

## Hypoxic hepatitis in an extensive aortic dissection: case report

Hepatitis hipóxica en una disección aórtica extensa: reporte de caso

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### ABSTRACT

Hypoxic hepatitis is an uncommon cause of hepatic damage characterized by a centrilobular necrosis. Its pathophysiology remains unclear. Aortic dissection is a rare but frequently catastrophic event. It is caused by an aortic intimal tear with propagation of a false channel in the media. Depending on the site and extension, it can cause hypoperfusion of any organ leading to cellular ischemia and necrosis. We are presenting a case of hypoxic hepatitis in a patient with an extensive aortic dissection who present to the emergency department.

**Keywords:** Hepatitis; Aortic dissection; Necrosis (source: MeSH NLM).

### RESUMEN

La hepatitis hipóxica es una causa poco frecuente de daño hepático caracterizada por una necrosis centrilobular. Su fisiopatología sigue siendo poco clara. La disección aórtica es un evento raro pero con frecuencia catastrófico. Dependiendo del sitio y la extensión, puede causar hipoperfusión de cualquier órgano lo que conduce a una isquemia celular y necrosis. Nosotros presentamos un caso de hepatitis hipóxica en un paciente con disección aórtica extensa que se presenta al servicio de emergencia.

**Palabras clave:** Hepatitis; Disección aórtica; Necrosis (fuente: DeCS BIREME).

### INTRODUCTION

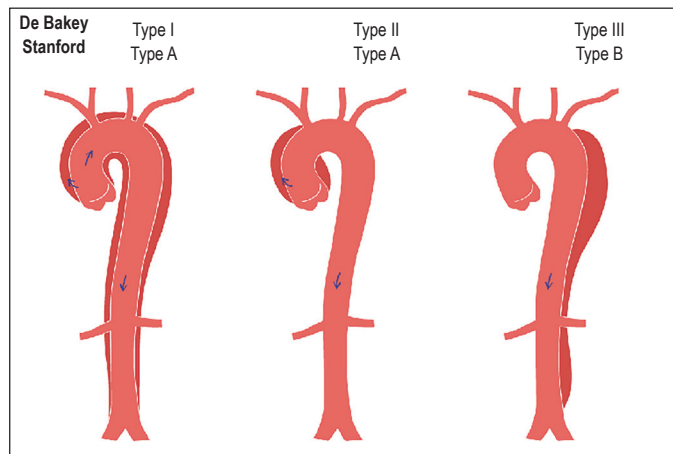
Hypoxic hepatitis (HH), also known as ischemic hepatitis or “shock liver”, is a cause of severe acute liver injury in the context of impaired oxygen delivery<sup>(1)</sup>. Studies report an incidence of 1% among all intensive care unit admissions<sup>(2,3)</sup>, 3% in cardiac care unit admissions<sup>(4)</sup>. Moreover, 50% of patients with transient elevation of transaminases in the inpatient setting was caused by this disease<sup>(5)</sup>. Mortality during hospitalization is 45%<sup>(2)</sup>.

The pathophysiology of HH is not well understood. There are different pathways to explain the HH: a decreased oxygen concentration, characterized by hypoxemia (severe respiratory failure and anemia); a decreased oxygen delivery (low cardiac index, and low hepatic blood flow); a decreased oxygen availability by peripheral vasoconstriction (septic shock); and increased oxygen consumption by hyperdynamic state hiperthermia. Only 51% of patients with HH had severe hypotension<sup>(2,3)</sup>. Given the multifactorial nature of this condition the best term to name it is hypoxic hepatitis<sup>(2)</sup>.

Aortic dissection (AD) is defined as a “disruption of the medial layer provoked by intramural bleeding resulting in a separation of the aortic wall layers”<sup>(6)</sup>. The incidence is 6 per 100 000 persons/year, with an average age of presentation of 72 years and a male predominance<sup>(7)</sup>. The Stanford classification, classifies the dissections into: type A (involves the ascending aorta) representing 60% of AD and type B (not involving the ascending aorta)<sup>(6)</sup> (Figure 1). The risk factors for AD are hypertension, preexistent aortic disease, family history of aortic diseases, prior cardiac surgery, smoking, thoracic trauma, and use of intravenous drugs<sup>(6)</sup>. AD is a rare cause of HH, and there are only few reports in the literature<sup>(8,9)</sup>.

### CLINICAL CASE

A 58-years-old Hispanic man presented to the emergency department with 1 month of dyspnea that gradually worsened during the last three days which was associated with a decreased urinary flow. He also presented an episode of non-specific abdominal pain that progressively worsened. He recalled an episode of chest pain associated with palpitations and profuse



**Figure 1.** Stanford classification of aortic dissection.

sweating a month prior to admission while he was doing weightlifting. He denied any medical condition and drugs use. No family history of connective tissue disorders.

On presentation, he was afebrile, blood pressure recordings of 130/80 mmHg in both arms, heart rate of 68 bpm, respiratory rate of 36 rpm and a blood oxygen saturation of 90%. The physical examination was remarkable for bilaterally decreased breath sounds and crackles. His abdomen was tender on the right upper quadrant. A liver span of 16 cm was found. His pulses were symmetrically decreased. The remainder of the physical examination was unremarkable. Table 1 shows the evolution of the laboratory values.

