

Letter to the Editor

Delayed cardiac tamponade in a patient with previous minor blunt chest trauma

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Abstract

Hemopericardium with cardiac tamponade after non-penetrating chest trauma is a very rare but life-threatening condition. If this complication develops after an interval of several weeks following the non-penetrating chest trauma, the causal relation with the traumatic event is less evident, which may delay proper diagnosis and adequate treatment. We describe diagnosing and therapeutic management of a patient in shock who suffered from cardiac tamponade four weeks after a minor blunt chest trauma.

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1. Introduction

Hemopericardium with cardiac tamponade is a life-threatening situation that can occur in patients with myocardial infarction, aortic dissection, and malignant or infectious pericardial diseases [1]. In addition, both penetrating and non-penetrating chest trauma can be associated with hemorrhagic cardiac tamponade that generally occurs within several hours to a few days [2]. However, after non-penetrating chest trauma, cardiac tamponade is very rare and occurs in less than 1/1000 [3]. If the tamponade develops after an interval of several weeks following a non-penetrating chest trauma, the causal relation with the traumatic event is less evident, which may delay proper diagnosis and adequate treatment. We describe diagnosing and therapeutic management of a patient in shock who suf-

fered from *cardiac tamponade four weeks after a minor blunt chest trauma*.

2. Case report

A 70-year old male patient with sudden onset of vigorous chest pain and dyspnoea was admitted. The patient had no history of cardiac disease and used no medication. Four weeks before admission the patient had an accident during which a heavy door was pushed against the left side of his chest which resulted in strong chest pain for several days. The patient did not attend his general practitioner but treated himself with analgesic medication until the pain vanished.

On physical examination, the patient had cold cyanotic extremities and low blood pressure (70/40 mm Hg). The pulse was fast (110 bpm) and showed pulsus paradoxus while central venous pressure was elevated. Pulsation was intact in all extremities, the chest showed no local bruising or tenderness, and heart sounds were hardly audible while there were a few crepitations at the bases of both lungs. The electrocardiogram (Fig. 1A) showed sinus tachycardia with minimal elevation of the ST-segment in leads I and AVL,

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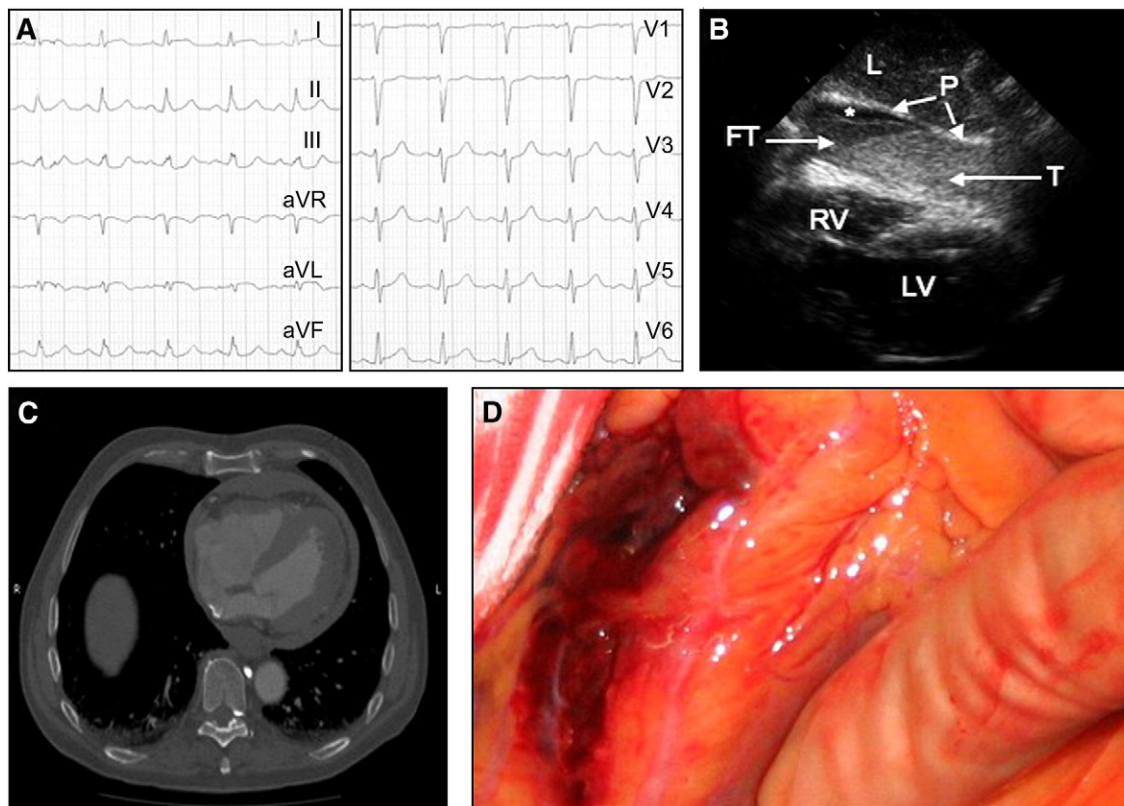


Fig. 1. Electrocardiography, echocardiography, and computed tomography. A: Electrocardiogram on admission showed sinus tachycardia and mild ST-segment elevation in the lateral leads. B: Transthoracic echocardiography in subcostal view demonstrated a hemopericardium with a large layered thrombus (T = thrombus; FT = fresh thrombus) and a small amount of fluid (*) between pericardium (P) and both right and left ventricle (RV and LV, respectively). C: Computed tomography revealed a massive hemopericardium. D: Surgical situs. A superficial hemorrhagic myocardial defect in the lateral wall of the left ventricle was found.

suggesting transmural ischemia of the lateral left ventricular wall.

