International Journal of Medical and Health Research
ISSN: 2454-9142
www.medicalsciencejournal.com
Volume 4; Issue 8; August 2018; Page No. 62-64

Complications of cirrhosis, and its management

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Abstract
Diseased liver has many complications which progress to cirrhosis. Cirrhosis is characterized by liver abnormal structure and functions where damage and death of some liver cells form scar tissues and the other living cells multiply to replace the dead cells resulting in regenerative nodules formation within the scar tissues. There are many causes of cirrhosis is caused by many factors like chemicals, viruses, accumulation of toxic metals in the liver and autoimmune liver disease.
The major complications of cirrhosis include varices, ascites, hepatic encephalopathy (HE), hepatopulmonary hypertension, hepatocellular carcinoma, hepatorenal syndrome, spontaneous bacterial peritonitis, and coagulation disorders. These can occur secondary to portal hypertension, abnormal synthetic function, or combination of both.

Keywords: variceal hemorrhage, ascites, SBP

Introduction
Cirrhosis results from chronic liver disease, and is characterized by advanced fibrosis, scarring, and formation of regenerative nodules leading to architectural distortion.
Major complications of cirrhosis include ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, portal hypertension, variceal bleeding, and hepatorenal syndrome [1].
Cirrhosis is considered the 12th leading cause of death in the United States. 29,165 of deaths in 2007 caused by cirrhosis which is the major risk factor for hepatocellular carcinoma development; this malignancy rates tripled from 1975 to 2005 [2].
This review will focus on cirrhosis and its complications. The major complications of cirrhosis include varices, ascites, hepatic encephalopathy (HE), hepatopulmonary hypertension, hepatocellular carcinoma, hepatorenal syndrome, spontaneous bacterial peritonitis, and coagulation disorders. These can occur secondary to portal hypertension, abnormal synthetic function, or combination of both.

Discussion
Variceal Hemorrhage
Cirrhosis represents a disease progression through different prognostic stages including compensated and decompensated stages. VH is a complication that results from portal hypertension denoting decompensation stage.
In case of VH management we need to to control acute hemorrhage preventing its recurrence within 5 days which lead to death. Therabies like antibiotic prophylaxis, ligation in lieu of sclerotherapy and the use of safe vasoactive drugs lead to decrease the VH mortality rate [3].
Other therapeutic strategies used to improve the survival rates like the restrictive transfusion strategy where the target hemoglobin level between 7–9 g/dl because as it was found that in patients with cirrhosis transfusing for a target hemoglobin of 9–11 g/dl has been associated with high mortality rates and increased hepatic venous pressure gradient [4].
The other strategy is done in patients at a high risk of failing standard therapy, where the use of ‘early’, preemptive, transjugular intrahepatic portosystemic shunt (TIPS) placement Showed reduction of therapy failure and to improve survival [5].
VH can occur in conjunction with other complications like ascites and/or encephalopathy The matter which has different prognostic implications and higher mortality rates [6].
VH screening is considered a preventive measure in patients with cirrhosis and depends on its stage. In case of compensated cirrhosis, screening endoscopy should be performed within 1 year and repeated every one to two years to detect any silent varices [7].
Where in case of complicated cirrhosis which shows bleeding, encephalopathy, ascites, hepatocellular carcinoma, or hepatopulmonary syndrome, screening should be performed within three months. Endoscopy should be performed again in one year if small varices are found [8].
Treatment with beta blockers used in the treatment of medium and large varices [9], where repeated sessions of endoscopic

variceal ligation is superior to beta-blocker therapy to prevent bleeding [10].

In case of acute variceal bleeding which considered as a fatal complication, it is important to achieve hemodynamic stability as soon as possible. But excess fluid administration or transfusions may cause other complications such as deterioration in liver function, ascites and pulmonary edema, so immediate medical therapy is administered in acute variceal bleeding like vasopressors. Also prophylactic antibiotics administered due to the high risk of bacterial infection, In case of esophageal variceal bleeding EVL is the primary therapy, where EVO with cyanoacrylate is recommended in gastric varices. In acute vaiceal bleeding endoscopic treatment is difficult to be applied due to the high amount of bleeding which prevents the clear visual field and the bad general status which causes rabid deterioration during the procedure, so in this case balloon-occluded retrograde transvenous obliteration Balloon tamponade, TIPS, and surgical treatment including liver transplantation may be attempted [11].