Chest-X-ray revealed a widening in the aortic knob with alveolar edema. The finding on ECG were sinus rhythm with t wave inversion in leads I, AVL. The transthoracic echocardiogram showed reduced ejection fraction 37%, a mild aortic and mitral regurgitation, and an intimal flap. The CT scan showed a hypodense lineal image dividing the aortic in two lumens that extend from the thoracic aorta to the iliac bifurcation (Figure 2). Given the high risk of surgical mortality, the patient

and family refused heroic measures, and he died on hospitalization day 17.

**DISCUSSION**

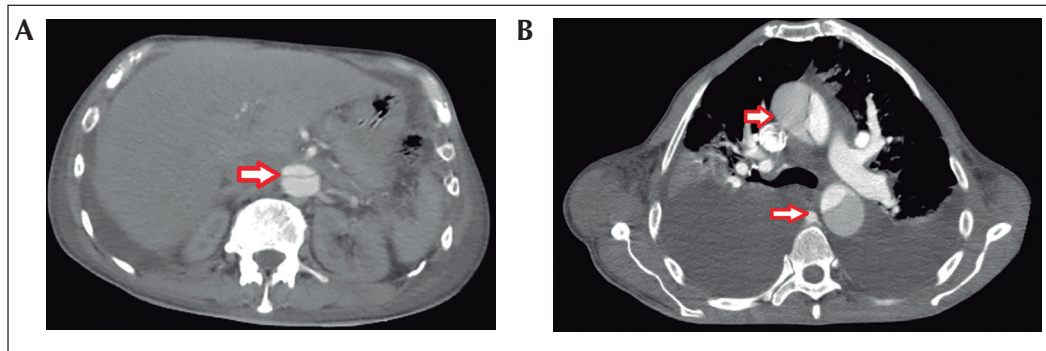
Although the histologic presence of centrolobular necrosis is an essential element in HH, it is usually identified by a significant transient elevation of serum aminotransferase levels (>10 times upper limit value)<sup>(5)</sup>, in a clinical context of a heart, pulmonary or vascular failure and in the absence of other potential causes of liver injury such as viral or drug induced hepatitis<sup>(4)</sup>.

Our patient meets the diagnostic criteria for HH, with a transient marked elevation of serum aminotransferases levels (AST 710 ui/l and ALT 1210 ui/l), no history of alcohol consumption, hepatotoxic drugs or herbs, and negative tests for viral hepatitis, in the context of an AD. Liver biopsy is not required nor is advised if the patient fulfills these three criteria<sup>(3,10)</sup>.

During the evaluation of his laboratory results, a transient elevation of aminotransferases, LDH and creatinine were noticed. These changes are described

**Table 1.** Evolution of the laboratory values.

	Day 1	Day 5	Day 6	Day 9	Day 16
Hemoglobin (mg/dl)	12,4				
Creatinine (mg/dl)	1,6	3,7	4,4	2,1	1,1
AST(IU/L)		721	580	268	82
ALT (IU/L)		1270	1000	741	241
LDH (U/L)	7207	2179		1008	1065
INR	1,76		1,69	1,45	1,53
Total Bilirubin (mg/dl)	1,8	1,9		1,2	



**Figure 2.** The CT scan showed: a) Linear image in abdominal aorta, b) Hypodense lines in ascending and descending thoracic aorta.

in patients with HH<sup>(10)</sup>. The pathophysiology of HH in our patient is related to a state of hypoperfusion secondary to AD, demonstrated by the liver, and kidney compromise<sup>(6)</sup>.

The most common clinical presentation in AD is knife-like severe acute chest pain (present in 80% of patients with AD) radiating to the dorsal region (40%). Other presentations are aortic regurgitation (40%), cardiac tamponade (20%), myocardial ischemia (15%), heart failure (10%), pleural effusion (15%), syncope (15%), mesenteric ischemia (5%), acute renal failure (20%)<sup>(6)</sup>.

The diagnosis of AD is based on imaging studies. The sensitivity and specificity of transthoracic echocardiography are 80% and 96% respectively. Transesophageal echocardiography has a sensitivity of 99% and specificity 89%. The diagnosis is based on the detection of intimal flaps. CT scanning is the most widely used technique due to its immediate availability in emergency room, with a sensitivity of 95%. The sensitivity and specificity of MRI is 98%<sup>(6)</sup>.

The diagnosis of AD in our patient was difficult, due to the atypical presentation mainly characterized by hepatic and renal involvement. However, when transthoracic echocardiography was performed, it showed an intimal flap and the CT scan confirmed the diagnosis. The treatment approach depends on the type of AD. For type A, surgery is required due to a high mortality 90% without treatment<sup>(6)</sup>. The 30-day mortality rate was 47% in patients with AD type A despite the surgery<sup>(7)</sup>.

In conclusion, HH is a rare condition, which is related to patients with significant comorbidities, with a complex and multifactorial pathophysiology. In the setting of a patient with sudden onset of HH, and renal failure, the physician should keep a high index

suspicion for AD. Diagnostic and therapeutic delays will negatively impact on outcome and survival of these patients.

**Conflicts of interest:** None

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