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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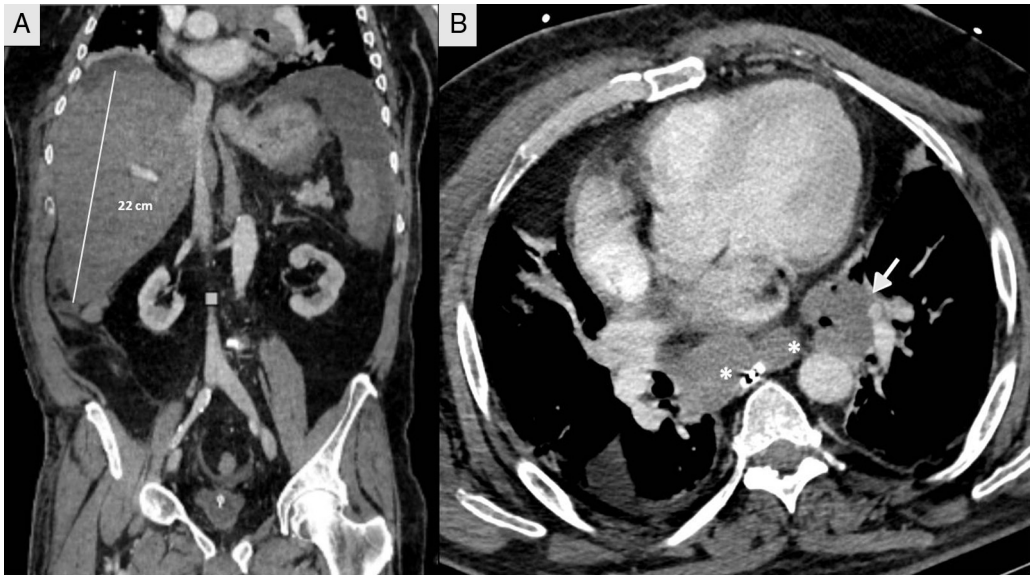

Figure 1 Thoracic/abdominal multi-slice CT with IV contrast. (A) Axial section of the thorax showing enlarged lymph nodes in the subcarinal region (*) and left hilar region (white arrow). (B) Coronal section showing a large homogeneous hepatomegaly.

substances. He had no general symptoms, weight loss or family history of liver disease. On admission, a chest-abdomen CT was performed, showing marked hepatomegaly with an anteroposterior axis in the axial section of 30 cm and 22 cm in the coronal section, no focal lesions identified, with normal biliary tract and large subcarinal mediastinal masses of enlarged lymph nodes (Fig. 1). He was admitted to the internal medicine department initially with the following blood results: blood glucose 104 mg/dl, creatinine 1.13 mg/dl, urea 67 mg/dl, sodium 142 mmol/l, potassium 4 mmol/l, AST 266 U/l, ALT 457 U/l, alkaline phosphatase 174 U/l, GGT 1033 U/l, total bilirubin 2.5 mg/dl, amylase 87 U/l; normal blood count, coagulation: PT 79%, INR 1.17, derived fibrinogen 550 mg/dl; arterial blood gases: pH 7.45, pCO_2 35 mmHg, pO_2 71 mmHg, bicarbonate 24.5 mM/l and base excess +1.6 mM/l. The patient made poor clinical progress, with liver failure (AST 4791 U/l, ALT 2301 U/l, alkaline phosphatase 286 U/l, GGT 739 U/l, total bilirubin 12.2 mg/dl, LDH 5470 U/l, ammonium 106 mmol/l, coagulopathy with PT 28% and INR 2.79), acute oliguric renal failure (creatinine 3.8 mg/dl, urea 105 mg/dl), amylase 165 U/l, lipase 185 U/l, CK 1866 U/l, potassium 7 mmol/l, blood glucose 34 mg/dl and metabolic acidosis with pH 7, lactate 13 mmol/l and base excess −20 mM/l. Given his poor progress, he was transferred to the intensive care unit (ICU), requiring orotracheal intubation and connection to mechanical ventilation due to acute respiratory failure, haemodynamic instability and neurological deterioration in relation to metabolic encephalopathy. On examination, the patient's abdomen was very distended, tympanic and diffusely painful on palpation. Viral serology for hepatitis A, B, C and E was negative. Cytomegalovirus, syphilis, Epstein–Barr virus and herpes simplex virus were all negative. Serology for *Leptospira* was negative. Monoclonal antibody CA 72.4 and squamous cell-associated antigen negative. Autoimmunity studies (rheumatoid factor, ANA, AMA, ANCA and anti-smooth muscle) negative. Blood and urine cultures were negative. Leucocytes 19,300 without marked neutrophilia and procalcitonin 4.47 ng/ml. The patient suddenly went into distributive shock refractory to the usual supportive treatment including vasopressor drugs, high-dose corticosteroids, continuous renal replacement techniques and empirical treatment with meropenem. He died 12 h after being admitted to ICU. A post-mortem examination was carried out and the Pathology report confirmed the proliferation in the liver of chaotically distributed nested tumours which had surrounded and destroyed portal spaces and central veins, widening the sinusoids, trapping the hepatocytes and replacing them with fibrosis (Fig. 2). No masses or obstructions of infra-or suprahepatic vascular structures were identified. Hilum appeared normal. Mediastinal masses of enlarged lymph nodes of tumour-like appearance; subcarinal of 6 cm × 4 cm, bilateral para-tracheal of 8 cm × 3 cm and para-aortic on the anterior side of the arch of 4 cm × 3 cm. There was no compression of venous circulation in this area. Growth of the same type of cells was also observed in the mediastinal and para-aortic lymph nodes. Immunohistochemical study (Fig. 2) was positive for chromogranin, CD56, PGP9.5 and focally with weak intensity for CK7, concluding that it was a WHO grade 2 of 3, large cell neuroendocrine carcinoma (2 mitosis/10 high power field [HPF] and Ki-67 of 5%), with massive infiltration of the liver and mediastinal lymphadenopathy, without identifying the primary lesion.

Neuroendocrine tumours are a very heterogeneous group of tumours that produce hormones and can arise in any organ. Within this type of cancer, neuroendocrine carcinomas are poorly differentiated tumours with a high rate of proliferation and very aggressive behaviour that tend to already have metastasis at the time of diagnosis. Some 13% begin with unknown primary tumour[9] and the most common location is the gastrointestinal tract. The Ki-67 index classifies them histologically, grade 2 tumours being those with a Ki-67 index of 3–20%. Other indicators of poor prognosis are high levels of chromogranin A (CGA) and the mitotic index.[10]