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Case Report

Internal Medicine Section

Severe Mitral Stenosis Presenting as Chronic Liver Disease: A Rare Case Report

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ABSTRACT

High Serum Ascites Albumin Gradient (SAAG) ascites in severe rheumatic mitral stenosis is very uncommon. Hereby, the authors present a similar case with this rare combination. The present case report describes a 42-year-old male with no history of hypertension or diabetes, but occasional alcohol consumption, who presented to department with complaints of recurrent anasarca over the past five years. The electrocardiogram showed an irregular R-R interval with no P waves, indicating atrial fibrillation. Ascitic fluid analysis revealed a high serum ascites albumin gradient with low ascitic protein levels. The patient responded well to treatment and was discharged with appropriate medications. He was referred to a higher centre with cardiothoracic surgical facilities for mitral valve replacement and tricuspid annuloplasty. The present case report highlights the uniqueness of the combination of severe rheumatic mitral stenosis and ascites.

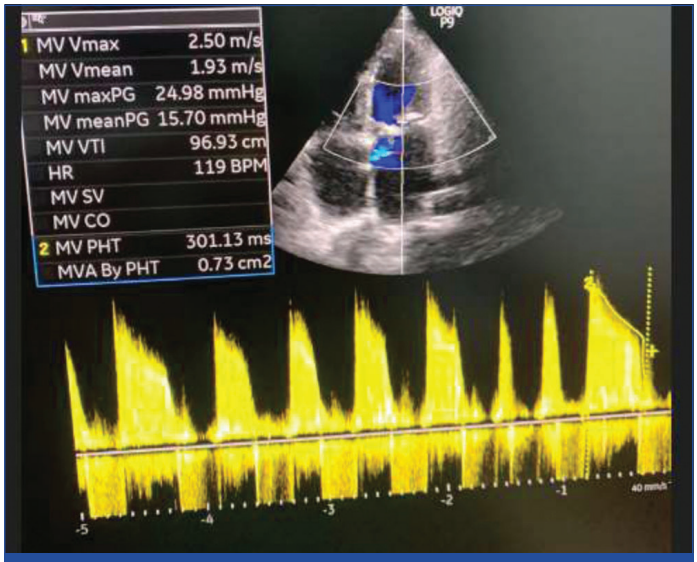
Keywords: Ascites, Atrial fibrillation, Liver disease

CASE REPORT

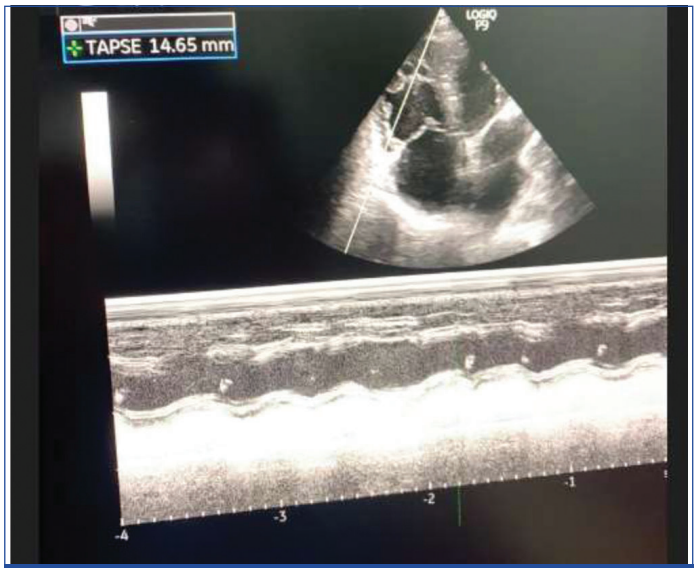
A 42-year-old male with no history of hypertension or diabetes, but occasional alcohol consumption, presented to Medicine ward with complaints of recurrent anasarca for the past five years. He denied any symptoms indicative of orthopnoea, paroxysmal nocturnal dyspnoea, oliguria, or frothy urine. Additionally, he denied any history of acute rheumatic fever during his childhood. The patient did not provide any treatment history for the past five years.

