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Case Report

Unusual case of liver cirrhosis presenting as a mass compressing the inferior vena cava: A case report☆,☆☆

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ABSTRACT

Liver cirrhosis is a significant global health burden, accounting for approximately 2 million deaths per year worldwide. The underlying etiologies of cirrhosis include viral hepatitis (hepatitis B, C, and D), toxins (such as alcohol and drugs), autoimmune diseases, cholestatic conditions (including primary biliary cholangitis and primary sclerosing cholangitis), vascular disorders (such as Budd-Chiari syndrome, sinusoidal obstruction syndrome, and cardiac cirrhosis), and metabolic disorders (including hemochromatosis, nonalcoholic steatohepatitis, and alpha-1 antitrypsin deficiency). Patients with liver cirrhosis typically present with symptoms such as jaundice, scleral icterus, nausea, and vomiting, accompanied by abnormal liver enzyme levels. Other defining features include spider angiomas, caput medusa, and esophageal and/or rectal varices. Abdominal imaging often reveals fibrotic changes within the liver.

In this article, we present a case of a 38-year-old female presenting with signs and symptoms of cirrhosis, with subsequent imaging revealing a Porta hepatis mass compressing the inferior vena cava (IVC). The patient underwent a biopsy consistent with liver cirrhosis. This case is unique in the presentation of her liver cirrhosis as a compressive mass rather than the usual fibrotic changes within the liver parenchyma.

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Introduction

Chronic liver disease accounts for approximately 2 million deaths each year [1]. The etiologies include viral hepatitis (HBV and HCV), alcoholic steatohepatitis, nonalcoholic steatohepatitis (NASH), as well as autoimmune and genetic diseases [1]. A common complication of chronic liver disease is eventual fibrosis, characterized by dense fibrotic septa within the parenchyma and the collapse of liver structures, which together cause distortion of hepatic vasculature [2]. Ultimately, this distortion is referred to as cirrhosis, resulting in portal hypertension and hepatic synthetic dysfunction.

Hepatocellular carcinoma (HCC) is a common complication of liver cirrhosis, preferentially metastasizing to the lungs, intraabdominal lymph nodes, bone, and vascular structures including the portal system, inferior vena cava (IVC), and right atrium [4]. Prior studies have shown that liver cirrhosis reduces the risk of liver metastases. One theory is related to abnormal hemodynamic blood flow in patients with portal hypertension.

Another explanation is the higher concentration of metalloproteinase inhibitors [3]. Tumor cells use collagenases for local invasion of tissues, and with collagenases inhibited by metalloproteinase inhibitors, fewer tumor cells are able to metastasize [3]. Another possible explanation is the reduced liver-specific lectins. These lectins recognize various glycoproteins on tumor cells, and without them, tumor cells cannot adhere to liver cells [3].

Case report

Patient is a 38-year-old female with a past medical history of anxiety, depression, hypertension, and alcohol use disorder presents to a community hospital with a 6-week history of nausea, vomiting, and bilateral abdominal pain located in the upper left and right quadrants. Since then, she has experienced increasing abdominal pain and pressure along with abdominal swelling. The patient also reports 3 weeks of night sweats and a yellow tint in her eyes and skin. Over the past 2 weeks, she has had decreased stool and urine output, as well as reduced oral intake due to nausea. She saw her primary care physician, who prescribed Ondansetron for the nausea, which did not resolve her symptoms. She was advised to go to the emergency department if her symptoms persisted. The patient denies similar symptoms in the past and states that the persistence of symptoms prompted her to come to the hospital for further evaluation.

Upon admission, the patient was evaluated and found to have severe scleral icterus and jaundice. On physical exam, patient endorsed tenderness to palpation in the upper left and middle quadrants of her abdomen. Laboratory testing revealed elevated liver enzymes listed below:

- Total Bilirubin of 23.2 mg/dL (Reference 0-1.0).
- Direct Bilirubin of 18.9 mg/dL (Reference 0-0.3).
- Indirect Bilirubin of 7.6 mg/dL (Reference 0-1.1).
- AST at 232 U/L (Reference 15-37).
- ALT at 102 U/L (Reference 10-49).

