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Review Article

Back to Basics in Cirrhosis of Liver

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Abstract

It is well said and proven in literature that important causes of cirrhosis of liver include alcohol intake, metabolic associated fatty liver disease and chronic hepatitis B and C. Less important causes include autoimmune and congenital liver disease, Wilson's and alpha1 antitrypsin deficiency disease. There can occur gradual progression from chronic hepatitis to cirrhosis and in certain cases can further reach stage of hepatocellular carcinoma. It is said that good clinician can recognize a cirrhotic patient from a distance, as he walks in for consultation, by certain characteristic clinical features associated with cirrhosis of liver. In this article, we have tried to highlight the same.

Keywords: Cirrhosis; Umbilical Hernia; Spider Naevi; Gynaecomastia; Hydrothorax; Caput Medusae

Introduction

The data pertaining to India regarding various clinical aspects of chronic liver disease (CLD) like aetiology, natural history, clinical presentation, treatment recommendations and its effect of public health, is limited [1-3]. Moreover, in comparison to developed countries, the burden of morbidity and mortality has not been analysed in depth in developing countries like India [3-5]. which is essential for determining the status of country's public health system [6,7]. The analysis of disease burden in particular geographical area helps in planning cost effective control measures [7]. The lack of exact data is detrimental in development of effective policies which can help in taming the menace of such deadly diseases and decrease the need of liver transplant [7]. The government bears huge expenditure on CLD patients which can be reduced substantially by investing on preventive strategies for developing chronic liver disease [8]. The important causes of cirrhosis of liver worldwide include alcohol intake, metabolic associated fatty liver disease and chronic hepatitis B & C followed by less important causes like autoimmune & congenital liver disease, Wilson's and alpha1 antitrypsin deficiency disease. There can occur gradual progression from chronic hepatitis to cirrhosis and hepatocellular carcinoma (H.C.C). There are certain typical clinical features in cirrhotic which can be easily recognized even on first visit.

Discussion

Caput medusae

It is one of the cardinal features of portal hypertension which is also seen in cirrhosis of liver and causes formation of caput medusae [9]. due to shunting of blood from the portal venous system through the umbilical veins into the abdominal wall veins, which manifest as the caput medusae [10]. Caput medusae in Latin means for 'head of Medusa' due to its resemblance to Medusa's hair once Minerva had turned it into snakes [11]. The appearance is due to cutaneous portosystemic collateral formation between distended and engorged paraumbilical veins that radiate from the umbilicus across the abdomen to join systemic veins. Dilated abdominal veins may be caused by clinical conditions such as inferior vena cava syndrome [12] or the superior vena cava syndrome with obstruction of the azygous system [13]. It usually requires no specific treatment and is less commonly seen in clinical practice these adays due to earlier diagnosis and treatment of portal hypertension. In some cases, complications such as bleeding of paraumbilical veins can occur which can be life-threatening and requires hemodynamic stabilization, correction of coagulopathy, direct compression or suture ligation of the bleeding varices should be performed to control bleeding and in refractory cases, therapeutic interventions such as

embolization or trans jugular intrahepatic portosystemic shunt may be used to decompress the collateral route

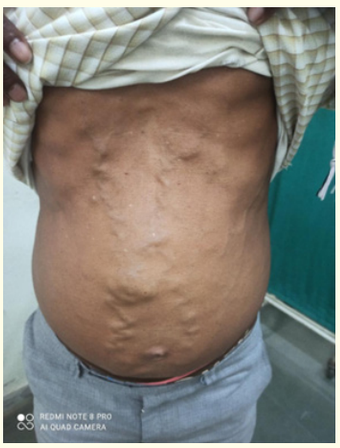

Figure 1: Caput Medusae.

Figure 2: Large Spider Naevi on Face.

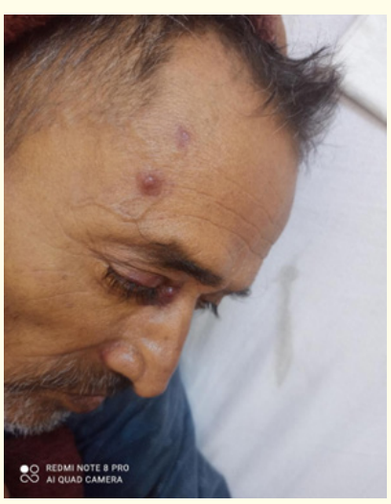

Figure 3: Spider Naevi on Temple.