On examination, authors found moderate icterus and bipedal pitting oedema. There was an irregularly irregular pulse with an apex pulse deficit greater than 10. The internal jugular vein was engorged, and there was moderate ascites and mild hepatomegaly. Cardiac auscultation revealed an outward shift of the apex of the heart and a mid-diastolic murmur not radiating towards the axilla or base of the heart. The murmur was best heard on the left lateral decubitus with a loud P2. On chest auscultation, authors heard diffuse bilateral rhonchi. Blood investigations showed a Haemoglobin (Hb) level of about 12.4 gm/dL and a Total Leukocyte Count (TLC) of about 7300 microliters. Liver Function Test (LFT) revealed a total bilirubin level of about 13.99 mg/dL, direct bilirubin level of about 11.08 mg/dL, Alanine Transaminase (ALT) value of about 41 U/L, Aspartate Transaminase (AST) value of about 98 U/L, Alkaline Phosphatase (ALP) value of about 126 U/L, and an albumin level of about 3.8 gm/dL. The electrocardiogram showed an irregular R-R interval with no P waves, suggesting atrial fibrillation. Ascitic fluid analysis showed a high serum ascites albumin gradient with low ascitic protein.

Ultrasonography of the whole abdomen with splenoportal Doppler showed mild hepatosplenomegaly with hypoechoic liver parenchyma, dilated hepatic veins, moderate ascites, cardiomegaly, and a normal splenoportal axis with dilated caliber of portal and splenic veins. Elastography showed 5.51 kPa, suggestive of normal to mild hepatic fibrosis. Echocardiography [Table/Fig-1] showed severe mitral stenosis with a mitral valve aperture of 0.8 cm^2, mild tricuspid regurgitation, Tricuspid Annular Plane Systolic Excursion (TAPSE) of 14.65 mm, and a hint of pericardial effusion [Table/Fig-2]. Ocular examination showed the absence of Kayser-Fleischer rings, and the serum transferrin saturation was 14.6, ruling out Wilson's disease and haeochromatosis as potential causes of liver failure. The differential diagnosis in present case were Chronic Liver Disease (CLD) with portal hypertension, constrictive pericarditis, dilated cardiomyopathy, and mitral valve disease. After a battery of investigations, authors



[Table/Fig-1]: Echocardiographic features of mitral stenosis.



[Table/Fig-2]: Echocardiographic features of TAPSE.

finally diagnosed him as a case of severe mitral stenosis causing right-sided Heart Failure (HF), resulting in passive hepatic congestion and transient liver failure. He was being treated with diuretics and spironolactone, Tab Digoxin (0.25 mg), laxatives, Tab Warfarin (1 mg), and inhalers. The patient responded to treatment in 14 days and was discharged with the mentioned medications. He was referred to a higher centre equipped with cardiothoracic surgical facilities for mitral valve replacement and tricuspid annuloplasty. The patient underwent the surgery (mitral valve replacement with tricuspid valve repair) and returned to hospital for follow-up, which showed no accumulation of abdominal fluid as revealed by abdominal Ultrasonography (USG).

DISCUSSION

In a clinical perspective, HF can present as a syndrome of reduced exercise tolerance, with symptoms such as dysaponea and fatigue, related to impaired cardiac output. Alternatively, it can present as a syndrome of fluid retention caused by elevated filling pressure [1]. HF, particularly right-Sided Heart Failure (RHF), can also lead to various hepatic abnormalities. Any cause of right ventricular dysfunction can be associated with severe hepatic congestion, even in patients who are asymptomatic, and this condition may only be suggested by abnormal LFTs during routine laboratory analysis [2].