The patient received a CT abdomen and pelvis with IV contrast, which revealed extensive malignant tumor within the liver, ascites, and streaky changes involving the abdominal fat. Peritoneal carcinomatosis could not be ruled out. There was also prominent submucosal fat involving most of the colon—a finding seen in chronic inflammatory bowel disease (Fig. 3). Radiology recommended a triple-phase CT of the liver to further evaluate the degree of liver pathology. The patient underwent the recommended imaging, which demonstrated hepatic steatosis and hepatomegaly as well as multiple geographic and round to ovoid areas of pronounced hypodensity. The largest area involved the entirety of the caudate lobe, which was markedly enlarged. Other foci, such as those in the periphery of the right lobe, were rounder and more akin to fluid collections (Fig. 4). The hepatic vessels were patent. Overall, the findings suggested fulminant hepatitis, with small hypodense lesions possibly representing developing fluid collections, such as abscesses. No evidence of malignancy was found.

The patient was recommended to have a biopsy of the liver, which was performed on a peripheral lesion from the right hepatic lobe. The biopsy revealed liver cirrhosis, macrovesicular steatosis, and severe reactive changes, including feathery degeneration (Fig. 1). With these findings, Gastroenterology was consulted and recommended an MRCP. The patient underwent the recommended imaging, which revealed moderate intraperitoneal free fluid and a porta hepatis mass measuring 10.3 × 5.7 cm, suspicious for cholangiocarcinoma, noncalcific gallstone, polyp, metastatic disease, or hepatocellular carcinoma.

Blood tests for tumor markers were also conducted, revealing the following results:

- AFP: normal at 2.8 ng/mL (Reference 0-8.3).
- CEA: normal at 1.89 ng/mL (Reference 0-5).
- CA 15-3: elevated at 27.6 U/mL (Reference 0-25).
- CA 19-9: elevated at 88 U/mL (Reference 0-35).
- CA 125: elevated at 1012 U/mL (Reference 0-38.1).
- HCG: normal at 2 mIU/mL (Reference 0-6).

Given these findings, the patient was recommended for liver transplant evaluation. Throughout the hospital stay, the patient developed worsening jaundice, bilateral lower extremity edema, dysphagia, and increasing bloating and tenderness to palpation of the abdomen. The transplant hospital evaluated the medical records and recommended further evaluation with a second liver biopsy from the porta hepatis mass as well as an ERCP.

The patient underwent an ERCP, which revealed no abnormalities. A second biopsy from the porta hepatis mass was also performed, which confirmed liver cirrhosis, macrovesicular steatosis, and severe reactive changes, including feathery degeneration (Fig. 2). Those findings were similar to the previous biopsy from the right hepatic lobe. After confirmation of liver cirrhosis with 2 separate biopsies from different locations, the transplant hospital was contacted and agreed to receive the patient for evaluation of liver transplantation. The patient was stabilized and transferred in stable medical condition.

