Concussion of an athlete´s heart – a case report of blunt chest trauma-associated loss of consciousness in a professional soccer player (RCD code: VIII)

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Abstract

We present a case of cardiac concussion (commotio cordis) resulting from blunt chest trauma injury in a professional athlete. The soccer player received frontal injury to the chest caused by another player's knee during a training session. A few minutes of spontaneously relapsing unconsciousness ensued and the patient was admitted urgently for observation. Surface electrocardiogram showed dynamic alterations in the precordial leads, cardiac necrotic markers were only mildly elevated and cardiac magnetic resonance could not detect any injury-related pathology. The athlete received conservative treatment. We discuss the differential diagnosis with cardiac contusion and Brugada syndrome. Electrocardiographic abnormalities persisted during the two-week follow-up. The patient successfully continues his professional soccer career. JRCD 2017; 3 (5): 171–175

Key words: cardiac contusion, chest injury, Brugada syndrome, sports-related injury, rare disease

Background

Blunt chest injuries are an important clinical problem in current practice, especially due to the increasing incidence of traffic accidents. The chest wall and the soft tissues in the area are the most common locations affected by blunt traumas [1].

Blunt cardiac injury covers the spectrum of myocardial concussion and contusion to myocardial rupture. The right atrium and right ventricle are the most frequently injured chambers because of their anterior positioning in the chest, followed by the left atrium and left ventricle [2]. Blunt cardiac injury is most frequently caused by road traffic accidents and result from a direct blow to the chest from a steering wheel or rapid deceleration [3]. Other, less frequent causes include falls from a great height, sports injuries, blast forces, and indirectly through compression of the abdomen with upward displacement of abdominal viscera [4].

Chest injuries in contact and collision sports are relatively rare, particularly those that are life-threatening [4]. As with every acute sports-related injury assessment, the mechanism of trauma must be considered, the initial life-threatening condition must be evaluated and managed accordingly, and the development of further symptoms must be monitored [5].
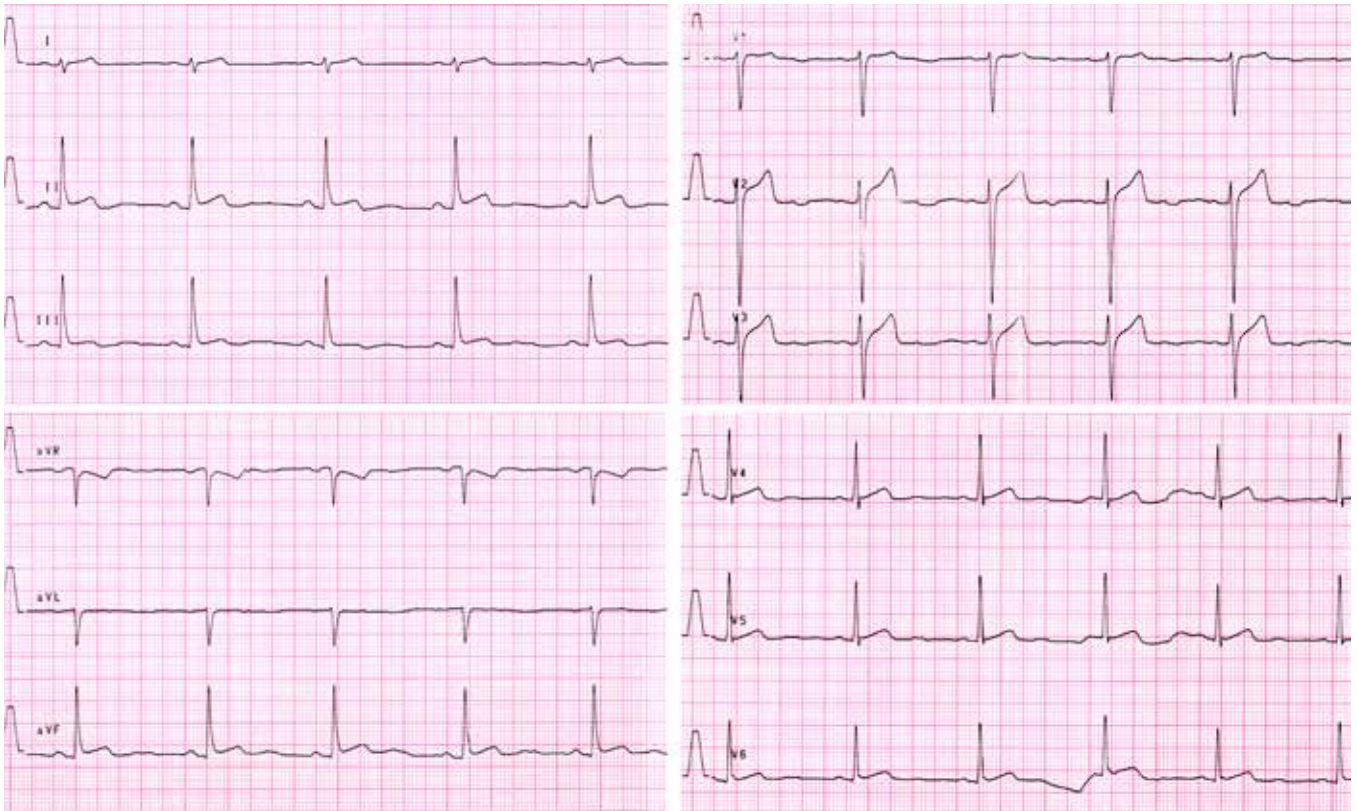
In this case report, we present a 26-year-old male professional soccer player who suffered blunt chest trauma with electrocardiographic alterations due to cardiac injury.

