Delayed sudden death determined by right atrial contusion - case report and literature review

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Abstract: The authors made a search for the blunt thoracic trauma complications in the literature stressing out the differences between commotio cordis and cardiac contusion as the blunt cardiac injury. Physiopathology, clinical and autopsy findings are presented for both conditions. A rare case of delayed death after right atrium contusion in a 6 years-old child is reported and documented. In this particular case lethal cardiac complications appeared 10-12 hours after sustaining the thoracic blow. When promptly diagnosed and appropriately monitored and treated, cardiac contusion may have a better outcome. Commotio cordis urge for a very rapid intervention of CPR and defibrillation as the sole chance of survival treatment.

Key words: Commotio Cordis, Cardiac contusion, Myocardial contusion

Blunt thoracic trauma may determine in some circumstances and in some persons (young, elder persons, etc.) severe complications such as fractures (sternum or ribs), lung contusions with haemorrhages and emphysema, mediastinum contusions with involvement of the major vessels (tears of aorta endothelium, etc.), pleural complications (haemotorax or/and pneumothorax, even subcutaneous emphysema) or cardiac trauma.

The heart itself may suffer as a direct consequence of the blow the cardiac arrest as a dramatically abrupt clinical symptom of commotio cordis (which is Latin for "disturbance of the heart"), essentially a concussion [1] which determine a disruption of the conductive system.

Nevertheless very similar circumstances may determine cardiac contusion which clinically may have a less dramatic onset but with visceral lesions (impact cardiopathy). This is the blunt cardiac injury, formerly known as myocardial contusion [2].

Commotio cordis was initially described as early as 1857; it is defined as an instantaneous cardiac arrest and almost simultaneous sudden death from a disruption to the conductive system produced by a witnessed, nonpenetrating localized impact to the chest, in the absence of pre-existing heart disease or identifiable morphologic injury to the sternum, ribs, chest wall, or heart [1]. The victim may collapse immediately after the blow secondary to a lethal arrhythmia, but in up to 50% of cases, there is a short delay between the times of impact and collapse [1].

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Although reported as the second most common cause of sudden cardiac arrest in young athletes (behind HOCM), commotio cordis is underreported and under-recognized [3].

The United States Commotio Cordis Registry (USCCR), in Minneapolis, Minnesota, reported that as of September 2001, only 180 cases had been documented. Up to 62% of these cases involved engagement in organized, competitive sports, with two-thirds of the patients being younger than 16 years of age and 80% being male [1].

Regardless of the mechanism, impacts resulting in commotio cordis are typically of low energy and velocity [4, 5].

In 1930, George Schlomka was the first to describe the factors that can lead to arrhythmia after a moderate precordial impact. He believed that the force, location, and type of object causing the impact determined the type of injury and the subsequent risk of arrhythmia [3].

The threshold speed of impact to cause ventricular fibrillation is between 25 and 30 mph, therefore a relatively little force but at a vulnerable period of the cardiac cycle [5, 6].

When the speed is over 50 mph, however, the likelihood of ventricular fibrillation actually decreases, although the possibility of myocardial contusion becomes greater [1].

Impact on the centre of the cardiac silhouette induced ventricular fibrillation in 30% of reported cases, compared with 13% and 4% at the left ventricular base and apex, respectively [5].

The impact must be delivered 10-30 milliseconds before the peak of the T wave in the cardiac cycle in order to induce ventricular fibrillation [1]. Induction is likely secondary to the activation of potassium-carrying ion channels via mechanic-electric coupling. The activation of these ion channels generates an inward current, thus locally augmenting repolarisation and resulting in premature ventricular depolarization and the initiation of unstable ventricular arrhythmias. If impact occurs during other portions of the cardiac cycle, different conduction disturbances, such as heart block, bundle branch block, or transient ST segment elevation, may be induced [3, 5, 7].

