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Case Report

Case Report on AKI on CKD and Hepatic Renal Syndrome

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Abstract:
Hepatorenal syndrome (HRS) is a severe form of kidney dysfunction associated with cirrhosis and portal hypertension. It is characterized by renal vasoconstriction without significant histological changes in the kidneys, leading to renal failure. This case report discusses a 72-year-old male with a history of decompensated chronic liver disease (DCLD) and HRS, presenting with abdominal pain, distension, decreased urine output, shortness of breath, and pedal edema. The patient had a history of gastrointestinal bleeding, recurrent blood transfusions, and poor dietary habits, including chronic alcohol consumption and smoking. Laboratory findings revealed elevated blood urea, serum creatinine, and uric acid, indicating impaired renal function. Severe anemia and neutrophilic leukocytosis suggested chronic disease and an active infection, likely spontaneous bacterial peritonitis (SBP). Imaging studies showed gallbladder wall edema, ascites, and Grade 3 esophageal varices, confirming portal hypertension. The patient was diagnosed with decompensated cirrhosis, acute kidney injury superimposed on chronic kidney disease, anemia of chronic disease, and suspected SBP. Management involved a multidisciplinary approach, including nephrology and hepatology specialists. The treatment plan included antibiotics (cefotaxime and metronidazole) to treat SBP, medications to control portal hypertension (octreotide and carvedilol), erythropoietin for anemia, and supportive treatments for hepatic encephalopathy. Despite aggressive management, the prognosis remains poor, emphasizing the need for early liver transplantation in patients with advanced HRS. This case highlights the complex interplay of liver and kidney dysfunction in cirrhosis and underscores the importance of early intervention and multidisciplinary care in improving patient outcomes.
Keywords: Hepatorenal Syndrome (HRS), Decompensated Chronic Liver Disease (DCLD), Acute Kidney Injury (AKI), Spontaneous Bacterial Peritonitis (SBP), Portal Hypertension

Introduction

Hepatorenal syndrome (HRS) is a form of kidney function impairment that characteristically occurs in cirrhosis. Recent changes in terminology have led to acute HRS being referred to as acute kidney injury (AKI)-HRS and chronic HRS as chronic kidney disease (CKD)-HRS (1). AKI-HRS is characterized by a severe impairment of kidney function owing to vasoconstriction of the renal arteries in the absence of substantial abnormalities in kidney histology. Pathogenetic mechanisms involve disturbances in circulatory function due to a marked splanchnic arterial vasodilation, which triggers the activation of vasoconstrictor factors (2). An intense systemic inflammatory reaction that is characteristic of advanced cirrhosis may

also be involved. The main triggering factors of AKI-HRS are bacterial infections, particularly spontaneous bacterial peritonitis. The diagnosis of AKI-HRS is a challenge because of a lack of specific diagnostic tools and mainly involves the differential diagnosis from other forms of AKI, particularly acute tubular necrosis. The prognosis of patients with AKI-HRS is poor, with a median survival of ≤3 months. The ideal treatment for AKI-HRS is liver transplantation in patients without contraindications. Medical therapy consists of vasoconstrictor drugs to counteract splanchnic arterial vasodilation together with volume expansion with albumin. Effective measures to prevent AKI-HRS include early identification and treatment of bacterial infections and the administration of albumin in patients with spontaneous bacterial peritonitis.

Hepatorenal syndrome is a form of functional renal failure arising from liver cirrhosis secondary to diminished renal bloodflow (3).Typically kidneys are histologically normal . Portal hypertension leads to vasodilatation of splanchnic vessels, decrease in systemic vascular resistance, and activation of the renin-angiotensin-aldosterone causing vasoconstriction of renal arteries. Characteristically, HRS affects cirrhotic patients with ascite. Two types of HRS have been described. Type 1 is characterized by acute rapid decrease in kidney function and progressive kidney failure in less than 2 weeks. Prognosis is poor with high mortality (90%) within 3 months (4). Type 2 HRS presents as a less severe kidney failure and gradual decline in renal function and longer survival than type 1 HRS. The rate of progression of renal impairment is the main diagnosis criteria between the two types of HRS.

Anemia causes microcirculatory tissue hypoxia. This phenomenon is called anemic hypoxia. Renal ischemia is the most common cause of HRS. Renal tissue hypoxia or ischemia can trigger the initial tubular damage. Anemia is frequently observed during the clinical course of cirrhosis. There are rare publications addressing the potential or additive effects of anemia on HRS in cirrhotic patients (5).