Figure 1. A baseline 12-lead surface electrocardiogram of the patient recorded two weeks before the incident; derivations as denoted in the picture, all stripes recorded at 10mm/mV, 25 mm/s

Case presentation

A twenty-six-year-old male patient of Haitian origin, a professional soccer player, was admitted after frontal collision with another player during training (struck with a knee to the precordial area), resulting in short-term loss of consciousness lasting between one and five minutes according to witnesses. On admission the patient was asymptomatic and physical examination was unremarkable. There was no personal or family history of cardiovascular diseases or sudden death.

Transthoracic echocardiography (TTE) showed normal chamber dimensions, preserved global and segmental contractility of the left ventricle with ejection fraction of 73%, mild pulmonary regurgitation, otherwise normal valve morphology and function, hyperechogenic ascending aorta and no signs of pericardial effusion. The initial radiologic evaluation included computed tomography (CT) scan of the head, performed to exclude potential intracranial causes or sequelae of the loss of consciousness, as well as abdominal ultrasonography, which were unremarkable.

Admission electrocardiogram (ECG) showed sinus bradycardia of 50 beats per minute and diffuse ST-segment elevations in anterior and inferior leads, however, similar alterations were previously observed in the resting ECG in this patient before trauma (Figure 1). Laboratory tests revealed elevated levels and dynamic changes of serum creatine kinase (CK) and CK-MB activity, without significant release of 5th generation high-sensitivity troponin T (hs-TnT).

Further electrocardiographic recordings showed dynamic changes in leads V1-V3, mimicking to a degree, an ST-elevation myocardial infarction evolution (Figure 2 a-c). The coronary CT angiography showed normal coronary arteries and no signs of thoracic aorta dissection. Furthermore, no fractures were identified in the patient's chest.

Cardiac magnetic resonance imaging (CMRI) showed neither ischaemic nor non-ischaemic myocardial damage, no pathological early or late gadolinium contrast enhancement (LGE) was observed (Figure 3 a-b). 24-hour Holter ECG monitoring registered sinus rhythm with an average frequency of 56 beats per minute (maximum 81/min, minimum 43/min) and a few sinus bradycardia episodes (the slowest recorded ventricular action was 37/min at night), no significant arrhythmia was observed.

The patient also experienced a transient febrile episode (38.7 °C) of unknown cause treated successfully with paracetamol and sodium dipyrone (metamizole). Blood inflammatory markers were within normal range and no clinical signs of infection were present.

Patient management and follow-up

Cardiac necrotic markers and initially mildly elevated hepatic aminotransferases quickly normalized throughout hospitalization. The patient was discharged from the hospital after 3 days of observation with persistent ECG changes, and advised to avoid

physical activity until ECG trace normalization and was scheduled for follow-up after 2 weeks.

