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Persistent Untreated Pericardial Effusion Leading to Congestive Hepatopathy

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Abstract Congestive Hepatopathy (CH) is an important differential diagnosis when evaluating patients who present with right upper quadrant pain, hyperbilirubinemia, and jaundice. We describe a 54-year-old male with a chronic pericardial effusion who develops cholestatic jaundice and acute hepatic injury as a result of early cardiac tamponade. This case report demonstrates the challenges in diagnosing congestive hepatopathy and reviews the pathophysiology that leads to variable presentations of the condition.

Keywords: *Congestive Hepatopathy, Pericardial effusion, Elevated liver enzymes, Abdominal pain*

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1. Introduction

Congestive hepatopathy (CH) is when passive venous congestion leads to liver dysfunction and subsequent injury. Such congestion often results from right heart failure, as seen in constrictive pericarditis, tricuspid regurgitation, mitral stenosis, cor pulmonale, and cardiomyopathy [1]. This case report documents an atypical presentation of CH that resulted from a chronic pericardial effusion. The patient presented with jaundice, right upper quadrant pain, and hyperbilirubinemia, which are findings consistent with CH [2]. Although the patient had a chronic, intermittent, dry cough, he did not demonstrate overt cardiac symptoms suggestive of right heart failure. In addition, the patient's laboratory findings, including elevated liver function tests, deviated from the collective descriptions of congestive hepatopathy. As a result, an extensive work up for liver pathology and extrahepatic sources of cholestasis delayed the patient's diagnosis of early cardiac tamponade.

2. Case Presentation

A 54-year-old male with past medical history of type 2 diabetes mellitus, hypertension, hypothyroidism, history of deep venous thrombosis (not on anticoagulation), and obstructive sleep apnea presented to the Emergency Department complaining of sharp right upper quadrant pain and dark colored urine for three days. He also admitted that his skin looked more yellow than normal. The patient had an intermittent dry cough, but he denied chest pain, shortness of breath, or palpitations. Review of systems was positive for fever and chills. The patient denied recent travel, family history of liver disease, alcohol consumption, tobacco or drug use, or recent blood transfusion.

On arrival, the patient was hemodynamically stable with visible jaundice and abdominal distension. He was found to have distant heart sounds, but no notable edema, jugular venous distension, or other significant cardiac findings upon physical exam. Initial laboratory values revealed leukocytosis, thrombocytosis, hyperbilirubinemia, and elevated alkaline phosphatase. Aspartate aminotransferase and alanine aminotransferase were within normal limits. Urinalysis was positive for bilirubin and protein. In addition, ferritin, gamma glutamyl transferase, erythrocyte sedimentation rate, and c-reactive protein were found to be elevated. Lipase, lactic acid, alcohol and acetaminophen levels were all within normal limits.

Our working diagnosis at the time was primary obstructive liver disease causing cholestasis, and this guided the following work up of additional laboratory tests and imaging. The acute hepatitis panel was negative, which made a viral etiology less likely. And the patient's autoimmune serology, which included anti nuclear antibody, anti mitochondrial antibody, and anti smooth muscle antibody, were all normal. Despite these findings, the patient had persistent leukocytosis and hyperbilirubinemia, his alkaline phosphatase remained above 450, and his liver enzymes began trending up. Ultrasound of the abdomen showed trace ascites but no acute intra-abdominal process. Computed tomography of the abdomen and pelvis with IV contrast showed a moderate to large size pericardial

effusion, which, when compared to past imaging, had been present for years. The etiology of this effusion was unknown, but it seemed to have occurred when the patient was diagnosed with untreated hypothyroidism. The effusion had not been drained in the past since the patient was asymptomatic, and it was presumed that the effusion would resolve once the patient's hypothyroidism was treated appropriately. The patient had been properly treated for hypothyroidism since diagnosis, but despite him being compliant with treatment, the effusion persisted. Given these findings, the patient was scheduled for an echocardiogram to evaluate his chronic pericardial effusion; however, the hepatic imaging and biopsy took precedence given his clinical picture.

Further imaging with magnetic resonance cholangiopancreatography (MRCP) showed a contracted gallbladder and enlarged left hepatic lobe, which was suggestive of early hepatocellular disease. There was no evidence of an extrahepatic biliary obstruction. Since the work up thus far showed nonspecific hepatocellular disease, we decided to proceed with liver biopsy. A liver biopsy was consistent with suspected liver disease, showing venous outflow obstruction, subtle mild bland lobular cholestasis, and mild, focal subsinusoidal fibrosis. However, the cause of the patient's hepatic injury was still unclear.

