Let's take a look at drinks, but also at solid foods

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Question

A 62-year-old Caucasian patient was admitted to the hospital for the management of alcohol dependence (approximately 280g of ethanol per day for the past 10 years). He had no other relevant medical history. He reported asthenia, physical deconditioning and severe inappetence with a marked decrease of food intake in the past months, eating a maximum of one meal per day. On clinical examination, there were significant oedema of the lower limbs, marked conjunctival jaundice and low brachial perimeter compatible with myopenia (26 cm). The results of the blood test were as follows: total bilirubin 7.2 mg/dL (direct 6.4 mg/dL), INR 1.31, CRP 25.2 mg/dL, albumin 31 g/L, prealbumin 0.13 g/L, ASAT 251 U/L, ALAT 77 U/L, gamma-GT 3718 U/L, alkaline phosphatases 374 U/L, ferritin 3777 µg/L (transferrin saturation 85%), triglycerides 344 mg/dL. IgG, IgA, IgM were normal. Platelets and neutrophils were normal. Viral serologies B and C were negative. Ultrasonography showed a hyperechoic liver without bile duct dilatation. Liver elasticity was high (32.2 kPa), as well as controlled attenuation parameter or CAP (342 dB/m).

Due to significant cholestasis, hepatocellular insufficiency and high liver elasticity, we measured the hepatic venous pressure gradient (9 mmHg) and performed a transvenous liver biopsy (Fig 1A-D).

What is your diagnosis? What do you recommend?

On liver biopsy (Fig. 1A), severe steatosis was readily identifiable, but unusually in ALD, portal inflammatory infiltration was also noted (Fig 1C). Contrasting with the results of elastometry, the fibrosis stage was only F2 (Fig. 1B). At higher magnification, cholestasis, ductular reaction and neutrophilic infiltration near the bile ductules were observed (Fig. 2A). Immunohistochemistry for keratin 7 evidenced intact interlobular bile ducts, ductular reaction and scattered keratin 7 positive hepatocytes dispersed throughout the lobule (Figure 2B). There were also some ballooned hepatocytes, discrete lobular inflammation and microvesicular steatosis but no satellitosis.

We retained the diagnosis of cholestatic steatohepatitis due to the synergistic effect of excess alcohol intake and protein energy malnutrition. Similar histological findings (steatosis, portal inflammation with neutrophils and macrophages, cholestasis, ductular reaction and periportal fibrosis) have been described in patients with intestinal failure-associated liver disease (IFALD) (1) or after bariatric surgery (2). These aspects are not pathognomonic and can be seen in other situations.

The exact pathophysiological mechanisms remain unclear, but seem to be related to rapid weight loss, protein energy malnutrition, high circulating levels of free fatty acids, sometimes combined with parenteral nutrition solutions, injured and hyperpermeable intestine with gut microbiota alteration and bacterial overgrowth as well as changes in secondary bile acids (2,3). Alcohol may play an additional role in the pathophysiology of liver damage in this case, as in some cases following bariatric surgery (2,3).

At the 3-month follow-up, after nutritional intervention and alcohol abstinence, we observed clinical improvement, disappearance of the hyper-echogenic liver and complete normalisation of all laboratory values. The patient maintains long-term abstinence from alcohol and had regained his appetite. An elastography check requested two years later showed normal liver elasticity (5.2 kPa) and normal CAP value (200 dB/m).

We therefore conclude that the combination of alcohol intake and protein energy malnutrition may explain the cholestatic steatohepatitis presenting as icteric-oedematous decompensation mimicking decompensated cirrhosis, even though the degree of fibrosis was moderate. This underlines the role of nutritional assessment and management in liver disease.