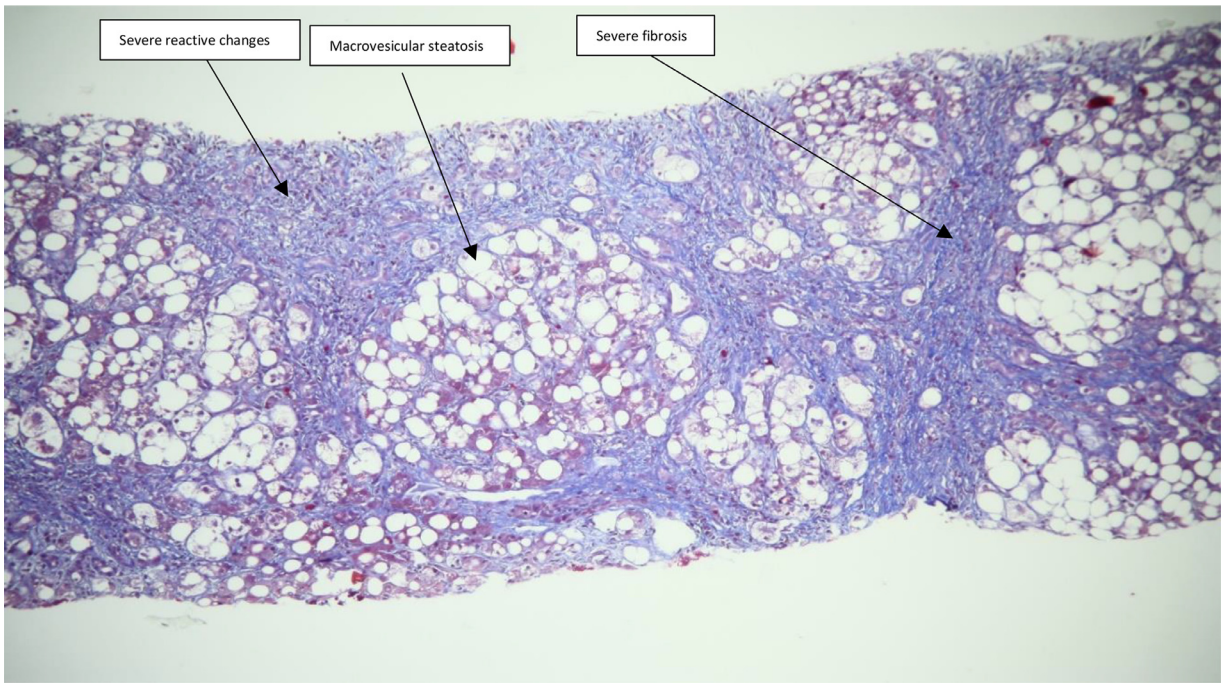

Fig. 1 – Trichrome stain of peripheral liver lesion revealing severe reactive changes, severe fibrosis with macrovesicular steatosis.

