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

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INTRODUCTION

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 hepatomegaly[1]. Hyperammonemic encephalopathy (HE) is rare in congestive heart disease, especially without known underlying hepatic pathology[2]. 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.

CASE REPORT

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

Total bilirubin	3.9 mg/dL	**Lactate**	2.7 mmol/L
Alkaline phosphatase	150 unit/L	**BNP**	458 pg/mL
AST	32 unit/L	**Ammonia**	183 umol/L
ALT	19 unit/L	**INR**	1.4

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.

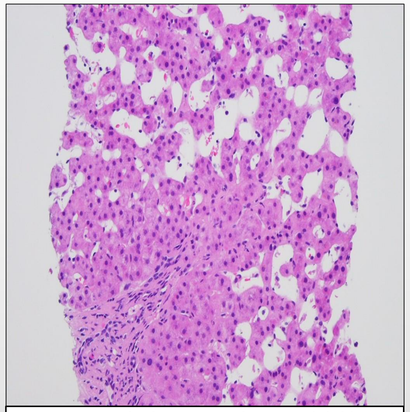

FIGURE 1: marked pericentral sinusoidal dilation

DISCUSSION

Liver disease accounts for 90% of HE cases while noncirrhotic causes, including heart failure, represent a minority of cases[3]. In heart failure, hyperammonemia is from increased venous congestion and hypoperfusion in addition to heart failure-induced myocardial injury, which creates ammonia itself[4]. 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 ammonia[5]. Additionally, diuresis for acute heart failure is also effective by relieving hypoperfusion secondary to systemic congestion[6]. 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.

TABLE 1: Reported cases of hyperammonemic encephalopathy secondary to congestive heart failure

Study	Age/Sex	Symptoms	Ammonia (ug/dL)	Treatment
Haider et al. (2013)[7]	69-year-old male	Lethargy, unresponsiveness, ascites	136	Lactulose
Kaida et al. (2015)[6]	48-year-old male	Coma and tremor	Unknown	Conventional HF mgmt.
Komiyama et al. (2016)[8]	78-year-old female	Consciousness disorder, pedal/facial edema	Unknown	Diuretics & BCAAs
Narita et al. (2020)[4]	81-year-old male	Cognition decline, decreased activity, asterixis	221	Lactulose, BCAAs, HF mgmt.
Current Report, 2021	59-year-old male	Encephalopathy, asterixis, ascites	183	Lactulose and diuresis

REFERENCES

SCAN ME

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