Ascites

It is the most common decompensating complication of cirrhosis causing the highest mortality rate. So we aim to prevent the progress of the pathophysiological mechanisms that lead to the formation of ascites like hyponatremia, acute kidney injury and the splanchnic and systemic circulations vasodilatation [12], which lead to renal sodium and water retention and increased intravascular volume, causing hyperdynamic circulatory state with low systemic vascular resistance and high cardiac output. In case of ascites diuretics used to increase sodium excretion but the use of transjugular intrahepatic portasystemic shunt (TIPS) showed higher survival rates especially in those with a MELD <15 [13].

In advanced stages of cirrhosis, progressive vasodilatation Leads to the non-osmotic release of antidiuretic hormone or arginine vasopressin (AVP) causing an increase in water reabsorption and dilutional hyponatremia. So the use of V 2 receptor antagonists improves hyponatremia but with transient effect [14].

Treatment of underlying etiology and causes like Alcoholic liver disease, hepatitis B, hepatitis C and autoimmune hepatitis leads to improvement in ascites. Also salt restriction through dietary sodium restriction Limiting salt intake to 2000 mg/d or 88 mmol/d is recommended.

Diuretic regimens in the treatment of ascites include a combination of spironolactone and a loop diuretic(furosemide), unless the sodium level in the serum is less than 125 mEq per. Ascites patients in early stages should have diagnosis of cell count, albumin level, total protein test and bacterial culture for sensitivity, where Serum-ascites albumin concentration is used to calculate the dL (11 g per L) or greater, serum-ascites albumin gradient measured to confirm ascites. As If the serum-ascites albumin gradient is 1.1 g per L of portal hypertension so it is indicated that cirrhotic ascites or heart failure–associated ascites is confirmed, where if the value less than 1.1 g per dL it indicates another cause of ascites like peritoneal carcinomatosis or nephrogenic ascites.

Higher response rates have been reported in another trial which showed that diuretic and albumin combination resulted in shorted hospital stay, decreased readmission rates and lower recurrence rate [15].

Other diuretics like Triamterene, metolazone and hydrochlorothiazide used as asecond line treatment where eplenerone, torasemide, and bumetanide diuretics are expensive and did not well studied in the setting of cirrhosis and ascites. hydrochlorothiazide in combination with spironolactone and furosemide result in hyponatremia. Amiloride is justified in patients with tender gynecomastia due to its lower efficacy and high cost to spironolactone [16].

in the case of tense ascites paracentesis is considered a safe and effective therapeutic treatment followed by sodium restriction and diuretics [15].

NSAIDS are avoided in this case as they can reduce sodium excretion [17]. Also angiotensin converting inhibitors (ACEIs) and angiotensin receptor blockers are avoided as Arterial pressure is an independent predictor of survival in cirrhosis patient but dependent on high levels of vasopressin, angiotensin, and aldosterone.

Beta blockers should be used with care. Propranolol decreased survival rates in patients with refractory ascites [18].

Spontaneous Bacterial Peritonitis (SBP)

Bacterial infections are a common complication in hospitalized patients with cirrhosis [19]. Where third of the patients are exposed to infection which found to increase the mortality rates [20], and it often discovered when paracentesis is performed through the cytologic analysis of the ascitic fluid acquired by paracentesis [21].

spontaneous infections are The most common bacterial infections in cirrhosis like spontaneous bacterial peritonitis (SBP), spontaneous bacterial empyema (SBE) and spontaneous bacteremia which leads to UTIs and pneumonia [22].

These infections may lead to development of further decompensation with development of AKI, jaundice, coagulopathy and/or encephalopathy through worsening of vasodilatation and a systemic inflammatory state [23].

Empirical antibiotic treatment (third-generation cephalosporin) and early resuscitation of patients with severe sepsis or septic shock decrease the mortality [24].

Pathogen identified through a special microbiologic procedure where enteric gram-negative rods and streptococci form the preponderance of SBP pathogens.

Several antibiotic options including cefotaxime and ceftriaxone cephalosporins used in SBP management, where Patients should be evaluated after 48 hours in order to determine the effect of the antibiotic therapy.

Antibiotic Prophylaxis should be administered to all SBP patients where some reports suggest a role for fluoroquinolones in patients with a low concentration of ascitic fluid protein [25].

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