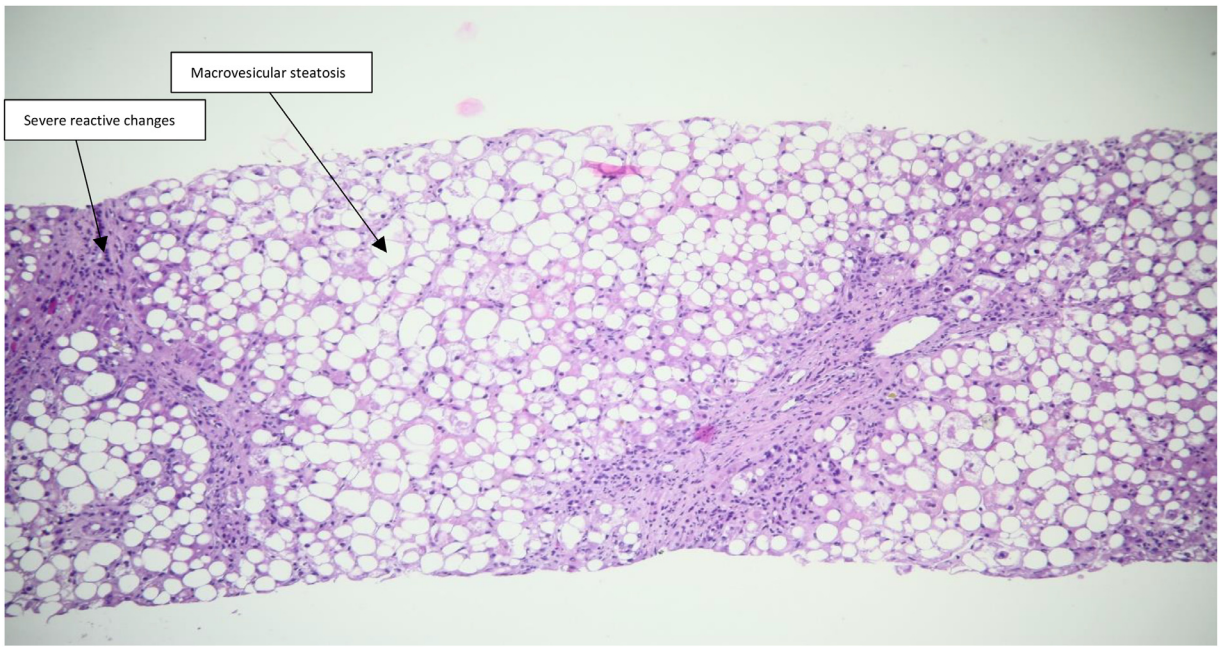

Fig. 2 – Hematoxylin and eosin stain of Porta Hepatis lesion biopsy revealing macrovesicular steatosis with severe reactive changes.

Discussion

Cirrhosis is characterized by fibrosis and nodule formation of the liver secondary to chronic injury. Multiple cells play a role in cirrhosis, including hepatic stellate cells (HSC), sinusoidal endothelial cells (SEC), and Kupffer cells (KC). HSCs line the walls of the liver sinusoids and function to store vitamin A. When exposed to inflammatory cytokines, HSCs transform into myofibroblasts, depositing collagen and resulting in fibrosis. SECs form the endothelial lining and have fenestrations that allow for fluid and nutrient exchange between the

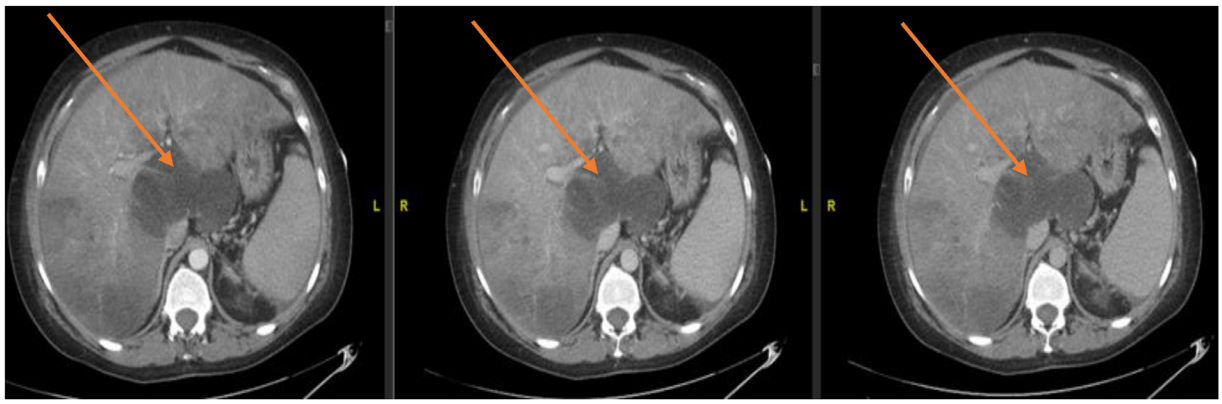

Fig. 3 – Axial CT in arterial phase (left), venous phase (middle), and delayed phase (right): Enlarged and hypodense caudate lobe without contrast enhancement. Round to ovoid hypodense lesions throughout the liver.

