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Hepatorenal Syndrome Precipitated by Pneumonia in Patients with Chronic Liver Disease - A Case Report

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Abstract: ***Hepatorenal syndrome (HRS) is defined as a potentially reversible kidney failure in patients with cirrhosis and ascites. It is characterized by a marked reduction in glomerular filtration rate (GFR) and renal plasma flow. Pneumoniaand any other infection can be precipitant factors of hepatorenal syndrome. We report the case of a patient diagnosed with HRS, with rapid increased of serum creatinine levels which is normal 2 weeks prior to hospitalization.Hepatorenal syndrome can be managed effectively with albumin and vasopressin, and such treatment can be started as early as in the emergency department. Acute care physicians should not be hesitant in diagnosing and treating hepatorenal syndrome as early as in the emergency department for appropriate patients.***

Keywords: Hepatorenal Syndrome, Liver Cirrhosis, Infection

1. Introduction

Cirrhosis is the leading cause of liver related death globally. Cirrhosis is a result of advanced liver disease and is characterizedby fibrosis of liver tissue and conversion of normalarchitecture into regenerative nodules, leading to a loss ofliver function.[1]Most patients are asymptomatic and compensated in the initialstages of cirrhosis. In patients with compensated cirrhosis and become decompensated, its usually defined as the first occurrence of ascites,oesophageal variceal bleeding, hepatic encephalopathy,and, in some individuals, increased bilirubin concentration.Release of vasodilators and blood pooling in thesplanchnic circulationresults in renal hypoperfusion,with consequent activation of the renal–angiotensin–aldosteronesystem and fluid retention.[2]

Hepatorenal syndrome (HRS), a rapidly progressive form of acute renal failure that occurs in patients withcirrhosis and ascites in the absence of other causes of renalfailure can happen as a maladaptive vasodilatory response. It is characterized by a marked reduction in glomerularfiltration rate (GFR) and renal plasma flow.[1]We presented a case of hepatorenal syndrome precipitated by pneumonia in patient with liver cirrhosis admitted at Wangaya Regional Hospital Denpasar. Its important to exclude other kidney disease associated with liver disease such as glomerulonephritis, IgA nephropathy (frequently associated in ethanolic patients) or renal distal tubular acidosis etc before diagnose HRS. The exclusion is done usually clinically, in the absence of proteinuria and in the absence of cells and casts in the urine.[4]

This case presentation is substantial as a reminder regarding the importance of risk factors identification in hepatorenal syndrome, as this complication is highly possible to be encountered especially by primary care provider in their practices, so the treatment can be started as early as in the emergency department.

2. Case Report

A 66-year-old female was admitted due to rapidabdominal distension. She also complaint a significant decrease of urine output. The patient has a past medical history that is significant for end-stage liver cirrhosissince4 years. She had also been recently admitted for altered mental status and had been treated for recurrent hepatic encephalopathy.She was alert and oriented and in apparent shortness of breath due to distended abdomen. Vital was within normal limit. Other physical examination findings showed stigmata of chronic liver disease. Abdominal examination showed grade III ascites with collateral vein formation (Figure 1). Liver and spleen were hard to evaluate due to marked abdominal distention. No peripheral oedema was found. Laboratory examination revealed leukocytosis (19.90x103 /µL) with neutrophil predominance (75.7%) and low lymphocyte (10.2%), mild normochromic normocytic anaemia (Hb=8.6 g/dl; MCV=94.1 fL; MCH: 33.7pg). Neutrofil/lymphocyte ratio was found to be high (NLR ratio=7.43). Liver enzyme test showed no marked elevation (SGOT=56; SGPT=18). Protein analysis showed low albumin level (2.1 g/dl). Urinalysis examination showedno proteinuria or haematuria. Bilirubin was also seen in the urine of our patient. Serum ureum and creatinin level was found to be high (ureum 92 mg/dl; serum creatinine 3.1 mg/dl). Chest x-ray revealed suspected pneumonia (Figure 2). The patient was started on branchedchain amino acid infusion, dextrose 10 % and sodium chloride infusion, proton pump inhibitor, lactulose, and third-generationcephalosporin antibiotic intravenously. Diuretic was also given to reduce water retention. Norepinephrine also given to the patient as a vasoconstrictor agent. The patient undergo paracentesis after 5 days hospitalization due to hard to breathe. Albumin transfusion were given prior to paracentesis procedure.

