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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.

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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.

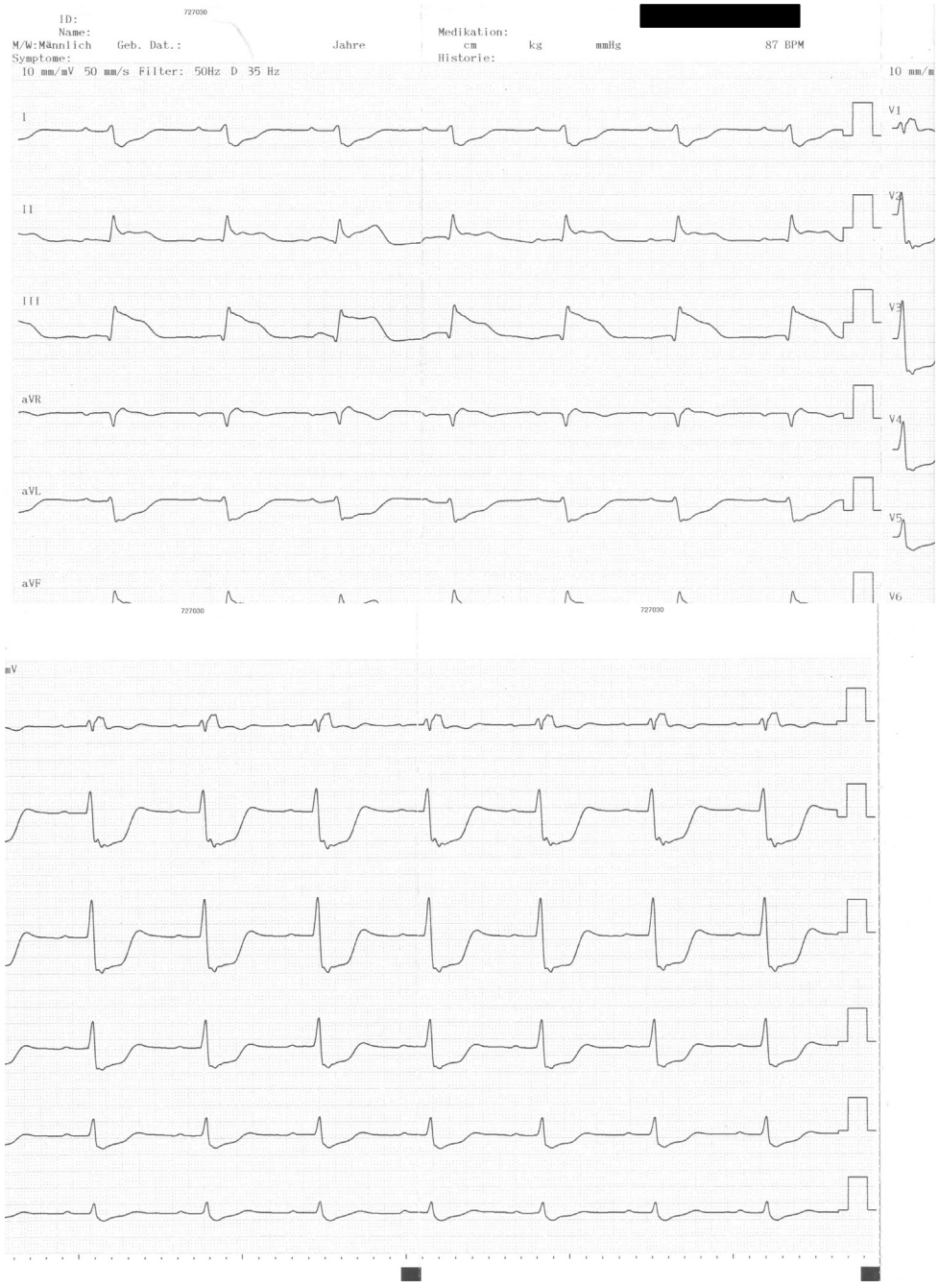

Fig. 1. 12-lead-ECG in trauma room.