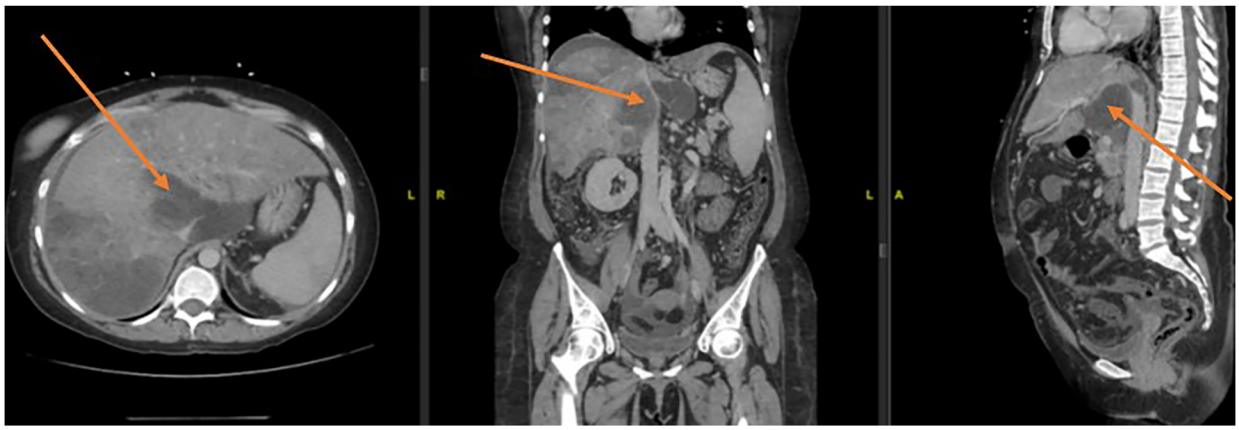

Fig. 4 – Axial (left), coronal (middle), and sagittal (right) CT abdomen and pelvis with contrast: Enlarged caudate lobe encircling and compressing inferior vena cava. Demonstration of diffuse hypodense tissue in the right hepatic lobe.

sinusoids and hepatocytes. Defenestration of the sinusoidal wall occurs secondary to chronic alcohol use, promoting perisinusoidal fibrosis. KCs are macrophages that line the wall of the sinusoids and release toxic mediators when exposed to foreign substances [5].

Intrahepatically, SECs synthesize nitric oxide and endothelin-1, which act on HSCs causing relaxation or contraction of the sinusoids. In patients with cirrhosis, there is an increase in endothelin-1 and a decrease in nitric oxide production, leading to increased intrahepatic vasoconstriction and causing portal hypertension [5]. Increased tumor markers in patients with liver cirrhosis are often considered unspecific, but the combined elevation of CA19-9 and CA125 is useful for identifying patients with advanced fibrosis or cirrhosis with high specificity [8]. For these reasons, the definitive diagnosis stems from a liver biopsy.

In alcoholic liver disease, histopathologic findings include micronodular cirrhosis, Mallory bodies, and fatty changes. Ischemia or other toxic injuries can cause centrilobular fibrosis; however, when hepatocytes are individually surrounded by collagenous stroma in the centrilobular region, the process is more likely due to alcohol [6]. Features of alcoholic cirrhosis include centrilobular sclerosis, pericellular, perivenular fibrosis, paucity of inflammation, and central-central or central-portal prominent bridging [6].

Radiologically, the presence of a large caudate lobe is highly suggestive of cirrhosis caused by alcohol abuse. The frequency of visualization of the right posterior hepatic notch in patients with alcoholic cirrhosis is significantly greater than in patients with viral cirrhosis [7]. The patient presented in this case had an alcohol use disorder, with pathologic findings revealing liver cirrhosis on 2 biopsies obtained from the right hepatic lobe and the porta hepatis mass. Although malignancy was high on the differential, it was subsequently ruled out as the tumor markers are nonspecific during liver cirrhosis. Liver cirrhosis does not normally present as a compressive mass, making this a unique scenario possibly related to long-term alcohol use.

Conclusion

Although alcohol consumption can cause liver cirrhosis, it is usually associated with caudate lobe enlargement and the

presence of a right posterior hepatic notch. The presence of an obstructive porta hepatis mass is a novel finding and can be seen in patients with long-term alcohol use. More research needs to be done and more cases need to be published to establish a clear association between alcohol use and the presence of an obstructive IVC porta hepatis mass.

Learning points

- Cirrhosis normally presents as multiple lesions scattered throughout the liver; in this report we present a case where cirrhosis is presenting as a distinct compressive mass resembling a tumor.
- Management is similar to alcoholic cirrhosis, and definitive treatment is liver transplantation.

Patient consent

This letter serves as a patient consent statement. This case report will be published after confirming that written, informed consent for publication of this case was obtained from the patient. The consent form was also submitted and approved by the hospital IRB.

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