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Review Article

The Emergency Medicine Approach to the Decompensating Cirrhotic Patient

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Abstract

Background: Cirrhosis is the 8th leading cause of death in the United State and has a variety of causes, most commonly Hepatitis C and alcohol. Liver fibrosis and nodule formation results in significant complications due to portal system hypertension. There are several deadly complications that emergency physicians must consider.

Objective of the Review: Provide an evidence-based update for the resuscitation of Decompensating cirrhotic patients and an overview of cirrhosis complications.

Discussion: Cirrhosis is a common condition in the U.S. and leads to several deadly complications including variceal hemorrhage, ascites, Spontaneous Bacterial Peritonitis (SBP), hepatic encephalopathy, hepatorenal syndrome, and hepatopulmonary syndrome. Key resuscitation techniques and care of the complications of cirrhosis can reduce morbidity and mortality.

Conclusions: Decompensating cirrhotic requires extensive resuscitation, and new techniques for intubation and restoring circulation show promise in reducing morbidity and mortality.

Keywords: Cirrhosis; Spontaneous bacterial peritonitis; Hepatic encephalopathy; Hepatorenal; Hepatopulmonary; Ascites; Liver; Airway

Abbreviations

SBP: Spontaneous Bacterial Peritonitis; GI: Gastrointestinal; DSI: Delayed Sequence Intubation; NG: Nasogastric; NC: Nasal Cannula; IV: Intravenous; ETT: Endotracheal Tube; SIRS: Systemic Inflammatory Response Syndrome; TXA: Tranexamic Acid; LMA: Laryngeal Mask Airway; LR: Likelihood Ratio; PMN: Polymorphonucleocyte; CBC: Complete Blood Count; VBG: Venous Blood Gas; ECG: Electrocardiogram; UA: Urinalysis; CT: Computed Tomography; TEG: Thromboelastography; UTI: Urine Tract Infection ; IPVDs: Intrapulmonary Vascular Abnormalities; Cr: Creatinine

Introduction

Cirrhosis is the late stage of progressive hepatic fibrosis and is considered irreversible. In 2010, cirrhosis was the 8th leading cause of death in the U.S., and it most commonly is due to Hepatitis C, followed by alcohol [1,2]. Acute failure can be due to drugs/medications (most commonly acetaminophen), viral hepatitis, autoimmune, ischemia (shock liver), and Wilson's disease, but the cause is often indeterminate (up to 14%). Compensated cirrhotics possess a five-fold increase in mortality and decompensate cirrhotics a ten-fold increase in mortality when compared to the standard population [1-3].

Decompensate cirrhosis is defined by the presence of cirrhosis and the development of a major complication such as variceal hemorrhage, ascites, Spontaneous Bacterial Peritonitis (SBP), hepatic encephalopathy, hepatorenal syndrome, or hepatopulmonary syndrome [2,3]. Bleeding, infection, increased alcohol intake, dehydration, electrolyte abnormalities (hypokalemia specifically), and constipation can worsen complications [2-5].

These complications are the end result of changes in the liver: fibrosis and regenerative nodules replace normal hepatic tissue, decreasing hepatic venous flow and elevating portal pressures. This in turn causes splenomegaly, resulting in anemia, hypoalbuminemia, thrombocytopenia, and often ascites [1-5].

Discussion

History and examination

The patient's chronic medical management (including medications, endoscopic assessment of varies, follow up visits, normal weight) should be assessed, as well as symptoms such as anorexia, fever, abdominal pain, pruritis, melena, hematemesis, and prior complications. After completing the initial assessment and primary survey, evaluate for any signs of cerebral edema and herniation such as altered mental status, focal neurologic deficit, and/or posturing. Volume status can be difficult to ascertain in these patients due to abnormal volume distribution. The presence of asterixis and jaundice should be assessed. Ascites can be difficult to diagnosis based on exam alone, but ultrasound can be helpful in this setting, as well as in the assessment of volume status. Signs of chronic failure include ascites, caput medusa (superficial periumbilical veins), muscular atrophy, gynecomastia, testicular atrophy, spider angiomas, palmar erythema, and parotid gland enlargement [5,6].

