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Hepatorenal Syndrome with Decompensated Cirrhosis of Liver with Portal Hypertension with Ascites

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ABSTRACT

The prognosis for hepatorenal syndrome, a dangerous side effect of liver cirrhosis, is quite poor. The pathophysiological hallmark is severe renal vasoconstriction, which is caused by intricate changes in the systemic, renal, and splanchnic circulations as well as the vasodilators and constrictors. Timely diagnosis and care are essential since novel therapeutic methods, such as vasoconstrictor therapy, can improve short-term outcomes and buy time for liver transplantation, which can result in full recovery. People with severe liver disease (HRS) develop renal failure. It is dangerous and has a poor prognosis. The exact mechanism underlying this condition is still unknown. Newer therapy techniques with better prognoses have been made available, though, thanks to recent scientific advancements. A patient may now have a chance of survival if they are diagnosed with HRS early, receive appropriate medical attention to control their renal failure and have access to a liver transplant if necessary. About 4% of patients with severely decompensated cirrhosis who are admitted experience HRS, with a cumulative probability of 18% at one year and 39% at five years. A patient's likelihood of developing HRS is one in three if they have spontaneous bacterial peritonitis. Patients with severe cirrhosis are typically at risk for developing hepatorenal syndrome, which can result in jaundice and other chronic liver disease stigmata such as spider naevi, palmar erythema, and finger clubbing. Splenomegaly, bleeding propensity, hepatic encephalopathy, edema, and ascites are further clinical characteristics. Patients typically exhibit bounding pulses, a broader pulse pressure, and low arterial blood pressure.

CONCLUSION: Here, we'd want to concentrate on the fact that a patient with cirrhosis of the liver is being treated by a primary care doctor. A primary care physician must identify risk factors, make the appropriate test requests, and perform imaging to identify illness problems. It helps in preventing mortality and further problems.

KEYWORDS: cirrhosis, prognosis, syndrome, splenomegaly, erythema.

INTRODUCTION:

Hepatorenal syndrome (HRS) is a multiorgan disease that mostly affects the liver and kidneys. It is one of the factors that can lead to acute kidney damage in patients who have either acute or chronic liver disease. In the late 1800s, the first correlation between cirrhosis and renal failure was found. Among the several possible Hepatorenal syndrome is one of the causes of acute kidney injury in patients with acute or chronic liver disease. In addition to fulminant hepatic failure from any cause, affected individuals typically have portal hypertension as a result of cirrhosis, severe alcoholic hepatitis, or (less frequently) metastatic tumors. The hepatorenal syndrome is the result of a sequence of declines in kidney perfusion caused by the increasing severity of hepatic injury. A bad prognosis is linked to the exclusion diagnosis of hepatorenal syndrome.[1]

HRS is a condition that can affect people who have liver failure, cirrhosis, or severe alcoholic hepatitis. It typically happens when liver function rapidly declines as a result of an abrupt insult like an infection, gastrointestinal bleeding, or excessive use of diuretics.[2,3] 18% of patients develop HRS within a year of diagnosis, and 39% do so within five years. HRS is a quite common cirrhosis consequence. It is thought that declining liver function affects the circulation that supplies the intestines, which in turn affects the kidneys' blood flow and blood vessel tone. Rather than resulting from kidney injury directly, these variations in blood flow are what cause kidney failure in HRS patients. Hepatorenal syndrome is diagnosed based on individual laboratory testing. The hepatorenal syndrome has been classified into two types: While type 2 HRS is linked to ascites, or fluid collection in the abdomen, which does not improve with conventional diuretics, type 1 HRS is characterized by a rapidly progressing deterioration in kidney function.[4]

CASE PRESENTATION:

The patient came to AVBRH with complaints of distension of the abdomen for 2 to 3 weeks. All routine investigations were done on 18/05/2023 Hb s/o 6.9, creatinine s/o 2.7, albumin s/o 2.0, ammonia s/o 124, and remaining values are within normal limits. surgery opinion was taken on 21/05/2023 and

