CASE REPORT IN INFECTIOUS DISEASES

Tuberculous cardiac tamponade presenting as severe hypoxic hepatitis

Valdes Roberto Bollela[1], Fernanda Guioti Puga[1], Rodrigo Carvalho Santana[1]

Affiliations:

[1]School of Medicine of Ribeirão Preto, University of São Paulo, São Paulo, Brazil.

Corresponding author:

Prof. Valdes Roberto Bollela, Hospital das Clínicas, Faculdade de Medicina de Ribeirão Preto, Universidade de São Paulo, Departamento de Clínica Médica. Av. Bandeirantes 3900 Monte Alegre, 14049-900 Ribeirão Preto, São Paulo, Brasil. Tel.: +55 16 3602-2468. E-mail: vbollela@gmail.com.

Abstract

A 57-year-old man was referred to the Emergency Department with epigastric and respiratory dependent pain for six days. The physical examination showed mild jaundice, painful liver and muffled heart sounds. Laboratory tests revealed alanine aminotransferase 14,620 IU/L, bilirubin 10.8 mg/dL and serum lactate 13.9 mmol/L. The chest radiograph revealed diffuse interstitial infiltrate predominantly in the right perihilar region with an increased cardiothoracic index. An abdominal ultrasonography confirmed the hepatomegaly and enlargement of inferior vena cava, while the echocardiogram showed a large pericardial effusion with signs of cardiac tamponade. The patient was transferred to the intensive care unit (ICU) where he underwent a pericardiocentesis. A total of 640 ml of hemorrhagic fluid was drained, with significant clinical improvement after the procedure. *Mycobacterium tuberculosis* was isolated from the gastric lavage and pericardial fluid cultures. Ten days after admission and cardiac tamponade drainage the patient was recovered, the liver aminotransferases were close to the normal values and the patient presented a progressive clinical and laboratory improvement with the tuberculosis treatment. Tuberculosis cardiac tamponade usually does not have an acute clinical presentation and is a rare but life-threatening cause of severe hypoxic hepatitis, which may lead to mal-perfusion secondary to blood stasis in the liver. As soon as the cause of liver hypoxemia is removed there will be a rapid and impressive improvement in the liver damage and function markers.

KEY WORDS: cardiac tamponade; pericarditis, tuberculous; hepatitis.

Riassunto

Un uomo di 57 anni è stato ricoverato nel Dipartimento di Emergenza per un dolore epigastrico accentuato dal respiro insorto 6 giorni prima. La visita medica evidenziava un lieve ittero, un fegato dolente e toni cardiaci ovattati. I test di laboratorio rivelavano valori di alanina aminotransferasi pari a 14.620 IU/L, di bilirubina pari a 10,8 mg/dL e di lattato sierico pari a 13,9 mmol/L. La radiografia del torace metteva in evidenza diffusi infiltrati interstiziali soprattutto nella regione peri-ilare destra con un indice cardiotoracico aumentato. L'ecografia addominale confermava un'epatomegalia ed una dilatazione della vena cava inferiore, mentre l'esame ecocardiografico mostrava un importante versamento pericardico con segni di tamponamento cardiaco. Il paziente fu trasferito in unità di terapia intensiva dove venne sottoposto ad una pericardiocentesi. Fu drenato un totale di 640 ml di liquido emorragico con un significativo miglioramento del quadro clinico al termine della procedura. Il *Mycobacterium tuberculosis* fu isolato dale colture effettuate sul lavaggio gastrico ed il liquido pericardico. Dieci giorni dopo il ricovero ed il drenaggio del tamponamento pericardico il paziente si ristabilì, le aminostransferasi epatiche si normalizzarono ed il paziente presentò un miglioramento progressivo del quadro clinico e di laboratorio con il trattamento della tubercolosi. Il tamponamento da tubercolosi cardiaca di solito non ha una presentazione clinica acuta ed è una rara ma potenzialmente mortale causa di epatite ipossica severa che può portare a deficit perfusionale da stasi epatica. Quando la causa dell'ipossiemia del fegato viene rimossa, si assiste ad un rapido e notevole miglioramento del danno epatico e dei markers di funzionalità epatica.

TAKE-HOME MESSAGE

Hypoxic hepatitis may occur due to tuberculous cardiac tamponade and removing the cause of liver hypoxemia lead to an impressive and rapid improvement in the liver damage and function markers.

Competing interests - none declared.

