Karate kick-induced myocardial contusion

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SUMMARY
Myocardial contusion should be suspected in a selected patient group with blunt thoracic trauma, who have elevated troponin, ECG changes and/or haemodynamical instability. Echocardiography is useful for direct visualisation of possible complications. In stable conditions, MRI allows for good visualisation of the heart and can confirm a suspected myocardial contusion as well as demonstrate the extent of myocardial damage. Based on the present literature, the authors developed a diagram for the diagnostic approach of a patient with suspected myocardial contusion.

BACKGROUND
Patients presenting with blunt thoracic trauma are common in the emergency department. In these patients, the question often arises whether myocardial contusion is present. This question remains quite challenging since there is no universal definition of myocardial contusion and no single test to determine the diagnosis in the emergency department.

CASE PRESENTATION
A man in his 40s with no medical history was presented at our emergency department after being assaulted. He received a ‘karate kick’ to the chest and suffered multiple hits to the head. On arrival at our emergency department, he reported dizziness, severe chest pain despite analgesics.

INVESTIGATIONS
Physical examination revealed a shoe-shaped abrasion on his sternum. Palpitation of the sternum was extremely painful. On auscultation, there were normal bilateral breath sounds. His oxygen saturation on presentation was 96% without extra oxygen. His pulse was weak and irregular, with a frequency of approximately 180/min. The initial blood pressure was too low to measure. The patient was somnolent without any other neurologic abnormalities. Further physical examination revealed multiple haematomas of the face and skull. His abdomen, pelvis, extremities and spine showed no abnormalities. The ECG indicated atrial fibrillation with a ventricular frequency of 176 bpm, with a normal heart axis, ST-elevation in III, aVF, V3-V6 and ST-depression in I and AVL (figure 1). Afterwards, the patient’s reports of chest discomfort slowly subsided, his blood pressure normalised and his consciousness improved. Direct damage to the right coronary was deemed unlikely because the patient’s chest reports disappeared, his ECG showed no signs of acute myocardial infarction and the CT did not show any hemopericardium or pericardial effusion. He was admitted to the intensive care for observation.

DIFFERENTIAL DIAGNOSIS
In patients with blunt thoracic trauma, there is a wide range of causes that can make a patient haemodynamically instable. The impact of the trauma can cause wall motion abnormalities due to intramural haematomas or lesions of the coronaries. Another possibility is the rupture of myocardial walls, at either the atrial level or ventricular level, which can cause pericardial effusion or tamponade. Valve dysfunction, papillary detachment or chordal detachment are also problems, which can be caused by blunt thoracic trauma. Finally, there is a possibility of aortic dissection as an effect of the trauma.

TREATMENT
The patient was in cardiogenic shock and directly received oxygen and in total 2 L saline 0.9%. Furthermore, 150 mg of amiodarone was given intravenously after which the atrial fibrillation converted to sinus rhythm without significant abnormalities (figure 1). Afterwards, the patient’s reports of chest discomfort slowly subsided, his blood pressure normalised and his consciousness improved. Direct damage to the right coronary was deemed unlikely because the patient’s chest reports disappeared, his ECG showed no signs of acute myocardial infarction and the CT did not show any hemopericardium or pericardial effusion. He was admitted to the intensive care for observation.

During his hospital admission, patient remained haemodynamically stable. No new rhythm disorders were observed. The troponin rose to a maximum of 2.6 μg/L and CK to 1840 U/L, normal range <171 U/L. Because of the supraventricular tachycardia, elevated troponin and low blood pressure, a myocardial contusion was suspected and the cardiologist was consulted. A transthoracic echocardiogram (TTE) showed a non-dilated left ventricle with good systolic function, a D-shaped septum, an enlarged right ventricle (RV) with reduced function, severe tricuspid regurgitation and a dilated right atrium. The TTE was strongly suggestive of a myocardial contusion of the RV with secondary RV dilatation and tricuspid regurgitation.

results showed an elevated troponin-T 0.105 mcg/L (normal range <0.014 μg/L) and CK 335 U/L, (normal range <171 U/L).

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Case report

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the outpatient clinic, patient was without complaints. His TTE showed a decrease in tricuspid regurgitation and RV diameter. Follow-up with TTE will be performed in a years’ time.

OUTCOME AND FOLLOW-UP
A cardiac MRI showed a dilated RV with diffuse oedema and LGE showed a D-shaped ventricular septum fitting the diagnosis of an RV myocardial contusion (figure 2). After his troponin levels started to decrease and the absence of rhythm disorders was confirmed, the patient was sent home. During follow-up 1 month later in the outpatient clinic, patient was without complaints. His TTE showed a decrease in tricuspid regurgitation and RV diameter. Follow-up with TTE will be performed in a years’ time.

