Case study

Case report on Cirrhosis of liver

Abstract

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, that leads to portal hypertension and end stage liver disease. The patient 56 years old who was apparently admitted in AVBRH on date 9/12/2020 in ICU with the chief complaint of abdominal distention, breathlessness on exertion, pedal edema, fever since 8 days. After admitted in hospital all investigation was done like blood test, ECG, fluid cytology, peripheral smear,ultrasonography, etc. All over investigation observe and then final diagnosis conformed as cirrhosis of liver. Patient was not having any history of communicable disease or any hereditary disease but he has history of hypertension and type II Diabetes mellitus since 12 years. Patient was COVID-19 negative and admitted in intensive care unit.Patient has been undergone with various investigations like physical examination, blood test, CSF fluid examination, ascitic fluid examination, fluid cytology, peripheral smear, ultrasonography, RT-PCR etc. Patient was treated with tab. farobact ER 300mg BD, tab. Lasix 40 mg OD, tab. Udilive 300mg BD, tab. Rifagut 300mg BD, tab. Metformin 500mg OD, tab. Amlo 5mg OD, syp. Duphalac 30ml HS. Monitor vital signs, maintain input output**,** Monitoring and managing potential complications like, bleeding and haemorrhage, hepatic encephalopathy, fluid volume excess, monitor laboratory tests as indicated, Identify and assess for pedal edema.

Conclusion: Patient was admitted to hospital with the chief complaint of abdominal distention, breathlessness on exertion, pedal edema, fever since 8 days his condition was very critical and patient was admitted in AVBR hospital immediate treatment was started by health team and all possible treatment were given and now the patient condition is satisfactory.

Key words: *Liver damage , Treatment, Nursing management*

Comment [DA1]: Patient is

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Introduction

Cirrhosis is a state that appears as a response of liver damage. It is characterised by destructions of normal liver tissue and it replaced by fibrous bands of tissue and nodules of regenerating liver tissue[1].It is late stage of scarring of tissue of the liver caused by various forms of liver diseases such as, chronic alcoholism and hepatitis. Liver is damaged whether by disease, excessive alcohol consumption or another because it tries to repair itself[2]. According to WHO, about 46% of global diseases and 59% of the mortality is because of chronic diseases and almost 35 million people in the world die of chronic diseases[3].Global

prevalence of cirrhosis from autopsy studies ranges from 4.5% to 9.5% of the general population [4].During 2001, the estimated worldwide mortality from cirrhosis was 771,000 people, ranking 14th and 10th as the leading cause of death in the world and in developed countries, respectively[5]. Deaths from cirrhosis have been estimated to increase and would make it as the 12th leading cause of death in 2020 [6].

CASE REPORT

Patient present history:

The patient 56 years old who was apparently admitted in AVBRH on date 9/12/2020 in ICU with the chief complaint of abdominal distention, breathlessness on exertion, pedal edema, fever since 8 days. After admitted in hospital all investigation is done like blood test, ECG, fluid cytology, CT scan etc. All over investigation observe and then final diagnosis conformedas cirrhosis of liver.

Past history:

Patient was not having any history of communicable disease or any hereditary disease like tuberculosis asthma but he has history of hypertension and type II Diabetes mellitus since 12 years.Patient was COVID-19 negative and admitted in intensive care unit. Patient was on oxygen support from 09/12/2020 and patient was having nasogastric tube from 10/12/2020 and removed on 22/12/2020 and shifted to male medicine ward 29 on 27/12/2020.

Causes:

Chronic alcohol abuse, chronic viral hepatitis (hepatitis B,C and D),fat accumulating in the liver (non-alcoholic fatty liver diseases),iron build-up in the body(hemochromatosis),cystic fibrosis, copper accumulated in liver (Wilson's disease),poorly formed bile ducts(biliary atresia),autoimmune hepatitis, Alagille syndrome(genetic digestive disorder),destruction of the bile ducts(primary biliary cirrhosis),infections such as syphilis or brucellosis, medications including methotrexate or isoniazid[7].

In this case cause is chronic alcohol consumption, Mr. sanjay taking alcohol in the past 20years.

Clinical findings:

Fatigue, loss of appetite, nausea, swelling in your legs, feet or ankles (pedal edema), weight loss, yellow discoloration of skin and eyes(jaundice), fluid accumulation in abdomen (ascites),fever, breathing difficulty.

Investigations :

Investigations	Patient value	Normal value	Inference
Renal function test			
1. Potassium (k+)-serum	4.5	3-5mEq/L	Normal
2. Creatine-serum	3.4	0.7-1.25mg%	Increased
3. Urea- serum	16.5	18-40mg%	Decreased
4. sodium-serum	141	136-145mEq/L	Normal

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Comment [DA9]: need to add prevalence and mortality because of of cirrhosis in of Liver in India

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Liver function test 1. Albumin 2. Bilirubin-total 3. Protein-serum 4. AST(GOT) 5. ALT(SGPT**)**	 3.1 5.0 8.6 42 34	 3-5gm% 0.3-1mg% 6-8gm% 17-59I.U/L 0-35I.U/L	 Normal Increased Normal Normal Normal
Complete blood count 1. Hb 2. Total RBC count 3. Total platelet count 4. Total WBC count	 10.9 3.47 1.52 14900	 13-15.5gm% 4.5- 6millions/cu.mm 1.5-4lacs/cu.mm 4000-11000/cu.mm	 Decreased Decreased Normal Increased
Total WBC count 1. Monocytes 2. Granulocytes 3. Lymphocytes 4. Eosinophils 5. Basophils	 04 80 18 01 00	 4-10% 40-60% 17-48% 0-5% 0-2**%**	 Normal Increased Normal Normal Normal

