H71 Alcoholic Hepatomegaly: A Case Report and Review of the Literature

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Learning Overview: After attending this presentation, attendees will be familiar with the pathologic features of alcoholic hepatitis and fatal alcoholic liver disease, in the absence of variceal bleeding, and will review the etiology of fulminant jaundice.

Impact on the Forensic Science Community: This presentation will impact the forensic science community by assisting in the recognition of fatal and non-fatal features of acute and chronic alcoholism, in particular hepatomegaly and alcoholic hepatitis, in the absence of ascites or esophageal variceal bleeding.

Excessive, prolonged alcohol intake has been shown to cause a variety of hepatic changes, including steatosis, inflammation, and fibrosis; inflammation may result in the development of alcoholic hepatitis in persons with a genetic predisposition.[1] Sequelae include resolution, hepatomegaly, cirrhosis, and/or progression to hepatocellular carcinoma. A host of systemic complications may include hiccups, abdominal pain, splenomegaly with thrombocytopenia, and portal hypertension leading to ascites and esophageal varices. Alcoholic liver damage may be recognized at gross autopsy from hepatomegaly, liver friability due to steatohepatitis, micronodular or macronodular cirrhosis, and jaundice. Although death is commonly due to variceal rupture and gastrointestinal bleeding, it may occur from alcoholic hepatitis alone.

Case History: A 36-year-old Caucasian male with a long-standing history of alcohol abuse was found dead in a supine position on the floor next to his bed, surrounded by empty alcohol bottles. Eight months earlier, he had presented to the emergency room with complaints of hiccups and right upper quadrant pain. He denied recent alcohol intake despite an odor of alcohol detected by emergency room personnel; the laboratory reported a blood alcohol concentration of 0.34g/dl. The patient's family stated that he was still drinking heavily on a regular basis. He admitted to consuming upward of 25 ounces (over 700g) of vodka per week. His liver panel results were consistent with liver damage (bilirubin 4.7mg/dL [direct 2.7mg/dL], AST 339 U/L, ALT 76 U/L, alkaline phosphatase 76 U/L) with thrombocytopenia (platelets 65K/uL). Despite impaired liver function, his albumin was normal at 4.1g/dL, and he did not have ascites; his kidney function appeared normal, without evidence for hepatorenal syndrome. An ultrasound examination showed hepatomegaly and fatty infiltration, characteristic indicators of alcoholic liver damage.[2] He was admitted to the hospital for a week with a diagnosis of alcoholic hepatitis, placed on withdrawal precautions, and subsequently discharged with a recommendation to abstain from alcohol. There was no further medical contact before he was found dead in his room.

At autopsy, he showed head-to-foot jaundice, including scleral icterus, and multiple purple contusions consistent with thrombocytopenia. The liver showed profound hepatomegaly (4475g/9.845lbs), about three times the normal weight, and measured 35cm x 26.5cm x 12cm, with both extreme steatosis and micronodular cirrhosis. Aspiration of the gallbladder resulted in 110cc of sludgy bile, although the biliary tree was patent. His spleen, at 582g, was also enlarged to about three times its normal weight. There was no ascites. Esophageal varices were unruptured, with no blood in the gastric pouch. Death was ascribed to fulminant liver failure, due to recurrent alcoholic hepatitis.

Discussion: The amount of alcohol intake required to initiate liver failure is approximately 100g-200g per day.[3] In this case, he admitted to an alcohol intake of 700 grams per day. Although this patient was only 36 years old at the time of death (below the average presenting age of 53 years for alcoholic hepatitis), he showed signs of fulminant liver failure superimposed on hepatic cirrhosis and recurrent alcoholic hepatitis. Acute fulminant jaundice may be due to alcoholic hepatitis, hepatotropic viruses, ischemic injury, drugs and toxins (e.g., acetaminophen, anticonvulsants, poisonous mushrooms), and unusual entities such as autoimmune hepatitis. He had no history of exposure to viral agents or hepatotoxic drugs (acetaminophen could not be ruled out). Although he never developed ascites or variceal bleeding, which are common causes of death from alcoholic cirrhosis, his autopsy findings correlated to his presenting symptoms on his prior hospital admission. His fatal liver disease resulted from many of the systemic alterations that have been described in association with alcoholic hepatitis.

Reference(s):

1. Yeluru A., Cuthbert J.A., Casey L., Mitchell M.C. (2016). Alcoholic Hepatitis: Risk Factors, Pathogenesis, and Approach to Treatment. *Alcoholism: Clinical and Experimental Research*, 40(2), pp246-255.
2. Fuster D., Garcia-Calvo X., Zuluaga P., et al (2018). Ultrasound Findings of Liver Damage in a Series of Patients Consecutively Admitted for Treatment of Alcohol Use Disorder. *Drug Alcohol Depend*, 190, pp195-199.
3. Anand A.C., Garg H.K. Approach to Clinical Syndrome of Jaundice and Encephalopathy in Tropics (2015). *J Clin Exp Hepatol*, 5(Suppl 1), pp S116–S130.

Alcoholic Hepatomegaly, Alcohol-Induced Liver Failure, Alcoholic Hepatitis

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