Case Report

A 72-year-old male with a known history of hepatorenal syndrome (HRS) and decompensated chronic liver disease (DCLD) presented to the hospital with multiple systemic complaints. The patient reported abdominal pain localized to the right iliac fossa, progressive abdominal distension, decreased urine output, and shortness of breath classified as Grade 3–4 on the Modified Medical Research Council (MRC) Dyspnea Scale. He also exhibited bilateral pedal edema, most pronounced around the ankles, and had a history of melena and hematuria. His past medical history was notable for recurrent blood transfusions, including a recent episode, in the context of chronic liver disease.

In light of his presenting symptoms and underlying medical conditions, a comprehensive diagnostic workup was initiated. The investigations included Liver Function Tests (LFT), Complete Blood Picture (CBP), Renal Function Tests (RFT), cytology, uric acid levels, iron profile, stool examination, ultrasonography (USG) of the abdomen and pelvis, and upper gastrointestinal endoscopy. The laboratory results revealed significant abnormalities, including elevated blood urea (172 mg/dL), elevated serum creatinine (2.7 mg/dL), and increased uric acid (9.6 mg/dL), indicating impaired renal function. A marked neutrophilic leukocytosis was evident with neutrophils at 90%. Hematological analysis showed severe anemia with hemoglobin at 3.8 g/dL and a low red blood cell (RBC) count of 1.59 million/cmm. The iron profile was deranged, showing low serum iron (40 µg/dL) and reduced iron saturation (18.6%). Additionally, hypoalbuminemia was noted, with a serum albumin level of 2.5 g/dL.

Imaging studies supported these findings. USG of the abdomen revealed gallbladder wall edema and gross ascites. Upper gastrointestinal endoscopy demonstrated features of portal hypertension and high-grade (Grade 3) esophageal varices. Based on the clinical presentation, imaging, and laboratory findings, a

final diagnosis was made of decompensated chronic liver disease (DCLD) with portal hypertension, Grade 3 esophageal varices, acute kidney injury (AKI) superimposed on chronic kidney disease (CKD), anemia of chronic disease, and ascites secondary to portal hypertension.

The patient was managed collaboratively by nephrology and hepatology specialists. Antibiotic therapy included intravenous cefotaxime 2 g for the treatment or prevention of spontaneous bacterial peritonitis and intravenous metronidazole 500 mg for anaerobic coverage. Supportive medications included intravenous vitamin K 10 mg to correct coagulopathy, intravenous octreotide 250 mcg to manage variceal bleeding and reduce portal pressure, and subcutaneous erythropoietin 4000 IU to stimulate erythropoiesis and manage anemia. Oral medications prescribed were carvedilol (Cardivas) 3.125 mg as a non-selective beta-blocker to control portal hypertension, rifaximin 550 mg to prevent hepatic encephalopathy, febuxostat (Feblar) 40 mg to address hyperuricemia, sodium bicarbonate (Nodosis) 500 mg for metabolic acidosis management, and Evion LC (a combination of vitamin E and L-carnitine) for nutritional support. Additionally, syrup sucralfate was administered for gastrointestinal mucosal protection, and syrup lactulose (Duphalac) was given to reduce ammonia levels and prevent hepatic encephalopathy.

Discussion:

Clinical Summary and Significance

The patient is a 72-year-old male with a known history of hepatic dysfunction attributed to poor dietary habits, chronic alcohol consumption, and smoking. He presented with complaints suggestive of progressive liver and renal deterioration, including right iliac fossa abdominal pain, abdominal distension, decreased urine output, shortness of breath (Grade 3–4 on the MRC Dyspnea Scale), pedal edema, melena, and hematuria. These symptoms indicate significant systemic decompensation, consistent with decompensated chronic liver disease (DCLD) and hepatorenal syndrome (HRS).

Clinical Presentation and Interpretation

The abdominal pain and distension are classical features of ascites, which is typically seen in patients with cirrhosis and portal hypertension. Decreased urine output in the setting of cirrhosis suggests acute kidney injury (AKI), likely superimposed on chronic kidney disease (CKD), consistent with HRS. The shortness of breath may be attributed to fluid overload or hepatic hydrothorax, often associated with advanced liver disease. Pedal edema is frequently caused by hypoalbuminemia and portal hypertension. A history of melena and hematuria raises the suspicion of gastrointestinal bleeding, likely from ruptured esophageal varices, a known complication of portal hypertension. The patient's history of multiple blood transfusions supports the presence of chronic anemia.

