

UDC 616.36:616.61

HEPATORENAL SYNDROME

Tsitko E. V., Somani Harshitha

Scientific adviser: Ph.D., Associate Professor E. G. Malaeva

**Educational Establishment
«Gomel State Medical University»
Gomel, Republic of Belarus**

Introduction

Hepatorenal syndrome (HRS) is the development of renal failure in patients with advanced chronic liver disease and occasionally, fulminant hepatitis, who have portal hypertension and ascites [1]. It is a diagnosis of exclusion. The histological and, or, structural appearance of the kidney is normal, and the kidneys usually begin to function normally after liver transplant in these patients. So, now, due to portal hypertension, splanchnic vasodilatation occurs leading to activation of RAAS which further leads to severe intra-renal vasoconstriction. Ultrasonography of the kidney and urine analysis are normal. Pre-renal impairments present excluding GI loss, diuretics overdose and decreased albumin [2].

Aim

The purpose of this article is to understand Hepatorenal Syndrome with the help of a case of HRS type 2 with the inclusion of the new treatment modalities.

Material and methods of research

Epidemiology. HRS is common, with a reported incidence of 10 % among hospitalized patients with cirrhosis and ascites. In decompensated cirrhotics, the probability of developing HRS with ascites ranges between 8–20 % per year and increases up to 40 % in 5 years. An estimated 35–40 % of patients with end-stage liver disease (ESLD) and ascites will develop HRS. Frequency is equal in both sexes.

Results and discussion

A male patient 45-year-old from Gomel, was admitted in regional hospital on 4/3/22 in ICU diagnosed as the case of hepatorenal syndrome. He complaints of abdominal pain, lower extremity, distention of abdomen, his hemoglobin level at the time of admission was 9.7 gm%. In the past medical history, he had HRS and alcoholic cirrhosis. He was hospitalized with swelling in his lower extremities and abdomen. Clinically, he also presents with nausea, vomiting, loose stool, cough, cold, loss of consciousness and chronic alcoholism since 22-year last intake, backache and fever. In physical examination, his temperature is 36 °C, BP 120/72, pulse 115, and weight is 51 kg and ascitic fluid was palpated in the abdomen. He appeared to be attentive and oriented, with no signs of discomfort. His pupils were circular and equal. The abdomen was swollen and the bowel sounds were regular. He developed edema and swelling in lower limbs. His albumin level was 2.3 and KFT - 128. Ultrasonography was found to be normal.

Pathophysiology In 2007, the International Club of Ascites (ICA) classified HRS into types 1 and 2 (HRS-1 and HRS-2). HRS-1 is characterized by a rapid deterioration of renal function that often occurs because of a precipitating event, while HRS-2 is a moderate and stable or slowly progressive renal dysfunction that often occurs without an obvious precipitant.

Diagnostics is done with the help of abdominal ultrasound, diagnostic paracentesis, and ascitic fluid cultures in the suspected cases. The diagnosis is based on the presence of a reduced GFR in the absence of other causes of renal failure in patients with chronic liver diseases. The new diagnostics criteria for HRS-1 (renamed HRS-AKI.7) is regarding the pathogenesis of the disease regards HRS as not a purely «functional» entity with hemodynamic derangements, but that systemic inflam-

mation, oxidative stress and bile salt-related tubular damage may contribute significantly to its development. That is, HRS has an additional structural component that would not only make traditional diagnostic criteria less reliable, but would explain the lack of response to pharmacological treatment with vasoconstrictors plus albumin that correlates with a progressive increase in inflammation.

Management: Medical — The patient admitted on 4/3/22 in regional hospital was prescribed medication Tab Lasix 40 mg, Tab Metoclopramide 40 mg, Tab Thiamine, Inj Vit K 10 mg OD, Tab Neurobim pan-OD. Further culture test was done of ascites fluid which revealed presence of bacteria confirming Spontaneous Bacterial Peritonitis. Inj Pantoprazole 500 mg BD, Inj Albumin 20 % BD, Inj optineurin 1 ampule OD, Inj Cefotaxime 2 gm TDS, Inj Dopamine 3 mcg/kg/min, Inj Octreotide 10 mg IM.

Surgical: Liver transplant is awaited. And a follow-up diagnostic paracentesis was done after 48 hours of initiation of treatment for testing the efficacy of treatment. TIPS (Transjugular Intrahepatic Portosystemic Shunt) procedure was consulted with the surgeon.

Follow up: Advice the patient to visit the hospital after one week. He's prescribed to take Tab Lasix 40 mg, Tab Metoclopramide 40 mg, Tab thiamine and Tab Neurobin pan OD.

Also, the following advice was given:

1. Prevent the infections.
2. Maintain personal hygiene.
3. Proper rest and sleep.
4. Intake of healthy diet.
5. Regular follow up until proper donor is found.

Conclusion

In most cases, clinically seen case is of HRS type I which has a bad prognosis, having a median survival rate of 4-6 weeks without transplant. Also, medicines like Terlipressin or Norepinephrine or Midodrine+ SC Octreotide can be used in patients awaiting liver transplant.

REFERENCES

1. Devuni D. Hepatorenal Syndrome: background, pathophysiology, etiology, 2021.
2. Harrison's principle of internal medicine.
3. Acevedo, J. G. Cramp ME. HRS: update on diagnosis and therapy / J. G. Acevedo // World J Hepatol. 2017.
4. International Club of Ascites 2007 guidelines.
5. Малаева, Е. Г. Гастроэнтерология : учеб. пособие / Е. Г. Малаева. Гомель : ГомГМУ, 2017. 122 с.

UDC 616.344-002-031.84-07-08-053

ASPECTS OF CROHNS DISEASE BASED ON AGE AND DIAGNOSIS ON ITS LOCATION AND CLINICAL TYPES

Tsitko E. V., Dontala Sowjanya

Scientific adviser: Ph.D., Associate Professor E. G. Malaeva

**Educational Establishment
«Gomel State Medical University»
Gomel, Republic of Belarus**

Introduction

Crohn's disease (CD) is a type of inflammatory bowel disease, it causes inflammation of your digestive tract, which can lead to abdominal pain, severe diarrhea, weight loss and malnutrition, inflammation caused by CD can involve different areas of digestive tract and it also affects different layers of bowel in different people [1]. CD is increasing in prevalence worldwide. It arises from a complex interplay between both genetic predisposition and environmental influence. The impact of CD