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## Case Report

# Ventricular tachycardia from intracardiac hematoma in the setting of blunt thoracic trauma



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## ARTICLE INFO

## Article history:

Received 12 August 2013

Accepted 4 December 2013

Available online 22 December 2013

## Keywords:

Cardiac hematoma

Premature ventricular complexes

Ventricular tachycardia

ECG

MRI

## ABSTRACT

In the victims of motor vehicle accidents, unrecognized myocardial injuries may pose diagnostic and therapeutic challenges. Herein, we present a case of a 17-year-old man who developed multiple ventricular premature complexes and nonsustained ventricular tachycardia in the setting of blunt chest trauma from a motor vehicle accident. We discuss significance of the electrocardiographic abnormalities in making an accurate diagnosis of cardiac hematoma and its management.

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## 1. Introduction

In the victims of motor vehicle accidents (MVAs), unrecognized myocardial injuries may pose diagnostic and therapeutic challenges. Both cardiac concussion and contusion may remain asymptomatic. However, due to development of parenchymal edema, hemorrhage and patchy necrosis, cardiac contusion may present with pain, hemodynamic compromise, and various electrocardiographic (ECG) abnormalities. Herein, we describe how morphologies and patterns

of multiple premature ventricular complexes (PVCs) and ventricular tachycardia (VT) inferred the diagnosis of cardiac hematoma in a patient who was presented following an MVA.

## 2. Case presentation

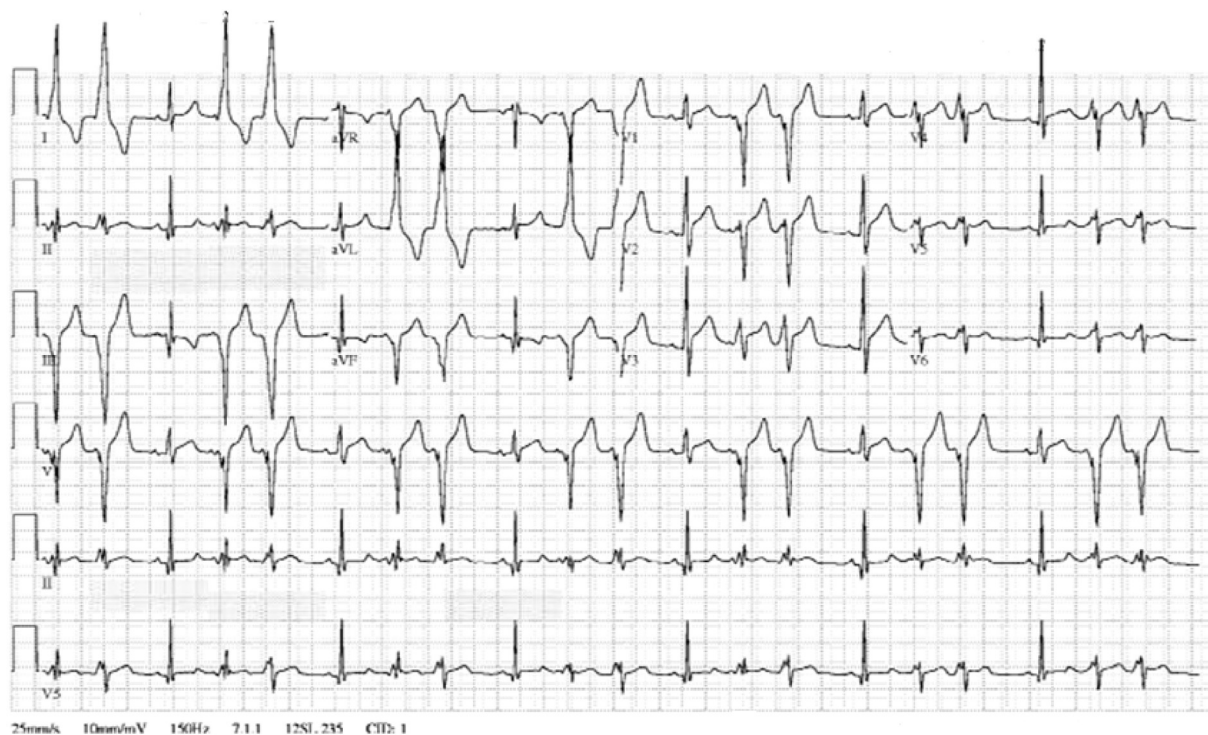
A previously healthy 17-year-old man who was a driver of the car skidded while driving at a high speed and fell into a ditch. The paramedical staff upon arrival to the scene promptly performed endotracheal intubation and needle compression

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<http://dx.doi.org/10.1016/j.ihj.2013.12.011>

