International Journal of Research in Medical Sciences
Yashfeen M et al. Int J Res Med Sci. 2023 Sep;11(9):3443-3446
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20232805

Case Report

A case report of an ethanol-related decompensated chronic liver disease with complications

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Received: 26 June 2023
Revised: 01 August 2023
Accepted: 02 August 2023

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ABSTRACT

Decompensated chronic liver disease, is a histopathologically defined condition with a variety of clinical symptoms and complications, some of which are associated with an increased risk of in-hospital mortality. Cirrhosis is predicted to affect 100 (range 25-400) per 100,000 people worldwide, with a male-to-female ratio of one. Patients with ethanol-related cirrhosis have a 5-year death rate ranging from 60-85%. One of the leading causes of cirrhosis is alcoholism. However, it can also be caused by non-alcoholic steatohepatitis (NASH), autoimmune disorders, and viral hepatitis. The decompensated chronic liver disease carries a 9.7 times greater chance of mortality. Cirrhosis is a histologic diagnosis, but a combination of clinical, laboratory, and imaging characteristics can help confirm a cirrhosis diagnosis. For evaluating liver cirrhosis, a liver biopsy continues to be the gold standard. A non-invasive approach to assessing liver cirrhosis is transient elastography (FibroScan). Patients with severe cirrhosis may experience several significant sequelae that complicate their clinical path. These include portal hypertension and related side effects, such as gastroesophageal varices, splenomegaly, ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome, hepatopulmonary syndrome, and hepatocellular carcinoma. In decompensated chronic liver disease, treatment focuses on underlying liver disease, dietary changes, and long-term medical management to control underlying problems. For patients who do not react to other medications, liver transplantation can be an effective long-term therapy option.

Keywords: Ethanol-related decompensated chronic liver disease, Portal hypertension, Oesophageal varices, Ascites, Hepatic encephalopathy, Liver transplantation

INTRODUCTION

Cirrhosis is a pathological condition characterized by the diffuse disruption of the liver's normal architecture, accompanied by fibrosis and nodule formation. It is the end pathway for a wide range of chronic liver diseases.[1] In ethanol-associated cirrhosis, the nodules are usually less than 3 mm in diameter. Cirrhosis of this type is known as micronodular cirrhosis. Obesity, smoking, chronic hepatitis C, and low vitamin D levels are linked to an increased incidence of alcohol-related cirrhosis in women compared to men. Many cirrhosis complications are caused by the development of portal hypertension, collateral variceal formation, and hyperdynamic circulation, which requires medical attention and management. Early liver transplantation can be a successful treatment for highly selected patients with alcoholic cirrhosis who have failed previous therapies.

CASE REPORT

A 35-year-old male who was diagnosed to be DCLD six months ago presented to the emergency department complaining of abdominal pain lasting for three days,

which was diffuse, and dull aching and loose stools (5 episodes per day) for two days, which were watery in consistency and not blood-stained, abdominal distension for one month and swelling of legs for two weeks. He had a one-day history of a low-grade fever and an altered sensorium with increased daytime sleepiness and a subtle lapse in concentration. On probing the past history, the patient was diagnosed with DCLD six months ago, besides which he does not have any other comorbidities. His personal history includes non-oliguric and irregular bowel habits and sleep patterns. He was an alcoholic for three years. His consumption of alcohol was 80 g/day until he quit last year. There was no family history of NAFLD or hepatitis B infection.

On examination, the patient was awake, responded to oral commands, was afebrile, and hydrated just adequately. His vital parameters were a pulse rate of 86 beats/minute, respiratory rate of 18 breaths/minute, blood pressure of 100/70 mmHg, and a SpO_2 of 97%. The patient had mild pallor, icterus, and bilateral pedal edema. On head-to-toe examination, severe sarcopenia and wasting of small muscles in the hand were found. Other features were uneventful.

System examination: Cardiovascular system: S_1 S_2 heard normally, no murmurs. Respiratory system: Bilateral airway entry equal and clear, with no added sounds. Central nervous system: Pupils equally react to light; flapping tremor is present.

Per abdomen examination: On inspection, the abdomen was uniformly distended, the flanks were full, engorged veins were present over the abdomen, and an umbilical hernia was present. On superficial palpation, there was generalized abdominal pain, the abdomen was warm and tenderness present over all quadrants. On deep palpation, the spleen was palpable. On percussion, shifting dullness was present, and a fluid thrill was appreciated.

Investigations

Blood investigations such as complete blood count (CBC), liver function test (LFT), renal function test (RFT), serum electrolytes, and PT INR were taken. His viral marker tests such as HIV, HBsAg, and anti-HCV were negative.