The in-hospital providers focused their attention on the ECG changes as the cause of accident without considering the possibility of a high energy blunt chest trauma. However the initial clinical assessment included the search for possible trauma associated findings without success. A standardized trauma patient transfer checklist based on the mnemonic MIST (Mechanism of Injury, Injuries, Signs, Treatment) can help in such circumstances. Lethal outcomes of thoracic trauma often result from complications and are not exclusively dependent on the severity of the pulmonary or cardiac trauma itself. Blood gas analyses, respiratory mechanism functional values, radiographic findings, and hemodynamics do not allow reliable determination of a patient's prognosis after thoracic trauma. Cardiac contusions and direct injury with dissection of the coronary arteries and secondary myocardial infarction must be routinely considered in addition to acute myocardial infarction. Along with typical accompanying injuries, arrhythmias often occur in conjunction with cardiac contusions. Attenhofer et al. found ECG changes in 32% of patients after blunt thoracic trauma, primarily tachycardia, ST segment changes, and bundle branch blocks [6]. ECG changes present after blunt force thoracic trauma can completely resolve within a few months. Determination of myocardial markers in cases of severe thoracic trauma also does not permit any valid differentiation. Cardiac enzyme levels do serve as an indicator of cardiac pathology, though individual laboratory values are less significant in determining a diagnosis than the patient's course and clinical condition. It can be pathophysiologically