On day five of the patient's hospital stay, he underwent echocardiogram and was found to have a large pericardial effusion with septations. There was diastolic collapse of the right ventricular outflow tract, and a respiratory variation of approximately 37% in mitral valve inflow velocity and approximately 61% in tricuspid valve inflow velocity (Figure 1 - Figure 3). These findings were suggestive for tamponade physiology and raised concerns of early tamponade, prompting emergent pericardiocentesis. The patient, however, remained largely asymptomatic. One liter of fluid was drained from the pericardial sac, and analysis of the fluid was negative for malignancy. The patient tolerated the procedure well and was started on colchicine and indomethacin post-operation.

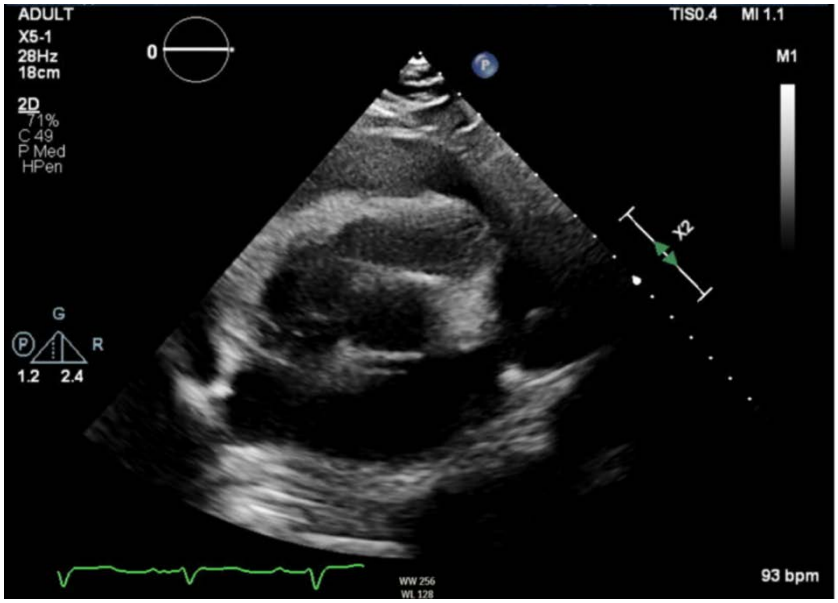

Figure 1. Diastolic collapse of the Right ventricle outflow tract

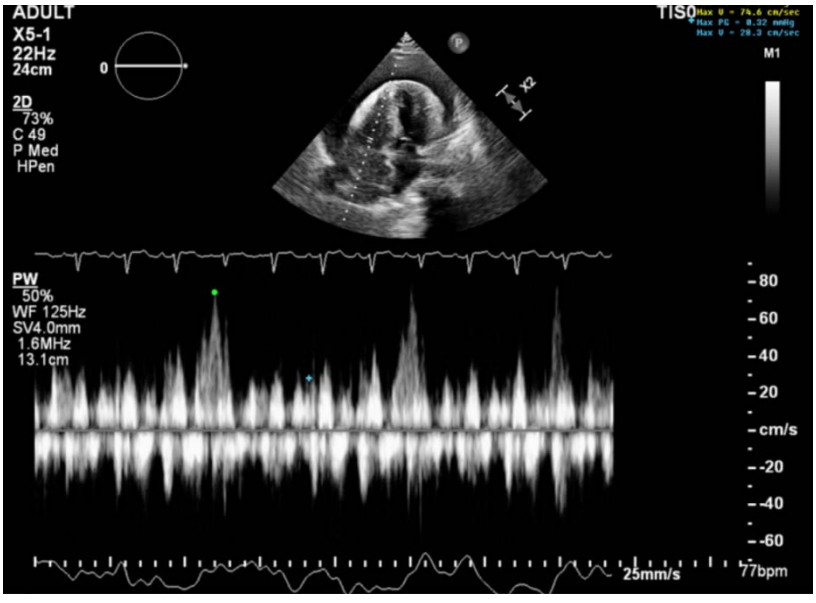

Figure 2. Respiratory variation of ~ 37% in mitral valve inflow velocity, suggestive for tamponade physiology