Emergency echocardiography (Fig. 1B) showed compression of the right ventricle as a result of a huge hemopericardium, consisting of several layers of thrombus of different echo density (i.e., thrombus of different age) and a small amount of pericardial fluid. Computed tomography (Fig. 1C) showed the hemopericardium but no aortic dissection and no evident fracture of the ribs. The troponin-I level was slightly elevated (0.98 g/l) while the other cardiac markers were within normal range.

Despite of rapid administration of colloid fluids, blood pressure gradually declined. Considering the differential diagnoses of hemorrhagic cardiac tamponade, we had suspicion of a rupture of the left ventricular wall and intended to perform emergency thoracotomy. However, due to an ongoing cardiac surgical emergency procedure, thoracotomy could not be performed immediately. Because of further hemodynamic deterioration, pericardial drainage was performed which drew 200 ml of blood. This resulted in a transient increase of the mean blood pressure by 15 mmHg.

In order to gain further information while waiting for surgery, we performed coronary angiography (Fig. 2A and B) which revealed multiple significant coronary stenoses in all three coronary arteries. Left ventricular angiography (Fig. 2C

and D) showed a good systolic left ventricular function but no signs of left ventricular wall rupture.

Surgery was performed with a delay of 1.5 h. More than 1 l of blood and thrombus was removed from the pericardial sac, which improved hemodynamic parameters immediately. There was only a small superficial hemorrhagic myocardial defect (Fig. 1D) in the lateral left ventricular wall, which was stitched and glued with a patch. In addition, coronary bypass grafting was performed on all three coronary arteries with saphenous vein grafts. Extensive exploration of the myocardium at the site of the hemorrhagic defect clearly excluded the presence of left ventricular rupture or transmural myocardial necrosis. In addition, based on shape and consistency of the defect, we can exclude that the hemorrhage may potentially be the result of an arterial bleeding.

The patient fully recovered without complication and was discharged after 9 days. Two weeks later, echocardiography showed a normal cardiac function.

3. Discussion

Blunt chest trauma may lead to various kinds of cardiac damage ranging from minor contusion to cardiac tamponade. However, cardiac tamponade after *minor* blunt chest trauma

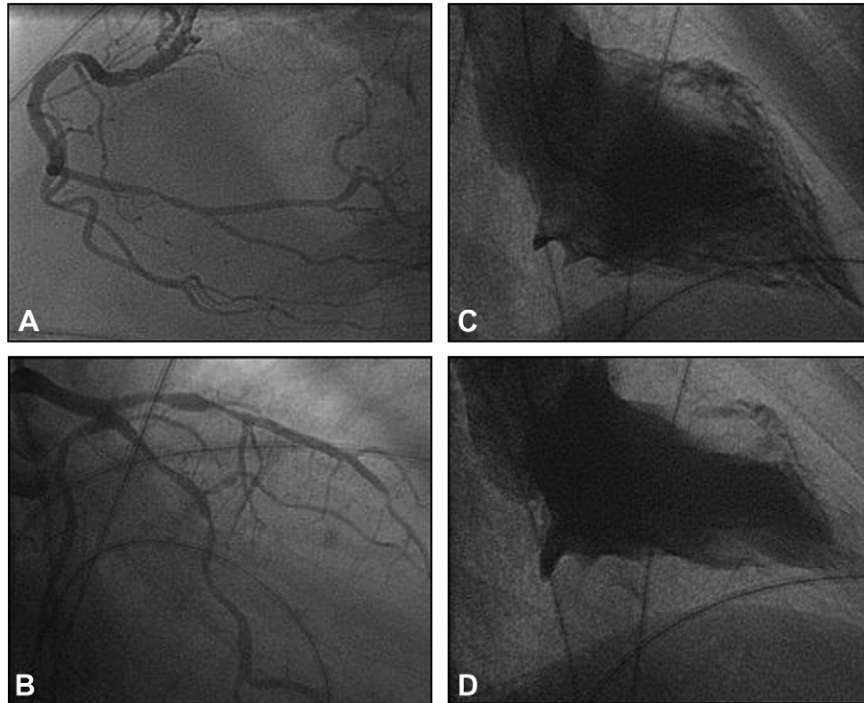


Fig. 2. Coronary and left ventricle angiography. A,B: Significant stenoses were seen in the right (A) and left coronary arteries (B). C, D: Left ventricular angiography revealed good systolic function and no evidence of ventricular rupture.

is a rare event [4]. Traumatic hemopericardium may develop secondary to cardiac rupture, vascular damage, or diffuse myocardial hemorrhage. In the present case, echocardiography showed a hemopericardium with layers of different echo density. This suggests that the myocardial defect, which was observed during surgery, may have bled discontinuously from a small non-arterial vessel.

Hemorrhagic tamponade generally occurs within hours to a few days after a blunt chest trauma, when the chest still shows typical marks. The present case presents an unusual life-threatening complication following minor non-penetrating chest trauma in a patient who suddenly became symptomatic after an interval of several weeks without complaints. In such a case, the relation with the traumatic event may be less evident which may impede clinical management.

Acute coronary artery occlusion following blunt chest trauma has previously been described, resulting from intimal tearing or subintimal hemorrhage [5]. In the present case, it is

most likely that the electrocardiographic abnormalities were a consequence of the compression of a severely diseased coronary branch by the evolving tamponade.

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