was advised USG local site and mgsulf dressing. USG abdomen and pelvis were done on 24/5/23 and findings s/o liver cirrhosis with dilated portal vein and cholelithiasis with edematous GB wall with gross ascites with splenomegaly with bilateral minimal pleural effusion. The patient is currently symptomatically better and hence is being discharged on request (DOR) and advised to follow up in gastro opd after 1 month for review. Medications are given during stay- The patient's condition has improved after receiving the following injections: monocef 1gm iv BD, emset 4mg iv TDS, terlipressin 0.5mg iv QID, albumin 20./. iv STAT, tab esomac 40mg po od, tab rifagut 550mg po BD, tab ultracet po SOS, tab cardivas 3.125mg po BD, tab torsemide plus po BD, and hep apoprotein powder 2tsp in 1 glass of water TDS. treatment on discharge: tab zifi 200mg po bd X 5 days, tab stomach 40mg od X 15 days, tab midodrine 2.5mg po tds X 1 month, tab meet 4mg po sos, tab right 550mg po bd X 15 days, tab ultracet po sos, tab torsemide plus po bd X 30 days, tab cardivas 3.125mg po bd X 30 days, hepapro protein powder 2tsp in 1 glass of water TDS. Follow up in gastro OPD after 30 days for review. follow up in medicine OPD for regular BP checkups maintain local hygiene, drink plenty of fluids, and avoid spicy food.

BRIEF PATIENT INFORMATION

Particulars relating to the patient: The patient complained of abdominal distension for two to three weeks when they arrived at AVBRH. All routine investigations were done on 18/05/2023 Hb s/o 6.9, creatinine s/o 2.7, albumin s/o 2.0, ammonia s/o 124, and remaining values are within normal limits. surgery opinion was taken on 21/05/2023 and was advised USG local site and magsulf dressing.

Primary concern symptoms of the patient:

Distension of abdomen, since 2 to 3 weeks, Patient was alright 2-3 weeks back when he developed distension in his abdomen in the last 3 weeks which was gradual in onset and progressive in nature, with no aggravating and relieving factors. C/o generalized weakness for 3 weeks c/o swelling in bilateral lower limb for 3 weeks h/o hematemesis /Malena. No h/o seizure/LOC, no h/o fever / cough/ cold, no h/o nausea/ vomiting, no h/o bladder complaints, no h/o trauma

Medical family and psychosocial history:

He belongs to joint family members who do not have any ailment, such as diabetes or hypertension. He kept up a solid rapport with the medical staff and nursing.

Lab Test	**Value on Admission**	**Normal Value**
COMPLETE BLOOD COUNT		
Hemoglobin	6.9	13-17g/dl
MCHC	32.8	31-36%
MCV	100.5	80-100um^3
MCH	33	25.4-34.6 pg
Total RBC COUNT	4.5	4.5 -6 million/mm^3
Total WBC COUNT	14400	4000-10000 .mm^3
Total PLATELETS COUNT	2.64	1.5-4
Hematocrit	17.4	40-55%
MONOCYTES	03	2.0-12.0%
LYMPHOCYTES	25	26-46%
RWD	13.9	11.5-14.5
EOSINOPHILS	02	0.0-5.0%
BASOPHILS	00	0.0-2.0%
BODY FLUID		
Lactic Dehydrogenase (LDH)	74	140-280IU/L
Ascitic Fluid –SUGAR	94	100 mg/dl
Ascitic Fluid –PROTEIN	2.0	0.3-4.0g/dl
PH	7.4	7.35-7.45
KFT		
UREA	25	13-45mg/dl
Creatinine	2.7	0.8-1.4mg/dl
Sodium (Na+)	135	135-145 mEq/L
Potassium(K+)	4.0	3.5-5.5 mEq/L
LFT		
Alkaline Phosphatase	296	65-306 IU/L
ALT (SGPT)	17	1-40 IU/L
AST(SGOT)	37	1-40 IU/L
TOTAL PROTEIN	5.6	6.5-8.5g/dl
ALBUMIN	2.0	3.5-5.3 g/dl
Total Bilirubin	3.1	0.3-1.0mg/dl

BC Bilirubin Conjugated	2.5	0.0-0.3mg/dl
Bilirubin Unconjugated	0.6	0.2-0.8 mg/dl
Lipase	50	0-160 U/L

PERIPHERAL SMEAR :- Reduced RBC mass RBCs –Anisocytosis showing macrocytic RBCs with microcytes and occasional feagmented RBCs Platelets –Adequate on smear . No Haemoparasites seen . Impression – Dimorphic Anemia .

C.S.F. Exam /Ascites fluid / other body fluid , cell count :- Received 4 ml of yellowish transluscent fluid in a clot activator bulb labelled as ascites fluid on wet mount RBCs 0.1 cells/HPF, WBC s 0.1 cells/HPF TLC –Approximately 122 cel;s /cumm DLC –Polymorphs -75% Lymphocytes -25%.