He underwent an ultrasonography of the abdomen and pelvis which revealed, the liver appeared coarse, with nodularity on the surface and irregular margins. The gall bladder was found contracted. The spleen was 13 cm in size. Pelvis was surrounded by free fluid.

He then underwent Doppler for the study of the portal venous system, which revealed, liver: measures 7.9 cm, shrunken in size, irregular in surface coarse echoes, and blunted margins. Gall bladder: wall thicken. Portal vein: at hilum-9 mm, at confluence-9 mm, velocity-17 cm/s. Spleen: measures about 13.5 cm. Pelvis: massive free fluid was noted in the pelvis and peritoneal cavity.

His report of ascitic fluid cell count and cytology revealed, total count: 45 cells/mm^3.

Direct count: 80% mesothelial cells, 20% lymphocytes.

Cellular smear studies showed reactive mesothelial cells admixed with lymphocytes in an eosinophilic fluid background.

Ascitic fluid had a high SAAG score with the low protein.

The ascitic fluid test showed protein levels of 0.8 g/dl, 0.7 g/dl of albumin, and 85 mg/dl of sugar.

A non-invasive approach transient elastography (FibroScan) was done to assess liver cirrhosis, which showed 28.6 kPa of advanced chronic liver disease with fibrosis and esophagogastroduodenoscopy was taken and found the patient had grade 2 oesophageal varices. His MELD Na score was 17.

Arriving at a diagnosis of an ethanol-related decompensated chronic liver disease with portal hypertension, oesophageal varices grade 2, massive ascites, and hepatic encephalopathy grade 2.

Management

Fluid restriction: <1 liter per day, salt restriction: <4.6 grams per day, backrest at an angle of 30°, soft, bland diet abstinence from alcohol.

Drug therapy: The patient was started on the IV antibiotic cefotaxime 1 g TDS. Syrup lactulose, 15 ml TDS was given. A lactulose enema BD was given. A beta-blocker, Tab. propranolol 40 mg BD, was given to reduce portal pressure.

An injection of human albumin (20%), 100 ml OD, was administered after the therapeutic paracentesis to replenish intravascular volume. An H_2 blocker, Tab. ranitidine 150 mg BD, was given.

The patient's sensorium improved with treatment and then the patient was started on diuretics (Tab. spironolactone 25 mg/day and Tab. furosemide 40 mg/day) and non-selective beta blockers.

Other liver-supportive measures were given, such as Tab. UDCA (ursodeoxycholic acid) 300 mg TDS. Tab. rifaximin 550 mg BD.

The patient improved with further treatment and was advised to register for liver transplantation in the outpatient department.

Table 1: Routine blood investigations.

Investigations	Admission day 01	Admission day 05	Admission day 10	Reference range	Units
Complete blood count					
White blood cells	8800	14800	12900	4000-11000	Cells/cu mm
Red blood cells	3.4	3.0	3.1	4.5-5.5	million/cu mm
Hemoglobin	11.1	10.3	10.4	15-17	g/dl
PCV	32	29	30	40-45	%
MCV	95	96	97	78-98	fl
MCH	33	33	33	25-35	Pg/cell
MCHC	35	34	34	30-35	g/dl
Platelets	1.1	88000	1.2	2-5	Lakhs/cu mm
Liver function test					
Total bilirubin	4.1	3.3	3.4	0.2-1.0	mg/dl
Direct bilirubin	1.2	1.5	1.1	0.1-0.3	mg/dl
SGOT	56	37	38	10-40	Iu/L
SGPT	25	15	21	10-40	Iu/L
Alkaline phosphatase	91	89	107	40-100	Iu/L
Protein (total)	5.0	5.0	6.1	6-8	g/dl
Albumin	1.9	2.4	2.9	3.5-5	g/dl
Renal function test					
Random blood sugar	42	77	140	70-140	mg/dl
Urea	32	44	21	20-40	mg/dl
Creatinine	1.0	0.7	0.5	0.5-1.2	mg/dl
Serum electrolytes					
Sodium	135	132	137	135-145	mEq/L
Potassium	3.8	4.8	4.2	3.5-5	mEq/L
Prothrombin time (PT)	24.6	19.2	19.4	10-15	Seconds
International normalised ratio (INR)	2.0	1.53	1.4	1.1 or below	No unit

DISCUSSION

Approach to a patient with decompensated chronic liver disease

A percentage of cirrhotic patients between 20% and 63% present with signs characteristics of decompensation, such as jaundice, ascites, variceal bleeding, or hepatic encephalopathy.[2] The child Turcotte Pugh (CTP) score and the MELD score are the clinical assessments that are most frequently used to assess prognosis in individuals with alcohol-associated cirrhosis.[3]