Figure 1: Grade III ascites with collateral vein formation

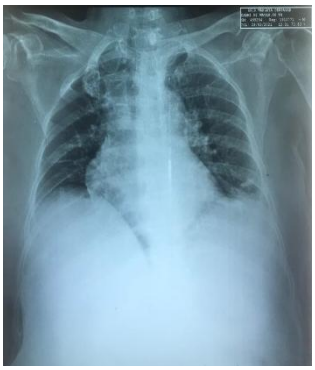

Figure 2: Chest x-ray

3. Discussion

Our case presented rapid abdominal swelling and significant decreasing urine output, that associated with a past medical history significant for end-stage liver cirrhosis. Review of systems was positive for jaundice and distended abdomen. Renal function test shows high level of serum creatinine which was normal 2 weeks ago. The chest x-ray shows infiltrate that suggestive for pneumonia. The workup disclosed probable pneumonia induced hepatorenal syndrome.

Hepatorenal syndrome (HRS) have been describe in types I and II. Type I HRS is characterized by acute onset and rapidly progressing kidney failure with a doubling of serum creatinine to > 2.5 mg/dL in less than 2 weeks. Type II HRS is less severe than type I and its renal function decline progression is more gradual.Type II HRS predisposes patients to the development of type I HRS after a precipitating event.[5]Ourpatient's condition fits the type I hepatorenal syndrome associated with her cirrhosis and is characterized by a significantly increased serum creatinine concentration and a no parenchymal kidney disease proven.

Splanchnic vasodilationas a consequence of portal hypertension is mediated principally by nitric oxide and other vasodilator substancessuch as carbon monoxide, glucagon, vasodilator peptides, and others. Splanchnic vasodilation sequesters bloodin the splanchnic vascular bed leading to a reduced effectivearterial blood volume. At a late stage, the cardiac output may also fall due to thedevelopment of cirrhotic cardiomyopathy. As the effectivearterial blood volume declines, renin-angiotensin-aldosterone system (RAAS), sympathetic nervoussystem (SNS), and arginine vasopressin are stimulated.This causes sodium and water retention, leading in turn toascites and hyponatraemia, as well as vasoconstriction of therenal. The end result ofthis process is a severe decline in renal blood flow leadingto reduced glomerular filtration rate (GFR) and the developmentof HRS.[3]

Oral Spironolactone and furosemide injection are given to the patient since first hospitalization. The use of furosemide as a single agent has been demonstrated to be less efficacious than spironolactone. In the largest, multicenter, randomized controlled trial performed in patients suffering from ascites, dietary sodium restriction and a dual diuretic regimen with spironolactone and furosemide has been shown to be useful in more than 90% of patients in accomplishing a reduction in the volume of ascites to satisfactory levels.[6]

Bacterial infections are a frequent trigger of HRS probably through a cytokine-mediated impairment of circulatory function, worsening effective hypovolemia. The severity of liver failure, bacterial infection is an important determinant of the high mortality rate in patients with type-1 HRS.[7]The commonest infection in cirrhotic patients is spontaneous bacterial peritonitis (SBP), followed by urinary tract infection, and in this patientsis pneumonia.[8] Third generation of cephalosporine were given to the patient as a broadspectrum antibiotics to treat the bacterial infections.

Elevated intraabdominal pressure may play a role in the development of hepatorenal syndrome in patients with refractory ascites. Therapeutic paracentesis may be required in patients who having severe (tense) ascites. Albumin may sometimes be given intravenously in the amount equal to the proportion of ascites removed, as this method may decline serum albumin levels in blood.[6]Serial large volume paracentesis (4–6 l/day) with albumin infusion (8 g/litre of ascites removed) was more effective and was associated with fewer complications and shorter duration of hospitalisation compared with diuretic therapy.[8]

Vasoconstrictive agent may be used to treat type I hepatorenal syndrome as splanchnicvasodilatation plays a key role in the pathogenesis of HRS. Terlipress insignificantly decreases plasma renin and aldosterone, with an improvement in glomerular filtration rate (GFR) in patients with type I HRS but its not available in some region, especially in our hospital. So, other vasoconstrictive agents currentlybeingused in HRS treatment are somatostatin analogues(octreotide), α-adrenergic agonists, midodrine andnorepinephrine.[9]Norepinephrine is used in this patient because of its availability in our region and is has proved effective in the treatment of HRS and must be associated with administration of albumin.[10]

4. Conclusion

Hepatorenal syndrome is a serious complication of cirrhosis of the liver and carries a high mortality. The pathogenesis of HRS is complex and incompletely understood, but it is postulated that splanchnic vasodilation reduces effective circulating volume, which in turn leads to renal hypoperfusion. Infection can be the precipitating factors of HRS in patient with cirrhosis. Preventing development of

hepatorenal syndrome can be done by preventing progression of liver disease in the well compensated patient, avoiding agents known to exacerbate AKI, and preventing factors that further impair circulatory status and reduce kidney perfusion. Clinicians should weigh the risks and benefits of continuation of nonselective b-blockers on an individual basis in patients with refractory ascites.[11]Medical treatment for hepatorenal syndrome is a supportive therapy until the definitive therapy for HRS which is liver transplant is provided.

5. Declarations

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