The primary pathophysiology implicated in hepatic dysfunction in HF is passive congestion resulting from increased filling pressures or reduced cardiac output, which affects perfusion. Elevated Central Venous Pressure (CVP) can cause passive hepatic congestion, leading to increased liver enzymes and both direct and indirect serum bilirubin levels. Additionally, decreased cardiac output and impaired perfusion can be associated with acute hepatocellular necrosis, resulting in significant elevations in serum aminotransferases. In patients with significant Tricuspid Regurgitation (TR), a prominent systolic pulsation of the liver, attributed to an enlarged right atrial V wave, is often observed. A presystolic pulsation of the liver, caused by an enlarged right atrial A wave, can occur in various other conditions, including pulmonary hypertension (primary or secondary), tricuspid stenosis, restrictive cardiomyopathy involving the right ventricle, and constrictive pericarditis [2].

Heart failure is characterised by the inability of systemic perfusion to meet the body's metabolic demands and is typically caused by pump dysfunction. Occasionally, it presents with features of non cardiac dysfunction, such as hepatic failure. This can be due to passive congestion resulting from increased filling pressures or low cardiac output and impaired perfusion [2]. Interestingly, ascites may be observed in up to 25% of patients, but splenomegaly is typically absent, even in the presence of ascites and lower extremity oedema [3]. Jaundice is commonly not reported.

Hepatic congestion can arise from various causes of right-sided HF, including constrictive pericarditis, severe Pulmonary Arterial Hypertension (PAH), mitral stenosis, Tricuspid Regurgitation (TR), cor pulmonale, cardiomyopathy, and as a postoperative consequence of the Fontan procedure for pulmonary atresia and hypoplastic left heart syndrome [4].

On the other hand, the course of mitral stenosis can be insidious and may span several decades, with most patients developing symptoms in their fourth or fifth decades of life. The onset of dysaponea is often related to the development of pulmonary hypertension In a study conducted by Harding MB et al., they reported a case of critical mitral stenosis in which the patient developed new-onset atrial fibrillation with low-output congestive cardiac failure and fulminant ischemic hepatic failure, leading to severe coagulopathy. Percutaneous mitral valvotomy helped in the recovery of all the patient's symptoms [5].

The SAAG is often an effective basic test for distinguishing between different causes of ascites. One of the key characteristics of HF is high-gradient ascites (SAAG >1.1 g/dL), while low-gradient ascites in HF is relatively uncommon and requires further testing to rule out more serious causes, including infection and cancer [6]. Typically, HF-related ascites is associated with a high SAAG and high ascitic protein levels. However, in rare cases, it has been observed that ascites due to HF can result in a low SAAG. In such cases, other causes of low SAAG ascites need to be ruled out, and the cause is usually passive hepatic congestion causing low SAAG ascites due to increased backflow in congestive HF [6]. However, the present case reported high SAAG ascites with low ascitic protein, which contradicts the earlier study by Trongtorsak A et al., [6].

In general, ascites is an abnormal collection of fluid in the peritoneal space. The most common cause of ascites is decompensated liver cirrhosis. However, various conditions could also lead to ascites, including congestive HF [7]. In other words, venous return is impeded in HF, resulting in an expansion of venous volume, higher hydrostatic pressure, and subsequent filtration of fluid into the peritoneal cavity [8].

Furthermore, the SAAG was introduced into clinical practice as a more useful parameter to help determine the aetiology of ascites, as it reflects the oncotic-hydrostatic balance [9,10]. A SAAG of 1.1 g/dL or greater indicates the presence of portal hypertension, which is a characteristic feature of HF-related ascites [6,11].

In the present case report, authors observed severe mitral stenosis presenting as chronic liver disease. The present study was the first-time endeavour in the eastern part of India, and authors did not find any similar studies previously.

CONCLUSION(S)

Heart failure related ascites is well known for high gradient ascites (SAAG >1.1 g/dL) related to portal hypertension, which can be obtained through electrocardiography for irregular R-R intervals. It can be concluded that the ascitic fluid analysis revealed a high gradient of serum ascites for albumin with low ascitic protein, indicating the occurrence of severe mitral stenosis along with chronic liver disease.

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