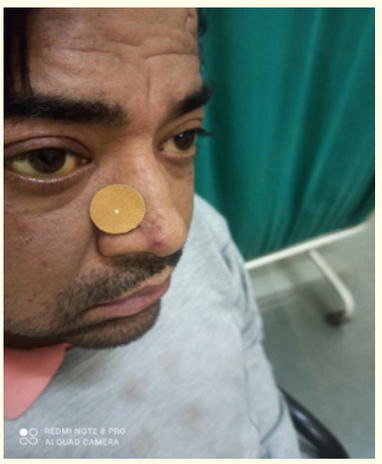

Figure 4: Spider Naevi on Nose.

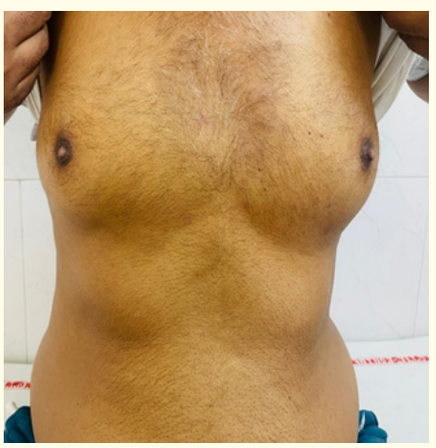

Figure 5: Bilateral Gynaecomastia.

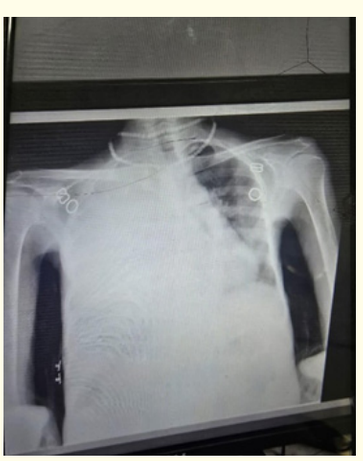

Figure 6: Right Hydrothorax.

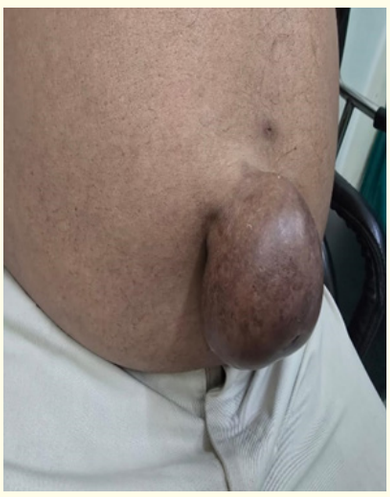

Figure 7: Umbilical Hernia.

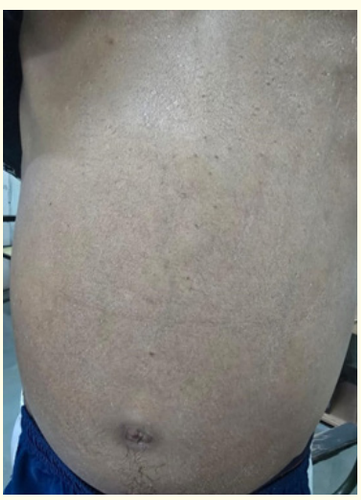

Figure 8: Tense Ascites.

Spider naevi

Spider angioma also called as spider naevus or spider telangiectasia, basically is a vascular lesion characterized by anomalous dilatation of end vasculature found subcutaneously. The lesion contains a central, red spot and reddish extensions which radiate outward like a spider's web and can be multiple or solitary lesions. A spider angioma has body, legs, and surrounding erythema. The body appears as a 1 to 10 mm central arteriole visible as a punctum or eminence. It is typically painless, resembles a spider's body, and is surrounded by attenuated capillaries radiating in a spider-legged fashion, decreasing in size toward the margins [14-16]. Spider angiomas can be seen in physiologic conditions like pregnancy or severe malnutrition but can be marker of an underlying systemic disease such as cirrhosis, rheumatoid arthritis, or thyrotoxicosis. Solitary spider angiomas are seen in 15% of young adults who usually have fewer than 3 lesions but multiple spider angiomas are characteristic of chronic liver disease with a specificity of 95% [17]. Spider angiomas are characteristically found on the face, neck, upper chest, and arms in adults, corresponding to the distribution of superior vena cava. They can also be seen in sites other than the skin, such as the mucosa of the oral cavity and gastrointestinal tract. Bleeding from these lesions is unusual unless picked or scratched. Rarely, fine-needle electrocautery, 585 nm pulsed, dye laser, 532 nm KTP (potassium-titanyl-phosphate) laser, or electro desiccation have been used to clear spider angioma for cosmetic concerns. They resolve after liver transplantation in cirrhotic patients.

