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Ischemic hepatitis triggered by cardiac tamponade: A case report

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ABSTRACT

Background: Ischemic hepatitis (commonly referred to as shock liver and hypoxic hepatitis) is an uncommon clinical condition associated with massive but transient elevation of liver enzymes up to more than 20 times the standard limit; without evidence of other causes of acute hepatitis such as viral, alcoholic, metabolic, and toxin-induced hepatitis. Clinically, it presents as an acute liver failure caused by decreased hepatic perfusion and oxygen delivery. It is often encountered in critically ill patients in intensive care units and can rarely be triggered by pericardial tamponade. ***Case presentation:*** On hemodialysis, a 33-year-old end-stage renal disease male patient with hypertension and cardiomegaly presented with right upper quadrant pain. Examination revealed elevated jugular venous pressure, displaced apical beat, tender right upper quadrant and epigastric areas, and enlarged liver with a round border and soft surface. Electrocardiography revealed P mitral and electrical alternans but no ischemic changes. Chest radiography showed an increased cardiothoracic ratio and flask-shaped heart. Extensive laboratory investigations showed elevated levels of aspartate aminotransferase (5000 U/L) and alanine aminotransferase (7000 U/L). Ultrasound showed passive congestion of the liver. Pericardial effusion was confirmed by echocardiography, and the diagnosis of cardiac tamponade was established. Pericardiocentesis was done, and 1200 ml of bloody fluid was aspirated. After pericardiocentesis, the patient demonstrated dramatic improvement. ***Conclusions:*** Ischemic hepatitis is an uncommon entity associated with a significant mortality rate, mainly when it develops on top of decreased cardiac output. Therefore, physicians must be familiar with the broad differential diagnosis for liver disease signs and symptoms and should always consider the possibility of ischemic hepatitis in such cases.

Keywords: Ischemic Hepatitis, Cardiac Tamponade.

BACKGROUND

Ischemic hepatitis, commonly referred to as shock liver and hypoxic hepatitis, is an uncommon clinical condition associated with a massive but transient elevation in liver enzymes up to more than 20 times the normal limit; without evidence for other causes of acute hepatitis, including viral, alcoholic, metabolic, and toxin-induced hepatitis. It presents clinically as an acute liver failure and as centrilobular necrosis with minimal inflammation on histological examination [1].

Ischemic hepatitis is usually preceded by systemic hypotension, with subsequent decreased hepatic perfusion and oxygen delivery. It is commonly encountered in critically ill patients admitted to intensive care units [1]. Ischemic hepatitis is the most common cause of extreme elevation (>1000 U/L) of liver aminotransferases, which can also be found in viral and toxin-induced hepatitis [2].

Pericardial tamponade is a rare trigger of ischemic hepatitis. Massive pericardial tamponade decreases cardiac output, impairs hemodynamics, and induces tissue hypoxia. Pericardial effusion is a known chronic renal failure complication that may lead to acute renal failure [3, 4], which is frequently associated with ischemic hepatitis [5]. Here, we present a case of ischemic hepatitis triggered by massive pericardial effusion in a patient with end-stage kidney disease.

Case presentation

A 33-year-old male patient with an established diagnosis of end-stage renal disease and hypertension for 1.5 years was undergoing hemodialysis when he started to experience sudden right upper quadrant (RUQ) pain. The pain was characterized as

stabbing and radiated to the back. The patient had been receiving 3 hemodialysis sessions per week regularly. His past medical history was also significant for cardiomegaly for 1.2 years, and his usual state of health was limited to moderate exercise. Following the onset of pain, he was transferred to the emergency department (ED), receiving a chest radiograph. He was subsequently treated as a case of chest infection and then discharged home. A few hours later, he returned to the ED due to worsening RUQ pain and was found to have elevated liver enzymes, and the patient was subsequently referred for further investigations and management.

Upon further investigation, the patient's physical exam showed borderline low blood pressure despite being off antihypertension medications, jugular venous pressure elevation, displaced apical beat, tender RUQ and epigastric area, and an enlarged liver (liver span was 22 cm) with a round border, soft surface, and no nodularity. Electrocardiography revealed P mitral and electrical alternans, but no ischemic changes were detected. Chest radiography showed an increased cardiothoracic ratio and a flask-shaped heart (Figure 1). Extensive laboratory investigations showed elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) (7000 and 5000 U/L, respectively), lactate of 4 mmol/L, total serum bilirubin (TSB) of 0.5 mg/dl, prothrombin time (PT) of 18.5 seconds, and international normalized ratio (INR) of 1.6. Additionally, he had elevated levels of blood urea nitrogen (BUN) and creatinine, which were 126.5 mg/dl and 18 mg/dl, respectively. Septic workup was significant for leukocytosis, elevated acute phase reactants levels, and moderate metabolic acidosis (pH value was 7.21 on blood gases testing), so he was started on broad-spectrum antibiotics.