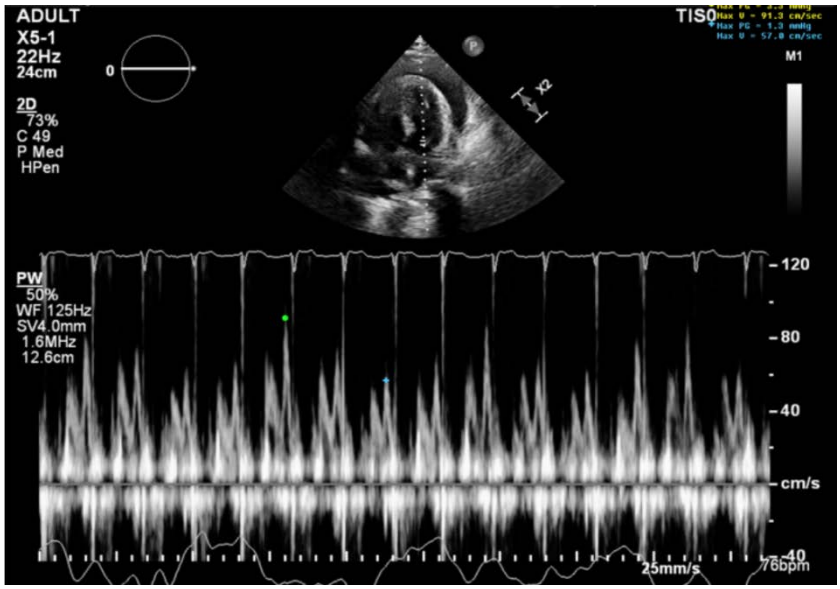

Figure 3. Respiratory variation of ~ 61% in tricuspid valve inflow velocity, suggestive for tamponade physiology

Over the following days post-operation, the patient's lab values normalized, with the liver enzymes returning to normal and alkaline phosphatase starting to trend down. The patient's white blood cell count returned to normal within three days following the pericardiocentesis. In addition, the patient reported resolution of his right sided abdominal pain and cough, and his urine was no longer discolored. Repeat echocardiogram prior to patient's discharge confirmed minimal pericardial effusion and no hemodynamic compromise. At a follow up appointment two months later, the patient underwent an echocardiogram which showed resolution of the effusion.

Table 1. Trend of Liver function tests

	Day 1	Day 3	Day 6	Day 8	7 week follow up
ALP	370	499	424	355	244
AST	24	41	33	21	21
ALT	46	70	52	37	30
Total Bilirubin	2.8	2.1	1.4	0.6	0.7

3. Discussion

This is the first documented case of congestive hepatopathy resulting from a subacute to chronic pericardial effusion that evolved into an early cardiac tamponade. While there is notable literature on conditions of right sided heart failure causing hepatic congestion, our patient's presentation was more subtle than what is described in review articles and other case reports. As a result, it was more challenging to identify the patient's underlying condition, and a diagnosis of congestive hepatopathy was only achieved after the patient underwent treatment.

Congestive hepatopathy is when passive venous congestion leads to liver dysfunction and subsequent injury. The congestion is due to right heart failure, such as constrictive pericarditis, tricuspid regurgitation, mitral stenosis, cor pulmonale, and cardiomyopathy [1]. Although our patient had an untreated pericardial effusion, his nonspecific symptoms obscured the association between his seemingly stable, chronic cardiac condition and his acute liver injury.

The first indication of congestive hepatopathy may be abnormal liver laboratory tests, since patients are often asymptomatic. Symptomatic patients can present with jaundice, which could be mistaken for biliary obstruction [2]. As seen in our patient case, one of his chief complaints was yellow skin, prompting the initial work up to focus on liver pathology and extrahepatic sources of cholestasis. Jugular venous distension is a key physical finding that helps distinguish congestive hepatopathy from primary liver disease and Budd-Chiari syndrome. While hepatojugular reflux is generally present with hepatic congestion, it was not apparent in our patient [2]. Additional physical findings associated with CH include ascites, hepatomegaly, and liver tenderness. This right upper quadrant discomfort, as seen in our patient case, is believed to be due to stretching of the liver capsule [2].

Mildly elevated serum bilirubin is the most common abnormal liver laboratory value and is seen in approximately 70% of patients. Total serum bilirubin tends to be less than 3 mg/dL and consists of mostly unconjugated bilirubin [3]. The precise cause of hyperbilirubinemia is uncertain, but in our patient's case, hepatocellular dysfunction and canalicular obstruction due to distended hepatic veins could have been contributing factors. According to the literature on congestive hepatopathy, bilirubin levels correlate with right atrial pressures but not with cardiac output [4].