The initiation of VF is related to the peak left ventricular pressure produced by the blow. Left ventricular pressure changes likely result in cell membrane stretch and mechanical deformation (may be even selective disruption of the cytoskeleton) which seems fundamental to the activation of ion channels and underlies the mechanism of VF in commotio cordis [8].

As an uncommon clinical entity commotio cordis may induce sudden death from cardiac arrest in previously healthy children or in young athletes after a blunt blow to the chest [9].

The detailed external/internal forensic examination of the body reveals no evidence of structural, pathologic, or histologic signs of trauma to the heart.

Cardiac concussions are clinically, pathologically, and chemically different from a cardiac contusion [10].

*Cardiac contusion*, actually *blunt cardiac injury*, usually imply delayed clinical complications and tissue lesions [11, 12, 13, 14].

Traumatic circumstances may involve sports activities, accidents (road traffic accidents –blunt blow or seat belt syndrome [15], etc.) or aggression.

Usually not well recognized partly because of a general lack of appreciation of the real trauma severity may develop lesions of impact and subsequent cardiopathy which may lead to heart failure and death even after 4 weeks [16].

Other late complications may manifest: pulmonary embolism (transmural contusion of the right ventricle leading to a mural thrombus which eventually became detached) [17], right ventricular failure and pulmonary emboli up two 2-3 days [18], fatal cardiac arrhythmias
several days after blunt chest trauma particularly when other severe injuries are present [19], a complete atrioventricular block [20], dysrhythmias and cardiogenic shock (in 5%, [19]).

Thus, a normal ECG on admission and absence of cardiac arrhythmias during the first 24 h of intensive care treatment do not necessarily exclude the occurrence of life-threatening arrhythmias in the further course.

Histologically, injuries determined by contusions were identified at subepicardial, myocardial or subendocardial layer as interstitial haemorrhage, disruption or coagulative necrosis as well as contraction band necrosis of the muscle fibers, which might be categorized into the hemorrhagic, necrotizing and mixed forms. Intravascular thromboses were occasionally discovered post-impact. Immunohistochemically myoglobin (Mb) is detected with large amount of Mb deposited between cells at 5 min post-impact and little deposition 60 min post-injury.

Loss of CK-MB with small amount of intercellular CK-MB deposition 5 min after impact and depletion of CK-MB 180 min post-impact. Focal accumulation of fibrinogen at the cell membrane 5 min after impact and a large quantity of fibrinogen within cardiac fibers 180 min [21].

Hours after the contusion cellular oedema may cause causing lethal bradyarrhythmias and hypotension [22]: 4-6 days after blunt cardiac injury histological findings may include severe interstitial oedema, haemorrhages and infiltration of lymphocytes and neutrophils, fresh myocardial necrosis and fatty degeneration [22].

During the healing process, haemorrhages are absorbed gradually in a patchy, irregular pattern with subsequent scar formation [23].

Cardiac markers such as myocyte structural proteins myoglobin, FABP, cardiac troponin, desmin and the three plasmatic proteins (fibrinogen, fibronectin and C5b-9) prove modifications in vital contusions. In these cases there is in addition an intrasarcomelial accumulation of plasma proteins [24].

Diagnostic must be suspected in witnessed and unwitnessed blunt thorax trauma because misrecognized usually lead to aggravated complications. Confusing symptoms crosslink the myocardial contusion and myocardial infarction due to many similarities in terms of pathophysiologic changes [24]. On 169 patients the interval between injury and diagnosis was 3.2 +/- 2.3 days (mean +/- SD) from injury and in six patients the diagnosis was made only at autopsy [25].

On a 4-year study Biffl WL et al. 1994, diagnosed myocardial contusion in 30% of patients with high-risk blunt chest trauma. Cardiac enzymes such as CPK-MB are disputable as sole predictor of a complication [26] and seems to be nonspecific [27]. Cardiac troponin I (cTnI), or T (cTnT) in serum (highly specific for the myocardium) have replaced classical markers, such as creatine kinase MB. Cardiac troponins are preferred markers because of their high specificity and sensitivity, elevation of troponins has been shown to be associated with a poor outcome.

