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THE SECOND TYPE OF HEPATORENAL SYNDROME – CASE REPORT AND LITERATURE REVIEW

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Abstract. Hepatorenal syndrome (HRS) is a severe complication that occurs during the decompensation of liver cirrhosis. Although the most characteristic feature of the syndrome is functional renal failure due to severe renal vasoconstriction, this process is a more general process that also affects the heart and brain. There are two types of HRS. Type 1 HRS is marked by a rapidly progressive deterioration of circulatory and renal functions associated with a very poor prognosis (median survival is less than 2 weeks). Type 2 HRS is characterized by stable impairment of blood circulation and renal function, with an average survival of 6 months The primary culprit behind HRS is the expansion of vessels in the portal system, leading to decreased blood flow to the heart and ultimately lower blood volume leaving it. Treating HRS includes administering albumin or vasoconstrictors intravenously or correcting portal hypertension with a transjugular intrahepatic portacaval shunt; each of these treatments may serve as a bridge to liver transplantation, which is the preferred solution for these patients.

Key words: hepatorenal syndrome, clinical case, cardiohepatorenal syndrome

Introduction.

Kidney failure is common in advanced chronic liver disease, particularly in those with ascites. This is often due to events that reduce renal perfusion However, we must take into account that there are multiple factors that decrease the accuracy of serum creatinine measurements in cirrhotic patients - including muscle wasting that is typical for these patients [1 Although hepatorenal syndrome (HRS) isn't the most common form of acute kidney injury (AKI) seen in those with liver disease, it still accounts for less than 10%. Other causes, such as volume-unresponsive AKI and acute tubular necrosis occur more frequently. HRS can be either spontaneous or due to known triggers such as spontaneous bacterial peritonitis (SBP), characterized by impaired renal function due to vasoconstriction of the renal arteries and normal renal histology [2]. Its pathophysiology includes hyperactivity of the sympathetic system and and renin-angiotensin-aldosterone systems RAAS; elevation of nitric oxide and systemic vasodilatation [3]. HRS is divided into two types: type I has a rapid elevation of serum creatinine (over twice the normal range) with a creatinine clearance down to 50%, usually reaching up to 2.5mg/dL [4]. HRS type II has a more insidious and less aggressive course, and its hallmark is ascites refractory to diuretics.

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The former usually presents with oliguria (less than 400-500ml/day) and can respond well to therapies for half of those affected [5]. .

In 1996, the International Ascites Club (IAC) released the initial diagnostic criteria for HRS, which was later revised and updated (2007). He 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 aier ≥ 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 an open access 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)" [6].

The clinical expression of HRS usually presents itself as pre-renal failure which does not respond to increased volume expansion. When diagnosing HRS, it is important to rule out other causes of AKI in cirrhotic patients. [7]. The latest data shows that HRS carries the worst prognosis among all complications associated with liver cirrhosis - mortality rate exceeding 50% within several months [8]Treatment of HRS nvolves the use of vasoconstrictors and volume expansion with albumin, , however, this approach has low success rate (40-50%) and recurrent episodes are common. Ultimately, orthotopic liver transplantation is often considered to be the most effective long-term treatment; it is restricted only by high mortality rates and lack of suitable donor organs [9].