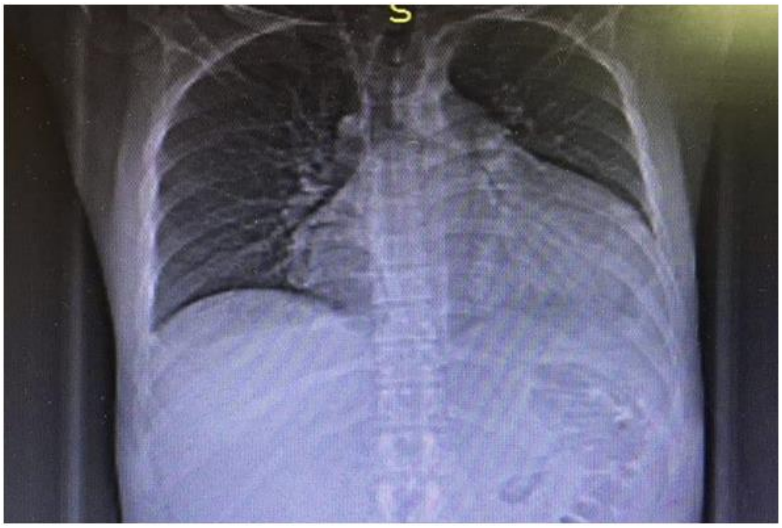

Figure (1): Chest radiograph showing flask-shaped heart.

Acute viral hepatitis, toxin-induced hepatitis, and shocked liver were considered differential diagnoses. Hepatitis profile came negative, toxin-induced hepatitis was unlikely given the patient's drug history, and Doppler ultrasound rolled out a vascular cause of acute liver injury. Ultrasound examination revealed passive congestion of the liver (Figure 2). A computed tomography scan followed this for a more accurate diagnosis of the liver abnormality, which coincidentally revealed pericardial effusion (Figure 3). This finding was confirmed by echocardiography, and the diagnosis of cardiac tamponade was established. Pericardiocentesis was done on the second day of admission, and 1200 ml of bloody fluid was aspirated. After pericardiocentesis, the patient demonstrated dramatic improvement. Based on the exclusion of other potential diagnoses and supported by the patient's improvement following pericardiocentesis, a diagnosis of ischemic hepatitis triggered by cardiac tamponade was reached. The patient was discharged after 8 days with an AST level of 21 U/L, ALT of 232 U/L, BUN of 37

mg/dl, and creatinine of 7.7 ml/dl as normal PT and INR values.

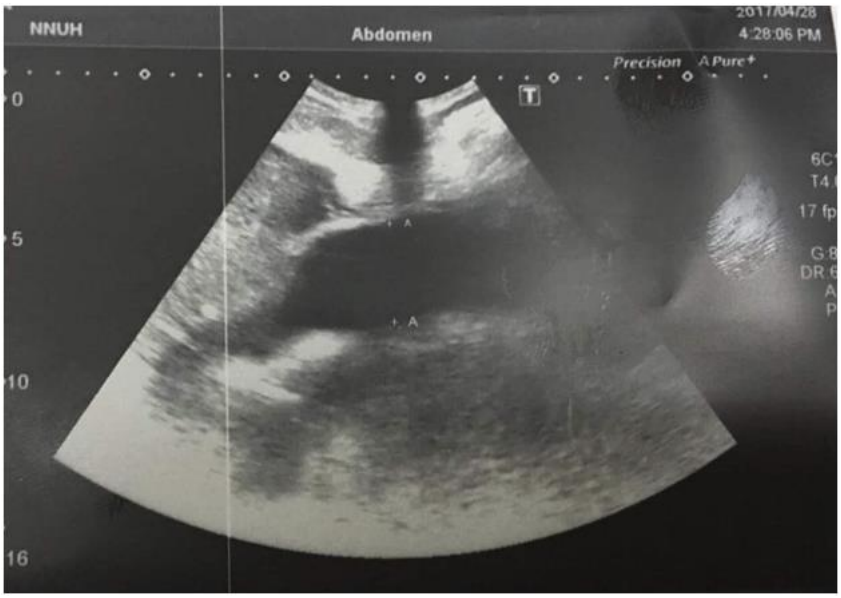

Figure (2): Ultrasound imagining showing passive congestion of the liver.

