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Recurrent gastrointestinal bleeding secondary to Dieulafoy's lesion successfully treated with endoscopic ultrasound-guided sclerosis☆

Hemorragia digestiva recurrente por lesión de Dieulafoy tratada con éxito mediante esclerosis guiada por ecoendoscopia

We present the case of an 82-year-old male who attended the Emergency Department with haematemesis and melaena. His previous medical history included hypertension, atrial fibrillation on anticoagulant therapy, an operated abdominal aortic aneurysm and a previous episode seven months earlier of upper gastrointestinal bleeding caused by a Dieulafoy lesion (DL) in the gastric fundus, which had been treated by sclerosis and placement of endoclips.

On arrival at our hospital, the patient had blood pressure of 85/50 mmHg, a heart rate of 120 bpm and haemoglobin of 6.5 g/dl, for which he was resuscitated with volume and transfusion of blood products. Subsequently, within the first 8 h of the patient arriving in the emergency department, a gastroscopy was performed in which a large clot was identified adhered to the greater curvature-fundus. It was not possible to clearly visualise the focus of the bleeding, but it was treated by injecting adrenaline and polidocanol through the clot. Another gastroscopy was performed the next day, showing a clot of less than 1 cm in size, as well as small adjacent punctate disruptions in the mucosa, probably corresponding to the sclerosis of the previous day and the endoclips placed in the previous admission. As there were no signs of re-bleeding and it was not possible to determine the exact point of origin of the bleeding, no treatment was carried out this time.

Back on the ward, the patient had melaena and anaemia once again. An endoscopic ultrasound (EUS) was performed in which a 2 mm calibre vessel was identified coming through the stomach wall at the level of the gastric fundus, highly compatible with Dieulafoy's lesion (Fig. 1). The lesion was sclerosed using an endoscopic ultrasound (EUS)-guided needle, injecting 0.5 mg of adrenaline and 2 cc of polidocanol, after which the flow at that level was seen to disappear. After that, over the next 6 months of follow-up, the patient had no external signs of bleeding and did not require further transfusions.

DL is a vascular malformation in the gastrointestinal tract, most commonly occurring in the stomach.[1–4] It consists of a submucosal artery that is histologically normal but follows a tortuous path and does not undergo normal branching, thereby becoming 10 times larger than the normal calibre of the capillaries in the gastrointestinal mucosa.[1,5–7]

Although uncommon, DL is important as a serious cause of upper gastrointestinal bleeding, representing approximately 1–2% of cases.[2,7] The aetiology and pathogenesis are unknown, although it typically affects adult males with multiple comorbidities, such as hypertension, diabetes mellitus and chronic kidney disease.[1,2] The use of NSAIDs has also been associated with DL, probably as a result of the ischaemia and atrophy they cause in the gastrointestinal mucosa, which makes bleeding in these patients more likely.[1,6,8]

DL can remain silent for years, starting in most cases as sudden-onset painless gastrointestinal bleeding, which can have high morbidity and mortality rates, and has a tendency to recur.[2,4,5,8]

Endoscopy is the diagnostic technique of choice in this disease.[4,8] However, in up to 33% of cases, repeat endoscopies are needed to reach a definitive diagnosis,[2,6,7] as DL can be difficult to detect if there is no active bleeding due to the small size of the lesion and the normal appearance of the surrounding mucosa.

From the point of view of treatment, endoscopy has proven to be a safe and effective technique, achieving