At follow-up, the resting ECG trace failed to return to baseline and resembled the admission ECG after collision (Figure 2 d). The patient reported no symptoms. There were no new echocardiographic changes (Figure 3 c-e) and the athlete presented with full exertional capacity. Due to the fact that the dynamic ECG changes were limited to the precordial leads V1-V3 (which at one point fulfilled the Corrado criteria [6] – Figure 2 b), and because the exact mechanism of syncope was unknown, a provocation test with class Ia antiarrhythmic ajmaline (1 mg/kg) was performed at follow-up to exclude the possibility of Brugada syndrome, despite the absence of family history for the disease.

Since no damage to the heart was identified, the patient was permitted to return to his training sessions with a recommendation to avoid precordial injuries. A routine program of cardiologic assessment (ECG, TTE and exercise ECG testing on a treadmill) was scheduled (every 6 months in our center). The patient continued his professional soccer career, however, further follow-up data are not available, since shortly after the incident he was transferred to a football club in another country.

Review of the literature

The presentation of non-specific symptoms with dynamic electrocardiographic alterations mimicking acute coronary syndrome, but no abnormalities in other diagnostic methods, including echocardiography and CMRI, as well as a non-specific pattern of cardiac biomarkers, made this a unique case. This is the first case report of blunt chest trauma with isolated ECG alterations in a professional soccer player to our knowledge.

The diagnosis of myocardial contusions in sports remains a challenge. Initial assessment of chest injuries includes determination of the mechanism of injury, evaluation of the athlete´s airway, breathing, circulation and level of consciousness. There are no ideal tests or specific criteria in the literature demonstrating the appropriate assessment for detecting myocardial damage, although Troponin I and T are considered highly sensitive for this condition and useful in the stratification of patients at risk of complications [7].

In general, mild injuries due to cardiac contusion are well tolerated by patients, followed by an uncomplicated recovery with excellent prognosis [7]. Mild symptoms, such as palpitations or precordial pain that may be produced by myocardial contusion, can also be associated with musculoskeletal injury, thereby obscuring the diagnosis. Life threatening ventricular arrhythmias and cardiac failure are some possible, although less common complications of cardiac injury. The reported incidence of cardiac contusion in pa-

Figure 2. Dynamic changes in the right precordial leads (V1-V3) of the surface 12-lead electrocardiogram following a frontal blunt chest trauma; stripes represent (in top-bottom order) a) baseline before the incident b) admission record c) further evolution of the curves d) two-week follow-up record with persisting alterations compared to baseline, resembling the admission record; all stripes recorded at 10mm/mV, 25 mm/s
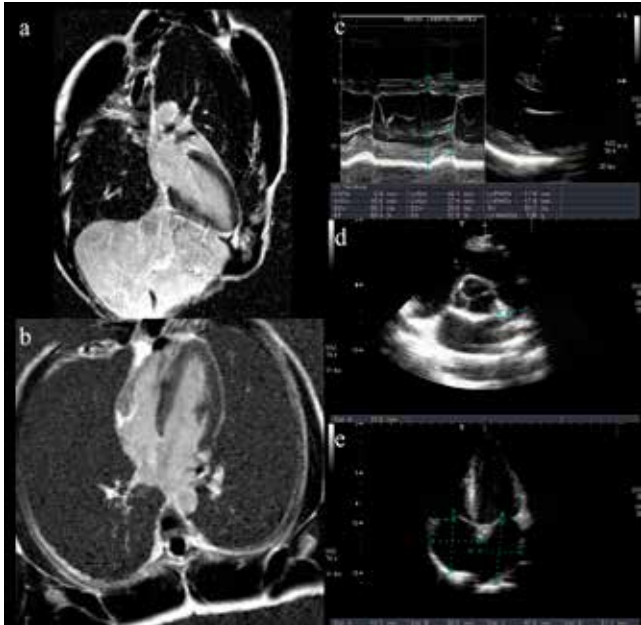
Figure 3. Cardiac magnetic resonance (a-b) and transthoracic echocardiography (c-e) imaging showing no structural damage to the heart and absence of late gadolinum enhancement (LGE), as well as the borderline thickness of the left ventricle (11 mm)

tients with blunt chest trauma ranges between 3–56% of patients, depending on the criteria used for establishing the diagnosis [7].

