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Case report

# Severe cardiac trauma or myocardial ischemia? Pitfalls of polytrauma treatment in patients with ST-elevation after blunt chest trauma

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## HIGHLIGHTS

- ECG changes after blunt chest trauma can be misleading.
- Only by knowing the cause of accident can avoid erroneous time-intensive diagnostics and ensure a proper overall assessment.
- A TEE can be a useful early adjunct in trauma management in the setting of ongoing instability because it can guide resuscitation efforts.
- A stantardized preclinical – trauma room protocol according to ALTS® should be used during transfer.
- A thromblysis should be avoided in the acute setting because a blunt chest trauma can mimic a myocardial infarction.

## ARTICLE INFO

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## ABSTRACT

*Introduction:* Thoracic injuries are the third most common injuries in polytrauma patients. The mechanism of injury and the clinical presentation are crucially important for adequate emergency treatment.
*Presentation of case:* Here we present a case of a 37-year-old male who was admitted to our level-1 trauma center after motor vehicle accident. The emergency physician on scene presented the patient with a myocardial infarction. During initial clinical trauma assessment the patient developed circulatory insufficiency so that cardiopulmonary resuscitation was necessary. Considering the preclinical and clinical course it was decided to proceed with thrombolysis. Despite consistently sufficient resuscitation measures circulatory function was not restored and the patient remained in asystole and passed away.
*Discussion:* The initial assessment showed cardiopulmonary instability. After applying thrombolysis a therapeutic point of no return was reached because surgical intervention was impossible but autopsy findings showed severe myocardial and pulmonary contusions likely due to shear forces.
*Conclusion:* This case outlines the importance of understanding the key mechanism of injury and the importance of communication at each stage of healthcare transfer. A transesophageal echocardiography can help to identify injuries after myocardial contusion.


## 1. Introduction

Thoracic injuries are the third most common injuries in polytrauma patients, after injuries to the head and extremities, with an overall fatality rate of 10.1% in the United States [1]. Thoracic injuries can significantly increase mortality in patients with multisystemic trauma and are directly responsible for 25% of all trauma-related deaths [2], more than two-thirds of those cases in developed countries result from motor vehicle collisions. Only 2.5%–10% of thoracic trauma patients require thoracotomy, usually those with penetrating injuries [3–5], and most potentially fatal injuries can be treated during the “golden hour” by simple, rapid procedures such as chest tube insertion [4,6]. Knowledge of the presence of acute, life-threatening thoracic injury, based on the mechanism of injury and the clinical presentation, is of crucial importance for adequate emergency treatment, particularly because acute post-traumatic impairment of pulmonary function can quickly lead to death [7]. To diagnose and treat cardiac arrhythmias quickly in

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multiple injured patients, continuous cardiac monitoring is very important. Troponins are regarded as markers of mild cardiac injury [8]. Knowledge of and compliance with clearly defined diagnostic and therapeutic guidelines have resulted in a significant reduction in early fatalities during the pre-clinical and trauma room phases of treatment [9–11]. According to the Advanced Trauma Life Support (ATLS®) algorithm, an accurate diagnosis of cardiac contusions can only be established by directly observing the myocardium. A previous study reported myocardial contusions at autopsy in 14% of blunt trauma victims [12]. Another study of patients with a mean "injury severity score" of 22.2 ± 13.1 resulting almost entirely from blunt trauma found thoracic trauma to be the most common significant injury [13]. Precise determination of the cause of trauma during history taking is crucial. Lateral impact, for example, results in clear differences in thoracic injuries and overall severity compared with frontal impact. Details communicated by the emergency physician on scene are crucially important for the subsequent diagnostic and therapeutic procedures in the trauma room. Below we present a case after motor vehicle accident with a blunt chest trauma in which an incorrect interpretation of ECG abnormalities, unknown cause of accident in the absence of external indications of injuries had serious consequences.

## 2. Patient history

A 37-year-old man was admitted to our trauma room. The patient had driven his vehicle into a pole in an urban center and collided with a traffic light. The airbag had deployed. The speed at which the accident occurred was unknown. Six minutes after the accident, the emergency physician arriving on scene found the patient responsive, though becoming increasingly disoriented. His blood pressure was 55/35 mmHg and his heart rate (HR) was 105/min. His pupils were initially moderately large. The initial on-scene ECG showed myocardial infarction with ST elevation. Following cervical spine protection, placement on a vacuum mattress, insertion of peripheral venous catheters, administration of fentanyl (0.25 mg) and propofol (150 mg total), and endotracheal intubation, the patient was transported to the trauma room of our level-I trauma center, arriving 13 min after the accident. At admission his blood pressure had stabilized at 105/55 mmHg and his HR was 103/min.

