

VIDEO SESSION

## Left Ventricle to Right Atrium Shunt Secondary to Blunt Chest Trauma. A Case Report

Hector Anninos, MD, Panagiotis Politis, MD, Pantelis Gounopoulos, MD,  
Apostolos Koroneos, MD, Panagiotis Dedeilias, MD

*Departments of Intensive Care  
Medicine and Cardiothoracic Surgery,  
Evangelismos Hospital, Athens, Greece*

### INTRODUCTION

Intracardiac shunts are rarely encountered as sequelae of non-penetrating heart trauma and their clinical manifestations may often be unrecognized in the multi-injured patient. However, they are serious complications and their diagnostic approach is not always feasible. We hereby present a case of a young man with a left ventricle to right atrium communication after blunt thoracic trauma sustained during a car accident.

### CASE REPORT

**KEY WORDS:** *intracardiac  
communication; non-penetrating  
injury; echocardiography*

A 26-year-old man with nonsignificant personal and family medical history, was admitted to the hospital after suffering a car accident. At admission he was fully awake and oriented, with a Glasgow coma scale (GCS) 15/15. He was slightly tachypneic, he had a heart rate of 110/min and a blood pressure 80/50 mm Hg. On auscultation there was a decrease in vesicular breath sounds in the lower areas of the lung and a systolic murmur 3/6 heard diffusely in the precordial area.

The electrocardiogram (ECG) revealed incomplete right bundle branch block (RBBB) with left posterior hemiblock (LPH) (Figure 1). The patient displayed elevated hepatic liver enzymes (aspartate aminotransferase-AST 455 IU, alanine aminotransferase-ALT 543 IU, normal <40 IU), creatine phosphokinase-CPK 1388 (normal <190), CPK-MB 71 (normal <18), high-sensitivity cardiac troponin T-hsTnT 245 ng/ml (normal <14, grey zone 14-53). The white blood cell count reached 22000/mm<sup>3</sup> and the hemoglobin was 14.5 g/dl. The rest of the blood analysis did not show any abnormal values. The patient underwent chest computed tomography (CT) scanning which displayed bilateral pleural effusions with compression atelectasis, fractures of the third, fourth and fifth right costal ribs, contusion in the right lung, and rupture of the aortic isthmus with the presence of pseudoaneurysm and mediastinal hematoma. The abdominal CT scan was normal.

The patient was transferred to the catheterization laboratory and aortography was performed which confirmed the CT findings. No other site of blood extravasation was found. A stent was inserted into the descending aorta at the isthmus level. Subsequently he was admitted in the ICU and placed under close monitoring. He was hemodynamically stable at the time (heart rate 100/min, blood pressure 105/65 mmHg) and arterial blood gas (ABG) examination showed pH 7.42, PCO<sub>2</sub> 30 mmHg, PO<sub>2</sub> 132 mmHg,

#### ABBREVIATIONS

CT = computed tomography  
ECG = electrocardiogram  
ICU = intensive care medicine  
LPH = left posterior hemiblock  
RBBB = right bundle branch block

