

## CASE REPORT

### Commotio Cordis Secondary to Aggression

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#### Case report

A 35-year-old man was admitted following a recovered cardiorespiratory arrest. He was attacked by another man with multiples blunt trauma by a wood stick in the chest and head. An eyewitness statement indicated that the victim had lost consciousness and collapsed after being hit in the chest. He was in cardiac arrest, and a nearby healthcare professional provided first aid with cardiopulmonary resuscitation. The first electrocardiogram (ECG) rhythm strip, fifteen minutes later, identified ventricular fibrillation (VF) (Figure 1A). Sixteen electrical shocks were delivered, always with VF, before restoration of sinus rhythm and circulation. A 12-lead-ECG revealed sinus rhythm without ST deviations and a corrected QT interval of 414 msec. On admission, he was under ventilatory support, with a heart rate of 80 beats per minute, blood pressure of 121/70mmHg, and no signs of shock. There was no previously known medical condition, besides being an active smoker and a binge drinker, and no family history of early coronary disease, cardiomyopathy, or sudden death. He was under the influence of alcohol, as was confirmed by blood tests (blood alcohol level of 1.31g/L), and had a discretely elevated cardiac troponin-T of 0.9ng/ml (normal range, 0 to 0.08 ng/ml). The remaining analyzes, cranial and cervical CT scan, chest X-ray, and abdominal eco FAST were normal. An initial echocardiogram in the emergency department demonstrated normal-sized chambers and global ventricular systolic dysfunction

explained by a prolonged cardiac arrest. He was admitted to the intensive care unit for post-resuscitation care with therapeutic hypothermia and temporary ventilatory support. At 48 hours of admission, there was a normalization of systolic function with a normal ejection fraction by echocardiogram (Figure 2A-B). Coronary angiography excluded coronary disease (Figure 2C-D). Serial electrocardiograms were not suggestive of an arrhythmogenic substrate (figure 1B). The sudden cardiac arrest was assumed secondary to a *commotio cordis* (CC), and for this reason, the patient was not proposed for an implantable cardioverter-defibrillator (ICD). The clinical outcome was favorable, and the patient was discharged. He remained asymptomatic during three years of follow-up, with normal ECG, echocardiogram with strain-rate imaging, exercise stress test, and 24-hour-Holter. During mid-term follow-up, a cardiac magnetic resonance imaging (MRI) showed normal-sized chambers, global and regional function, and no myocardial edema or myocardial scar by late focal hyperenhancement (Figure 2E-F).

#### Discussion

CC is a rare cause of sudden death provoked by a blunt impact to the chest, inducing malignant dysrhythmia in the absence of cardiac damage. The Latin translation means “*agitation of the heart*,” and it appears to be a primary arrhythmic event initiated by mechanical stimulation that predominantly affects young males and frequently in association with sports practice.<sup>1</sup> Our patient was older, and the CC was secondary to aggression. With low recognition, atypical cases, such as those secondary to aggression, are even more challenging to recognize. As described by Mu J et al.,<sup>2</sup> in a witnessed assault, the association between the blow to the chest and the subsequent collapse strongly suggests CC. The circumstances surrounding the event and statements of

#### Keywords

Atrioventricular Block; Cardiovascular Diseases/ complications; Ventricular Fibrillation; Arrhythmias, Cardiac/complications; Heart Arrest; Diagnosis, Differential; Commotio Cordis.