## 3. Clinical findings

We assumed care of the intubated and ventilated patient, first addressing his hemodynamic situation. According to ATLS® the clinical trauma assessment showed no indication of osseous instabilities in the thorax, pelvis, or extremities. Initial venous blood gas analysis showed the following values: pH 7.139, $pCO_2$ 64.9 mmHg, $pO_2$ 24.9 mmHg, $HCO_3$ 21.1 mmol/L, Hb 5.9 mmol/L, potassium 2.8 mmol/L, sodium 133 mmol/L, lactate 6.3 mmol/L, and base excess −6.6 mmol/L. Focused Assessment with Sonography for Trauma (FAST) initially showed no abdominal free fluid, pericardial effusion, or organ rupture. There were no signs of increased intracranial pressure. We then administered 1500 mL Ringer acetate and 500 mL colloid infusion solution and 200 mmol of bicarbonate. Massive pleural fluid collection was excluded. Twelve-lead ECG showed right axis deviation, right bundle branch block, and ST-segment depression in V2–V6 (Fig. 1). Still during initial assessment, the patient developed circulatory insufficiency with barely measurable arterial blood pressure. Cardiopulmonary resuscitation was started immediately. The patient received 1 mg of adrenaline. His circulation briefly improved but he quickly required resuscitation again. Central venous and arterial catheters were placed in the groin. Return of spontaneous circulation was again achieved using high doses of adrenaline. Emergency echocardiography performed in the trauma room showed overall impaired ventricular contractility and a left ventricular ejection fraction close to zero. Based on the clinical findings, ECG changes and transthoracic echo findings, acute transmural posterior wall myocardial infarction with cardiogenic shock was assumed to be the cause of circulatory arrest.

Still showing short-term return of spontaneous circulation, the patient developed ventricular fibrillation that was immediately defibrillated. Resuscitation including several defibrillations continued. 250 mg of Dobutamine was added after 3rd defibrillation. A short period of electromechanical dissociation ended spontaneously. Considering the preclinical and clinical course, the patient's young age, and based on the working diagnosis of a posterior wall infarction it was decided to proceed with thrombolysis. Laboratory testing showed increased troponin T at 50.67 pg/mL (<14 pg/mL) and myoglobin at 770.6 μg/L (28–72 μg/L). 50 mg Actilyse® (Alteplase, Boehringer Ingelheim Pharma, Germany) was administered intravenously. This was the "point of no return" (Fig. 2) in which no surgical invasive intervention e.g. resuscitative thoracotomy could be performed. Repeat ultrasound after thrombolytic therapy revealed pleural effusion in the right thorax. Oxygenation quickly deteriorated and blood appeared in the endotracheal tube. After suctioning, oxygenation significantly improved. Hemoglobin dropped to 3.6 mmol/L, and we transfused three packed red cell units (300 mL each), after which hemoglobin increased and remained stable. The patient's initially small, light-responsive pupils became wide and non-responsive. Despite consistently sufficient resuscitation measures, with high-dose administration of catecholamines and thrombolysis therapy, circulatory function was not restored and the patient remained in asystole. The patient died in the emergency room of hemodynamically significant circulatory failure.

## 4. Autopsy findings

In summary, the patient suffered multiple injuries resulting from massive frontal impact against the torso. The impact caused severe myocardial and pulmonary contusions and tears in the pulmonary arteries and veins, likely due to shear forces. The patient experienced massive internal hemorrhage, resulting in his death.

## 5. Discussion

This is a case of a young patient involved in a high energy motor vehicle accident. The initial assessment according to ATLS® showed cardiopulmonary instability, without evidence of intra-abdominal, intrathoracic or pelvic bleeding or open femoral fractures. The 12-lead ECG performed during standard trauma care showed an abnormality consistent with an ST-elevation myocardial infarction. The FAST examination was negative initially. Fig. 3 shows the treatment algorithm with performed diagnostics and patient-specific findings. A CT scan could not be performed due to patients' instability. ECG changes are non-specific and are not regarded as a strong indicator of myocardial contusion in trauma cases, though they can indicate cardiac involvement and certain complications [14,15]. A meta-analysis by Maenza et al. showed a concordance between important cardiac complications and abnormal ECG findings [16]. Previous studies report a high likelihood of abnormal FAST results, ECG findings, and myocardial markers in patients with cardiac injuries secondary to chest trauma [17,18].

Witnesses reported that the decedent drove into a pole under unobstructed road conditions. Bystanders pulled the patient from the vehicle. He was initially conscious, but became increasingly somnolent and lost consciousness before the emergency physician arrived. Bystanders immediately initiated chest compression.