Gynecomastia

It is benign enlargement of male breast tissue due to imbalance between oestrogen and testosterone levels and is commonly seen in men with cirrhosis because the liver's ability to metabolize and regulate these hormones is compromised. In cirrhosis, the metabolism of oestrogen is reduced, causing elevated oestrogen-to-testosterone ratio, which promotes breast tissue growth. It is commonly seen in old age and obese persons and can be painful or asymmetrical. Aldactone used as diuretic for treating ascites and pedal edema in cirrhotic can cause gynecomastia in some cases and if it occurs then it has to be substituted by other drugs like triamterene. Maintaining a healthy diet and regular exercise routine can help manage cirrhosis and reduce the severity of gynecomastia. Medications such as anti-oestrogens or aromatase inhibitors may be prescribed to counteract the effects of elevated oestrogen levels. In severe and refractory cases mastectomy may be considered [18].

Umblical hernia

Decompensated cirrhotic with ascites are at risk of developing umbilical hernia to the tune of 20%, which is ten times more than in the general population. The major causative factor is formation of ascites as it causes muscle wasting and increased intra-abdominal pressure. Due to low physiologic reserve in cirrhotic, there is increased risk of death from pre-operative, operative and post-operative complications of umbilical hernia during repair. Thus, umbilical hernia repair is usually done in complicated cases, such as strangulation of the bowel or rupture of the skin with leakage of ascitic fluid [19,20] and majority are managed conservatively. The risk of death with hernia repair in emergency condition is seven times higher than for elective hernia repair in cirrhotic patients [21]. In general population, female sex and obesity are risk factors for umbilical hernia but in cirrhotic patients, umbilical hernias is more likely seen in men with ascites [22-24]. Umbilical hernia recurrence rate in cirrhotic with ascites after surgery ranges from 0% to 40% [23,24]. The effective management of ascites is essential to repair success as well as to reduce recurrence rate. The elective hernia correction in relatively less decompensated liver, is associated with less complication, and is thus recommended.

Hepatic hydrothorax

It is defined as presence of a pleural effusion (usually >500 mL) in a patient with cirrhosis in the absence of other underlying causes like cardiac, pulmonary, kidney, pleural or malignant disease. It is right-sided in 85% of cases and left-sided in approximately 13%; whereas, only 2% of patients have fluid in the pleural cavity on both sides [25]. Hepatic hydrothorax (HH) occurs in approximately 5 to 15 percent of patients with cirrhosis and accounts for 2 percent of all pleural effusions. It occurs more frequently in cirrhotic patients having ascites, Porto-systemic hepatic encephalopathy and acute kidney injury (AKI). Rarely, it can be seen in absence of ascites. In a study of 495 patients with cirrhosis and pleural effusion, hepatic hydrothorax was seen in 16 percent. Cirrhotic with ascites usually tolerate up to 5 to 10 L of fluid with minimal symptoms, in contrast to those with a pleural effusion who can develop shortness of breath, cough, and hypoxemia, with as little as 500 mL of fluid. HH pathogenesis in cirrhotic patients have many explanations but the most probable one is ascites formation, due to portal hypertension related pathophysiological disorders [26]. Concomitant splanchnic and systemic arterial vasodilation, along with the activation of various neurohormonal signalling pathways, cause kidney dysfunction and hence decrease Na^+ and water excretion, as well as the glomerular filtration rate [27]. The negative pressure in intrathoracic cavity causes movement of ascites in peritoneal cavity to shift into the pleural space through small defects located mainly on the right side of the diaphragm. In current clinical practice, the therapeutic aim is to treat ascites and if necessary to perform thoracentesis. The target is to completely eliminate pleural effusion and signs of respiratory failure [28].

Conclusion

The scenario of bed side teaching has been replaced by battery of tests to which patients are subjected and only then final diagnosis is made. It has led to deterioration of clinical skills in the learners and more dependence on investigations. Ideally, a good clinician should be able to make diagnosis on basis of minimum and justified investigations. The awareness and good knowledge about clinical symptoms and signs of a disease like cirrhosis, helps in early diagnosis, progress and if required timely referral for liver transplantation.

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