Approach to airway management

The resuscitation of the cirrhotic patient presenting in extremis

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has several important aspects that may differ from the management of other patients. First, airway protection is paramount due to risk of rapid decompensation. Upper Gastrointestinal (GI) bleeding and decreased mental status may mandate airway protection. If the patient has massive hematemesis or demonstrates poor mentation, a low threshold to intubate the patient is advised [7].

a. These patients presents multiple challenges for emergency physicians due to the patient's tendency to rapidly desaturate and difficulty obtaining clear view of glottic structures. The concepts of NO DESAT and Delayed Sequence Intubation (DSI) can be beneficial in decreasing morbidity/mortality and increasing first-pass success. Immediately place the patient on Nasal Cannula (NC), and if needed, add a facemask with $15LO_2$ as well [7-9]. If the patient continues to demonstrate low oxygen saturation, noninvasive positive pressure ventilation may be considered, but use caution especially if the patient has active hematemesis. Ketamine with DSI can assist preoxygenation and denitrogenation, providing an oxygen reservoir during intubation. This allows placement of a nasogastric (NG) tube, which will decrease aspiration risk and clear the stomach of any present blood, improving the intubation attempt [7,10,11]. NG placement should only be used to clear the stomach of potential aspiration material and not to diagnose upper GI bleed in the immediate resuscitation efforts [7,10-13].

b. Several methods of intubation have been suggested for these tenuous patients. One approach involves several logical steps designed to optimize patient hemodynamic status and oxygenation, as well as provider view of glottic structures. The first step involves emptying the stomach with NG tube, followed by administration of metoclopramide 10mg IV [7,11,14]. An NG tube has low risk of further disrupting varices and can assist in clearance of blood from the stomach [7,10,15,16]. The patient is then intubated with the head of the bed elevated to 45 degrees and suction device ready. Adequate preoxygenation is best, as these patients will rapidly desaturate once sedative and paralytic medications are provided [7,8]. Smaller doses of sedative medications are recommended to protect hemodynamic status. As many of these patients will be in a shock state, a fraction of the dose of the sedative but larger doses of paralytics will be needed, such as rocuronium 2mg/kg IV [7,16-19]. Ketamine with its intrinsic sympathomimetic effects has lower risk of producing hemodynamic instability during intubation [7,16-19]. Paralytics such as rocuronium will also increase tone of the lower esophagus, potentially decreasing risk of aspiration [7,20]. Airway adjuncts including video scope, bougie, Laryngeal Mask Airway (LMA), suction set up, and a meconium aspirator are recommended [7]. If the first attempt fails, ventilate the patient gently with bag-valve mask, and consider placing a LMA. If the patient vomits, place him/her in Trendelenburg position. The meconium aspirator can be attached to the Endotracheal Tube (ETT) for improved suction if the baseline suction device is insufficient. If aspiration does occur during the intubation, the patient will likely experience pneumonitis and Systemic Inflammatory Response Syndrome (SIRS) [7].

GI bleeding/Hematemesis

Patients with GI bleeding in the setting of ascites typically present with hematemesis and/or melena due to variceal hemorrhage, gastritis, or ulcer. Varices form at a rate of 5-15% per year in cirrhotics, with one third of patients suffering hemorrhage over their lifetime. Variceal hemorrhage has an initial mortality rate upwards of 20%, which is decreased from prior rates of 30% [3-6,21-25]. Rates at one year though reach 70%. In cirrhotic patients, varices are the cause of bleeding in 50-90% of patients. As described above, airway protection is vital along with control of bleeding and avoidance of complications. Only 50% of patients with variceal bleeding will stop bleeding spontaneously, unlike other sources of GI bleeding [21-24].

One function of the liver is production of clotting factors and anticoagulants, and these proteins are reduced due to decreased production in cirrhotics. Levels of clotting factors and anticoagulants are reduced in a similar manner, meaning the intrinsic clotting tendency in these patients is approximately normal. Thus, INR often fails to predict bleeding associated with procedures [25,26]. Monitoring can be difficult, but Thromboelastography (TEG) does have potential to best characterize enzymatic coagulation in cirrhosis. In particular, the R-time is useful as a reflection of pro- and anti-coagulant agents [26-30]. Platelet and fibrinogen levels will also decrease in cirrhotics. With cirrhosis, proteins involved in fibrinolysis accumulate, leading to hyper-fibrinolysis in up to 50% of patients. These patients are at risk for bleeding and thrombus formation due to these physiologic changes [25-30].