RELEVANT PAST INTERVENTIONS AND OUTCOMES:

In the present case. Medications are given during stay- in monocef 1gm iv bd, inj emcet 4mg iv tds, and terlipressin 0.5mg iv QID, and albumin 20./. iv stat, tab esomac 40mg po od, tab Xifaxan 550mg po bd, tab ultracet po sos, tab Cardiovasc 3.125mg po bd, tab dytor plus po bd, hep apoprotein powder 2tsp in 1 glass of water tds then patient health is better than before.

DIAGNOSTIC FINDING:

A general checkup reveals a poor state of health. The state of consciousness is conscious, but the overall condition is unsatisfactory. The vital parameter's values are as follows: temperature is FEBRILE, pulse is 72/min, resp is 20/min, and bp is 130/90mmhg.

HEMATOLOGICAL INVESTIGATION:

The Hb is 11.4%, In the complete blood count, the total platelet count is 2.62, the total WBC count is 14400, the total RBC count is 4.5, and the calcium level is 9.5. The LIPID PROFILE readings for UREA, CREATININE, and SODIUM in the KIDNEY FUNCTION TEST are 25, 2.7, and 135, respectively.

THERAPEUTICINTERVENTION: In the present case medications were given during the stay- The patient's condition has improved after receiving the following injections: monocle 1gm iv BD, emset 4mg iv TDS, terlipressin 0.5mg iv QID, albumin 20./. iv STAT, tab esomac 40mg po od, tab rifagut 550mg po BD, tab ultracet po SOS, tab cardivas 3.125mg po BD, tab dytor plus po BD, and hep apoprotein powder 2tsp in 1 glass of water TDS.

NURSING MANAGEMENT:

The IV fluid was used to keep the electrolyte and hydration levels stable. Every two hours, vital signs are monitored, and the cardinal assessment is evaluated together with intake and output.

CONCLUSION: Timely treatment and management

DISCUSSION:

The patient came to AVBRH with complaints of distension of the abdomen for 2 to 3 weeks. All routine investigations were done on 18/05/2023 Hb s/o 6.9, creatinine s/o 2.7, albumin s/o 2.0, ammonia s/o 124, and remaining values are within normal limits. surgery opinion was taken on 21/05/2023 and was advised USG local site and mgsulf dressing. In the present case, medications were given during stay- inj monocle 1gm iv BD, injemeset 4mg iv TDS, injterlipressin 0.5mg iv QID, inj albumin 20./. iv STAT, tab esomac 40mg po OD, tab rifagut 550mg po BD, tab ultracet po SOS, tab cardivas 3.125mg po BD, tab dytor plus po BD, hepapoprotein powder 2tsp in 1 glass of water TDS then patient health is better than before.

The condition known as hepatorenal syndrome (HRS) is linked to cirrhosis and abrupt liver failure. By excluding alternative causes of renal failure, HRS is diagnosed. Transplanting the liver is the only conclusive treatment. Excessive circulatory dysfunction manifests as hepatorenal syndrome (HRS), which has a significant morbidity and fatality rate.[5] The International Club of Ascites has proposed a revised definition that bases the diagnosis of HRS on variations in serum creatinine rather than a set high number. The Centers for Disease Control estimate that cirrhotic liver disease causes 15,000 deaths annually in the US. Cirrhosis is characterized by an elevated hepatic venous pressure gradient (≥ 5 mm Hg) and portal hypertension.[6,7] The condition arises from a complex interplay between inflammation, necrosis, collagen deposition, and the regeneration of nodules in the liver parenchyma. In the end, elevated portal pressure lowers portal blood flow, which causes vasodilators to be released and splanchnic circulation blood to pool.[8] Renal hypoperfusion follows, which activates the renal-angiotensin-aldosterone system and causes fluid When reduced oncotic pressures from hypoalbuminemia are combined with fluid leakage from the splanchnic circulation, it surpasses the lymphatic drainage system's capability, and ascites, a typical consequence of decompensated cirrhosis, arise.[9,10]

CONCLUSION:

When a patient is brought into the medical facility. The patient's health was really poor, however following some research according to the finding investigation, he was diagnosed with diagnosis hepatorenal syndrome with decompensated cirrhosis of the liver with portal hypertension with ascites then medical attention was given right away. Patients receive prompt treatment and are at lower risk because of competent nursing care. The patient's health situation is safer and better than it was before.

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