Question 42 – May 20

You are asked to consult on a 72-year-old female patient in the emergency department. She presented with cellulitis of the right lower extremity and bilateral peripheral edema. She has a history of NASH cirrhosis and has been managed as an outpatient with diuretics. She has mild ascites and spontaneous bacterial peritonitis has been ruled out. She does not have a lower extremity DVT. She has not had variceal bleeding or hepatic encephalopathy symptoms in the past.

In the ED, she is alert, responsive, without asterixis and has a swollen and tender RLE and evidence for cellulitis. She is febrile. Her WBC is 22,000 k/mcL, her creatinine is 2.2 mg/dL (baseline 1.5 mg/dL) and venous ammonia level is 72 mcmol/L (normal 35-65).

Which of the following is true regarding the ammonia level and its management in this patient?

A. The high ammonia level is diagnostic of minimal hepatic encephalopathy (MHE) and should be treated with lactulose as a first line agent
B. The high ammonia level is diagnostic of overt hepatic encephalopathy (OHE) and should be treated with lactulose and rifaximin combination therapy
C. Because of the high ammonia level a non-contrast head CT should be obtained to rule out intracranial pathology
D. No treatment for the elevated ammonia level is necessary as the patient does not have hepatic encephalopathy symptoms
E. Lactulose should be started and dose titrated until the ammonia level decreases into the normal range

Answer: D

This patient has cirrhosis but no history or symptoms of hepatic encephalopathy. High blood-ammonia levels alone do not add any diagnostic, staging, or prognostic value in HE patients with chronic liver disease and therefore should not be checked in the absence of confusion or signs of HE. Ammonia levels are dependent on the source of the sample (venous or arterial), renal function, the level of WBC’s in the blood sample, and the delay between phlebotomy and laboratory analysis. The treatments for HE can be toxic and expensive and primary prophylaxis for HE has not been proven beneficial and is not recommended based on ammonia levels alone. MHE and OHE diagnoses are based on clinical or psychometric assessments and do not require ammonia levels. Treatment for HE, when indicated, should be based on the patient’s HE response not serum ammonia level. In this case, the patient has no signs of HE and has other reasons for hyperammonemia and no HE specific therapy is indicated.

References:
Lockwood AH. Blood ammonia levels and hepatic encephalopathy. Metab Brain Dis 2004;19:345-349