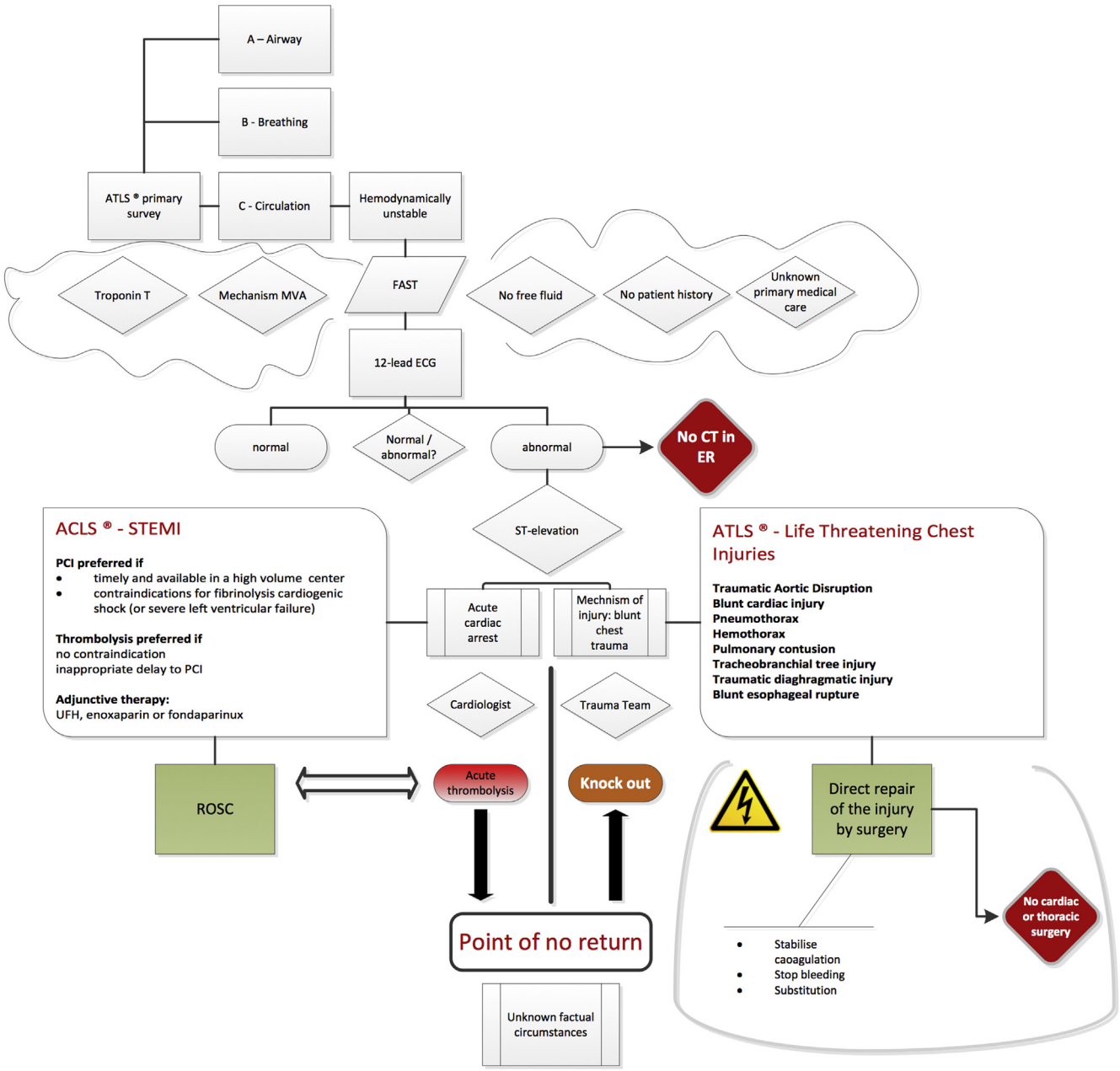

Fig. 2. Diagnostic and therapeutic work-flow.

assumed that blunt force thoracic trauma results in compression of the sternum and dorsal-paravertebral displacement of the heart, the ascending aorta, and the aortic arch. The resulting distraction of the descending aorta where it attaches to the spinal column (Fig. 3) can result in the typical distal tear in the exit of the left subclavian artery outlet. The history and ECG findings of this case prompted the cardiology staff to take over and initiate additional measures and transthoracic echocardiography (TTE). Limitation in acquiring adequate windows for TTE in the setting of blunt chest trauma makes transesophageal echocardiography (TEE) a valuable tool in hemodynamic assessment. TEE is especially useful in the unstable patient because it can be performed in the trauma room when transport for diagnostic imaging can be life threatening. Contrast-enhanced echocardiography may aid in the diagnosis of vascular and cardiac injuries, particularly if the presentation is subacute as from myocardial infarction or due to iatrogenic causes [19]. The cardiologist recommended to perform thrombolysis as a last resort. This was the 'point of no return' and led to the exclusion of further surgical intervention. Therefore, resuscitative thoracotomy could not be performed because the in-hospital providers assumed that firstly hypotension was related to cardiac decompensation and that secondly the low hemoglobin was a dilution effect of high liquid substitution and not a trauma related finding. Cardiac tamponade may not be a presenting feature if pericardial disruption and decompression of blood into the mediastinal or pleural space occur. However, even if initial FAST examination result is negative, tamponade may still develop as clot formation obstructs these pathways. In this case, this may have been the reason for the discrepancies between the initial FAST examination and the later autopsy finding. The injuries found at autopsy would have required