Other liver biochemical tests are typically only mildly elevated, including alkaline phosphatase. Alkaline phosphatase tends to be normal or minimally elevated in cases of acute heart failure, helping distinguish hepatic congestion from biliary obstruction [5,6]. However, this was not the case for our patient, who consistently had elevated alkaline phosphatase three to four times the upper limit of normal.

Aminotransferases can be elevated in one third of patients with congestive hepatopathy. The elevated levels are secondary to acute hepatic ischemia, and the degree of elevation is found to correlate with the extent of necrosis seen on liver biopsy [7]. When jaundice is accompanied

by elevated aminotransferase levels, the patient's presentation can resemble acute viral hepatitis [8,9]. Unless cardiac output is impaired, aminotransferase levels are usually not more than three times the upper limit of normal [2].

While congestive changes are reportedly seen on abdominal imaging of CH, such findings were not apparent in our patient's imaging. Ultrasound of the liver typically demonstrates dilation of the inferior vena cava and suprahepatic veins, as well as hepatomegaly with a homogeneous increased echogenicity. Similarly, computed tomography and magnetic resonance imaging show hepatomegaly, distended hepatic veins and inferior vena cava, and early reflux of contrast material from the right atrium to the inferior vena cava [4]. Since liver fibrosis could develop before congestive hepatopathy is clinically recognized, it is crucial to be able to recognize the condition on imaging [10].

When the diagnosis of congestive hepatopathy is indeterminate, a liver biopsy can help confirm the condition. Identifying the severity of histologic injury from passive congestion is also beneficial when patients have coexisting liver disease. Under normal physiologic conditions, low hydrostatic pressure in the liver is maintained by free blood flow through sinusoidal fenestrations. In hepatic congestion, increased hydrostatic pressure from right heart failure causes sinusoidal edema and hemorrhage. This in turn decreases circulating oxygen and leads to necrosis of the hepatic acinus in zone 3 and hepatocyte atrophy [4]. Ischemic injury and subsinusoidal fibrosis were noted on our patient's liver biopsy, along with venous outflow obstruction. Liver pathology can also demonstrate variable degrees of cholestasis and occasionally bile thrombi in the canaliculi, however this is more commonly seen in CH patients with severe jaundice [11]. While no bile thrombi were visualized in our patient's biopsy specimen, there was subtle cholestasis.

Management of congestive hepatopathy entails treating the underlying heart disease. Prognosis is predicted by the severity of the underlying cardiac condition, since most patients with congestive hepatopathy die of cardiac causes. Liver disease rarely contributes to morbidity and mortality in these patients [2]. In addition, early histologic changes associated with hepatic congestion may resolve with treatment. Diuresis is noted to aid in the resolution of jaundice and ascites, however excess diuresis should be avoided as it could impair hepatic perfusion [12].

Hepatic congestion should be suspected in patients with jaundice, hyperbilirubinemia, and cardiac conditions associated with elevated central venous pressure. Congestive hepatopathy may be missed in patients with overt hepatic congestion and vague cardiac symptoms. Considering that our patient did not present with distinct congestive hepatopathy, it was particularly challenging to diagnose his condition. The markedly elevated alkaline phosphatase, the lack of characteristic findings in the patient's presentation, and the indistinct hepatic imaging results made this patient case unconventional. In addition, the patient did not exhibit hepatojugular reflux, which is seen in both congestive hepatopathy and cardiac tamponade. This case demonstrates the importance of maintaining a wide differential and considering cardiac problems (i.e., right heart failure, pericardial effusion, or tamponade) as an underlying source for elevated liver function tests. For if this association had been recognized earlier in our patient's case, it could have prevented the undertaking of invasive procedures such as MRCP and liver biopsy.

4. Conclusion

- Congestive hepatopathy (CH) is hepatic injury from passive venous congestion within the liver that arises secondary to right-sided heart failure (ie, cardiomyopathy, tricuspid regurgitation, cor pulmonale, constrictive pericarditis, mitral insufficiency).
- While CH can be asymptomatic, moderate congestion causes right upper quadrant pain (due to stretching of the liver capsule), hepatomegaly, and jaundice.
- It is important to keep cardiac problems (i.e., right heart failure, pericardial effusion, or tamponade) as a differential for patients presenting with elevated liver function tests.
- There should be a low threshold for an in-depth cardiac work up if other etiologies have been ruled out.

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