Three management goals exist: 1) hemodynamic resuscitation, 2) prevention/treatment of complications, and 3) bleeding cessation. Resuscitation including IV access and blood product transfusion is key in initial management, but beware of over-transfusion and volume overload. These patients often demonstrate low blood pressure at baseline, and over-correction may worsen bleeding, as the predominant source in variceal bleeding is venous [26,28]. A dilutional coagulopathy can also develop with over-resuscitation due to low baseline platelets and coagulation factors in cirrhotic patients [26,28]. Goals of transfusion if active exsanguinations are present include minimum hemoglobin (Hgb) of 7g/dL, fibrinogen 150-200mg/dL, and platelets 50,000/mm^3 [26,28]. A low threshold to reach these goals with a transfusion ratio of 1:1:1 and the addition of cryoprecipitate due to low fibrinogen levels are warranted [27-35]. As discussed above, platelets and fibrinogen are decreased in cirrhotic states [26-30]. Platelets will assist in thrombus formation, with a target of 50,000/mm^3 warranted based on in vitro studies [29,30]. Fibrinogen assists with formation and stabilization of the thrombus. One study demonstrated a decrease in the need for transfusion products in liver transplant patients undergoing fibrinogen supplementation [36]. Guidelines support a target fibrinogen level of 150-200mg/dL [37]. Cryoprecipitate is the best product to increase fibrinogen. Due to the increased risk of hyperfibrinolysis, Tranexamic acid (TXA) may be of use at 1g IV. This treatment does show promise in decreasing mortality of upper GI bleeding, as shown in a recent Cochrane Review [38]. The recent Villanueva article in NEJM with goals of Hgb 7g/dL did not include patients with active, severe hemorrhage, though target hemoglobin of 7g/dL is preferential for these patients [39].

Complications most commonly include infection, and provision of antibiotics in this setting will dramatically decrease mortality and even recurrent bleeding risk. Infections include UTI (29%), SBP (23%), and respiratory source (11%). Antibiotics include ceftriaxone 1g or cefotaxime 2g, as up to 20% of patients with cirrhosis will have

an infection at time of admission, with up to half developing infection over hospitalization. These patients are at high risk for aspiration, and although controversial on whether it increases risk of aspiration, endotracheal intubation should be completed to protect the airway in the setting of severe hematemesis. Placement of a NG tube can assist in decompressing the stomach and clearing blood. These patients are also at increased risk of encephalopathy and renal failure with variceal bleeding [21,22,40-43].

Control of bleeding is essential. Ultimately, Endoscopic therapy (EGD) is required, as the source of bleeding must be targeted. EGD should be completed within 12 hours. Variceal ligation or sclerotherapy are definitive treatments [22,43-45]. This warrants immediate Gastrointestinal (GI), general surgery, and Interventional Radiology (IR) consultation. These three services each have interventions that may reduce patient hemorrhage. Erythromycin can assist gastric emptying and improve EGD [7]. Vasoactive agents include octreotide, vasopressin, somatostatin, and terlipressin, and these have been shown to improve hemostasis, transfusion requirements, and duration of hospitalization. Octreotide at 50mcg bolus with 50mcg/hr drip can decrease portal pressures and azygos blood flow, while increasing MAP. Vasopressin at 0.2 to 0.4 units/minute with nitroglycerin at 40 micrograms/min is another regimen. Terlipressin (2mg IV bolus every four hours), not available in the US, is the only actual agent beyond antibiotics that decreases mortality [41-48].

If unable to control bleeding with initial measures (endoscopic therapies fail 10% of the time), balloon tamponade is an option, but placement is associated with serious complications such as esophageal rupture. Three systems are available: Sengstaken-Blakemore tube (with a 250cc gastric balloon, esophageal balloon, and single gastric suction port), Minnesota tube (modified Sengstaken-Blakemore tube with an esophageal suction port above the esophageal balloon), and Linton-Nachlas tube (single 600cc gastric balloon) [43,45,49].

