Case Report On Cirrhosis Of Liver

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Abstract

Introduction: - The liver is irrevocably damaged and replaced with scar tissue as a result of the late-stage liver disease cirrhosis. Scar tissue prevents your liver from functioning properly. Cirrhosis typically develops decades after the onset of chronic liver disease. Men experience it twice as frequently as women. Clinical Findings: - Weakness, loss of appetite, easy bruising, skin yellowing (jaundice), itching, and weariness are all symptoms of cirrhosis. Diagnostic Evaluation: - Blood test: - HB % = 9.3gm%, total RBC count = 2.99cu.mm, total WBC count = 11800cu.mm, total platelet count = 1.23 lac cu.mm, total protein = 6.5g/L, total bilirubin = 15.8g/L, lymphocytes = 10%, monocytes = 0.2%, albumin =2.2. Peripheral Smear: - RBCs - Normocytic mildly hypochromic Platelets - Reduced on smear APC - 38000 cells/mm³ as per cell counter no Haem parasite seen. Ultrasonography: - Options include liver needle biopsy, ultrasonography, and magnetic resonance imaging (MRI), an innovative imaging technique called electrography. Therapeutic Intervention: - Inj. Albumin 20% × OD, Inj. Vitamin K 10mg ×OD, Inj. Optineuron ×OD, Inj. Pan 40mg × OD, Inj. Emset 4mg × SOS, Inj. Levofox 500mg × OD.

Outcome: - After treatment the patient show improvement. His appetite is improved and skin condition is also improved and his Weakness is reduced.

Conclusion: - With a known case of liver cirrhosis, the patient was admitted to the hospital with complaints of weakness, loss of appetite, easy bruising, jaundice, itching, and weariness. His condition improved after receiving the proper care.

Keywords: - Cirrhosis of the liver, scar tissue, easy bruising, chronic liver disease.

Introduction

Cirrhosis is a late stage of liver scarring (fibrosis) brought on by a variety of liver disorders and conditions, including prolonged alcoholism and hepatitis (1). Your liver strives to heal itself if it sustains damage from a disease, too much alcohol, or any other cause. As time goes on, more and more scar tissue develops, making the liver's ability to function problematic (decompensated cirrhosis)(2). Life-threatening cirrhosis has advanced stages. Cirrhosis usually leaves permanent liver damage that cannot be reversed, however, if the source is addressed and liver cirrhosis is identified early. Damage that has already occurred can usually only be limited (3).

Patient Identification: - On February 22, 2021, a male patient from Chandrapur who was 60 years old and had liver cirrhosis was admitted to the hospital. He weighs 55 kg and is 175 cm tall.

Present Medical History: - Patient with complaints of swelling on the bottom part of his body for a week, abdominal distension, yellowish discoloration of the sclera, nausea, and black stools.

Past Medical History: - Patient was diagnosed to have
cirrhosis of the liver at the age of 60 years. He has no past medical history.

Family History:- The family consists of five people. It was determined that patient had liver cirrhosis. Except for the patient, who was being admitted to the hospital, none of the other family members had health issues.

Past intervention and outcome:- A patient was found to have liver cirrhosis. None of the other family members had any health difficulties outside the patient, who was being admitted to the hospital.

Clinical Findings: -weakness, loss of appetite, easy bruising, skin yellowing (jaundice), itching, and weariness are all symptoms of cirrhosis.

Etiology: -There are numerous illnesses or circumstances that contribute to cirrhosis continuing. Although there may be more than one cause in some situations, the following are the most typical ones in the US:

Years of excessive alcohol consumption.

Hepatitis B and C are infections that persist over time.

Non-alcoholic fatty liver disease, also known as non-alcoholic steatohepatitis, is a more serious disorder.

Drinking a lot of alcohol for a long time can damage and chronically harm the liver. Two to three drinks per day, including beer and wine, for women and three to four for men, can damage the liver and cause cirrhosis. Protein deficiency is a prevalent issue in alcoholics.

Physical Examination: - There are hardly many anomalies found during a complete physical examination, except for the abdomen, which may reveal markers for splenomegaly, caput medusa, abdominal discomfort, and dullness to percussion on the flanks,

Disease diagnosis Assessment: - Blood test: - Hb% = 9.3gm%,

total RBC count = 2.99cu.mm, total WBC count =

11800cu.mm, total platelet count = 1.23 lac cu.mm, total protein = 6.5g/L, total bilirubin = 15.8g/L, lymphocytes = 10%, monocytes = 0.2%, albumin =2.2. Peripheral

Smear: - RBCs - Normocytic mildly hypochromic Platelets -Reduced on smear APC - 38000 cells/mm³ as per cell counter no Haemoparasite seen.

Ultrasonography: - Through a liver needle biopsy, magnetic resonance imaging (MRI), ultrasound, or even both. an important criterion method is known as electrography,

Therapeutic Intervention: - Inj. Albumin 20% × OD, Inj.

Vitamin K 10mg ×OD , Inj. Optineuron ×OD , Inj. Pan 40mg × OD , Inj. PEMset 4mg × SOS , Inj. Levofox 500mgOD

Discussion: The true incidence of AD-induced liver cirrhosis is unknown because liver biopsy has not been routinely performed in AD-treated individuals(4). Only five instances of AD-induced liver cirrhosis resulting from daily low dosage therapy have been documented in the literature, and almost all occurrences have involved patients who were on higher maintenance doses(5). Some researchers have hypothesized that when determining the likelihood of irreparable liver damage, the cumulative dose in its entirety might be more important. The previously described patients received cumulative dosages ranging from 165 to 213 g over 12-48 mo of therapy(6-14). The current patient had received a cumulative dose of 528 g of AD administered orally once daily for 84 months. Hepatic problems were not found during
treatment, despite the dosage being higher than in prior reports. As patients undergoing long-term AD therapy may experience greater silent liver disease development, to identify liver damage, routine liver function tests and/or biopsies should be carried out. Gilinsky et al. stated that if the damage to the liver was already irreparable, stopping the medicine did not fully restore the liver functionally histological screening for hepatic abnormalities is helpful to prevent permanent liver changes (15-27).

Conclusion: Alcohol-related morbidity and mortality are largely attributed to alcoholic liver disease. It is thought that 10% to 15% of alcoholics will develop cirrhosis. Alcohol addiction and excessive drinking can produce. The likelihood of developing ALD is, in large part, a result of both cirrhosis, hyperlipidemia, and alcoholic hepatitis. frequency and duration of heavy drinking, and it has been demonstrated that alcohol consumption per capita within populations is a significant predictor of cirrhosis mortality rates.

Ethical Clearance: - Taken from an institutional ethics committee.

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