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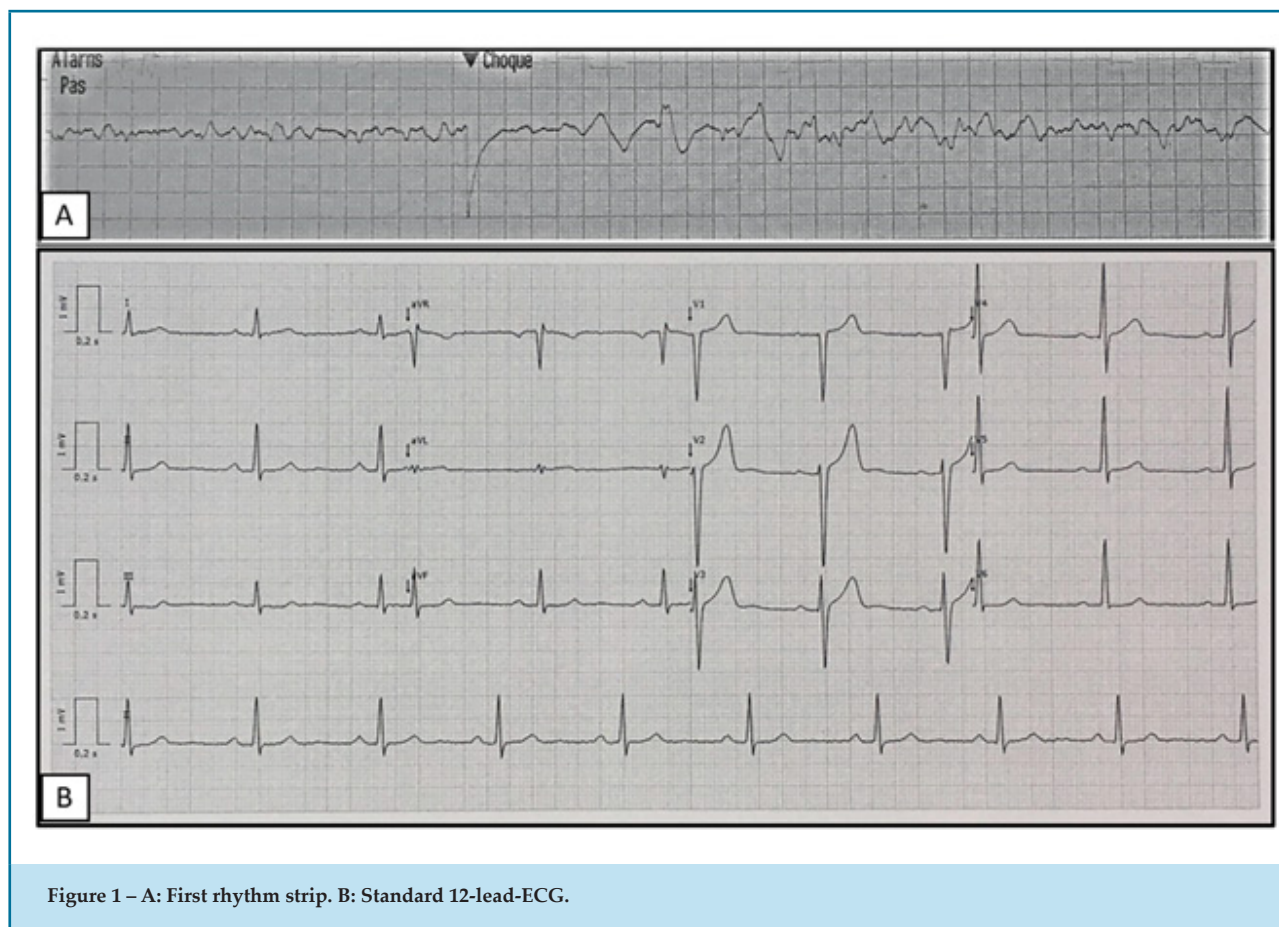


Figure 1 – A: First rhythm strip. B: Standard 12-lead-ECG.

a witness are essential in forensics and have significant implications in the criminal justice system. Very few cases associated with violent attacks have been reported, with estimates of 5% of the total events, and some have even led to homicide charges.<sup>3</sup>

Physiologically, the mechanical energy generated by the impact, within a specific 10-30 millisecond portion of the cardiac cycle - upslope of the T wave - alters the myocardium's electrical stability, by abruptly increasing the left ventricular pressure with a subsequent activation of ion channels and a dispersion of repolarization. In this abnormal setting, following depolarization results in a spiral wave that quickly breaks down into ventricular fibrillation. Time plays a significant role because of the small window of myocardium vulnerability. Still, other variables like speed, location of impact, shape, and hardness of the impact structure are relevant contributors based on animal models.<sup>4</sup> For example, impact energy superior to 50 joules will likely cause structural damage (contusio cordis). It is questionable if there is a component of individual susceptibility that might be modulated

by gender, chest wall pliability, or genetics.<sup>5</sup> The overwhelming male preponderance and victim's young age are easily explained by a preference for sports and an increased chest wall pliability in young individuals.

