphy (Fig. 1).

The histopathology report for the gastric mass biopsies described a mucosa with proliferation of atypical cells distributed in a sheet occupying the lamina propria. The nucleus–cytoplasm ratio was high; hyperchromatic, irregular nuclei were identified, as well as frequent mitotic figures, many atypical. Immunohistochemistry studies were positive for CD3, CD4, CD43 and CD79a, and negative for CD19, CD20, CD30, CD56 and CD2. The Ki67 proliferation index was very high, at close to 100% cellularity. Neither *Helicobacter pylori* nor Epstein–Barr virus was detected. Overall, the immunohistochemistry study was consistent with primary gastric T-cell lymphoma.

☆ Please cite this article as: Rodríguez-Martín L, Aparicio Cabezudo M, Álvarez-Cuenllas B, Linares Torres P, Hernando Martín M, López-Cuesta D, et al. Hemorragia digestiva alta secundaria a un proceso linfoproliferativo infrecuente. Gastroenterol Hepatol (Engl Ed). 2016;39:279–281.

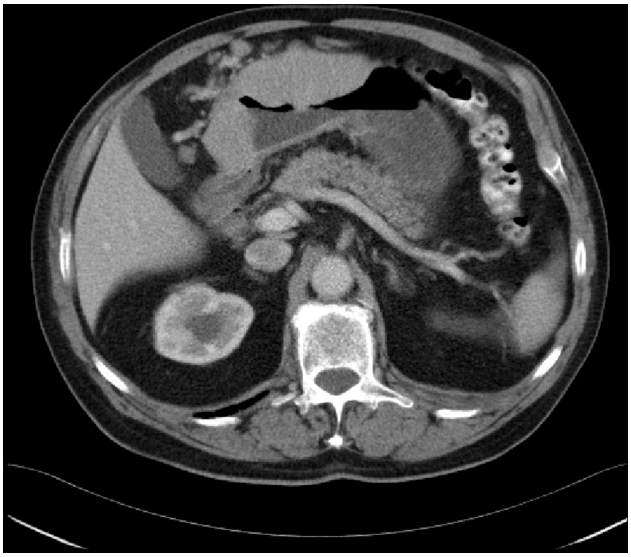

Figure 1 Abdominal computer tomography image showing a large mass on the anterior side of the stomach.

The patient was referred to the haematology department of our hospital, where bone marrow aspiration and bone marrow and peripheral blood flow cytometry were performed. Serology for human T-cell lymphotropic virus type 1 (HTLV-1) was not determined. The patient was classified as stage IIIE according to the Ann Arbor staging system. B-cell chronic lymphocytic leukaemia was detected as an incidental finding, with no data suggesting T-cell clonality.

Given his good baseline status (Eastern Cooperative Oncology Group (ECOG) 1), the patient underwent 5 cycles of a CEOP (cyclophosphamide, etoposide, vincristine and prednisone) chemotherapy regimen. However, despite oral iron treatment, anaemia was detected after the last cycle. Gastroscopy revealed progression of the gastric mass. Second-line treatment with gemcitabine and oxaliplatin was prescribed, but discontinued after 2 cycles due to neurological toxicity and deterioration in the patient's general condition. Now, 7 months after diagnosis, he is receiving palliative care.

Extranodal lymphomas, which make up around 40% of all lymphomas, are most often located in the gastrointestinal tract, especially at gastric level, where they account for 3% of primary neoplasms.[2,3] Most frequently—96% of cases—these are B-cell tumours, mainly MALT lymphoma, large-cell lymphoma, follicular lymphoma and Hodgkin lymphoma.

Clinical presentation is usually non-specific—fundamentally epigastric pain, vomiting and constitutional symptoms. The presence of haematemesis or melaena as a manifestation of gastrointestinal haemorrhage is rare, although occult gastrointestinal bleeding is observed in up to 19% of cases.[1,4] There is no pathognomonic endoscopic image, and a mass, a lesion suggestive of peptic ulcer or fold thickening may be observed.[5] Multifocal distribution is common, so it is important to take multiple biopsies, even from apparently unaffected areas.[6] Differential diagnosis based on immunohistochemistry should encompass the main gastric lymphomas. In our patient, the positive CD3 and negative CD19 and CD20 results enabled a B-lymphocyte neoplasm to be ruled out and confirmed the diagnosis of a T-cell lymphoproliferative process.

Table 1 summarises the main characteristics of gastric lymphomas that facilitate their differential diagnosis. T-cell lymphomas are a heterogeneous group of tumours that are difficult to diagnose, given their immunohistochemical

Table 1 Differential diagnosis of the most common gastric lymphomas.

Lymphoma	Diffuse large cell	MALT	Follicular	Hodgkin	Peripheral
Percent (%)	38–48	45–59	0.5–2	1	1.5–4
Cell type	B	B	B	B	T
Genetic alterations	BCL-2, BCL-6, c-MYC t(14;18),	Trisomy 3, BCL-2, t(14;18), t(11;18), t(1;14), t(3;14)	t(14;18), t(2;18), t(18;22)	BCL-2, t(11;14)	t(7;14), t(11;14), t(14;14), inv(14)
Immunohistochemistry					
B-cell: CD19, CD20, CD22, CD79a	+	+	+	+ CD79−	
T-cell: CD2, CD3, CD5, CD7 CD5	− If +, more aggressive (10%)	−	−	− +	Variable. Most have loss of CD5/CD7
Immunoglobulins	Surface/cytoplasm	Surface	Surface		
Other	CD45, CD10, MUM1/IRF4 CD30 anaplastic	*H. pylori*+	CD10, CD23	Cyclin D1, CD10, CD49d, FMC7	CD4, CD8, CD103, CD45RO, TCR γ, TCR β

BCL-6: B-cell lymphoma 6 protein; c-MYC: myelocytomatosis oncogene homolog; FMC7: formation of mitochondrial complexes 1 homolog; inv: inversion; MALT: mucosa-associated lymphoma tissue; MUM1: multiple myeloma oncogene 1, also called IRF4: interferon regulatory factor 4; t: translocation; TCR: T-cell receptor.

variability, as even typical T-cell markers such as CD5 and CD7 may be absent.[7] This rare disease accounts for 1.5% of gastric lymphomas. *H. pylori* is not involved in its pathogenesis—as happens with more common lymphomas such as MALT—so eradication therapy is not an effective treatment option. Prognosis is generally poor due to the aggressiveness of the tumour and frequent relapses.[8]

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