CSF Examination -RBC-plenty of RBCs/HPF, WBC-2-3cells/HPF, TLC-75cells/cu.mm, DLC-polymorphs-70%, Lymphocytes-30%

Ascitic fluid examination-Ascitic fluid suger-194, Ascitic fluid PROTEIN-1.8, Albumin-0.7, LDH-79, PH-7.5

Peripheral smear-platelets reduced on smear APC-84000cells/mm3 as per cell counter**.**

Fluid cytology-smear shows occasional scattered polymorphous and occasional reactive mesothelial cells in clear background **IMPRESSION:** serous fluid

Ultrasonography

Impression: cirrhosis of liver with gross ascites, grade II B/L RPD, Rt sided plural effusion

MEDICAL THERAPY

1. **Pharmacological therapy**
   - Antibiotics
   - Laxatives
   - Vasopressin
   - Diuretic therapy
   - Human albumin solution
2. Replace fluid and electrolytes
3. Paracentesis

4. Therapeutic Intervention

Now patient treatment given in the ward i.e. tab. farobact ER 300mg BD, tab. Lasix 40 mg OD, tab. Udilive 300mg BD, tab. Rifagut 300mg BD, tab. Metformin 500mg OD, tab. Amlo 5mg OD, syp. Duphalac 30ml HS.

Nutritional therapy: Malnutrition can be present up to 20% of patients with cirrhosis, due to decreased protein and energy consumption nutritional assessments in cirrhotic patients are challenging and complicated by the hypervolemia, which can interfere with body weight and body mass index measures, as well as the reduced production or dilution of biomarkers such as albumin. Adequate protein intake, defined as 1.2 to 1.5 g/kg/d of protein per clinical guidelines[8].Studies have shown that vegetable and dairy protein is better tolerated than meat protein in patients with cirrhosis. The high fibre content of a plant-based diet can also help to reduce nitrogen waste products from the gastrointestinal tract[9].sodium restriction is also an important in patients with cirrhosis and ascites[10].Sodium-restricted diet, when combined with diuretic therapy, it is more effective for controlling fluid overload in 90% of patients[11]. High-sodium prepared foods, specifically deli meats, canned soups, frozen meals, and packaged snacks, should be avoided. Fruits, vegetables, legumes, raw nuts, and whole grains are naturally low in sodium and should be encouraged.

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Home care/Follow up care

- Advice patient to stop drinking alcohol
- Advice to check Weight daily and keep a weight log, if any sudden change in weight inform to physician
- Limit dried, packaged, and fast foods and don't add salt to your food at the table
- Advised to take medication exactly as directed and ask physician about getting vaccines for viruses that can cause liver diseases
- Follow up to health care provider or as advised
- Advised to call physician right away if you have, extreme tiredness, weakness, vomiting (with or without blood), yellowing skin or eyes, swelling on belly or legs, black stools, confusion or trouble thinking properly.

Discussion

Cirrhosis is the end stage of liver disease in which healthy liver tissue is replaced with scar tissue and liver is damaged permanently. Liver is not working properly because of scar tissue[12].current treatment for cirrhosis are limited to removing the injurious stimulus and eradicating viruses by using interferon, ribavirin andlamivudine in viral hepatitis and liver transplantation.Transplantation is the very successfultreatment for last stage cirrhosis[13].

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Liver cirrhosis can cause gastrointestinal bleeding, liver cancer and other severe clinical complications; therefore, it is very important to prevent and treat the disease. It is reported that approximately 57% of liver cirrhosis is due to hepatitis B virus (HBV) (30%) and hepatitis C virus (HCV) (27%) globally, and the etiologies differ geographically. Many studies have reported that different etiologies tend to result in different complications; for example, hepatocellular carcinoma (HCC) occurs more often in viral hepatitis liver cirrhosis (LC) cases. However, the exact relationship between the LC etiology and complications has

not been verified in large sample studies.Viral hepatitis remains a major cause of liver cirrhosis, but the ratio has decreased to less than 80%. Meanwhile, cases of autoimmune, cryptogenic, and mixed etiology are increasing gradually, from which an increase in incidence and prevalence can be expected in the future. Liver cirrhosis caused by alcohol consumption accounted for significantly more patients with upper gastrointestinal haemorrhage. HBV has long been considered a strong carcinogenic agent in liver cirrhosis patients, and our study confirmed this point of view, suggesting that more measures should be taken to prevent and manage viral hepatitis cirrhosis.Research study found that viral hepatitis is the most prevalent cause of liver cirrhosis in our hospital, accounting for more than 80% of all liver cirrhosis patients. In addition, most of the viral hepatitis liver cirrhosis was caused by HBV (nearly 96% of all viral hepatitis liver cirrhosis cases) [14]

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Conclusion

Liver disease is complex and not well understood many substance misuse professionals, yet liver disease is a common cause of illness and death among drinkers. Stoppingor reducing drinking can improves outcome from alcoholic liver disease, even in advanced stage of liver disease. Effective treatment for advanced alcoholic liver disease and its complications, in addition to stopping/reducing drinking. The two should be tackled together.

Comment [DA21]: should include the main finding of the study

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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