Portal hypertension

Portal hypertension is evident when the rise of the hepatic venous pressure gradient (HVPG) is greater than 10mm Hg. According to the location of increased resistance to portal blood flow, the etiology can be prehepatic, intrahepatic or posthepatic.[4] To evaluate patients with portal hypertension, imaging tests like USG, CT, and MRI with Doppler vessel analysis have been routinely used. The goal of treating portal hypertension is to either decrease intrahepatic resistance (e.g., nitrates) or reduce portal blood flow (beta blockers), either using pharmacological drugs or by radiologically or surgically creating a portosystemic shunt.[5]

Coagulopathy

Due to "cirrhotic coagulopathy," patients with chronic cirrhosis are more vulnerable to bleeding. Prothrombin time (PT), international normalized ratio (INR), and activated partial thromboplastin time (aPTT) are common coagulation system measurements. Viscoelastic assays (TEG, ROTEM, serum fibrinogen, and emerging sonorheometry) and thrombin generation assay (TGA) are whole-blood 'global' markers of coagulation and are used for decision on fresh frozen plasma transfusion.[6] Vit K can be used to treat hypo-prothrombinemia brought on by malnutrition and vit K deficiency. Enormous amount of fresh frozen plasma is used to reduce any outer bleeding episodes.[7] Cirrhosis patients may experience more bleeding when thrombocytopenia is severe.

Esophageal varices

Varices develop in 5-15% of cirrhotic patients annually. Development of variceal bleeding requires HVPG larger than 12 mmHg. Patients often present with upper gastrointestinal bleeding, such as malena/hematemesis. Most widely used procedure to identify oesophageal varices is esophago-gastroduodenoscopy. Preferred endoscopic modality for controlling acute oesophageal variceal bleeding is endoscopic variceal ligation. Other

therapies include balloon tamponade, stents, endoscopic sclerotherapy, and trans-jugular intrahepatic portosystemic shunt (TIPS).

Ascites

Ascites is the most frequent decompensating event in cirrhotic patients. Pathogenesis of ascites, which is the buildup of fluid in the peritoneal cavity, is explained by portal hypertension, sodium retention due to vasodilation, and following activation of sodium-retaining mechanisms.[8] Patients present with generalized abdominal pain and distension, often accompanied by peripheral edema. Shortness of breath, muscle wasting, and umbilical hernias are evident in a patient with large ascites. Physical examination (flank dullness, ballotable liver) and abdominal imaging (USG, CT) are used to make diagnosis. Cause for ascites is assessed from ascitic fluid analysis to differentiate between transudative and exudative ascitic fluid. Range of therapeutic interventions includes salt restriction alone (rarely used), use of diuretics (spironolactone and furosemide), therapeutic paracentesis with concurrent intravenous albumin administration, and for the most severe patients, TIPS, and finally liver transplantation. Ascitic fluid can get infected spontaneously in spontaneous bacterial peritonitis.

Hepatic encephalopathy

The term "hepatic encephalopathy" refers to complicated and irregular changes in the neuropsychiatric state that exacerbate liver disease. Pathogenesis of the disease is significantly influenced by ammonia.[9] West Haven classification is used to grade hepatic encephalopathy. Factors determining severity include acute bleeding (coagulopathy), infections, hyperglycemia, constipation, electrolyte imbalance, and surgical shunts which should be addressed to abate further progression. Increased serum ammonia levels in a cirrhotic patient with a changed mental status provide evidence for the diagnosis.[10] It is advised to conduct an EEG along with a clinical assessment and psychometric testing. Eliminating or correcting triggering variables is the goal of treatment. Lactulose or enemas are used as first-line treatments to reduce the nitrogen load, followed by antibiotics as second-line treatments and rifaximin as a prophylactic antibiotic to prevent repeated encephalopathy.

CONCLUSION

Patients with cirrhosis have a decreased chance of survival, with a median survival time of two years when decompensation is present. The liver will never restore its normal structure once it has been disorganized, as in cirrhosis. Symptomatic treatments are quite effective. With rates of 3-5% annually for alcohol-associated cirrhosis, patients are at significant risk of developing hepatocellular carcinoma. When cirrhosis patients are referred early in course of disease, liver transplantation significantly improves their prognosis.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Yashfeen M, Thilakavathi R. A case report of an ethanol-related decompensated chronic liver disease with complications. Int J Res Med Sci 2023;11:3443-6.