Cite this article as - Bollela VR, Puga FG, Santana RC. Tuberculous cardiac tamponade presenting as severe hypoxic hepatitis. J Health Soc Sci. 2016;1(3):279-286

DOI 10.19204/2016/tbrc28

Recived: 10/08/2016 Accepted: 15/10/2016 Published: 15/11/2016

INTRODUCTION

Acute ischemic liver injury, termed hypoxic hepatitis (HH), is the most frequent cause of markedly raised aminotransferase levels in critically ill patients [1]. The characteristic histopathological finding in this syndrome is centrilobular hepatocytes necrosis, due to reduced oxygen supply to the liver cells. Several mechanisms are involved in the pathogenesis of HH: hepatic hypoperfusion (circulatory shock), systemic hypoxia (respiratory failure), low oxygen extraction by the liver (sepsis) and passive venous congestion (right ventricular insufficiency). Heart failure and septic shock are among the most common conditions related to HH [2]. In the typical scenario, HH develops in a patient with known chronic heart failure who seeks medical attention due to an acute condition that predisposes to hypoxemia and/or hepatic hypoperfusion. Among the cardiologic conditions, pericardial effusion has been related to a small proportion of HH cases, where passive hepatic congestion plays a central pathophysiological role [3, 4]. This report presents a patient without previous cardiac disease who arrived in the Emergency Department with HH due to tuberculous pericardial effusion.

CASE REPORT

A man, aged 57 years, was referred to the Emergency Department complaining of epigastric respiratory dependent pain that had begun six days previously. The physical examination revealed increased abdominal girth and generalized mild jaundice. The liver was painful and enlarged, being palpable 10 centimeters beyond the right rib cage. The respiratory rate was 30 breaths per minute and the oxygen saturation was 90%. With cardiac auscultation, muffled heart sounds were detected, presenting a cardiac heart rate of 80 beats per minute. The blood pressure in the supine position was 90/60 mm Hg. Laboratory tests performed upon admission revealed aspartate aminotransferase (AST) 10,030 IU/L, alanine aminotransferase (ALT) 14,620 IU/L, gamma-glutamyl transferase (GGT) 248 U/L, total bilirubin (BRB) 10.8 mg/dL, international normalized ratio (INR) 2.78, creatinine 2.7 mg/dL, lactate dehydrogenase (LDH) 5.735 U/L, and serum lactate 13.9 mmol/L. The chest radiograph showed diffuse interstitial infiltrate predominantly in the right perihilar region and an increased cardiothoracic index. An abdominal ultrasonography confirmed the hepatomegaly, while the echocardiogram showed a large pericardial effusion with signs of cardiac tamponade (Fig. 1). The patient was transferred to the Intensive Care Unit (ICU) where he underwent a pericardiocentesis. A total of 640 mL of hemorrhagic fluid was drained, with significant clinical improvement after the procedure.

A chest computed tomography showed micronodules bilaterally associated with bronchiectasis and ground-glass opacification more marked in the right lung. Bilateral pleural effusion, pericardial effusion, and perihilar and paraaortic lymph nodes were also evidenced (Fig. 1). As the patient was unable to produce sputum specimens, a gastric lavage specimen was obtained in order to carry out an acid-fast bacilli search, which produced a positive result. At that time the patient was being treated with ceftriaxone, for bacterial pneumonia, and an anti-tuberculosis (TB) treatment was added, with levofloxacin, streptomycin, and ethambutol due to hepatitis. Subsequently, *Mycobacterium tuberculosis* was isolated from the gastric lavage and pericardial fluid cultures, obtained during the pericardiocentesis. Serologic tests for HIV, Cytomegalovirus, viral hepatitis (A, B, and C), Epstein-Barr virus, Dengue virus, leptospirosis, and Hantaviruses were all negative.

The patient presented a progressive clinical and laboratory improvement (Fig. 2) and was discharged from the ICU to the ward. As soon as the aminotransferases levels returned to values close to normal, the TB treatment scheme was modified to the standard daily rifampicin/isoniazid/pyrazinamide/ethambutol. A chest radiograph performed 14 days after discharge revealed a significant improvement in pulmonary opacifications and a return to normal cardiac dimensions.

DISCUSSION

Hypoxic hepatitis was formerly termed 'ischemic hepatitis' as it was believed that insufficient hepatic perfusion was the sole factor responsible for centrilobular [5]. With the evolution of knowledge on the subject, Henrion et al., in a pioneering study considering hemodynamic aspects in liver cell necrosis, suggested the term 'Hypoxic hepatitis' as more embracing, since other factors besides liver hypoperfusion were involved in centrilobular hepatocytes necrosis [6]. Currently, it is well known that HH also occurs associated with other conditions, such as severe hypoxemia, reduced capacity of oxygen extraction by the liver, and passive venous congestion [2].