DISCUSSION
Myocardial contusion is a complication of blunt thoracic trauma and is suspected to be an important cause of fatal traumatic accidents. The reported incidence of myocardial contusion in blunt thoracic trauma ranges from 3% to 76%, reflecting the wide range of diagnostic criteria used. Because myocardial contusion can be a challenging diagnosis, we will provide an overview of the current literature on the diagnostic approach of a patient with blunt thoracic trauma.

Myocardial contusion is defined by the presence of injury to myocardial cells. The primary cause is motor vehicle accidents and it may already occur while driving 30 km/hour. Other causes of myocardial contusion include falls from more than 6 m, chest compressions and as shown in our case, direct impact to the chest. The myocardium can be injured by direct forces on the heart, by impact of the heart on adjacent structures or by increased intrathoracic pressure.

On a cellular level, the impact on the myocardium results in an intramyocardial haematoma along with oedema and necrosis, which is quite similar to a myocardial infarction. Damage to the myocardial tissue can produce electrical instability, potentially resulting in atrial and ventricular arrhythmias. Because of its anterior location in the chest, the RV is most susceptible to direct impact and injury can result in dysfunction or wall rupture. The mitral and aortic valves are more prone to lesions due to the higher pressure in the left ventricle. In addition, thrombosis and laceration of the coronaries can occur as well as pericardial effusion, hemopericardium and damage to the great veins.

Myocardial contusion should be suspected in patients with blunt thoracic trauma, presenting with sternum and rib fractures, chest pain not reacting to analgesics, patients who are haemodynamically unstable or present with arrhythmias. However, patients can also present without visible signs of trauma and symptoms can be obscured by other injuries. A high level of suspicion, therefore, is paramount.

The ECG is abnormal in 40%–83% of the patients with myocardial contusion. A recent systematic review described a pooled sensitivity and specificity of 71% and 75% for identifying myocardial contusion. The ECG may show pericarditis-like ST-segment changes, prolonged QT time, right bundle branch block (RBBB) or arrhythmias as described above. If

Figure 1 Initial ECG of the patient at presentation to the emergency department.

Figure 2 MRI showing a dilated right ventricle and D-shaped ventricular septum.
Figure 3  Diagnostic approach of a patient with suspected myocardial contusion. Created by R.N. Doornkamp, D.F.M. van Winden, M.S. Buiten and D. Josephus Jitta. TTE, transthoracic echocardiogram. ICU; intensive care unit, CCU; cardiac care unit.
ECG changes are present, we recommend (telemetric) cardiac observation and further analyses for the presence of myocardial contusion (figure 3).

High-sensitivity troponin-T (hs-TnT) directly reflects myocardial injury, and a negative troponin, therefore, is a strong predictor for the absence of myocardial contusion. The reported sensitivity (77%) and specificity (85%) for hs-TnT is high. The ideal timing for the measurement of troponin is unclear, but measurement after 3 hours has been proposed, comparable to myocardial infarction. In case of elevated troponin levels, cardiac observation is recommended and a rising troponin level should prompt further cardiac evaluation. Patients with a normal ECG and troponins can safely be discharged home.

Echocardiography in myocardial contusion allows for direct visualisation of possible complications. However, TTE is limited by access, which can be a problem in patients with severe thoracic trauma. In case of insufficient access, trans-esophageal echocardiography can be a viable alternative in the emergency room. However, injury to the larynx, oesophagus and spine needs to be excluded. We recommend performing a TTE in all are haemodynamically unstable patients with blunt thoracic trauma, in patients with rising troponin levels and/or persisting ECG changes or arrhythmias.

Little is known about the use of MRI in patients with myocardial contusion. However, in stabilised patients, the MRI is an excellent method to separate traumatic from ischaemic myocardial injury and demonstrate the extent of myocardial injury. In stable cases with a high suspicion of myocardial contusion and a normal chest CT, MRI could be used to rule out myocardial contusion, especially if echocardiographic windows are limited.

Treatment of myocardial contusion depends on the severity of injury. In case of severe trauma to the (intra)cardiac structures or great arteries, haemodynamic stabilisation and thoracic surgery is necessary. Arrhythmogenic complications such as ventricular and supraventricular tachycardias can usually be treated medically or with aneclectrical cardioversion. In case of myocardial contusion without (severe) complications, patients should be monitored for 24–48 hours after the trauma, since most cardiac complications occur in this period of time.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

**REFERENCES**


**Case report**

An in-hospital observation period of 24–48 hours is recommended in myocardial contusion without complications, with a follow-up echo after 6–12 months.