Kidney failure due to liver cirrhosis – hepatorenal syndrome: clinical case

Madina Jumayeva*, Dilafruz Ubaydova, Gulrux Xayrullayeva, Zarina Kadirova, Bobur Ergashov, and Dildora Narzullayeva

Bukhara State Medical Institute, Bukhara, Uzbekistan

Abstract. These days, kidney failure is a big problem globally and can even lead to death for patients. There are many things that can cause kidneys to stop working properly, and liver diseases are especially important. This condition is called hepatorenal syndrome and happens when the kidneys stop working properly after liver cirrhosis. Recent information shows that there are two types of hepatorenal syndrome, which are named type 1 and type 2. Type 1 hepatorenal syndrome usually changes quickly and can be very serious, potentially leading to death if not treated within 2 weeks. Type 2 hepatorenal syndrome changes more slowly over time and has a better outlook, but can still lead to death within 4–6 months if not treated. Cases of type 2 hepatorenal syndrome (HRS) are not often talked about in medical papers, and studies about this serious problem of liver cirrhosis involve a small number of people.

1 Introduction

In our case think about, we portray the confirmation within the nephrology division of a 59-year-old male with direct azotate maintenance and serious hyponatremia. He displayed too with cryptogenic liver illness (show for end-stage liver malady Merge 35, Child-Turcotte-Pugh 12), headstrong ascites and spontaneous bacterial peritonitis, at the side serious unremitting hyponatremia and direct renal brokenness. Since the understanding upon affirmation met the major criteria for diagnosis of hepatorenal disorder sort 1, he was treated with repeated huge volume paracentesis beside anti-microbial treatment, in combination with plasma mixture and noradrenaline, accomplishing a moderate but favorable advancement, with fractional recuperation of kidney work. Within the taking after 9 months, serum creatinine remained moderately steady, between 1.38 and 1.55 mg/dL, making us put the positive conclusion of Type-2 HRS. The persistent is now beneath multidisciplinary monitorization (gastroenterology and nephrology divisions).

The details of this situation were: there was a moderate improvement in a quiet with type 2 hepatorenal syndrome. The patient wasn't following the treatment plan and had social issues. It was difficult to include him on the waiting list for a liver transplant because he had a history of chronic alcohol abuse. In conclusion, hepatorenal syndrome is mainly about avoiding mistakes, but the process can be complicated, serious, and prone to errors.

* Corresponding author: farxodjonjumayev@gmail.com

© The Authors, published by EDP Sciences. This is an open access article distributed under the terms of the Creative Commons Attribution License 4.0 (https://creativecommons.org/licenses/by/4.0/).
Advanced chronic liver disease, especially in individuals with ascites, frequently results in kidney failure, which is typically brought on by one or more situations that further impair renal perfusion. However, we must take into account that there are a number of factors, particularly the muscular wasting that is characteristic of cirrhotic individuals, which reduce the diagnostic adequacy of serum creatinine [1].

In fact, less than 10% of all occurrences of acute kidney injury (AKI) in patients with liver illness are due to hepatorenal syndrome (HRS), with other causes such as volume unresponsive AKI and acute tubular necrosis being significantly more prevalent.

Vasoconstriction of the renal arteries causes functional impairment of the kidneys in HRS, although the tubular function is retained and the renal histology is nearly normal. It can happen without any precipitating events and spontaneously, as a result of well-known triggers such as spontaneous bacterial peritonitis (SBP) [2].

In 1996, the International Ascites Club (IAC) published the diagnostic criteria for HRS, later revised and updated (2007). The revised IAC criteria include: “(I) cirrhosis with ascites, (II) serum creatinine level >1.5 mg/dL, with removal of creatinine clearance, (III) no improvement in serum creatinine after ≥ 2 days with diuretic withdrawal and volume expansion with albumin, rather than saline for plasma expansion, (IV) absence of shock or recent use of nephrotoxic drugs, (v) absence of parenchymal kidney disease, indicated by proteinuria >500 mg/day, microscopic hematuria (>50 red blood cells per high power field), and/or abnormal renal ultrasonography, and last, but not least, removal of the previous minor diagnostic criteria (urine volume, urinary and serum sodium)” [3].

There are two different forms of HRS that are described based on their progression and the existence of triggering factors: Prerenal AKI, or Type-1 HRS, which is characterized by an acute decline in renal function and is typically brought on by a bacterial infection (SBP being the most frequent trigger), and Type-2 HRS, which is thought to be a more chronic form of renal dysfunction and is frequently linked to a diuretic-resistant ascites [4].

HRS manifests clinically as a pre-renal failure that does not respond to volume expansion. In cirrhotic people with disabilities, HRS must be regarded as a diagnostic of exclusion of alternative causes of AKI. The most recent data suggest that HRS has the worst prognosis of all liver cirrhosis complications and the greatest death rate (more than 50% of such patients pass away within a few-month period). Vasoconstrictors and volume expansion with albumin are used to treat HRS, although their efficacy is limited (40–50% of patients) and recurrences are common. Thus, orthotopic liver transplantation (OLT) is currently the only long-term treatment strategy; nevertheless, it is constrained by a high mortality rate and a dearth of suitable grafts [5,6].

