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Atrial Tamponade Causing Acute Ischemic Hepatic Injury after Cardiac Surgery

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Summary: A patient developed late cardiac tamponade after aortic valve replacement and coronary artery bypass grafting. Nausea and dramatic elevations of serum aminotransferases were the initial clinical manifestations of cardiac tamponade. Severe acute ischemic hepatic injury secondary to isolated compression of both atrial cavities by two loculated thrombi was diagnosed.

Key words: cardiac surgery, tamponade, acute hepatitis

Introduction

Late cardiac tamponade is a rare but potentially serious complication following open heart surgery. The tamponade is often insidious in onset and may not be recognized until the circulation is compromised. Although liver damage can be associated with acute and/or chronic heart failure,[1] acute circulatory failure of the liver following cardiac surgery has seldom been reported. According to Rex's literature review,[2] here we present the third reported case of a patient with severe acute hepatic injury as initial manifestation of a postoperative cardiac tamponade.

Case Report

A 77-year-old man with long-standing severe hypertension underwent double-vessel coronary artery bypass grafting and aortic valve replacement with a 21 mm Carpentier-Edwards valve for aortic stenosis. Three weeks earlier, he had undergone endarterectomy of the right common carotid artery without complications. He had no previous history of blood transfusion, use of hepatotoxic drugs, ethanol ingestion, or intravenous drug abuse. Seven days before surgery, aspirin was discontinued and his preoperative liver tests were normal. Isoflurane-fentanyl anesthesia was used without complications. The first 7 days after surgery were uneventful, with normal blood drainage by the chest tubes and normal coagulation tests. Routine transthoracic echocardiography performed on Day 5 showed left and right ventricular hypertrophy and normal function of the prosthesis. On postoperative Day 8, he developed sudden nausea, vomiting, dizziness, and diaphoresis. Two days later, moderate jugular distention (15 mmHg) with systolic blood pressure of 100 mmHg and oliguria were present. Abdominal examination was nontender, but painful hepatomegaly and disminution of the bowel sounds were detected. Laboratory evaluation included aspartate aminotransferase (AST), 1,753 IU/l (normal < 40 IU/l); alanine aminotransferase (ALT), 1,528 IU/l (normal < 40 IU/l); alkaline phosphatase (ALP), 80 IU/l (normal < 50 IU/l); lactic dehydrogenase (LDH), 3,228 IU/l (normal < 225 IU/l); creatine kinase, 28 IU/l; total bilirubin, 0.7 mg/100 ml; hemoglobin, 10.1 g/100 ml; amylase, 132 IU/l; lipase, 68 IU/l; white blood count, 20,830/mm3; prothrombin time, 24 s (normal < 13 s); blood urea nitrogen (BUN), 32 mg/100 ml; and creatinine, 2.0 mg/100 ml.

Twelve hours later, the patient became hypotensive (systolic blood pressure 70 mmHg), tachycardiac at a heart rate of 120 beats/min, with moderate jugular distention (14 mmHg) and without bowel sounds. A chest roentgenogram without interstitial lung edema and absence of significant hypoxemia discarded a predominant compromise of the pulmonary venous return.

Ischemic acute hepatic injury and mesenteric ischemia due to aortic abdominal dissection were suspected, but computed tomographic angiography disproved it. In the same study, a small pericardial effusion surrounding the right ventricle anteriorly with selective bilateral atrial compression by two masses was observed (Fig. 1A). Transesophageal echocardiogram (TEE) showed normal right and left ventricular systolic function. The right and left atrial cavities were severely compressed by two separate thrombi measuring 3.5 × 4.0 and 3.0 × 4.5 cm, respectively (Fig. 1B). Doppler examination showed that the mean gradient across the biological prosthesis was 7 mmHg.