The incidence of HH is low in general wards, where it is estimated at approximately 1 per 1,000 admissions [7]. However, its incidence increases in intensive care units, where rates of up to 21.9% have been reported in cardiac care units [8]. As can be seen, HH typically develops in critically ill patients, especially in those suffering from chronic congestive heart failure. In addition, it can occur in patients without previous cardiac or respiratory diseases, such as those with acute conditions like myocardial infarction or pericardial effusion. The latter condition is reported as a predisposing condition in only 1% to 8% of HH cases [3, 4, 9].

The typical aminotransferase pattern during the course of HH shows a dramatic rise in both ALT and AST, with higher AST levels, within 12–24 hours after the initiation of the event. Usually, the aminotransferase levels fall more than 50% within three days after the stabilization and elimination of the underlying HH-causing condition [2]. The diagnostic criteria are: a) clinical setting of acute cardiac, circulatory, or respiratory failure; b) dramatic but transient increase in serum aminotransferase activity, reaching at least 20 times the upper normal limit; and c) exclusion of other putative causes of liver cell necrosis, such as viral or drug-induced hepatitis [1].

Jaundice is an uncommon finding. The case reported here shows an unusual presentation with ALT level higher than AST, overt jaundice, and high bilirubin levels upon admission, which is an unsual finding in HH. This laboratory and clinical pattern could be explained by late hospital admission. Therefore, the patient was first assessed in a late stage of installed HH when, due to its longer high-life, ALT levels predominated over AST. The enlarged liver seen in this patient strengthens the hypothesis that the patient was first seen at a later stage of the underlying disease. Another relevant aspect, in this case, is that liver disease signs predominated over those of pericardial effusion upon admission and guided the diagnostic workup in the first moment. Typically, the clinical findings of HH are concealed by those of the underlying predisposing condition. Unlike the typical HH setting, where the predisposing condition is related to an acute myocardial event, septic shock or respiratory failure, in cardiac tamponade, the presence of non-specific symptoms and the compensatory response may lead to a delay in seeking a medical care and diagnosis [10].

The evolution observed in this reported case followed what is usually described in HH, where the main therapy is to treat or eliminate underlying conditions. An emergency pericardiocentesis was performed, and circulatory function immediately improved. Liver and renal function normalized over the following 10 days. Bilirubin reached the serum peak level ten days after the initiation of symptoms and went back to normal levels one month after the pericardiocentesis. The ischaemic liver injury in this case probably occurred due to a combination of factors, such as hepatic congestion caused by cardiac tamponade and decreased hepatic arterial perfusion, secondary to hypotension.

Hypoxic hepatitis usually develops in the intensive care unit context and the prognosis depends on the underlying diseases. When it's due to an acute and reversible condition, as the cardiac tamponade, it presents with an unusual clinical and laboratory pattern. In these situations, the tamponade drainage reverses the liver damage in few days, with a good prognosis.

Figure 1. Day 0: Thorax X ray showing lung opacification and increased cardiothoracic index (A); Day 2: Computed tomography showing nodules, ground-glass opacification, and bilateral pleural effusion (B); Day 0: Liver ultrasonography showing huge enlargement of inferior vena cava (C); Day 5: Liver ultrasonography showing a normal exam five days after pericardial drainage (D); Day 0: Echocardiographic image with a huge pericardial effusion (E); Day 0: Echocardiographic image just after pericardial drainage showing total resolution of pericardial effusion (F).