Case presentation:
On January 5, 2023, a 59-year-old patient was admitted to the hospital with complaints of general weakness, increased fatigue, pain in the right hypochondrium, decreased appetite, an increase in the volume of the abdomen, swelling in the legs, nausea, frequent nasal and gingival bleeding, itching, yellowness of the skin and mucous membranes, the appearance of red painless spots on the skin of the trunk, both hands, with clinical manifestations of hepatocellular insufficiency, hepatic encephalopathy, hypersplenism, cholestasis, moderate edema, portal hypertension, ascites. In 2005, the patient was diagnosed with hepatitis C, but was treated irregularly. In 2015, diabetes was diagnosed as a concomitant disease. In 2019, he was diagnosed with cirrhosis of the liver. After that, the patient began to take antiviral drugs. General condition of moderate severity. Consciousness is clear. The skin is clean. Peripheral lymph nodes are not enlarged. The subcutaneous fat layer is moderately developed. Breathing through the nose, number 18 in 1 minute. Vesicular breathing in the lungs. The borders of the heart are not changed. Heart sounds are muffled, systolic murmur at the apex of the heart. Pulse 88 in 1 minute. blood pressure 110/80 mm Hg. Tongue wet, crimson. The abdomen is soft, enlarged in volume with...
chemochemical blood test on January 6, 2023

In January 2023, the patient underwent a biochemical blood test. The laboratory data showed a glucose level of 7 mmol/l, calcium level of 2.0 mmol/l, sodium level of 147 mmol/l, potassium level of 4.0 mmol/l, total protein level of 65 g/l, creatinine level of 118 g/l, and urea level of 77 mmo / l. The patient was diagnosed with diabetes mellitus type 2. The laboratory data also showed a total bilirubin level of 3.7 mmol/l, AST level of 28 mmol / l, GFR level of 25.7 mmol. /l, total protein level of 65 g/l, creatinine level of 96 mmol/l, direct bilirubin level of 17.6 (group 4) glucose level of 34.7, Hb level of 139 IU/l, ALT level of 139 IU / l, total protein level of 63%.

Heart sounds are muffled, systolic murmur at the apex of the heart. Pulse 75 in 1 minute. blood pressure 110/80 mm Hg. Tongue wet, epigastric region and unde

crimson. The abdomen is soft, ascitic fluid has decreased significantly, painful in the epigastric region and und

e.

Peripheral lymph nodes are not enlarged. The subcutaneous fat layer is moderately developed. Breathing through the nose, number 18 in 1 minute. Vesicular breathing in the lungs. The borders of the heart are no


Laboratory data: Biochemical blood test 04/09/23

The patient was also diagnosed with an umbilical hernia, for which the surgeon recommended surgical treatment, as well as diabetes mellitus.

Diabetes mellitus type 2. Umbilical hernia.

Liver cirrhosis of viral etiology. Class C by Child Pugh.

Complication:

Concomitant pathology:

Competitive disease:

Main disease:

Echocardiography

EKG

Ultrasound of the general abdominal cavity for the presence of ascites. Portal hypertension. Ascites. Diffuse increase in the echogenicity of the pancreatic parenchyma.


Liver cirrhosis of viral etiology. Class C by Child Pugh.

Expansion of varicose veins of the esophagus. 2 degrees.

2 Result

Traditionally, kidney and heart have been thought to represent two separate complications of progressive liver disease with distinct pathophysiological pathways; however, new evidence suggests that there is an association between the two organs, known as cardiorenal syndrome (CRS), which may affect kidney function in some patients [10]. In HRS type 2, renal failure remains relatively stable, making it difficult to conduct large-scale studies. A recent study showed that only 5% of hospitalized patients with cirrhosis of the liver and kidney failure have HRS type 2 [11].

In 2007, the International Ascites Club (ICA) classified HRS into types 1 and 2 (HRS-1 and HRS-2). HRS-1 is characterized by a rapid deterioration in renal function, which often occurs due to a triggering event, while HRS-2 represents moderate and stable or slowly progressive renal dysfunction. Clinically, HRS-1 is characterized by acute kidney injury, while HRS-2 is mainly characterized by refractory ascites [12]. The most common trigger for the development of HRS-1 is a bacterial infection, mainly spontaneous bacterial peritonitis, as well as paracentesis with the removal of a large volume of ascitic fluid without adequate administration of albumin. [13,14].

HRS has no specific clinical data. His physical findings generally reflect underlying progressive liver disease, acute kidney injury, and underlying circulatory disturbances. In the clinic prevail, respectively: ascites, jaundice, signs of liver failure and encephalopathy, gastrointestinal bleeding. Also, one of the important indicators is a decrease in diuresis <500 ml [6,8,15]. The accumulation of ascitic fluid, decreased diuresis, increased blood bilirubin, ALT, AST, urea, creatinine, decreased protein levels in this patient indicate the development of the syndrome. The fact that these indicators decreased by a certain amount due to the application of legal measures, but did not return completely to the norm, indicates its stability.

3 Conclusion

HRS is a rare but severe form of kidney dysfunction found in people with cirrhosis of the liver and is a common cause of death in these patients. The diagnostic process can be complex and lengthy, so in some cases where a patient does not meet all of the diagnostic criteria for the IAC, we may initiate treatment for "suspected" HRS based on clinical suspicion. Patients with HRS type 2 have slower progression of kidney failure and a better prognosis if they are eligible for liver transplantation. In this case, the patient rapidly developed HRS due to damage to the heart and liver; fortunately, over time there was some improvement, but not a complete remission, as laboratory changes in the blood show that the kidney function is impaired and the process has deepened.

References

3. F. Nurutdinova, Z. Tuksanova, Y. Rasulova, E3S Web of Conferences 474, 01002 (2024)
M. F. Jumaeva, Asian Journal of Pharmaceutical and Biological Research 11(3), 72-77 (2022)

M. F. Jumayeva, Asian Journal of Pharmaceutical and Biological Research 11(1), 2022

M. F. Jumayeva, Problems Biology and Medicine 6(140), 80-82 (2022)


N. B. Mukhamadieva, European Journal of Molecular and Clinical Medicine 7(11), 418-426 (2020)


G. Sobirova et al., Journal of Critical Reviews Evaluation of the gastrointestinal mucosa by the OLGA system in chronic atrophic gastritis (2021)


