American Journal of Emergency Medicine 36 (2018) 689–698

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Review

The emergency medicine evaluation and management of the patient with cirrhosis

Brit Long, MD [a,*], Alex Koyfman, MD [b]

[a] San Antonio Military Medical Center, Department of Emergency Medicine, 3841 Roger Brooke Dr, Fort Sam Houston, TX 78234, United States
[b] The University of Texas Southwestern Medical Center, Department of Emergency Medicine, 5323 Harry Hines Boulevard, Dallas, TX 75390, United States

ARTICLE INFO

Article history:
Received 8 November 2017
Received in revised form 21 December 2017
Accepted 22 December 2017

Keywords:
Liver
cirrhosis
peritonitis
varices
hemorrhage
encephalopathy
hepatorenal
hepatopulmonary
infection
ascites

ABSTRACT

Background: Cirrhosis is a significant cause of death in the U.S. and has a variety of causes, most commonly Hepatitis C and alcohol. Liver fibrosis and nodule formation result in significant complications due to portal system hypertension. There are several deadly complications 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. The disease develops from liver fibrosis, elevating portal pressures and modifying patient hemodynamics. Cirrhosis results in significant anatomic and physiologic modifications involving the gastrointestinal, cardiopulmonary, neurologic, renal, immunologic, and hematologic systems. The disease can be divided into compensated and decompensated states, with decompensation associated with significant morbidity and mortality. Complications include variceal hemorrhage, ascites, increased risk of bacterial infection, spontaneous bacterial peritonitis (SBP), hepatic encephalopathy, hepatorenal syndrome, hepatopulmonary syndrome, umbilical hernia, and hepatic hydrothorax. Resuscitation including airway and circulation measures is paramount in these patients, and several new techniques are offered for the approach to intubation and resuscitation for patients with severe cirrhosis.
Conclusions: Decompensating cirrhotics may require extensive resuscitation, and knowledge of the evaluation and management of complications associated with cirrhosis can improve care for patients with severe liver disease.
Published by Elsevier Inc.

1. Introduction

Cirrhosis is the late stage of progressive hepatic disease and a significant cause of mortality in the U.S. The disease most commonly arises from Hepatitis C and alcohol [1-6]. Patients with this disease are increasingly presenting to emergency departments (EDs) for their care, many requiring significant management for complications arising from cirrhosis [1,4,6-8]. Knowledge of this disease pathophysiology, complications associated with cirrhosis, and the evaluation and management of decompensated cirrhosis can assist emergency clinicians in optimizing care for these patients.

1.1. Pathophysiology and definitions

Cirrhosis is associated with fibrosis and regenerative nodules replacing normal hepatic tissue, which decreases hepatic venous flow and elevates portal pressures [6-9]. This causes splenomegaly, resulting in anemia, hypoalbuminemia, thrombocytopenia, and often ascites [6-10]. An increase in portal pressure causes splanchnic vasodilation and increased splanchnic blood flow, which elevates portal pressure further. A complicated hemodynamic state is associated with cirrhosis, with decreased effective blood volume and decreased mean arterial pressure. Any loss in vascular tone or volume from sepsis, gastrointestinal (GI) bleed, or overdiuresis can result in further organ dysfunction [5-10].

Cirrhosis can be divided into compensated and decompensated cirrhosis. Compensated cirrhosis, defined by lack of major complication due to cirrhosis, is associated with a 5-fold increase in mortality and decompensated cirrhotics a 10-fold increase in mortality when compared to the standard population [1-5]. Compensated cirrhosis is often associated with no symptoms. Complications occur in cirrhosis due to portal hypertension, plasma volume expansion, increased cardiac output, and imbalance of biochemical factors (vasoconstrictors, vasodilators, vascular endothelial growth factors, nitric oxide) [5-10]. A complication such as jaundice, ascites, spontaneous bacterial peritonitis (SBP), or hemorrhage drastically affects mortality rate. Management of compensated cirrhosis focuses on diagnosis and treatment of the specific underlying condition.

* Corresponding author.
E-mail address: brit.long@yahoo.com (B. Long).

https://doi.org/10.1016/j.ajem.2017.12.047
0735-6757/Published by Elsevier Inc.

Decompensated cirrhosis is defined by the presence of cirrhosis and the development of a major complication such as variceal hemorrhage, ascites, SBP, hepatic encephalopathy (HE), hepatorenal syndrome (HRS), or hepatopulmonary syndrome [4-7]. Infection, hemorrhage, increased alcohol intake, dehydration, electrolyte abnormalities (hypokalemia specifically), and constipation can worsen complications [6-10]. Infection (SBP, urinary tract infection) is the primary cause of decompensation, and infection due to UTI, SBP, or *Clostridium difficile* colitis is the number one cause of mortality [11-13]. These patients are at significant risk for mortality. If a major complication occurs, survival decreases to 50% over 5 years, compared to patients with compensated cirrhosis with survival rate 90% at 5 years [8,14-17].