*Corresponding author:*  
Hector Anninos, MD  
E-mail: ekanninos@yahoo.com

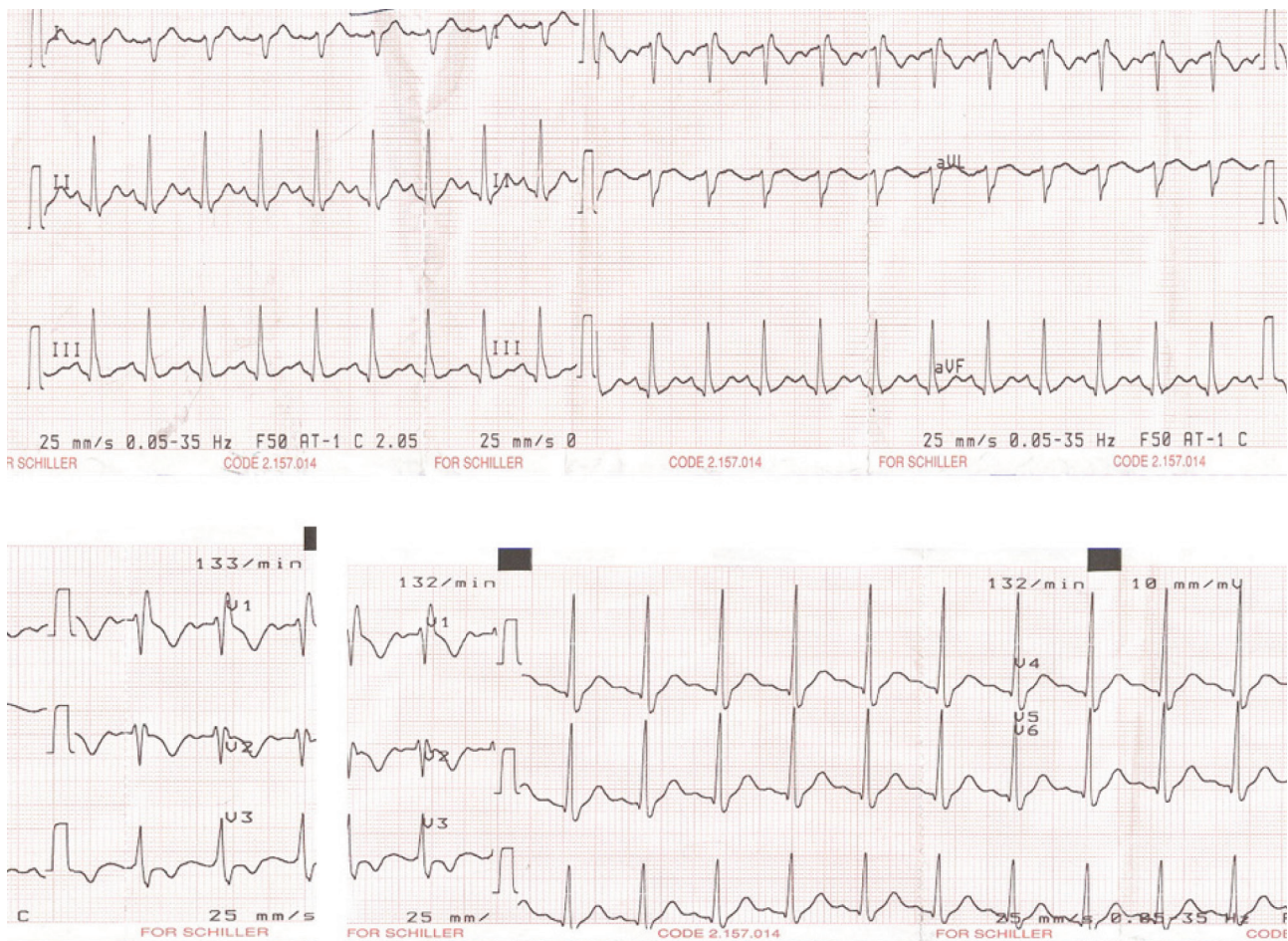


FIGURE 1. Electrocardiogram showing incomplete right bundle branch block (RBBB) with left posterior hemiblock (LPH).

and a bicarbonate level of 22mEq/L while receiving fraction of inspired O<sub>2</sub> of 60%. The oxygen saturation in blood drawn from the superior vena cava (ScvO<sub>2</sub>) was measured at 60%. Due to this borderline value and the systolic murmur heard on cardiac auscultation, a transthoracic cardiac ultrasound was performed, which disclosed an abnormal blood flow between the left ventricle and the right atrium without any remarkable findings regarding the right chambers (Figure 2). A traumatic rupture in the aortic root creating a shunt towards the right atrium was presumed. A right heart catheterization followed demonstrating O<sub>2</sub> step-up at the right atrium level (hemoglobin oxygen saturation in superior vena cava, right atrium, right ventricle and pulmonary artery was 46%, 80%, 85% and 85% respectively).

With the diagnosis of the intracardiac shunt confirmed, surgery was undertaken by the cardiac surgeon who repaired the shunt with placement of a synthetic patch. Following the operation, the patient had an uncomplicated course and was discharged after 28 days of hospitalization.

## DISCUSSION

Acquired left ventricular-to-right atrial communications are relatively uncommon complications, most often attributable to surgical procedures, trauma, endocarditis, myocardial infarction or even endomyocardial biopsy.<sup>1-3</sup> Most cases represent postoperative complications. Blunt chest trauma is scarcely reported in the literature as a cause of such an injury.<sup>4-6</sup> Usually myocardial contusion dominates the picture in non-penetrating heart traumas, although ventricular septal ruptures have been reported. Unfortunately, even in cases of intracardiac rupture the clinical presentation is non-specific and can be underestimated in the context of more predominant injuries. The ECG shows findings that can be attributed to electrolyte disturbances, acid-base imbalance, hypoxia or hypovolemia, which are all relatively common in thoracic injuries. Atrio-ventricular conduction disturbances have been reported although this was not the case in our patient.<sup>7,8</sup>