Ascites

Ascites is the most common complication of cirrhosis and is the accumulation of fluid in the peritoneum. Close to 60% of cirrhotic patients will have ascites within 10 years of diagnosis. Portal hypertension is the first step in fluid accumulation [5,6]. When a patient presents with ascites, question on weight gain, which as has positive likelihood ratio (+LR) of 3.2, and increase in abdominal girth (+LR 4.16). The absence of ankle swelling has a negative LR of 0.10, and absence of change in abdominal girth has a -LR of 0.17. In terms of physical exam, fluid wave has the highest +LR at 9.6, with shifting dullness also supportive (+LR 5.76). Ultrasound can assist in diagnosing ascites, as it will demonstrate free pockets of intra-abdominal fluid [5,6,50].

Ascites is managed chronically with diuretics (specifically spironolactone and furosemide in a 100:40 ratio) and sodium restriction. Abstaining from alcohol should be encouraged, as this will drastically improve ascites. These patients ultimately require GI/primary care for medical management [6,50-53].

In the ED setting, classic indications for paracentesis include new onset ascites, suspected SBP, and/or relief of cardio respiratory or GI manifestations. Specifically, removal of more than 5L can assist in improving dyspnea and early satiety these patients often experience. Paracentesis may also be associated with collateral advantages, such as a reduction in hepatic venous pressure gradients, intravariceal pressure, and variceal wall tension, which can in turn decrease risk of GI bleeding. There is no level of INR or platelets that serves as a contraindication for paracentesis [52,53].

Spontaneous bacterial peritonitis

SBP is an infection of ascitic fluid, and diagnosis requires the presence of ascites. Most cases are due to *E. coli* and/or *Klebsiella*. This is usually a sign of end-stage liver disease. Classic signs of this disease include fever (the most common sign), abdominal pain, and altered mental status. Abdominal pain and AMS are often subtle, and because these patients are baseline hypothermic, a temperature of 37.8°C should be used for fever. On exam, any abdominal tenderness, increase in ascites, or hypotension should raise suspicion of SBP. Other symptoms/signs include diarrhea, ileus, hypothermia, acidosis, and azotemia. If the patient has temperature greater than 37.8°C, abdominal pain/tenderness, change in mental status or ascitic Polymorphonucleocyte (PMN) count greater than 250 cells/mm^3, perform paracentesis and start antibiotics [5,53-57]. Each hour in delay of paracentesis increases the mortality by 3.3% [55].

SBP is diagnosed with an ascites neutrophil count ≥250 cells/mm^3, >1000 WBC, positive culture results, and exclusion of a secondary cause (such as surgical infection) [5,53-57]. The ascitic fluid should be sent to the laboratory for cell count/differential, culture, Gram stain, albumin, glucose, protein, LDH, amylase, and bilirubin. At least 20 cc of ascitic fluid in two separate blood culture bottles should be obtained, which can increase yield by 25% [56,59-61].

Once cultures are obtained, start antibiotics immediately (either cefotaxime 2g IV or ceftriaxone 1g IV). Cefotaxime 2g IV every 8 hours provides high ascitic fluid levels of the antibiotic [57]. Renal failure develops in up to 40% of patients with SBP, and this can be decreased with IV albumin infusion at 1.5g/kg with antibiotics [57,58].

There has been literature on utilizing urine dipstick to quickly diagnose SBP if the dipstick returns positive for leukocyte esterase and/or nitrates. However, sensitivities vary from 31% to 100%, with specificities of 81% to 100%. This may help rule in the diagnosis of SBP, but it cannot be used to rule out disease [58-60]. A dipstick device specifically designed for ascitic fluid is in development [61].

Other tests of peritoneal fluid have shown promise. An ascitic lactate level greater than 25mg/dL has demonstrated sensitivity and specificity approaching 100% in the diagnosis of SBP. Other tests including peritoneal fluid pH < 7.35 have shown promise when combined with fluid neutrophils [60,61]. Lactoferrin (an iron binding protein present in PMN's) presence in ascitic fluid has been shown in one study to have a sensitivity of 96% and specificity of 87%, but this test requires further validation [62].