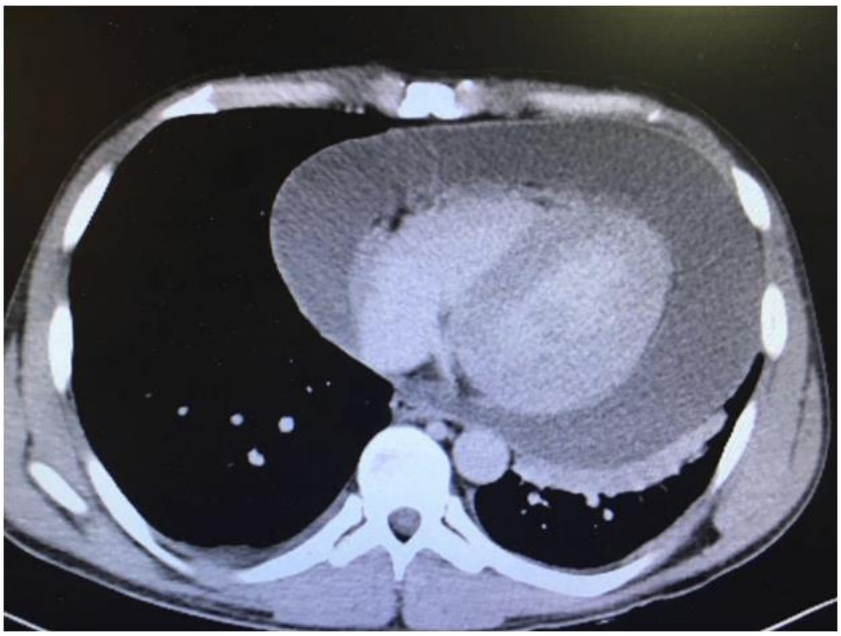

Figure (3): CT scan showing pericardial effusion.

DISCUSSION

Ischemic hepatitis is not a rare disease, as it occurs in 2.5% of admissions to the intensive care unit [6]. Generally speaking, the pathogenesis of ischemic hepatitis could be attributed to forward arterial failure and backward venous failure [7], both of which exist in the case of cardiac tamponade and heart failure. According to the relevant literature, the most commonly identified triggers of hepatic hypoxia are acute and congestive heart failure (especially right-sided failure), chronic respiratory failure, septic shock, and cardiac arrhythmias [8, 9]. Other uncommon implicated conditions include post cardiopulmonary surgery, hepatopulmonary syndrome, severe anemia, gastrointestinal hemorrhage, postpartum hemorrhage, obstructive sleep apnea, hepatic artery or complicated aortic aneurysm, and aortic dissection, vascular steal in celiac stenosis, and acute

limb ischemia [10-19]. Surprisingly, ischemic hepatitis has also been reported in patients with liver cirrhosis [20].

Before this case, few reports have described ischemic hepatitis triggered by a pericardial disease. For example, one case report described a case of ischemic hepatitis precipitated by constrictive pericarditis [21], whereas another presented a case of ischemic hepatitis caused by cardiac tamponade in a chronic kidney failure patient, similar to our patient [22].

Physical examination usually reveals enlarged liver, a sign that is usually absent in other causes of acute hepatitis. Signs of right-sided heart failure can also be observed. Additionally, passive congestion of the liver is another important finding in ischemic hepatitis [7]. It is worth mentioning here that congestive hepatopathy is a different clinical entity [23]. Liver congestion impairs oxygen diffusion due to edema, which makes hepatocytes more susceptible to hypoxic injury. This could also be linked to the liver enlargement observed in these patients.

Ultrasound is usually normal but may reveal hepatic vein dilatation due to passive congestion. Diagnosis is established based on clinical and laboratory findings. Typically, liver biopsy is undesirable [24]. Regarding liver function tests, the massive elevation of AST, ALT, and lactate dehydrogenase is expected within a short period (<72 hours). Normalization usually occurs within 7 to 10 days. Other less striking laboratory findings include mildly elevated total bilirubin, decreased prothrombin activity, and abnormal blood glucose levels [9].

Management mainly consists of supportive care and correction of the underlying cause. One study suggested that N-acetylcysteinecytoprotective may also be beneficial [25]. Another study reported that pretreatment with a statin could prevent the development of new-onset ischemic hepatitis in critically ill patients [26]. Calcium channel blockers and antiarrhythmic drugs could be associated with higher mortality in ischemic hepatitis due to increased inotropic support resistance or worsened preexisting cardiac disease [24]. Ischemic hepatitis is associated with high mortality during an acute presentation that could be as high as 50% [9]. The prognosis largely depends on the underlying condition that triggered the hypoxic episode. Developing hepatic encephalopathy and high serum phosphate level were poor prognostic factors for short-term survival [27].

CONCLUSION

Ischemic hepatitis is an uncommon entity associated with a significant mortality rate, mainly when it develops on top of decreased cardiac output. Therefore, physicians must be familiar with the broad differential diagnosis for liver disease symptoms and signs and should always consider the possibility of ischemic hepatitis in such cases.

Abbreviations

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; INR, international normalized ratio; PT, prothrombin time; RUQ, right upper quadrant; TSB, total serum bilirubin.

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Availability of data and materials

All relevant data are included in this manuscript.

Authors' contributions

QA was responsible for the clinical care of the patient. SM, ES, and AS contributed to the manuscript's design, drafting, and revision for the case report and the patient's data collection. QA critically reviewed the manuscript. All authors read and approved the final manuscript.

Ethics declarations

Ethics approval and consent to participate

There was no ethics committee approval as the data has been analyzed retrospectively and has no effect on treatment.

Consent for publication

Written informed consent was obtained from the patient's legal guardian to publish this case report, including all image data.

Competing interest

The authors declare that they do not have any competing interests.

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