C-reactive protein and/or other inflammatory biomarkers may add independent information in this context [28].

Computed tomography [29], radionuclide angiography (gated radionucleotide angiography RNA, multiple gated acquisition angiography MUGA, technetium pyrophosphate scanning, single photon emission computed tomography SPECT) [30], electrocardiography may be useful in the identification of the patients at the greatest risk and predictive of the severity of the myocardial injury [31, 32]. Echocardiography (transoesophageal echocardiography, transthoracic echocardiography) is useful in the
management of myocardial decompensation but not as a primary screening tool in blunt cardiac injury [27].

**Case report**

A 6 years-old child was playing in the courtyard with his 3 brothers (aged between 5 and 9 years). During the afternoon (somewhere between 17-18 hours) an apparently minor accident occurred: the child was hit by a swing chair in the chest. The incident was not reported to the parents. Apparently everything went well during the evening and after having their dinner at around 20.00 p.m., the children went to bed at about 21.00 pm. In the morning the 6-years-old child was found dead.

The autopsy was performed the same day. No external traumatic injuries were found. The internal examination revealed the presence of marked cerebral oedema, 2 small haemorrhages on the internal surface of the sternum without any visible fracture (Fig. 1), and a cardiac contusion with intense haemorrhage of the right atrium (Fig. 2).

**Histopathology**

The microscopic examination revealed the presence of extensive interstitial haemorrhages in the right atrium (pericardial, subepicardial fatty tissue and between myocardial fibres) (Fig. 3, 4), moderate oedema of the myocardial fibers, capillary leukostasis and focal interstitial leukocitic infiltration at the level of the right atrium (Fig. 5). Fragments from other regions of the heart were normal.
Fig. 3 Right atrium: leukocytic extravasates at the epicardium – myocard border. (HE, 40x)

Fig. 4 Right atrium: extensive interstitial haemorrhages unravelling the myocardial fibres (HE, 20x)
Discussion

Among cardiac contusions, right atrium myocardial contusion is less frequent due to the protection offered by the sternum [25].

Certain areas of the heart are more exposed to concussive trauma. The right ventricle is more vulnerable due to the location beneath the sternum, and any impact can be transmitted immediately to the right ventricle [23].

Our study presents a rare case of delayed blunt cardiac injury of the right atrium with myocardial contusion injury in a 6 years-old child. Both the region (right atrium) and the delayed complications represent a rarity in the literature.

According to information from the police investigation, death could have occurred in the interval 9.00 p.m.-8.00 a.m. next day, that is at least 3-4 hours after the traumatic event. However rare, similar delays have been reported [18, 19, 20, 21, 22, 25]. The cardiac lesions are important sustained by the histopathological modifications such as oedema of the myocardial fibers and extensive haemorrhages. The leukocytic infiltration (neutrophils and lymphocytes) of the myocardial contusion area suggest an even longer delay between the thoracic blow and death, most probably 10-12 hours: therefore the sudden death might occurred around 4-5 a.m.

In our view the final event was a major arrhythmic incident (ventricular fibrillation/cardiac arrest), triggered by the electric instability of the contused myocardial area (and probably favoured by the vegetative nocturnal pattern). Cardiogenic shock, considered by many authors the most probable cause of death in cases with long delays between the traumatic event and death, is not supported by the evolution or by the autopsy findings.
According to ICD-10 the cause of death statement will include Ia: cardiac arrest, Ib: cardiac contusion, Ic: thoracic blunt trauma, Id:-.

**Conclusion**

Cardiac contusion must be clearly differentiated from the *comotio cordis* which has a dramatically clinical onset by instantaneous cardiac arrest.

Isolated blunt injury of the right atrium is an extremely rare traumatic event. Delayed death (with an asymptomatic interval of 3-12 hours) after right atrial contusion is even less frequent, but nevertheless possible.

**References**