Hepatic encephalopathy

Patients with encephalopathy usually have advanced liver disease. Encephalopathy is a spectrum of neurologic abnormalities in patients with cirrhosis and is thought to be due to excess cerebral ammonia elevation, leading to increased glutamine. Overt encephalopathy

develops in up to 45% of patients with cirrhosis, but minimal encephalopathy with subtle findings will be present in 80% of patients [63-66]. Grading encephalopathy is based on four levels: Grade I involves a change in behavior and sleep with little change in consciousness, Grade II involves gross disorientation and possible asterixis, Grade III involves marked confusion and incoherent speech, and Grade IV involves a comatose patient with posturing [63-66].

One of the earliest markers is disturbance in sleep pattern, specifically hypersomnia during the day and insomnia at night. Other signs include asterixis, hyperactive reflexes, and decerebrate posturing. Asterixis may be present in Grade I, but it is often pronounced at Grade II. This is defined as flapping motion of outstretched, dorsiflexed hands [63-67]. Focal neurologic deficits (most commonly hemiplegia) can also be present in up to 17% despite normal cerebral imaging including computed tomography (CT) [68]. Overt encephalopathy, if present with severe cerebral edema, can lead to coma and death [63,64,68].

Precipitants of encephalopathy include GI bleeding, infection (SBP, UTI), hypokalemia, renal failure, dehydration, hypoxia, sedatives, hypoglycemia, and constipation. Evaluation should target these causes [63,64,68].

Diagnosis requires the following: history/exam supportive of the disease, exclusion of other causes of mental status changes (laboratory assessment and head CT are recommended), and search for precipitating cause. If possible, ask the patient's caregiver about baseline mental status, as well as new changes in sleep patterns and mental capacity [63,64,68]. Elevated ammonia is not required for the diagnosis, though the level is often elevated. Ammonia is elevated in liver disease due to decreased liver function and clearance from the blood, but many other causes of ammonia elevation can be present [63,67-70]. In general, ammonia levels do not correlate with degree of encephalopathy [71].

Laboratory assessment should include Complete Blood Count (CBC), liver enzymes, renal function, ammonia, Venous Blood Gas (VBG), Electrocardiogram (ECG), urinalysis (UA), and bilirubin. Noncontrast head Computed Tomography (CT) is indicated, as it may reveal generalized or localized cerebral edema.

Treatment focuses on reversing predisposing conditions such as infection (SBP, UTI), GI bleeding, electrolyte abnormalities (hypokalemia in particular), and constipation. After finding and correcting the precipitant, decreasing cerebral and blood ammonia concentrations should follow. Lactulose at 30 to 45ml 2-4 times per day by mouth if is most efficacious. If oral intake is not feasible, a lactulose enema can be used (1-3L of 20% solution). If not improved, rifaximin 400mg three times daily added to lactulose can be beneficial. Neomycin can be used as second line therapy dosed at 4-12g/day divided every 6 hours by mouth, but it does not have randomized trial support and can cause ototoxicity and nephrotoxicity [68,70-72]. Polyethylene glycol does have support for excreting ammonia in the stool with 4L given over 4 hours [52]. Other treatments under study include flumazenil, L-carnitine, acarbose, sodium benzoate, melatonin, serotonin antagonists, and opioid antagonists [68-74].

These patients are often agitated and can be a hazard to redirection if possible. Patients with liver disease are sensitive to benzodiazepines due to poor hepatic metabolism of the medications, but lorazepam has few metabolites and is likely the benzodiazepine with fewest complications. Haloperidol based on small studies has utility [64,68,71-73]. New generation antipsychotics do not currently have much evidence for use in this population [68,71-73].

Most patients with Grade I encephalopathy can be discharged with outpatient management, but disposition of patients with Grade II depends on the patient's clinical status. If the patient will not be able to adhere to treatment based on social situation, inpatient admission is warranted. Grades III and IV warrant admission, usually to an ICU [68,72].

Hepatorenal syndrome

This diagnosis requires renal failure in the setting of advanced liver disease and is a diagnosis of exclusion. This disease will develop in 39% of patients within five years and is a marker of liver failure. It is due to reductions in renal blood flow caused by splanchnic vasodilation and increased nitric oxide. Renal perfusion decrease in turn reduces glomerular filtration rate and sodium excretion. Precipitants include GI bleeding, infection, dehydration, electrolyte derangements, and renal injury [75,76].