2. Discussion

2.1. History and examination

A focused history and physical examination provide essential information. Evaluation of patient airway and hemodynamic status should be conducted as soon as possible in the patient with suspected decompensated cirrhosis, along with obtaining intravenous (IV) access and monitor placement. After completing the initial assessment and primary survey and initiating resuscitation as needed, focused history should be obtained. Along with the chief complaint, the patient's baseline neurologic status, etiology for liver disease, and prior complications (infection, variceal hemorrhage, encephalopathy) should be elicited. The patient's chronic medical management (including medications, endoscopic assessment of varices, follow up visits, normal weight) should be assessed, as well as symptoms such as anorexia, fever, vomiting, abdominal pain, pruritis, melena, altered mental status/confusion, and hematemesis [8-10,16,18]. Current medications often include diuretics if the patient has ascites. Physical examination should evaluate for signs of hemodynamic instability, sepsis, and cerebral edema and herniation (altered mental status, focal neurologic deficit, and/or posturing). New onset fever is an important finding, as is increased ascites or abdominal girth and abdominal pain or tenderness. Volume status can be difficult to ascertain in these patients due to abnormal volume distribution, and these patients often have decreased baseline mean arterial pressure with abnormal vasodilatation [6-10]. The presence of asterixis and jaundice should be assessed. Ascites can be difficult to diagnosis based on examination alone, but ultrasound can be helpful in this setting. Signs of chronic failure include ascites, caput medusa (superficial periumbilical veins), muscular atrophy, gynecomastia, testicular atrophy, spider angiomas, palmar erythema, and parotid gland enlargement [6,16,18].

2.2. Approach to airway management

Resuscitation in the decompensated cirrhotic requires consideration of several key aspects, specifically in the setting of upper GI bleeding from suspected gastroesophageal varices. Airway protection is paramount due to risk of rapid decompensation [6,7]. Diffuse bleeding from the oropharynx and/or decreased mental status may mandate airway protection [6-9,19]. Oxygen supplementation should be provided upon initial evaluation [19-22].

a. Patients with upper GI bleeding can present a “difficult airway” scenario. Patients with upper GI bleeding and cirrhosis tend to rapidly desaturate during intubation attempts, and due to extensive hemorrhage, obtaining clear view of glottic structures is difficult [9,10,19,22]. The concepts of NO DESAT and delayed sequence intubation (DSI) are vital to decrease morbidity/mortality and increase first-pass success [19-21]. The patient should be placed on nasal cannula (NC) with facemask to optimize preoxygenation, with NC continued during intubation [19-22]. If the patient continues to demonstrate hypoxemia, noninvasive positive pressure ventilation may be considered, though close monitoring is required with active hematemesis [19]. Ketamine for DSI can assist preoxygenation and denitrogenation, though ketamine should be provided in doses less than normal induction doses (0.2–0.5 mg/kg IV instead of 1.0–2.0 mg/kg IV) if the patient demonstrates signs of hemodynamic instability [23-28].
b. Several measures may improve intubation. A nasogastric (NG) tube may decrease aspiration risk and decrease gastric material (blood), improving the intubation attempt [19,22,29,30]. Placement of an NG tube can remove material that may be aspirated during intubation, but the NG tube should not be used to diagnose an upper GI bleed in the immediate resuscitation efforts [19,22,29-33]. An NG tube has low risk of further disrupting varices and can assist in clearance of blood from the stomach [19,26,34]. Metoclopramide 10 mg IV or erythromycin 250 mg IV can act to further empty the stomach of blood [19,35,36]. Of note, erythromycin is supported by the American College of Gastroenterology guidelines for upper GI bleeding, whereas metoclopramide has less clear utility [7,33,35,36].
c. The head of bed should be elevated to 45 degrees, with multiple suction devices ready. Suction-assisted laryngoscopy and airway decontamination (SALAD) can be beneficial, which consists of utilizing a rigid suction catheter as a tongue lifter/depressor to allow optimal laryngoscope blade position [19,22,37,38]. The suction catheter tip is then placed into the esophagus on the left side of the month during intubation, effectively providing continuous suction during intubation [22,37,38]. While attempting intubation, NC should remain on the patient due to concern for rapid desaturation. Direct or video laryngoscope with ability for direct view is recommended [19-22]. Airway adjuncts including videoscope, bougie, laryngeal mask airway (LMA), suction set up, and meconium aspirator are recommended [19-22,37,38].
d. If the patient has not already received ketamine, a sedative and induction agent should be provided. Smaller doses of sedative medications are recommended to protect hemodynamic status, such as ketamine 0.2–0.5 mg IV or etomidate 0.1 mg/kg IV [19,26-28,39,40]. As many of these patients will be in a shock state if actively bleeding, a fraction of the dose of the sedative but larger doses of paralytics will be needed, such as rocuronium 2 mg/kg IV [19,26-28,39,40]. Paralytics such as rocuronium will also increase tone of the lower esophagus, potentially decreasing risk of aspiration [19,41].
e. If the first attempt fails, the patient should be gently ventilated with bag-valve mask. 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) [19].

2.3. 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. No matter the source, a patient in extremis requires bilateral IV placement and cardiac monitoring [6,7,9,10,19]. Varices form at a rate of 5–15% per year in cirrhotics, with one third of patients suffering hemorrhage over their lifetime [6-10,42,43]. Gastroesophageal varices correlate with severe liver disease, and variceal hemorrhage has an initial mortality rate upwards of 20%, which is decreased from prior rates of 30% [5,7,14,43-48]. Hemorrhage from gastroesophageal varices is the most dangerous complication in cirrhosis, with risk of hemorrhage predominantly due to vessel wall diameter and variceal wall tension [6,7,49]. In cirrhotic patients, varices are the cause of bleeding in over 50% of patients [6-10,42-45]. Varices form as result of increased portal blood flow and increased portal flow resistance. Increased cardiac output, splanchnic arteriolar vasodilatation, and vascular collateral formation also contribute to variceal formation. As described above, airway protection is vital along with