References

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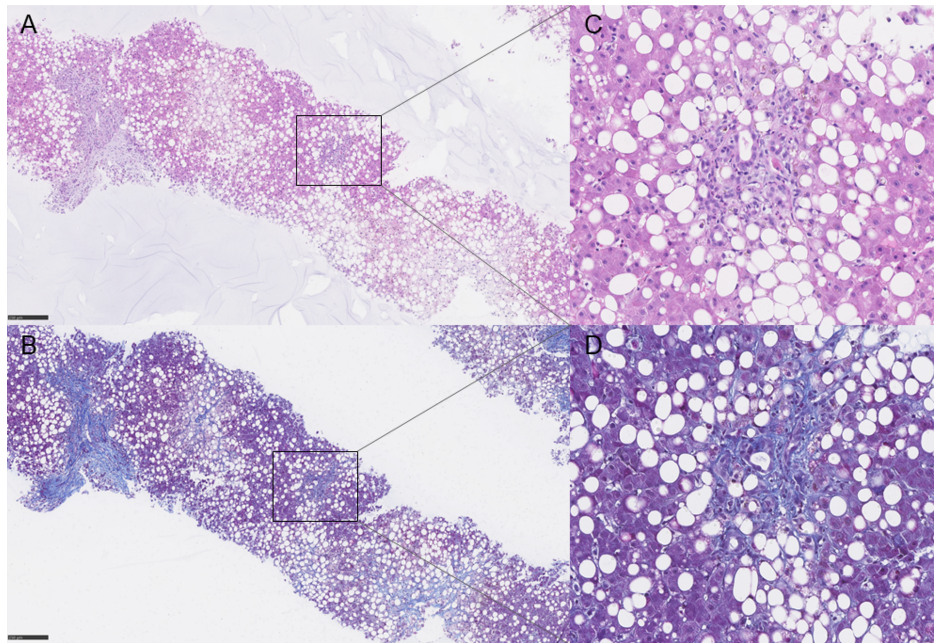

Figure 1. — Sections of liver biopsy at low magnification (10x) with haematoxylin and eosin (A) and trichrome blue (B) stainings. Severe steatosis, portal inflammation and absence of cirrhosis were noted. Inserts with 40x magnification provide better visibility of one portal tract (C,D). Scale bar: 250 µm.

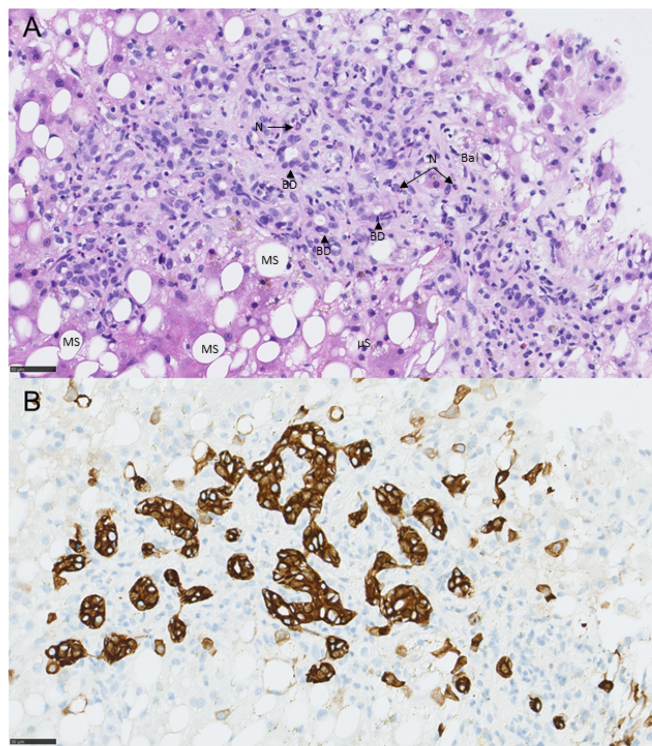

Figure 2. — The high-magnification (50x) liver biopsy stained with haematoxylin and eosin (A) shows predominant macrovesicular steatosis (MS) and inflammatory infiltration with some neutrophils (N) with the nucleus divided into three parts, near the bile ductules (cholangiolitis). Hepatocyte microvesicular steatosis (µS) and ballooning (Bal) are also noted. Keratin 7 staining reveals biliary epithelium with cholestatic injury and bile duct reaction (B). This type of injury is unusual in alcohol-related liver disease and may be compatible with cholestatic steatohepatitis associated with malnutrition. Bile ducts: BD. Scale bar: 50 µm.