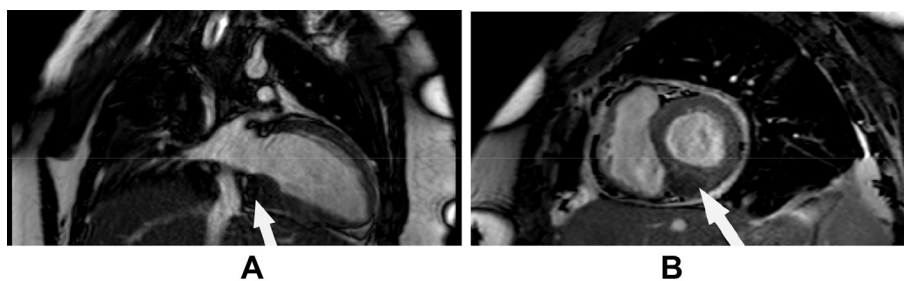


**Fig. 1** – A 12 lead electrocardiogram (ECG) shows multiple monomorphic premature ventricular complexes (PVCs) that have superior axis with a QS pattern in the inferior leads, tall R waves in leads I and aVL, and poor R wave progression in the lateral precordial leads.

of the left pneumothorax en route to the hospital. On physical examination upon arrival, he was unconscious with a Glasgow Coma Scale of 3 and required mechanical ventilation. His temperature was 99.0 °F (37.4 °C), heart rate was between 110 and 120 beats per minute, and blood pressure measured 95–110/65–80 mm Hg. He had soft tissue injuries, multiple rib fractures and a left-sided pneumothorax. Emergent computerized tomographic scans ruled out intracranial hemorrhage or neck trauma. Routine hematological and biochemical parameters were normal. Toxicological screening for alcohol and illicit drugs was negative. Family members reported no history of inheritable disorders or sudden cardiac death in the family. No major surgical intervention other than drainage of the pneumothorax by a chest tube was required to manage his

condition. The patient's clinical condition improved remarkably within 48 h leading to removal of the endotracheal and chest tubes and transferring him to a step-down unit.

On day 3, he developed episodes of asymptomatic monomorphic PVCs and nonsustained VT. The complexes demonstrated QS pattern in the inferior leads, tall R waves in leads I and aVL, and poor R wave progression in the lateral precordial leads (Fig. 1). These findings were suggestive of the inferior and basal lateral wall of the left ventricle as a focal site of origin of PVCs and VT. Based on the ECG findings, presence of a cardiac hematoma at the specific site causing PVCs and VT was suspected. Transthoracic echocardiographic examinations showed no major abnormalities except for trivial and stable pericardial effusion. Coronary angiography was



**Fig. 2** – Two views (A) and (B); a 2-chamber view long axis and a short axis view respectively, of cardiac magnetic resonance imaging (MRI) scans, show a large 3.2 × 3.0 × 2.5 cm intramural cardiac hematoma (white arrows) involving the inferior and basal lateral wall of the left ventricle.

normal. To further elucidate etiology, and a possible cause–effect relationship of PVCs and VT to the accident, an MRI examination of the heart was performed with and without intravenous administration of gadobenate dimeglumine (MultiHance<sup>®</sup>, Bracco Diagnostics Inc., USA). Multiplanar Fast Field Echo (FFE) images were obtained before and after contrast administration. These images showed a 3.2 × 3.0 × 2.5 cm intramural cardiac hematoma with hemopericardium at the inferior and basal lateral wall of the left ventricle (Fig. 2).

### 3. Discussion

Cardiac contusion may present with pain, hemodynamic compromise, and various ECG abnormalities that include ST segment and T wave changes, PVCs and atrial fibrillation. Although ECG changes may be nonspecific to diagnose cardiac contusion, it is important to bear in mind that as for almost all cardiac conditions, surface ECGs remain first-line and most widely used bedside non-invasive investigation. It is the inference from the ECG data that leads to more specific and confirmatory investigations. Transthoracic echocardiography has been reported as efficient for diagnosing a myocardial contusion. However, echocardiographic imaging modality has several limitations due to trauma related insufficient echo windows and poor quality examinations. Radionuclide imaging bears similar limitations of low sensitivity and specificity, and has no clinical utility.<sup>1</sup>

As shown in our case, cardiac MRI scan can be an important imaging modality to demonstrate intraparenchymal hemorrhage.<sup>2</sup> Cardiac MRI scan can also demonstrate preserved myocardium and differentiate between hematoma and pseudoaneurysm, which can be difficult to determine by echocardiography due to similar echogenicities between the hematoma and the ventricular cavity.<sup>3</sup> Follow-up MRI scans can also be used to define prognosis based on progression or regression of hematoma. Surgical intervention with implantation of a cardiac defibrillator may be warranted if MRI scans show significant scars as they may pose serious risk to sudden cardiac death from ventricular tachyarrhythmias.<sup>4</sup>

We managed our patient conservatively; the cardiac rhythm was closely monitored, oral metoprolol and supplemental potassium and magnesium were used to suppress PVCs and VT. Due to the extent of hematoma and potential formation of a large scar which may risk the patient to sudden cardiac death from ventricular tachyarrhythmias, we discharged him home with a wearable cardiac defibrillator (LifeVest<sup>®</sup>, Zoll Medical, USA). During his subsequent regular outpatient follow-ups for 6 months, his cardiac physical examinations, ECGs, ambulatory Holter monitoring and a transthoracic echocardiograms remained normal. Given his excellent recovery and no signs of PVCs or VT, we did not feel compelled, although desired, to repeat a follow-up MRI scan that the patient could not afford due to high cost.

In summary, ECGs findings of PVCs and VT should raise the suspicion of cardiac contusion and hematoma in patients with blunt chest trauma even in the absence of significant symptoms.

### Conflicts of interest

All authors have none to declare.

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