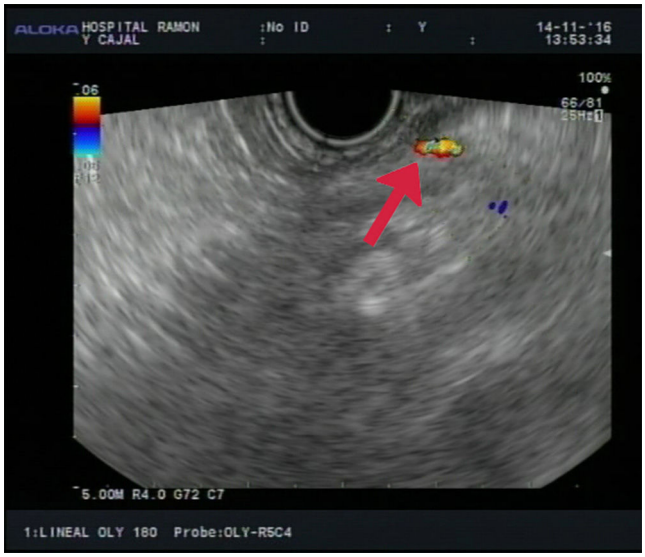

Figure 1 Endoscopy image. Vascular image crossing through the gastric wall compatible with Dieulafoy lesion.

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primary haemostasis in 90–100% of cases.[2] There are several endoscopic therapeutic options available,[2,5,6] with better results reported with mechanical methods, as well as with combination therapy compared to monotherapy.[2,4,8]

However, despite advances in endoscopy in this field having led to a dramatic reduction in the mortality rate from DL-related bleeding (from 80% to 8%), relegating surgery to the background,[4] the risk of re-bleeding in these patients is estimated to range from 9% to 40%.[2]

This makes the diagnosis and treatment of DL extremely challenging in a not insignificant number of cases, which explains the growing interest in the study of other minimally invasive techniques as alternatives to endoscopy, such as arteriography with selective embolisation[4,7] or EUS.[1,2,5,8]

In recent years, EUS has been singled out as a technological resource of great utility in the management of DL,[2,3,5,9] particularly in cases where endoscopy has previously failed. With Doppler ultrasound, the trajectory of the malformed submucosal vessel can be demarcated with great precision, and selective sclerosis performed on it by injecting substances such as adrenaline or ethanol.[3,4,9] Furthermore, Doppler ultrasound can also be used to confirm successful ablation of the lesion after treatment, using Doppler to demonstrate absence of flow in the malformed artery.[2,3]

Our case provides a clear example of the role of EUS in the management of recurrent DL-related bleeding, as this was the decisive technique in our patient in controlling DL refractory to endoscopic treatment.

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An unusual cause of acute liver failure: Tumour infiltration by neuroendocrine carcinoma☆

Una causa inusual de fallo hepático agudo: infiltración tumoral por carcinoma neuroendocrino

Acute liver failure (ALF) is a rare condition with rapid onset and a high mortality rate characterised by coagulopathy, with INR > 1.5, and any degree of encephalopathy in a patient without prior liver disease.[1] Some of the most common causes are toxicity to drugs such as paracetamol, viral hepatitis, autoimmune hepatitis, Wilson's disease and ischaemic hepatitis.[1,2] Although haematogenous spread of solid tumours often affects the liver, ALF caused by the spread of cancer is very uncommon, with few cases reported in the literature. In a study conducted in the United Kingdom over 18 years (1978–1995), this situation was documented in only 0.44% of patients, with cancers of haematological origin being the most common cause.[3] In another more recent American multicentre study conducted by the Acute Liver Failure Study Group, which reviewed 1910 patients with ALF from 1998 to 2012, only 27 patients (1.4%) were identified with ALF secondary to tumour infiltration.[4]

The liver parenchyma is replaced by cancer cells which propagate sinusoidally, causing hepatocellular ischaemia and releasing pro-inflammatory cytokines. The increase in AST, LDH and hepatomegaly support this hypothesis; fulminant hepatic failure triggers multiple-organ failure, with a high mortality rate of 60–80%.[5] The confirmation diagnosis is made by liver biopsy and in most cases is performed *postmortem*.[3,6–9]

We present the case of a 66-year-old male shepherd, ex-smoker (40 pack/years), moderate drinker, who had chronic bronchitis with home CPAP and hypertriglyceridemia. He had a ten-day history of upper respiratory tract infection, with no pyrexia, treated with levofloxacin and prednisone, with abdominal distension, meteorism and oliguria. He did not report the use of drugs of abuse or other hepatotoxic

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