The patient was immediately transferred to the operating room for exploration of the pericardium. A loculated hematoma compressing the right atrium laterally and another one

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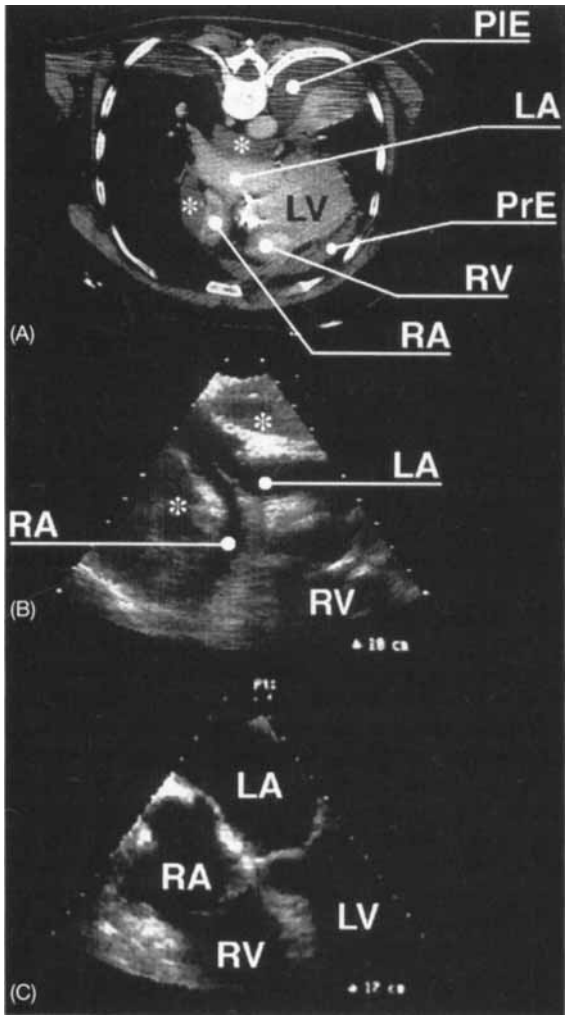

FIG. 1 (A) Computed tomographic angiography of the chest. A small pericardial effusion surrounding the right ventricle anteriorly is seen with selective bilateral atrial compression by two masses. (B) Transesophageal echocardiogram, four-chamber view, showing two large pericardial clots (*) compressing both atrial cavities. (C) Reexpansion of the right and left atria is noted after evacuation of the clots. LA = left atrium, RA = right atrium, LV = left ventricle, RV = right ventricle, PlE = pleural effusion, PrE = pericardial effusion.

compressing the left atrium posteriorly were found. The right atrial thrombus was severely compromising drainage of the inferior vena cava. After evacuation of the clots, blood pressure returned to normal and right atrial pressure decreased from 16 to 2 mmHg. No source of bleeding was identified. Repeat TEE after 2 h of evacuation of clots revealed reexpansion of both atrial cavities (Fig.1C).

Twelve hours after removal of the hematoma, laboratory data included AST, 5,297 IU/l; ALT, 3,434 IU/l; ALP, 99 IU/l; LDH, 1,166 IU/l; prothrombin time, 26 s (after 500 cc of fresh plasma); total bilirubin, 1.1 mg/100 ml; BUN, 74 mg/100 ml; and creatinine 4.8 mg/100 ml. The patient made an excellent postoperative recovery and transaminases and kidney function rapidly returned to normal. Nine days after reexploration of the pericardium, AST was 47 IU/l, and ALT was 65 IU/l. One month after first surgery, the patient was discharged in good condition. Hepatitis A, B, and C serologic tests drawn on the day of tamponade were all negative.