Laboratory and Diagnostic Findings

Laboratory evaluations showed elevated levels of blood urea (172 mg/dL) and serum creatinine (2.7 mg/dL), confirming renal impairment. Uric acid levels were also raised (9.6 mg/dL), supporting renal dysfunction. Severe anemia was evident with hemoglobin at 3.8 g/dL and RBC count of 1.59 million/cmm. Iron studies revealed low serum iron (40 µg/dL) and reduced iron saturation (18.6%), indicating iron deficiency anemia on a background of chronic disease. A marked neutrophilic response (90%) suggested an ongoing infection, such as spontaneous bacterial peritonitis (SBP). Hypoalbuminemia (2.5 g/dL) further indicated poor hepatic synthetic function.

Ultrasound imaging revealed gallbladder wall edema and gross ascites, findings typical in advanced cirrhosis. Endoscopic examination showed Grade 3 esophageal varices with evidence of portal hypertension, confirming a high risk for gastrointestinal bleeding.

Clinical Diagnosis

Based on clinical symptoms, laboratory data, and imaging findings, the patient was diagnosed with:

- Decompensated Chronic Liver Disease (DCLD)
- Portal Hypertension with Grade 3 Esophageal Varices
- Acute Kidney Injury (AKI) on Chronic Kidney Disease (CKD)
- Anemia of Chronic Disease with iron deficiency
- Suspected Spontaneous Bacterial Peritonitis (SBP)

Management and Therapeutic Plan

The multidisciplinary team, including nephrologists and hepatologists, initiated an integrated treatment regimen:

1. **Infection Management**:
    - Inj. Cefotaxime 2 g IV and Metronidazole 500 mg IV were prescribed empirically for SBP and gut-related infections.
    - Vitamin K 10 mg IV was administered to correct coagulopathy commonly seen in liver failure.
2. **Management of Portal Hypertension**:
    - Octreotide 250 mcg IV, a somatostatin analogue, was used to reduce portal pressure and manage variceal bleeding.
    - Carvedilol (Cardivas) 3.125 mg was initiated as a non-selective beta-blocker to further control portal hypertension and prevent bleeding recurrence.
3. **Renal and Anemia Support**:
    - Erythropoietin 4000 IU SC was given to stimulate erythropoiesis, particularly important due to compromised kidney function and chronic anemia.
    - Febuxostat (Feblar) 40 mg was prescribed to manage elevated uric acid levels.
    - Iron supplementation and supportive measures were considered to address the iron-deficiency component of anemia.
4. **Gastrointestinal and Hepatic Encephalopathy Management**:
    - Rifaximin 550 mg was used to prevent hepatic encephalopathy by modulating gut microbiota.
    - Sodium Bicarbonate (Nodosis) 500 mg helped manage acid-base imbalances.
    - Sucralfate syrup was used for mucosal protection, while Lactulose (Duphalac syrup) reduced systemic ammonia levels to prevent encephalopathy.
    - Evion LC, a combination of Vitamin E and L-carnitine, was included as nutritional support and antioxidant therapy.
5. **Supportive and Nutritional Care**:
    - Focused nutritional support and monitoring were emphasized, given the patient's hypoalbuminemia, anemia, and general debility.

Prognostic Considerations

The patient's outcome depends on multiple interrelated factors:

- Prompt and effective treatment of suspected SBP is crucial.
- Control of portal hypertension and prevention of further gastrointestinal bleeding significantly impacts survival.
- Renal recovery is uncertain, and long-term dialysis may be considered if renal function continues to decline.
- Ongoing management of liver function and prevention of encephalopathy are vital for improving quality of life.
- The severity of anemia and nutritional deficiencies also require regular reassessment.

Conclusion:

This case shows the complex interplay between hepatic and renal dysfunction in patients with advanced cirrhosis, highlighting the diagnostic and therapeutic challenges of hepatorenal syndrome (HRS) in the setting of decompensated chronic liver disease (DCLD).

The patient presented with multiple life-threatening complications including acute kidney injury on chronic kidney disease, severe anemia, spontaneous bacterial peritonitis, and high-grade esophageal varices all indicative of multi-organ involvement and systemic decompensation. Timely recognition of HRS, along with a comprehensive and multidisciplinary approach involving targeted antibiotic therapy, hemodynamic support with vasoconstrictors, correction of coagulopathy, and management of portal hypertension and hepatic encephalopathy, was essential in stabilizing the patient's condition. Nutritional support and anemia correction played a key role in improving overall systemic function. Given the poor natural prognosis of HRS, particularly in the context of Type 1 disease, this case reinforces the critical need for early intervention, ongoing monitoring, and consideration of liver transplantation as a definitive treatment. Early identification and treatment of infections, regular surveillance for variceal bleeding, and optimization of renal function are pivotal in improving both survival and quality of life in such patients.

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