Alcohol-related arrhythmias, usually atrial fibrillation, have a favorable prognosis if drinking habits are withdrawn. High serum ethanol levels may prolong the QT interval due to changes in ion channels. Together with an increase in sympathetic activation during a situation of aggression, the QT interval can sensitize the myocardium to ventricular arrhythmia. Although our patient had a normal QT interval on the first ECG, alcohol intoxication may have played a role in the initial refractory VF.

The diagnosis is challenging, and other conditions must be ruled out. The differential diagnosis includes: hypertrophic cardiomyopathy, coronary artery abnormalities, arrhythmogenic right ventricular cardiomyopathy, long-QT syndrome, Brugada syndrome, Wolf-Parkinson-White syndrome, dilated cardiomyopathy, Marfan syndrome, aortic valve stenosis, mitral valve prolapse, coronary artery disease, myocarditis, asthma,

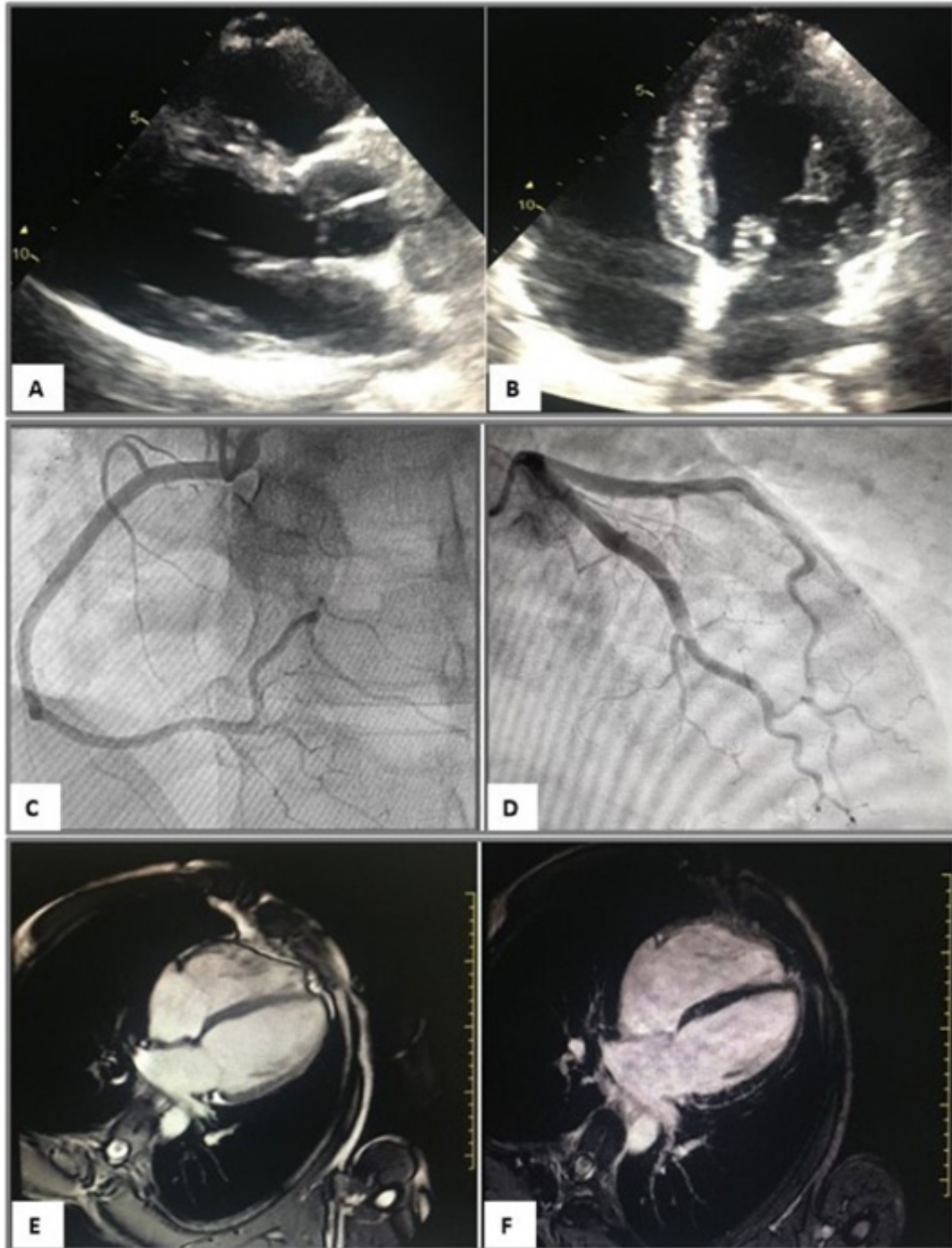


Figure 2 – Transthoracic echocardiogram. A: parasternal long-axis view. B: apical 4-chamber view. Coronary angiography. C: Right coronary artery. D: Left coronary artery. Cardiac magnetic resonance imaging. E: cine imaging (SSFP): 4-chamber view. F: late gadolinium enhancement sequences: 4-chamber view.



heat stroke, drug abuse, and a ruptured cerebral artery. Our patient underwent a complete study that excluded all other causes. Contusio cordis is often due to significant chest trauma, originating a contusion of the myocardial muscle, a cardiac chamber rupture, or heart valve disruption.<sup>6</sup> Presence of delayed enhancement in cardiac MRI may translate into myocardial structural damage (scar).<sup>7</sup> Contusio cordis was dismissed because there was no structural damage on the echocardiogram or scar in cardiac MRI during follow-up.