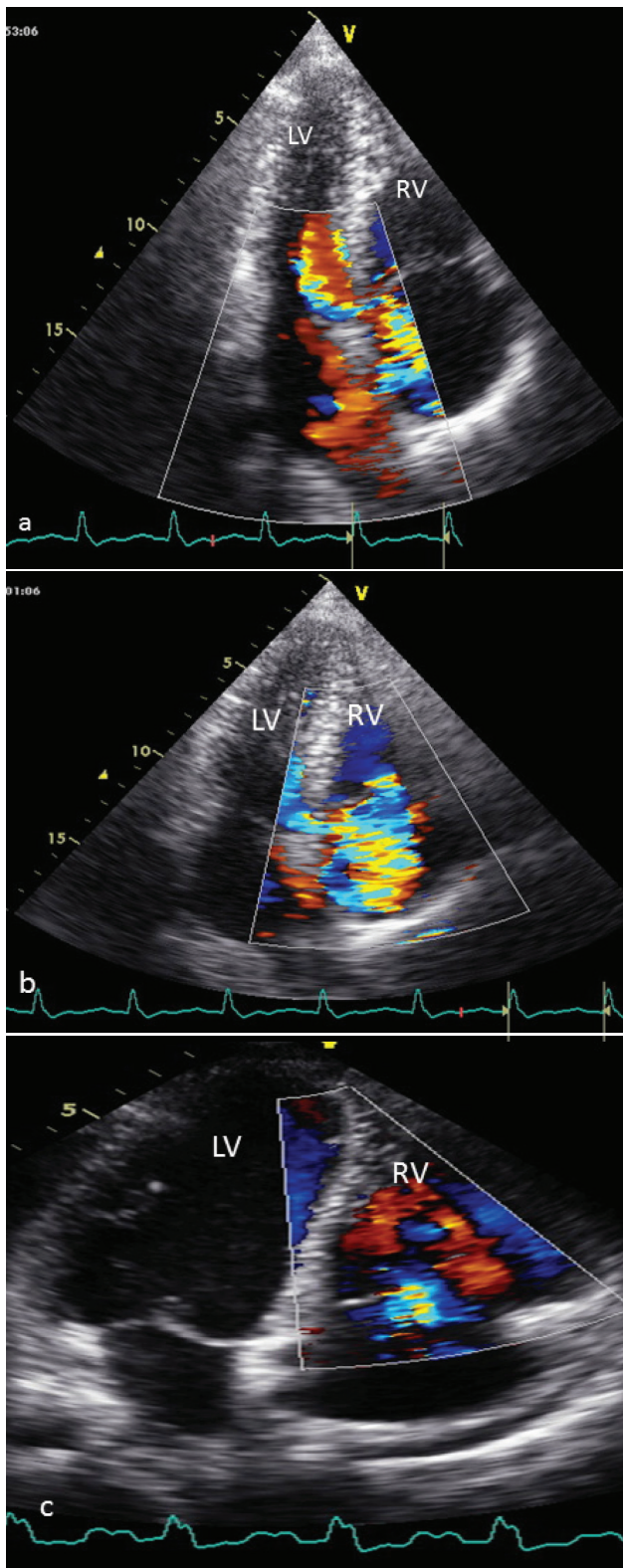


FIGURE 2. Pathological flow between left ventricle and right atrium (a, b) and mild tricuspid regurgitation (c). LV=left ventricle, RV=right ventricle

Though non-diagnostic, ECG findings can raise high degree of suspicion and prompt further evaluation by ultrasonography which can demonstrate a pathological flow and establish the diagnosis. The echocardiographic study should be detailed to discriminate tricuspid regurgitation jet from the left ventricle to right atrium shunt. However, transthoracic examination may be flawed due to suboptimal visualization especially in multi-trauma patients and in that case the transesophageal approach can be helpful, after the stenting procedure in case of aortic trauma.<sup>9,10</sup> In selected cases, contrast enhanced CT can be of diagnostic value.<sup>11,12</sup>

If the shunt is significant, hemodynamic deterioration may occur and this should prompt rapid surgical closure of the communication. In our case the initial hemodynamic instability was attributed to the aortic trauma and after the stent was positioned, the patient indeed recovered from hypotension and shock. The intracardiac shunt was presumed mostly on the basis of the physical findings.

In conclusion, non-penetrating cardiac trauma can sometimes cause significant intracardiac damage leading to rupture of cardiac structures and producing pathological shunts. With respect to their size and location, these lesions can produce a variety of clinical features ranging from complete absence of symptoms to rapid hemodynamic compromise and shock. After blunt chest trauma one should always be alert for the presence of such complications and regular physical examination is necessary to detect new cardiac murmurs which will guide further evaluation. Transthoracic ultrasound is a keystone examination but transesophageal views or CT may be needed in some cases.

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