Hyperammonemic encephalopathy secondary to right-sided heart failure: A case report

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DISCUSSION

Congestive hepatopathy is the chronic, passive congestion within the liver primarily due to a cardiopulmonary condition, which can manifest in several ways including hyperbilirubinemia, ascites, hyperammonemia, and hepatomegaly¹. Hyperammonemic encephalopathy (HE) is rare in congestive heart disease, especially without known underlying hepatic pathology². This is a case of a 59-year-old male presenting with hyperammonemic encephalopathy in the setting of chronic heart failure and in the absence of acute liver failure or known cirrhosis history.

A 59-year-old male with chronic heart failure presented with acute encephalopathy, jaundice, ascites, and grade III asterixis.

Patient had not been prescribed any medications contributing to current presentation. CT abdomen demonstrated small volume ascites with normal hepatic contour and size. Ascitic fluid revealed a high serum-ascites albumin gradient (SAAG) and ascitic fluid total protein – suggestive of cardiac ascites with evidence of portal hypertension. Transjugular liver biopsy was performed, exhibiting pericentral sinusoidal dilation consistent with outflow obstruction, complicated by early cirrhosis (Figure 1). The sinusoidal pressure was normal despite significant dilation and portal venopathy, indicating venous flow compromise secondary to solely cardiac disease.

The patient was treated with lactulose and aggressive diuresis with resolution of symptoms within three days. Serum ammonia decreased to 91 umol/L one day after initiation and was no longer trended given clinical improvement. Our patient's newly diagnosed early cirrhosis was determined not to be a major contributing factor to initial presentation of HE given normal sinusoidal pressure on biopsy and minimal liver injury reflected in lab values.

Liver disease accounts for 90% of HE cases while noncirrhotic causes, including heart failure, represent a minority of cases³. In heart failure, hyperammonemia is from increased venous congestion and hypoperfusion in addition to heart failure-induced myocardial injury, which creates ammonia itself⁴. A review of all reported cases of heart failure-induced HE was performed (Table 1). All treatments consisted of lactulose and/or branched chain amino acids (BCAAs) as well as diuresis, and all patients fully recovered prior to discharge. Lactulose improves HE by facilitating removal of ammonia⁵. Additionally, diuresis for acute heart failure is also effective by relieving hypoperfusion secondary to systemic congestion⁶. Chronic heart failure can present to the hospital with multi-organ dysfunction, including congestive hepatopathy. While uncommon, it is important to consider heart failure as the etiology for hyperammonemic encephalopathy especially in the absence of acute liver failure or known liver disease, as this can guide successful treatment of HE by incorporating heart failure management.

REFERENCES

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