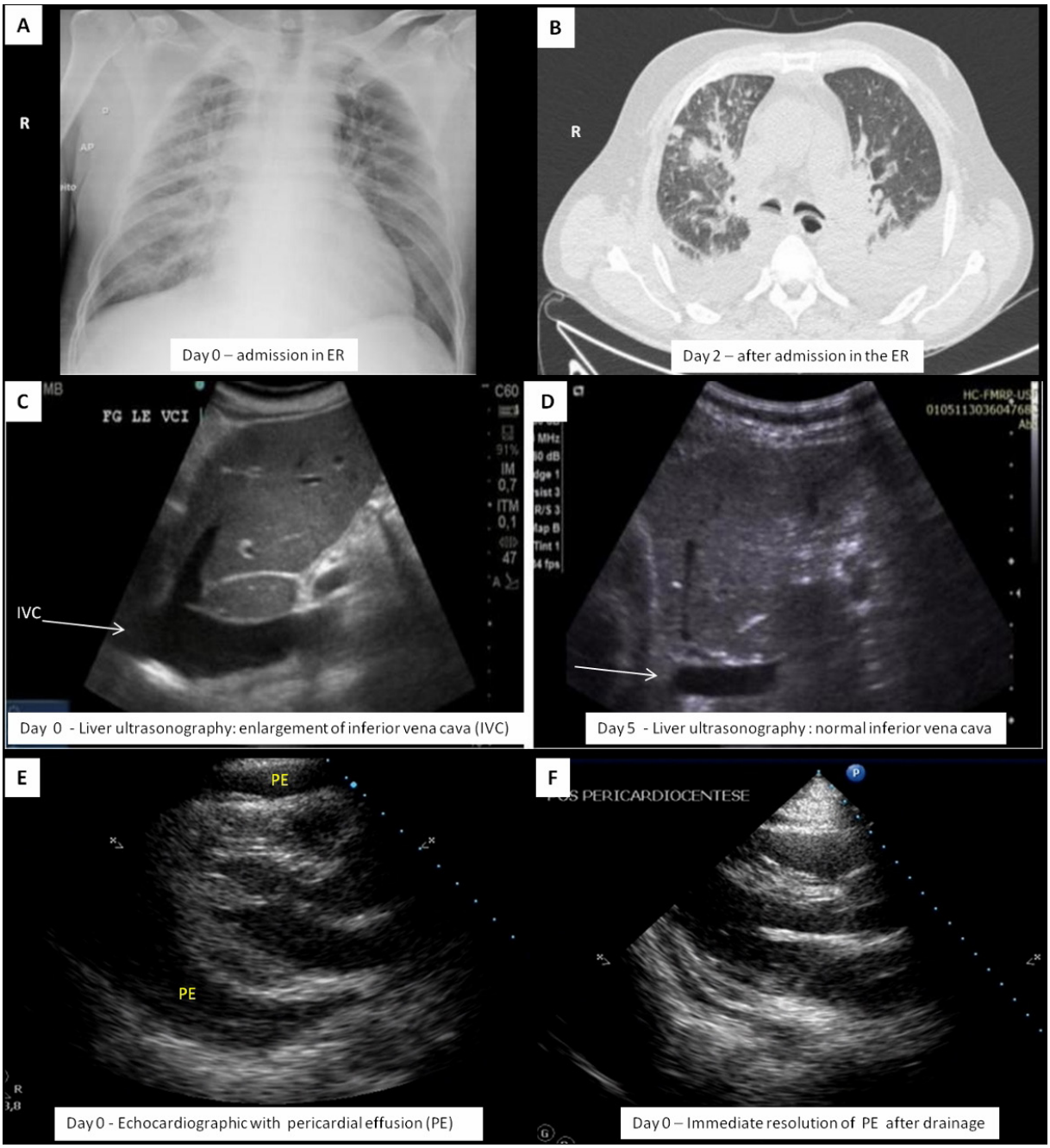
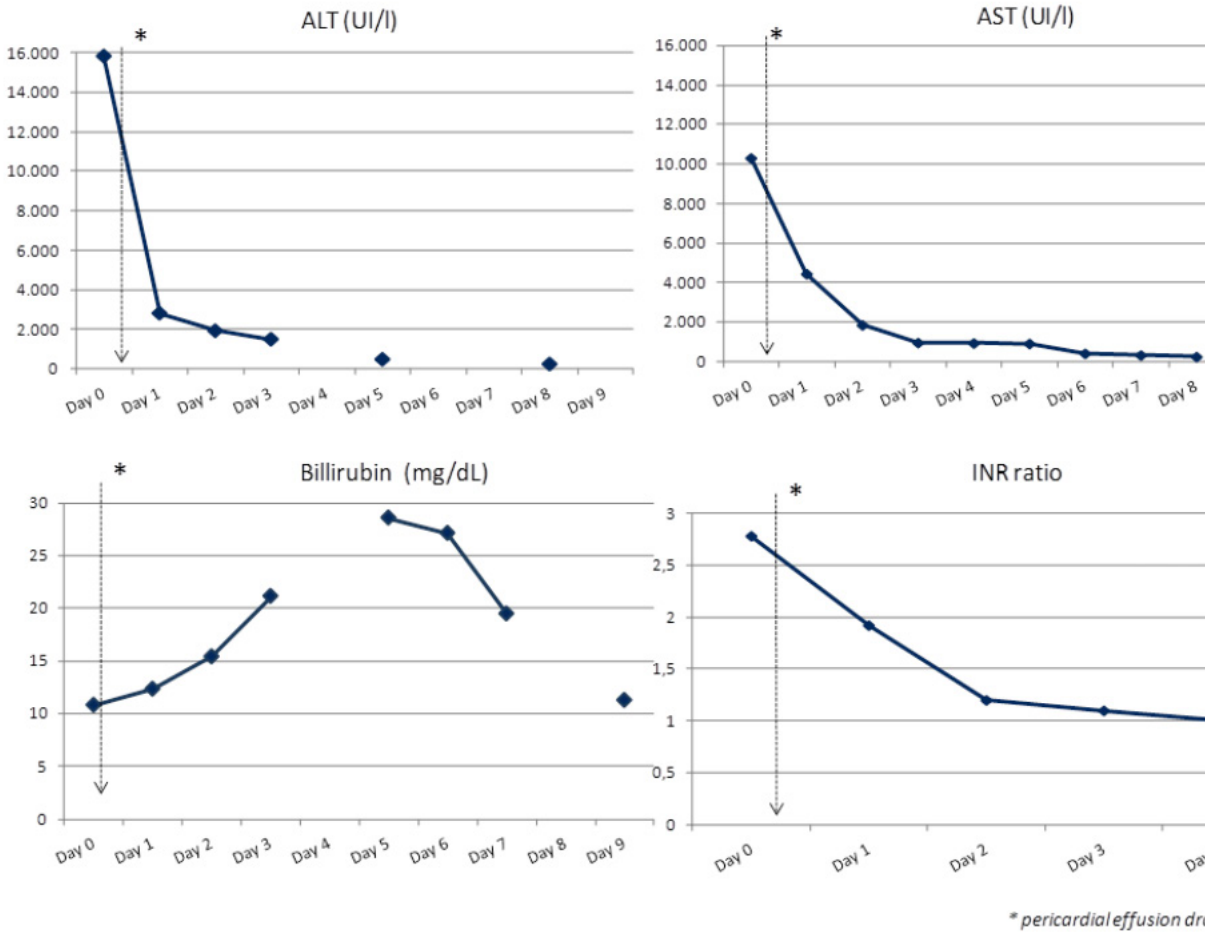