Increased awareness of this phenomenon in recent decades, especially among those who may be first responders and readily available defibrillators and medical staff on hand on sports grounds, have improved the mortality rate. The survival rate is estimated at 25% if cardiopulmonary resuscitation is initiated within the first three minutes.<sup>8</sup> Resuscitation is often unsuccessful, and early defibrillation is critical.<sup>1,8</sup> Our patient was lucky to be treated immediately and continuously by a nearby health professional until defibrillation was possible. Although the initial refractory VF, he recovered without sequelae. Given this particular case, the authors suggest that CC's victim should be submitted to aggressive resuscitation, keeping in mind that a normal heart structure and function are presumed.

CC survivors should undergo a comprehensive cardiac evaluation, including a 12-lead-ECG, ambulatory Holter monitoring, ECG stress test, echocardiogram, and cardiac catheterization.<sup>9</sup> Electrophysiologic testing and ICD are not generally recommended unless a secondary cause is suspected.<sup>9</sup> About 29% of CC survivors had mild to moderate residual neurologic disability or reduced left ventricular ejection fraction on long-term follow-

up.<sup>10</sup> During the 3-year follow-up, our patient remained asymptomatic with normal complementary exams.

CC is a rare and frequently fatal event. During a witnessed event, prompt initiation of cardiopulmonary resuscitation and defibrillation is essential for survival without sequelae. An atypical scenario of sudden death after chest impact secondary to aggression should raise suspicion of CC.

## Author contributions

Conception and design of the research: Gonçalves ML, Pires MI, Santos JM. Acquisition of data: Gonçalves ML, Santos JM. Analysis and interpretation of the data: Gonçalves ML, Pires MI. Writing of the manuscript: Gonçalves ML. Critical revision of the manuscript for intellectual content: Gonçalves ML, Pires MI, Santos JM, Correia J, Moreira D, Almeida I.

## Potential Conflict of Interest

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## Study Association

This study is not associated with any thesis or dissertation work.

## Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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