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Short-communication

Decompensated Liver Cirrhosis for the Acute Physician

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Short Communication

Liver cirrhosis (LC) is a disease of progressive fibrosis with regenerative nodules resulting in distortion of liver architecture. Early compensated LC can be managed or even reversed by specific treatment; however, decompensated LC has multiple complications leading to increase in morbidity and mortality [1].

Doctors and patients should be aware of the multiple risk factors which can influence compensated LC leading to decompensated LC, most common factors are infection, gastrointestinal bleeding (GIB), medications like benzodiazepines and alcohol, constipation, and dehydration with electrolyte abnormalities [2,3].

Distortion of liver architecture in LC is the main cause of increase in portal venous system pressure leading to portal hypertension (PH) which cause high pressure venous collaterals (varices) and ascites among other complications which will be mentioned below.

Variceal haemorrhage has a significantly high mortality even after introduction of endoscopic band ligation, mortality can reach 20% in one-month, other possible treatment for variceal

haemorrhage includes endoscopic sclerotherapy and Trans Jugular Intrahepatic Portosystemic Shunt (TIPS) [4].

Portal Hypertensive Gastropathy (PHG) also known as congestive gastropathy is very common in patients with PH, however, it usually presents with mucosal oozing and anaemia rather than significant bleeding events.

Ascites is fluid in the peritoneal cavity secondary to PH and low albumin in serum resulting in reduction of the oncotic pressure, ascites is considered the most common complication of LC. Treatment is a combination of sodium and water restriction plus diuretics, and if ascites is refractory to this treatment, then repeated therapeutic paracenteses or TIPS is required.

Spontaneous bacterial peritonitis (SBP) is infection in the ascitic fluid, and usually presents with abdominal pain and tenderness, encephalopathy and fever but can be asymptomatic, therefore all patients with known LC and ascites should have a diagnostic ascitic tap to rule out SBP as part of their work up in the hospital. Diagnosis of SBP is made if these is a positive bacterial culture or elevated ascitic fluid absolute polymorphonuclear leukocyte count ($\geq$250 cells/mm^3).

Hepatorenal syndrome (HRS) is an acute kidney injury in a patient with known advanced LC which is refractory to treatment (intravenous fluid and omitting nephrotoxins for example). The arterial splanchnic vasodilatation in patients with PH and advanced LC cause a reduction in renal perfusion which is usually refractory to fluid resuscitation. Two thirds of patients with HRS are oliguric, urine sediment is unremarkable, but sodium excretion is characteristically below 10.

Hepatic hydrothorax is commonly a right-side pleural effusion from the transfer of ascitic fluid via the defects in the diaphragm, it is important to rule out other common causes of pleural effusion like infection, tumor, or heart failure. Treatment of hepatic hydrothorax is like ascites, starting from water and sodium restriction to diuretics and pleural therapeutic thoracocentesis and TIPS in refractory cases. Pleural drain is contraindicated as it increases the risk of infection, renal failure, and electrolyte abnormalities.

Hepatopulmonary syndrome (HPS) is hypoxia due to intrapulmonary vascular abnormalities leading to Increase in alveolar-arterial gradient. HPS is more common with advanced liver disease. Cardiomyopathy and pulmonary hypertension also can commonly occur in patients with advanced LC and portal hypertension [5,6].

Hepatic encephalopathy (HE) is a common presentation in patients with decompensated LC, early sign of HE can be easily missed (disturbance in the diurnal sleep pattern-insomnia and hypersomnia) and if treatment is delayed, HE can progress to asterixis, hyperactive deep tendon reflexes, and coma. Treating HE starts with

treating the cause of decompensation and adding lactulose and rifaximin to reduce the level of ammonia.

Hepatocellular carcinoma (HCC) is common in patients with LC, risk increase with cirrhosis rather than the pathology of the liver disease except in chronic hepatitis B where HCC can occur before LC. HCC can present as decompensated LC; doctors should have a low threshold in requesting alpha-fetoprotein (AFP) and ultrasound.

Portal vein thrombosis due to unbalanced haemostasis and slow portal flow can occur and is another possible cause of decompensated LC presenting to the acute physician, ultrasound abdomen with dopplers can rule in/out this possible complication.

After mentioning the most common complications in patients with LC presenting to hospital, the researcher would like to remind the readers with the optimum care plan required for this vulnerable group of patients, as decompensated LC has a high mortality and early management can save lives.

Recommendations are early clinical assessment including ABC approach and national early warning score, a cannula or two if concern regarding bleeding and send for routine bloods including full blood count, renal and liver profile, coagulation screen, glucose, and electrolytes ($Ca/Mg/PO_4$), and CRP. A full septic screen including blood and urine culture, ascitic tap, a chest X-ray and Ultrasound liver with doppler study [8].

If suspicion of alcohol dependence and risk of withdrawal, prescribe Pabrinex and start chlordiazepoxide as per CIWA score. In case SBP is suspected or confirmed, start empirical broad-spectrum antibiotics with intravenous 20% human albumin solution 1.5 g/Kg.

Patients who are presenting with acute kidney injury or hyponatraemia <125 mmol/L and not overloaded, recommendations are to give up to two liters of crystalloid or 5% human albumin solution, omit nephrotoxins including diuretics and hypotensive agents to maintain a reasonable mean arterial pressure of 65 mmHg to aid perfusion with close monitoring for urine output. HRS is likely if renal function has not recovered and no evidence of obstruction, treatment of HRS is adding terlipressin to induce splanchnic vasoconstriction and human albumin solution [7].

If variceal bleed is suspected urgent blood transfusion if Hb below 70 or below 10 if active bleeding, haemodynamically unstable and a higher target (90) if known ischaemic heart disease. Terlipressin 2 mg QDS (unless ischaemic or severe peripheral vascular disease), empirical intravenous antibiotics (cefuroxime for example). Correct coagulopathy if prothrombin time is prolonged with INR > 1.5 give vitamin K to correct deficiency and fresh frozen plasma 15 mls/Kg, platelet transfusion if level is below 50,

and cryoprecipitate (unless FFP given) if fibrinogen is below 1.5.

Patients presenting with HE can benefit from lactulose 30 mls QDS- aiming 2-3 loose motions per day or phosphate enema if concerns regarding oral intake, and low threshold for CT brain if history of fall or trauma [7].

Venous thromboprophylaxis is indicated unless active bleeding or platelets below 50 even if INR is raised, and Dietician role is essential to maintain adequate nutrition supply [8].

After resuscitation, inform Gastroenterologist on call for endoscopy and further advice or intensive care for organ support if required.

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