Figure 2. Changes in the liver enzymes levels and international normalized ratio before and after cardiac tamponade drainage. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin (BRB) and international normalized ratio (INR) levels before and after cardiac tamponade drainage.



References

1. Henrion J. Hypoxic hepatitis. Liver Int. 2012 Aug;32(7):1039-52. PubMed PMID: 22098491.
2. Fuhrmann V, Jager B, Zubkova A, Drolz A. Hypoxic hepatitis-epidemiology, pathophysiology and clinical management. Wien Klin Wochenschr. 2010 Mar;122(5-6):129-39. PubMed PMID: 20361374.
3. Birrer R, Takuda Y, Takara T. Hypoxic hepatopathy: pathophysiology and prognosis. Intern Med. 2007;46(14):1063-70. PubMed PMID: 17634701.
4. Henrion J, Schapira M, Luwaert R, Colin L, Delannoy A, Heller FR. Hypoxic hepatitis: clinical and hemodynamic study in 142 consecutive cases. Medicine. 2003 Nov;82(6):392-406. PubMed PMID: 14663289.
5. Bynum TE, Boitnott JK, Maddrey WC. Ischemic hepatitis. Dig Dis Sci. 1979 Feb;24(2):129-35. PubMed PMID: 428301.
6. Henrion J, Luwaert R, Colin L, Schmitz A, Schapira M, Heller FR. [Hypoxic hepatitis. Prospective, clinical and hemodynamic study of 45 cases]. Gastroenterol Clin Biol. 1990;14(11):836-41. PubMed PMID: 2276565.
7. Johnson RD, O'Connor ML, Kerr RM. Extreme serum elevations of aspartate aminotransferase. Am J Gastroenterol. 1995 Aug;90(8):1244-5. PubMed PMID: 7639223.
8. Henrion J, Descamps O, Luwaert R, Schapira M, Parfonry A, Heller F. Hypoxic hepatitis in patients with cardiac failure: incidence in a coronary care unit and measurement of hepatic blood flow. J Hepatol. 1994 Nov;21(5):696-703. PubMed PMID: 7890882.
9. Fuhrmann V, Kneidinger N, Herkner H, Heinz G, Nikfardjam M, Bojic A, et al. Hypoxic hepatitis: underlying conditions and risk factors for mortality in critically ill patients. Intensive Care Med. 2009 Aug;35(8):1397-405. PubMed PMID: 19506833.
10. Sagrista-Sauleda J, Merce AS, Soler-Soler J. Diagnosis and management of pericardial effusion. World J Cardiol. 2011 May 26;3(5):135-43. PubMed PMID: 21666814. Pubmed Central PMCID: 3110902.