Case Report

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Constrictive Pericarditis: A Rare Cause of Cardiac Cirrhosis

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ABSTRACT

Hepatic dysfunction secondary to cardiac causes is a challenging clinical scenario which is often multifactorial and requires meticulous approach for early diagnosis. The liver disease can be secondary effect of any cause of right-sided heart failure including constrictive pericarditis, tricuspid regurgitation, mitral stenosis, cor pulmonale, or cardiomyopathy. Constrictive pericarditis is a rare cause of hepatic derangement and early diagnosis and management is crucial for improved patient outcomes.

Introduction

The circulatory system is a complex balance between multiple organs. Liver is a highly vascular organ and receives around 25% of the cardiac output [1]. Disruption in the circulatory system from heart diseases inadvertently affect the liver leading to hepatic decompensation. Acute as well as chronic liver injury can be seen in various heart conditions. Ischemic hepatitis, congestive hepatopathy, cardiac cirrhosis from long standing retrograde pressure, Fontan liver diseases and liver injury from cardiac medications are commonly encountered liver pathologies in setting of cardiac ailments [2]. Often patients with cardiac conditions present with an array of symptoms and multitude of laboratory abnormalities. The liver dysfunction is multifactorial, and it is often perplexing for clinicians to identify the inciting factor. This case highlights liver impairment in the setting of constrictive pericarditis and the subsequent improvement after pericardiotomy.

Case

A 51-year-old Caucasian woman with a history of atrial fibrillation and ischemic stroke status post thrombectomy presented with a complaint of bilateral lower extremity edema for three years that had worsened over the last year. The patient also reported abdominal distension and a 40-pound weight gain over the last year. She denied any chest pain, palpitations or syncope. Physical examination revealed jugular venous distension of 18cm, an irregularly irregular rhythm and 3+ pitting edema. Abdominal examination revealed significant wall edema with positive hepatojugular reflex. Laboratory data showed an elevated total bilirubin of 6.9mg/dL, INR 2.5, platelets of 76*10^3, and albumin of 3.6g/dL. AST, ALP and ALT were unremarkable. Hepatitis panel was negative. CT abdomen/pelvis showed dense pericardial calcifications and severe hepatic congestion along with anasarca, moderate ascites and a left pleural effusion. Hepatic morphology consistent with borderline cirrhosis and portal hypertension was also noted. Abdominal ultrasound showed hepatic steatosis with probable cirrhosis and portal hypertension. A transthoracic echocardiogram revealed severely reduced right ventricular systolic function with a moderately thickened pericardium and dilated inferior vena cava (IVC). Trans esophageal echocardiogram showed LVEF>55% and no evidence of thrombus in left atrial appendage. Right and left heart catheterization revealed markedly elevated filling pressures with diastolic equalizations. The patient was diagnosed with idiopathic constrictive pericarditis and underwent pericardiotomy. Pericardial biopsy showed dystrophic calcification and ossification. After an uncomplicated operation she required aggressive diuresis, ionotropic support and amiodarone for atrial

fibrillation. Five days post-procedurally, her liver function initially worsened. Her ALP increased to 196U/L, her ALT was 32U/L and her AST rose to 93U/L. Her total bilirubin worsened to 42.3mg/dL with a direct bilirubin of 30 mg/dL. Patient developed altered mental status and significant jaundice was noted. Amiodarone was discontinued, and she was started on lactulose for hepatic encephalopathy. Repeat abdominal US revealed sludge with no evidence of cholestasis. The patient underwent a liver biopsy which was consistent with cirrhosis with marked bile stasis and minimal inflammation. Her creatinine also rose to 2.4mg/dL from 1.1mg/dL. Gradually, her liver function tests improved over time and she was transferred out of the intensive care unit. She was discharged after being hospitalized for around two months, with regular follow ups to trend her liver function tests which trended downwards on subsequent office visits. Table 1 shows the trend in liver enzymes.

Discussion

Diagnosing the cause of liver injury in the setting of heart diseases is often a clinical dilemma. Our case is unique as cirrhosis was observed in the setting of right heart failure due to constrictive pericarditis. Primarily cardiac cirrhosis is a term use for hepatic derangements occurring in the context of cardiac dysfunction [3]. Cardiac conditions that cause cirrhosis due to hepatic congestion include valvular disease, severe pulmonary hypertension, cor pulmonale, heart failure and pericardial diseases such as constrictive pericarditis and tamponade [3]. Amongst the causes of cardiac cirrhosis, diagnosing constrictive pericarditis requires a high degree of suspicion to its rarity [4]. Constrictive pericarditis may be idiopathic or occur in patients with a history of pericarditis, trauma, cardiothoracic surgery, thoracic radiotherapy, or connective tissue disease [5] Cardiac hepatopathy has diverse clinical and histological manifestations that evolve throughout the natural history of the underlying cardiac disease. Liver associated enzymes in patients with cardiac diseases often vary [6,7]. However, when elevated indicate a poor prognosis [8,9]. Hepatic fibrosis is commonly seen and cirrhosis is rare [6]. Histological changes in the setting of heart failure involves sinusoidal dilatation progressing to fibrosis and development of regenerative nodules. Inflammation is minimal or absent [10,11]. Patients with cardiac cirrhosis are usually asymptomatic. Symptoms commonly occur in the setting of acute right ventricular decompensation, acute decompensation of valvular disease or constrictive pericarditis as reported in our patient with longstanding undiagnosed pericarditis. Symptomatic patients often present with abdominal pain, distension, hepatomegaly, ascites, nausea and vomiting. Peripheral edema and jugular venous distension are often noted [3]. Jaundice is less commonly reported typically occurs only in severe disease [12]. Once constrictive pericarditis is diagnosed, urgent pericardiotomy remains the treatment of choice [13]. In cardiac cirrhosis, treatment of the cardiac problem is the key to improvement in hepatic dysfunction [14]. Constrictive pericarditis is a rare cause of cirrhosis and hepatic derangements. The treatment lies in correcting the underlying cardiac problem. This case highlights the challenges faced in identifying the causative agent in the setting of multiple risk factors and focuses on obtaining relevant history, physical exam and data to differentiate potential causes of liver cirrhosis. It illustrates that clinicians should consider cardiac etiology in the workup for cirrhosis, especially in the absence of common etiologies and with liver biopsy consistent with hepatic congestion and inconclusive of primary liver disease.

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