Discussion

The incidence of cardiac tamponade as a delayed complication of open heart surgery is reported to be 0.3 to 2.6%.[3] This potentially lethal complication may be misdiagnosed because of frequent atypical presentations or selective chamber compression. It occurs more habitually in patients receiving systemic anticoagulation. In our case, although with moderate increase of the venous central pressure, the diagnosis of cardiac tamponade was masked by the abdominal clinical findings. Abnormal results of liver function tests are not infrequent after severe postoperative low cardiac output, but may be clinically inapparent, yet resulting in aminotransferase elevations. In this way, significant changes of the hepatic tests as the initial manifestations of tamponade have been reported infrequently.[2] Abdominal complications in these situations have been well recognized entities that carry significant mortality and morbidity. Intestinal ischemia has the highest mortality, but peptic ulcer disease is the most frequently reported.[4]

In our case, clinical and biochemical evidence of liver damage was associated with late selective bilateral atrial tamponade after aortic valve replacement and coronary artery bypass grafting. Explanations for aminotransferase elevations other than liver injury associated with tamponade are unlikely, as serologic tests for viral hepatitis were negative and no hepatotoxic drugs were used. On the other hand the enzymatic movement of the present case was typical of ischemic hepatic injury[5] (Fig. 2). In this case, the classical meteoric elevation and resolution of the AST and ALT with minor changes in the bilirubin and ALP were accompanied by a significant rise in

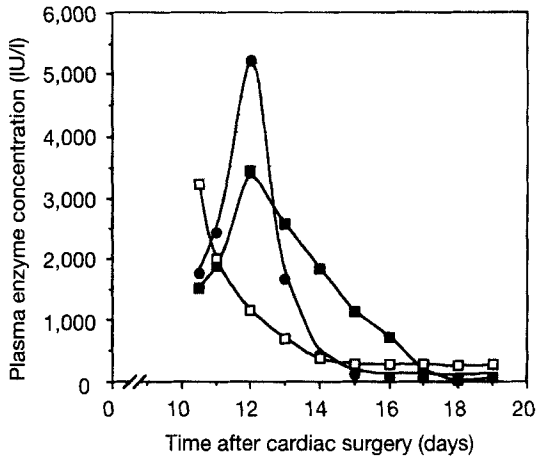

FIG. 2 The course of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactic dehydrogenase (LDH) in the patient reported. ● =AST, ■ =ALT, □ =LDH.

LDH and a rise in BUN and creatinine. Reversible renal failure reflects the renal ischemia that occurs concurrent with the hepatic ischemia. The early severe elevation of LDH and the massive decline in transaminases reaching control values 5 to 8 days after evacuation of the hematoma appear to be equally useful indicators for excluding viral hepatitis. In viral hepatitis, LDH elevation of a lesser severity, a more gradual fall in transaminases over weeks, and renal insufficiency occur later in the sequence of the disease process. Ischemic hepatic injury is characterized morphologically by centrilobular necrosis with little or no inflammatory response. It results from decreased hepatic blood flow, resulting in reduction of oxygen supply to the liver and increased hepatic venous pressure that leads to central venous and sinusoidal congestion.[5]

Normally, the prothrombin time is rarely prolonged by more than 3 s with the ischemic damage of the liver.[1] In our case, the patient was transfused with fresh plasma before surgical reexploration because of severe prothrombin time prolongation. We believe that congestive hepatomegaly secondary to right cardiac tamponade may be the main factor for the prolongation of prothrombin time. If severe hypotension is one of the requirements for the diagnosis of the ischemic hepatic injury,[1] our patient was actually hypotensive (blood systolic pressure of 70 mmHg) for only 6 h before confirmation of the elevation of aminotransferases. However, systolic blood pressure of 100 mmHg two days earlier might be considered to be too low for a postoperative patient with long-standing severe hypertension. In our case, we believe that right atrial tamponade with obstruction of the systemic venous drainage was the predominant cause for the low cardiac output and finally the main cause of liver injury. However, we also believe that hypotension due to compression of the left atrium by the thrombus, shown by TEE, could have contributed to a worsening of the ischemic damage to the liver.[1]

Conclusion

We have described the unusual presentation of a late postoperative cardiac tamponade producing an ischemic hepatic injury in a patient not receiving anticoagulation therapy. Severe rise in serum aminotransferases was the diagnostic key. Urgent cardiac evaluation with transesophageal echocardiography, and reexploration of the pericardium were determinants of an excellent postoperative recovery.

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