Brugada syndrome is a disorder characterized by sudden death associated with one of the ECG patterns involving an incomplete right bundle-branch block and ST elevations in the anterior precordial leads. Many clinical situations have been reported to unmask or exacerbate the ECG pattern of Brugada syndrome. Examples include a febrile state, hyperkalaemia, hypokalaemia, hypercalcemia, alcohol or cocaine intoxication, and the use of certain medications, including sodium channel blockers, vagotonic agents, alpha-adrenergic agonists, beta-adrenergic blockers, heterocyclic antidepressants, and a combination of glucose and insulin [8]. Therefore, we suspected that it could have also been triggered by a blunt chest injury and myocardial contusion, since the resulting ECG pattern was borderline diagnostic. However, no associated ventricular arrhythmia was ever recorded. The negative provocation test with ajmaline and no family history effectively excluded the Brugada syndrome.

Commotio cordis (concussion of the heart) is a condition that involves a similar mechanism of trauma, but differs from myocardial contusion because the chest blow occurs at the beginning of the T-wave in the cardiac cycle, thereby causing the heart to go into ventricular fibrillation [5,9]. This could not be excluded in our case, since the mechanism of the loss of consciousness remains unresolved. Minimal structural damage in imaging studies and laboratory tests, more suggestive of a skeletal rather than heart muscle injury, as well as preserved heart function seem to support the hypothesis of arrhythmic aetiology. Another characteristic feature differentiating concussion from contusion is the instantaneity of arrhythmia leading to loss of consciousness (in contusion potentially fatal arrhythmias occur usually within 24 hours) [9]. Hence, a spontaneously aborted ventricular tachycardia or fibrillation re-

sulting from commotio cordis seems to be a feasible explanation in this patient.

The American Heart Association and American College of Cardiology recommend comprehensive evaluation for underlying cardiac pathology and susceptibility to arrhythmias in survivors of commotio cordis (recommendation class I B), which should include resting and Holter ECGs, TTE, CMRI and stress testing as well as consideration of pharmacologic testing for Brugada and long-QT syndromes if indicated by ECG abnormalities [9]. We have performed all of the recommended assessments, with the exception of stress testing which was performed 2 weeks earlier and showed excellent exercise capacity without ECG abnormalities. In our case, we observed two distinct features that are not routinely mentioned in the literature of commotio cordis: (1) unusual ECG changes that could represent complications of ventricular arrhythmia due to local ionic and metabolic disturbances and (2) a non-infectious febrile state that may be explained by the release of inflammatory cytokines from injured tissues in the precordial area.

CMRI is a very useful tool in evaluating blunt cardiac injuries, which can provide detailed structural and functional imaging complementary to TTE. In T2-weighted sequences, evidence of acute myocardial injury and oedema can be found in the course of cardiac contusion [10,11]. Presence of LGE, especially in the sub-endocardial region, is believed to be highly specific for myocardial infarction and allows for differentiation between contractility impairment due to infarction or hibernation/stunning [10,12,13]. Southam et al. used CMRI to successfully differentiate myocardial contusion from infarction [13], however, there is growing evidence that contusion can be associated with the presence of LGE more often than not [10–12]. LGE was found to correspond with myocardial blush on angiography [12] and to surround a region of intramural haemorrhage following blunt chest trauma [11], however LGE is not present in all instances of intraparenchymal haemorrhage [14]. By definition, cardiac concussion, as opposed to contusion, occurs without structural damage to the heart [9], and therefore CMRI can help to differentiate between these two conditions [14], especially when loss of consciousness is not instantaneous or circumstances of the accident are unclear.

It is recommended, that if no underlying cardiac abnormality is identified, the individuals can safely resume training and competition after resuscitation from commotio cordis (class IIa C), since recurrence of heart concussion has a very low probability, although data from animal studies indicate some individual susceptibility [9]. This soccer player successfully continues his professional career.

In conclusion, blunt chest trauma associated with loss of consciousness or persistent chest pain requires a multidirectional diagnostic approach to assess for presence of heart damage, differentiate between cardiac contusion and concussion, and to exclude any underlying predisposing conditions that may lead to potentially fatal arrhythmias. In most cases of commotio cordis the underlying arrhythmia is ventricular fibrillation, which warrants immediate cardiopulmonary resuscitation and defibrillation. However, in our case, the exact arrhythmia was unknown and self-limiting.

Acknowledgements

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