Case presentation : The patient is 59 years old come to hospital with complaints like general weakness, fatigue, pain in the right hypochondrium, decreased appetite, increased abdominal volume, swelling on the legs, nausea, frequent nasal and gingival bleeding, itching, jaundice of the skin and mucous membranes, the appearance of red painless spots on the skin of the trunk, both hands, having clinical manifestations of hepatic cell insufficiency, hepatic encephalopathy, hypersplenism, cholestasis, moderate edematous, portal hypertension, ascites. The patient was diagnosed with hepatitis C in 2005, but received irregular medical treatments. In 2015, he was diagnosed with diabetes mellitus. In 2019, he was diagnosed with liver cirrhosis. Diabetes was diagnosed in 2015. In 2019, liver cirrhosis was diagnosed. After that, the patient started taking anti-viral drugs. The patient grew up in satisfactory conditions. The patient had the following examination results: Biochemical blood analysis 06.01.23 — total protein — 65 g/l, albumin —34 g/l , creatinine — 187 mkmol/l, glucose — 7.8 mmol/l, urea — 25,7 mmol/l, ALT— 66 ME/l, AST —139 ME/l, total bilirubin — 77 mmol/l, direct bilirubin — mmol/l 28, alkaline phosphatase — 187 ME/l, alpha-amylase — 32 U/l, calcium — 2.2 mmol/l , magnesium — 0.6 mmol/l . General blood analysis — Hb-118g/l, erythrocytes — 3,7,0x10*12/l, leukocytes — 9,0x10*9/l, platelet — 254,0x10*9/l, ESR — 7 mm/h. Blood clotting time — beginning : 2-50 min, ending : 3-10 min EGDS — Acute

hemorrhagic ulcer of duodenal bulb. Hemorrhagic erosive duodenitis. Dilatation of esophageal varicose veins. 2 degrees. Ultrasound general abdominal cavity — Hepatosplenomegaly. Cirrhosis of the liver. Portal hypertension. Ascites. Diffuse increased echogenicity of the pancreatic parenchyma. ECG — sinus rhythm, correct. heart rate - 63. hypoxic T wave in the anterior-disseminated area. EchoCG — moderate aortic valve stenosis. Dilatation of the right heart and left atrium. Moderate pulmonary hypertension. Pronounced hypertrophy of the wall of the left ventricle. calcification of the aortic valve leaflet. Sclerosis of the mitral valve . Dilatational dysfunction of the left ventricle, mitral regurgitation 1 degree, aortic regurgitation 1-2 degree.

Discussion. It has been traditionally taught that the kidney and heart are two separate complications of worsening liver disease with distinct pathophysiologic pathways; however, emerging evidence suggests that there is a connection between these two organs, known as cardiorenal syndrome (CRS), which could be impacting renal function in a subset of patients. [10].Here we will discuss the potential of CRS being present in hepatorenal syndrome (HRS) and its implications for treatment. In type 2 HRS, renal impairment remains relatively stable, making it difficult to conduct large-scale studies. A recent study found that only 5% of hospitalized patients with cirrhosis and kidney failure had type 2 HRS. [11] On top of the protocol treatment for underlying disease, the patient was additionally prescribed bisoprolol, veraspirone and ursosan; however, their condition did not improve. The patient was also diagnosed with umbilical hernia for which surgical treatment was recommended by their surgeon as well as diabetes for which diaglizid was prescribed by an endocrinologist and hepatorenal syndrome by the nephrologist Laboratory data: : Biochemical blood analysis 09.01.23 — total protein — 68 g/l, creatinine — 306 mkmol/l, glucose — 5.9 mmol/l, urea — 34,7 mmol/l, total bilirubin — 96 mmol/l, direct bilirubin — 25mmol/l , calcium — 2.0 mmol/l , sodium — 4,0 mmol/l , potassium — 4,0 mmol/l . Ultrasound general abdominal cavity for the presence of fluid — In the interstitial space and small pelvis is visualized ascitic fluid.

Conclusions. HRS is a rare, yet severe form of kidney dysfunction found in those suffering from liver cirrhosis and is a common cause of death for these patients. The diagnosis process can be complex and time-consuming, so in certain cases where the patient does not fulfill all IAC diagnostic criteria, we may initiate treatment for “presumed” HRS based on clinical suspicion. Patients with type 2 HRS have a slower progression of kidney failure, and a better prognosis if they are eligible for liver transplantation. In this case, the patient experienced rapid HRS development due to involvement of the heart and liver; fortunately, there was some improvement over time but not complete remission.

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