This condition should be suspected in patients with known cirrhosis and decreased urine output. Hepatorenal syndrome is marked by little to no urine sediment, low urine sodium, increase in serum creatinine (Cr), and oliguria. Oliguria is controversial, as some patients will maintain urine output of greater than 400ml/day. As these findings are similar to the diagnosis of acute tubular necrosis and prerenal azotemia, hepatorenal syndrome is also diagnosed with urine red cells less than 50/high power field, protein excretion less than 500mg/day, and no improvement with IV albumin [75,77-79]. Biopsy may be needed for definitive diagnosis.

There are two types of hepatorenal syndrome. Type one is the most serious with a creatinine (Cr) above 2.5, or doubling of Cr in a period of less than 2 weeks. Type two is less severe and usually develops over a period of greater than two weeks [75,77-79].

For treatment of hemodynamically unstable patients, norepinephrine at 0.5-3mg/hr to raise mean arterial pressure by 10mmHg with albumin at 1g/kg per day for two days is warranted. If the patient is stable, a combination of midodrine at 7.5mg PO TID, octreotide 50mcg/hr IV, and albumin 1g/kg can be utilized. Transjugular Intrahepatic Portosystemic Shunt (TIPS) is sometimes successful for medical nonresponders, but ultimately liver transplant is required with hemodialysis as a bridge to transplant [75,77-79].

Hepatopulmonary syndrome

This syndrome requires known liver disease, increased A-A gradient while on room air, and evidence of Intrapulmonary Vascular Abnormalities (IPVDs), which are diagnosed with nuclear scanning, contrast echocardiography, and/or pulmonary arteriography [80,81]. The IPVDs include increased pulmonary dilation of vessels and increased number of pulmonary vessels. The prevalence ranges from 4-47% of cirrhosis patients, with mortality at 5 years of 75%. The cause of death is usually due to complications of cirrhosis rather than respiratory failure surprisingly. Unfortunately, mild hypoxemia is common in ascites due to diaphragm elevation, decreasing

Functional Residual Capacity (FRC) and increasing ventilation/perfusion mismatch, making diagnosis difficult [80-83].

Classic findings include platypnea (increase in dyspnea when upright, improved with recumbency) and orthodexia (decrease in arterial O_2 when moving from supine to upright position). These are thought to occur due to increased blood flow to IPVDs [83,84]. Liver transplant is the only means of treatment. TIPS does have support with case reports. There is no medical treatment shown to treat this condition besides optimizing ascites and other medical conditions. Oxygen supplementation can be used to treat baseline hypoxemia and symptoms such as shortness of breath [82-86].

Liver transplant criteria

For transplantation consideration, cirrhotic patients must have a complication of portal hypertension or severe disease. Severe variceal hemorrhage, ascites, encephalopathy, and hepatorenal disease are manifestations of end stage disease and transplant markers. Tools including the Model for End-stage Liver Disease (MELD) and Child-Turcotte-Pugh system can be used to evaluate patients for transplant. MELD incorporates age, creatinine, bilirubin, INR, and hemodialysis twice in the prior week. Scores greater than 10 warrant evaluation for transplant, while patients with scores greater than 15 are candidates [87]. The Child-Turcotte-Pugh (CTP) system is also used to assess prognosis in chronic failure but was initially developed to assess risk with shunt surgery in variceal bleeding. This contains classes A, B, and C (C has the worst mortality), with points ranging from 1-3 for the following: encephalopathy, ascites, bilirubin, albumin, and PT/INR. Five to six points is Class A, 7-9 Class B, and 10-15 Class C [88].

Conclusion

Cirrhosis results in portal hypertension and leads to several deadly complications including variceal hemorrhage, ascites, Spontaneous Bacterial Peritonitis (SBP), hepatic encephalopathy, hepatorenal syndrome, and hepatopulmonary syndrome. Resuscitation including airway and circulation measures is paramount in these patients, and several new techniques are offered for the approach to intubation and resuscitation of these patients. Diagnosing complications of cirrhosis requires clinical suspicion and specific testing, and emergency providers can decrease patient morbidity and mortality with rapid intervention.

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