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Case Report on Cirrhosis of Liver

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

ABSTRACT

Liver is the second largest organ in human body, more than 5,000 separate bodily functions including helping blood to clot, cleansing the blood of toxins to converting food into nutrients to control hormone levels, fighting infections and illness, regenerating back after injury and metabolizing cholesterol, glucose, iron and controlling their levels. A 56- years old patient was admitted in AVBRH on date 9/12/2020 in ICU with the chief complaint of abdominal distension, breathlessness on exertion, pedal edema, fever since 8 days. After admitted in hospital all investigation was done including blood test, ECG, fluid cytology, peripheral smear, ultrasonography, etc. All investigation conducted and then final diagnosis confirmed 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 for 12 years. Patient was COVID-19 negative and admitted in intensive care unit. Patient had 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 300 mg BD, tab. Lasix 40 mg OD, tab. Udilive 300 mg BD, tab. Rifagut 300 mg BD, tab. Metformin 500 mg 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: Cirrhosis of the liver is one of the final stages of liver disease. It is a serious condition, causing scarring and permanent damage to the liver. Life expectancy depends on the stage and type of cirrhosis of liver. Cirrhosis progresses, more and more scar tissue forms, making it difficult for the liver to function (decompensated cirrhosis). Advanced cirrhosis is life-threatening. If liver cirrhosis is diagnosed early and the cause is treated, further damage can be limited and, rarely, reversed.

Keywords: Liver damage; treatment; nursing management.

1. 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]. Chronic liver disease (CLD) is a progressive deterioration of liver functions for more than six months, which includes synthesis of clotting factors, other proteins, detoxification of harmful products of metabolism, and excretion of bile. CLD is a continuous process of inflammation, destruction, and regeneration of liver parenchyma, which leads to fibrosis and cirrhosis [2].

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 [3]. 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 [4]. Global prevalence of cirrhosis from autopsy studies ranges from 4.5% to 9.5% of the general population [5]. Deaths from cirrhosis have been estimated to increase and would make it as the 12th leading cause of death in 2020 [6].

Around 10 lakh patients of liver cirrhosis are newly diagnosed every year in India. - Liver disease is the tenth most common cause of death in India as per the World Health Organization. Liver disease may affect every one in 5 Indians [7].

2. CASE REPORT

2.1 Patient Present History

A 56 –years- old patient was 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 including blood test, ECG, fluid cytology, CT scan etc. All investigations conducted and then final diagnosis confirmed as cirrhosis of liver.

2.2 Past History

Patient was not having any history of communicable disease or any hereditary disease but he has history of hypertension and type II Diabetes mellitus for 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.

2.3 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 [3].

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

2.4 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.

Table 1. Investigations of patient

Investigatio	ns	Patient value	Normal value	Inference
Renal function test				
 Potass 	ium (k+)-serum	4.5	3-5mEq/L	Normal
Creatin	e-serum	3.4	0.7-1.25mg%	Increased
Urea- s	erum	16.5	18-40mg%	Decreased
4. sodium	-serum	141	136-145mEq/L	Normal
Liver function test				
1. Albu	min	3.1	3-5gm%	Normal
Biliru	bin-total	5.0	0.3-1mg%	Increased
Prote	ein-serum	8.6	6-8gm%	Normal
4. AST	(GOT)	42	17-59I.U/L	Normal
5. ALT(SGPT)	34	0-35I.U/L	Normal
Complete blood count				
1. Hb		10.9	13-15.5gm%	Decreased
Total	RBC count	3.47	4.5-6millions/cu.mm	Decreased
Tota	l platelet count	1.52	1.5-4lacs/cu.mm	Normal
4. Tota	WBC count	14900	4000-11000/cu.mm	Increased
Total WBC count				
1. Mon	ocytes	04	4-10%	Normal
Gran	ulocytes	80	40-60%	Increased
3. Lym _l	ohocytes	18	17-48%	Normal
4. Eosi	nophils	01	0-5%	Normal
5. Basc	phils	00	0-2%	Normal

2.5 Investigation

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.

2.6 Medical Therapy

2.6.1 Pharmacological therapy

- Antibiotics
- Laxatives
- Vasopressin

- Diuretic therapy
- Human albumin solution
- 1. Replace fluid and electrolytes
- 2. Paracentesis
- 3. 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.

2.7 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 is 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.

2.8 Home care/Follow up care

- Advised patient to stop drinking alcohol
- Advised
- 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.

3. 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 [3] current treatment for cirrhosis are limited to removing the injurious stimulus and eradicating viruses by using interferon, ribavirin and lamivudine in viral hepatitis and liver transplantation. Transplantation is the very successful treatment for last stage cirrhosis[12].

Researcher found that Liver cirrhosis (LC) is an important cause of death globally, and prevention and treatment based on etiology is fundamental. The study illustrates that the major etiology of LC in Southern China is viral hepatitis, and the proportions of viral hepatitis and hepatitis B virus (HBV) are decreasing; whereas autoimmune, cryptogenic, and mixed etiology cases are increasing. Alcoholic LC patients exhibit a suffering risk of from gastrointestinal bleeding, and HBV LC patients exhibit greater risk of developing hepatocellular carcinoma [13].

Researcher reported that UGIB is the leading cause of death for patients with alcoholic liver cirrhosis [14].

Researcher found that among the major causes of LC, the OR of alcoholic LC is highest (OR = 1.89), indicating that alcoholic LC patients may have the highest risk of developing UGIB [15]. Researcher suggested that recent alcohol intake favors the development of gastro duodenal erosions. Above all, the cases of alcoholic LC may have more serious varices, higher HVPG, and more gastro duodenal erosions, resulting in a higher prevalence of UGIB [16].

4. CONCLUSION

Cirrhosis of the liver is one of the final stages of liver disease. It is a serious condition, causing scarring and permanent damage to the liver. Life expectancy depends on the stage and type of cirrhosis of liver. Cirrhosis progresses, more and more scar tissue forms, making it difficult for the liver to function (decompensated cirrhosis). Advanced cirrhosis is life-threatening. If liver cirrhosis is diagnosed early and the cause is treated, further damage can be limited and, rarely, reversed.

CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline Patient's consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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