"My Brother Won't Wake Up!"

DALHOUSIE UNIVERSITY Inspiring Minds

Tim Gash; Bijon Das MD, CCFP(EM)

Scott's Case

Scott, a 46-year-old male, was brought to the ED via ambulance after he was discovered unresponsive by his family, who then called 911. They reported that he had a history of significant alcohol abuse, diabetes and hypertension.

Paramedics found him sitting up in bed drooling and non-verbal. His vital signs, including blood sugar, were normal. They reported that he would slap at anyone that touched him and required restraints for transport to the hospital. He had been incontinent of both feces and urine. The family was concerned that he had been either abusing alcohol or using drugs. On presentation to the ED his GCS was seven, and he would not follow commands. His vitals were: HR 84 bpm; BP 112/80 mm Hg; RR 20 per min; blood glucose 6.8 mmol/L.

Upon examination, the patient was noted to have jaundice and scleral icterus. Both his legs and arms were noted to have pitting edema bilaterally. The patient was noted to have a distended abdomen. There was no evidence of asterixis.

Read on for more on Scott.

Questions and Answers

1. What is the most likely diagnosis?

Based on the history and physical exam findings the patient was suspected of having hepatic encephalopathy. The patient was empirically started on lactulose to treat the suspected underlying cause (see question 3). The patient was intubated to protect his airway. Tests that were ordered included ammonia level, ABG, blood cultures, EKG, urine dip, cardiac enzymes, liver enzymes, coagulation and routine blood. A CT head and chest x-ray were performed to rule out an extra-hepatic cause of his decreased level of consciousness. Both the Intensive Care and Gastroenterology services were consulted. The patient was admitted to the ICU and continued lactulose therapy. Antibiotic prophylaxis was also commenced for the treatment of hepatic encephalopathy, as well as to prevent and treat spontaneous bacterial peritonitis, a common cause of serious morbidity and mortality among hospitalised patients with liver disease.[1] Further studies included ultra-sound (US) and paracentesis (which was both diagnostic and therapeutic). The US revealed ascites, splenomegaly and cirrhosis. The patient improved with lactulose and was extubated the following day and transferred to the GI service. A liver biopsy was performed prior to discharge and revealed Stage IV fibrosis consistent with liver cirrhosis.

2. What are the pathophysiology and treatment of hepatic encephalopathy?

The pathophysiology of hepatic encephalopathy is currently unknown; however it is generally attributed to the toxic effects of nitrogenous compounds, both dietary and those produced by gut bacteria.

3. How should we approach hepatic encephalopathy in the ED?

The treatment of hepatic encephalopathy has focused on mechanisms to reduce nitrogenous compounds, more specifically, ammonia levels.

Specific therapies have included the use of non-absorbable antibiotics and cathartics. The proposed mechanism of antibiotics is the reduction of urease-producing intestinal bacteria, which results in the decreased production of ammonia in the gut.[2] Cathartic disaccharides (CD) are proposed to work via several mechanisms. These include reduced transit time through the GI tract; thereby limiting the time bacteria can act to form ammonia, and altering the pH of the colon resulting in more ammonia being incorporated into bacteria. This increases stool volume and the cathartic effect.[2] It has also been reported that CDs reduce small intestine production of ammonia by interfering with glutamine uptake by the intestinal wall.[3]

In North America, standard treatment involves the use of lactulose.[4] Other places in the world, including some European countries, use antibiotics such as Rifaximin, which some studies have shown to be at least equally, if not more, effective than lactulose.[4] Some controversy over the use of lactulose has occurred recently, based in large part on the findings of a Cochrane Review that was published in the BMJ.[5] The study put forth the idea that lactulose should not be used as standard treatment in controlled trials and that antibiotics are a superior treatment to lactulose; however, it was noted that the difference in effectiveness may not be clinically significant. The authors also indicated that the evidence for, or against the use of non-absorbable disaccharides is currently insufficient.[5] Some researchers have found benefits from zinc supplementation; however, to date, there is no consensus on whether this is in fact a valid treatment and what the dosing of zinc should be.[4] Dx

Back To Scott

A diagnosis of decompensated cirrhosis in the form of hepatic encephalopathy, ascites, jaundice, and cryptogenic cirrhosis secondary to alcohol was made, and a work-up for liver transplant was initiated.

The patient was discharged from the hospital on a regimen of furosemide 120mg q.o.d, amiloride 20 mg q.d, ursodiol 500 mg a.m. and 1 g p.m., lansoprazole 30 mg q.d., lactulose 30ml b.i.d., Zinc 50 mg q.d. and one multivitamin tablet q.d. He was vigorously implored to avoid alcohol consumption.

References

1. Hoefs, JC: Spontaneous Bacterial Peritonitis: Prevention and Therapy. Hepatology 1990;12(4 pt 1):76-81.
2. Morgan MY, Blei A, Grüngreiff K, Jalan R, Kircheis G, Marchesini G, Riggio O, Weissenborn K: The Treatment of Hepatic Encephalopathy. Metab Brain Dis 2007;22(3-4):389-405.
3. Conn HO, Lieberthal M. 1979. The Hepatic Coma Syndromes and Lactulose. Baltimore: Williams and Wilkins.
4. Bass NM: Review Article: The Current Pharmacological Therapies for Hepatic Encephalopathy. Aliment Pharmacol Ther 2007;25(Suppl 1):23-31.
5. Als-Nielsen B, Gluud LL, Gluud C: Non-absorbable Disaccharides for Hepatic Encephalopathy: Systematic Review of Randomized Trials. BMJ 2004;328(7447):1046–1051

Mr. Gash is a Senior Medical Student at Dalhousie University, Halifax, Nova Scotia.

Dr. Das is an Assistant Professor of Emergency Medicine, Dalhousie University, and an Emergency Physician at the Queen Elizabeth II Health Sciences Centre, Halifax, Nova Scotia.

Publication Mail Agreement No.: 40063348
Return undeliverable Canadian addresses to:
STA Communications Inc.
955 boulevard St-Jean, Suite 306
Pointe-Claire, QC, H9R 5K3