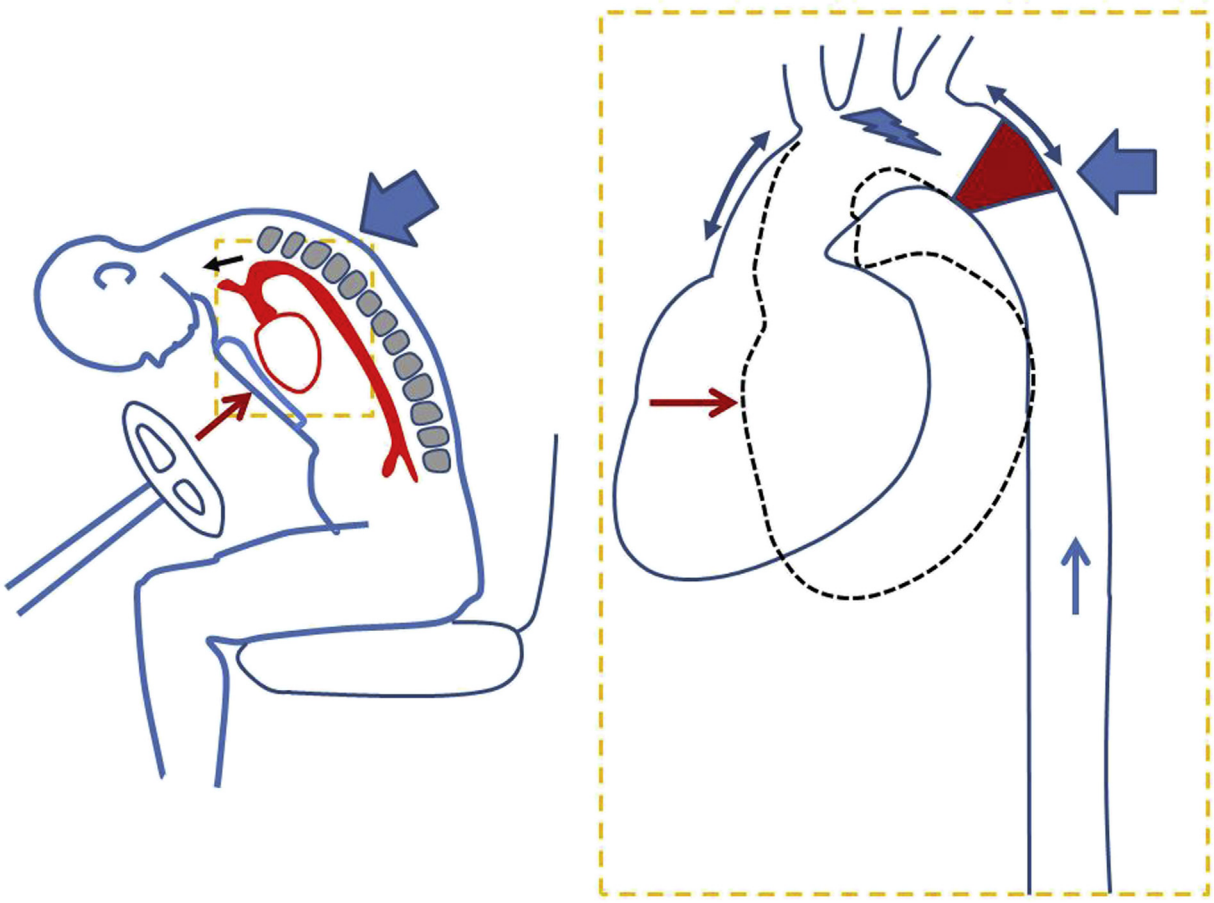

Fig. 3. Pathophysiology of thoracic trauma during motor vehicle accident (mod. Jauch and Heberer).

treatment with acute thoracic surgery. However, the combination of lack of a CT scan due to instability of the patient, an inadequate preclinical history and a missing TEE can negatively affect the outcome of patients with multiple injuries.

Essential information during patient transfer includes the cause of the accident, the patient's condition upon arrival of the emergency physician on scene, details concerning peripheral paralyses caused by the trauma, medical measures instituted at the site of the accident and during transport, prior diseases, complications, and a thoroughly completed standardized protocol such as the German Interdisciplinary Association of Critical Care Medicine [DIVI] emergency physician report. Only with this information is it possible to ensure a proper overall assessment and to avoid erroneous time-intensive diagnostics. TEE is a useful early adjunct in trauma management in the setting of ongoing instability because it can reveal concurrent injuries and guide resuscitation efforts. In this case, a TEE was not performed. In the absence of external indications of injuries and causes of injury, complete trauma history and proper transfer by the emergency physician to the trauma team leader are of critical importance.

However, even in cases of clear indications to a myocardial infarction, a thrombolysis should be avoided if a blunt chest trauma could be the reason for the ECG changes and if trauma diagnostics is not completed.

Human errors in medicine especially in incorrect diagnosis and treatment are among the ten major causes of death in healthcare systems [20]. There are two approaches to the problem of human mishaps: the person and the system approach with different philosophies. The person approach represent a “culture of blame” focused on mistakes of individuals. A “systems” approach emphasize a “safety culture”, that sees errors and critical incidents as a problem of the whole organization. The accepatance of human fallability and an open-minded non-punitive analysis of errors in the sense of a “preventive and proactive safety culture” should lead to solutions at the systematic level. There are different strategies to avoid systemic errors: to teach people how errors evolve in complex working domains and how types of errors are classified; the promotion of continuous medical education; and the training of problem-solving skills. Patient safety must have the highest priority.

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Author contribution

OÖ performed the analysis and drafted the manuscript. All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version for publication.

Conflicts of interest

The authors declare that there are no conflicts of interest.

Consent

The University Hospital of Leipzig Department of Trauma Surgery obtains informed consent for participation in research and authorization to collect, use and disclose protected health information from all patients.

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