Contusio Cordis Associated with Atrioventricular Block and Tricuspid Regurgitation

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Introduction

The expression "*contusio cordis*" defines a lesion of the cardiac myocyte, hemorrhagic or fibrotic, due to the application of violent kinetic force resulting from a blunt trauma to the precordial region[1]. Syncope is almost always the first clinical manifestation observed, and commonly precedes death. *Contusio cordis* differs from *commotio cordis,* the latter representing a cardiac electromechanical disorder consequent to a blunt trauma to the precordial region, at a critical moment of the cardiac cycle, which occurs within 10 to 30 ms from the T-wave peak, a period susceptible to the development of ventricular fibrillation[2-5]. Both forms are fatal most of the time[2,3,6-9]. *Commotio cordis* is a form of ventricular fibrillation, rather than a lesion in cardiomyocytes or adjacent organs[2,3]. It is a rare condition, leading most cases to death[6], and being more prevalent among men, because of its relationship to high-impact sports[3,6]. The accurate incidence of both entities is unknown, because there is no systematic way to register all cases; however, although rare in the literature, they are believed to be more common than reported[3,6]. *Contusio cordis* associated with complete atrioventricular block (CAVB) is an even rarer condition, with few cases reported[7]. The objective of the present study is to report a case of *contusio cordis* associated with CAVB and tricuspid regurgitation (TR).

Case report

The patient is a 37-year-old male with a history of admission to an emergency unit nine years before due to syncope after blunt trauma to his precordial region. That chest trauma was caused by a stone that hit the patient while inside a train at high speed. At the time, he received local care, denied cardiovascular symptoms and previous diseases, and was discharged. However, the patient reported dyspnea one year later and a new syncopal episode. He was then diagnosed with CAVB and submitted to permanent VVI pacemaker implantation. The patient reported being asymptomatic and skipping follow-up and pacemaker review for seven years, until one year ago, when dyspnea, fatigue and syncope resumed. On examination, he had dyspnea, bradycardia, pathological jugular venous distension, cannon A wave, lateral displacement of the apex beat, and heart murmur compatible with TR. Chest teleradiography evidenced heart enlargement. The electrocardiogram (Figure 1) showed CAVB. The echocardiogram (Figure 2) confirmed important TR secondary to rupture of tricuspid *chordae tendineae* and right ventricular dysfunction, allowing free communication between the right atrium and right ventricle, with significant right ventricular enlargement (5.0 cm). The other echocardiographic measures were as follows: left atrium, 3.4 cm; left ventricular systolic and diastolic volumes, 113 mL and 154 mL, respectively. Important systolic dysfunction was evidenced, with ejection fraction of 27%.

Complete failure of the pacemaker generator was detected, motivating its replacement. Heart failure (HF) treatment was optimized, being the patient discharged asymptomatic (NYHA functional class I) on carvedilol (12.5 mg/day), enalapril maleate (10 mg/day), and spironolactone (25 mg/day).

Discussion

Cardiac lesions resulting from chest trauma vary widely as follows: cardiac contusion; free wall rupture; septal rupture; and valvular lesion. Traumatic TR is rare[8,9], and often underestimated, because it is hemodynamically well tolerated and lesions in other organs are more valued.

Commotio cordis is frequently reported in the practice of competitive sports, such as martial fights and soccer, in car accidents, torture, and other high-impact traumas[6,9]. That is an electromechanical disorder of the heart due to a blunt trauma on the precordial region, at a critical point of the cardiac cycle. Usually, when chest trauma occurs during the QRS, a trend towards CAVB exists; however, when chest trauma occurs within the 10 to 30 ms preceding the T wave, ventricular fibrillation usually results.

Only at that particular point in time, a mechanical energy of at least 50J can cause ventricular fibrillation. The small window of vulnerability explains the rarity of *commotio cordis* manifesting as severe arrhythmia. Considering a sinus rhythm of 60 bpm, the cardiac cycle lasts approximately 1000 ms; thus, the likelihood of a trauma occurring at the particular point of ventricular repolarization ranges from 1% to 3%.

Keywords

Myocytes, Cardiac / pathophysiology; Atrioventricular Block; Tricuspid Valve Insufficiency; Commotio Cordis; Ventricular Fibrillation.

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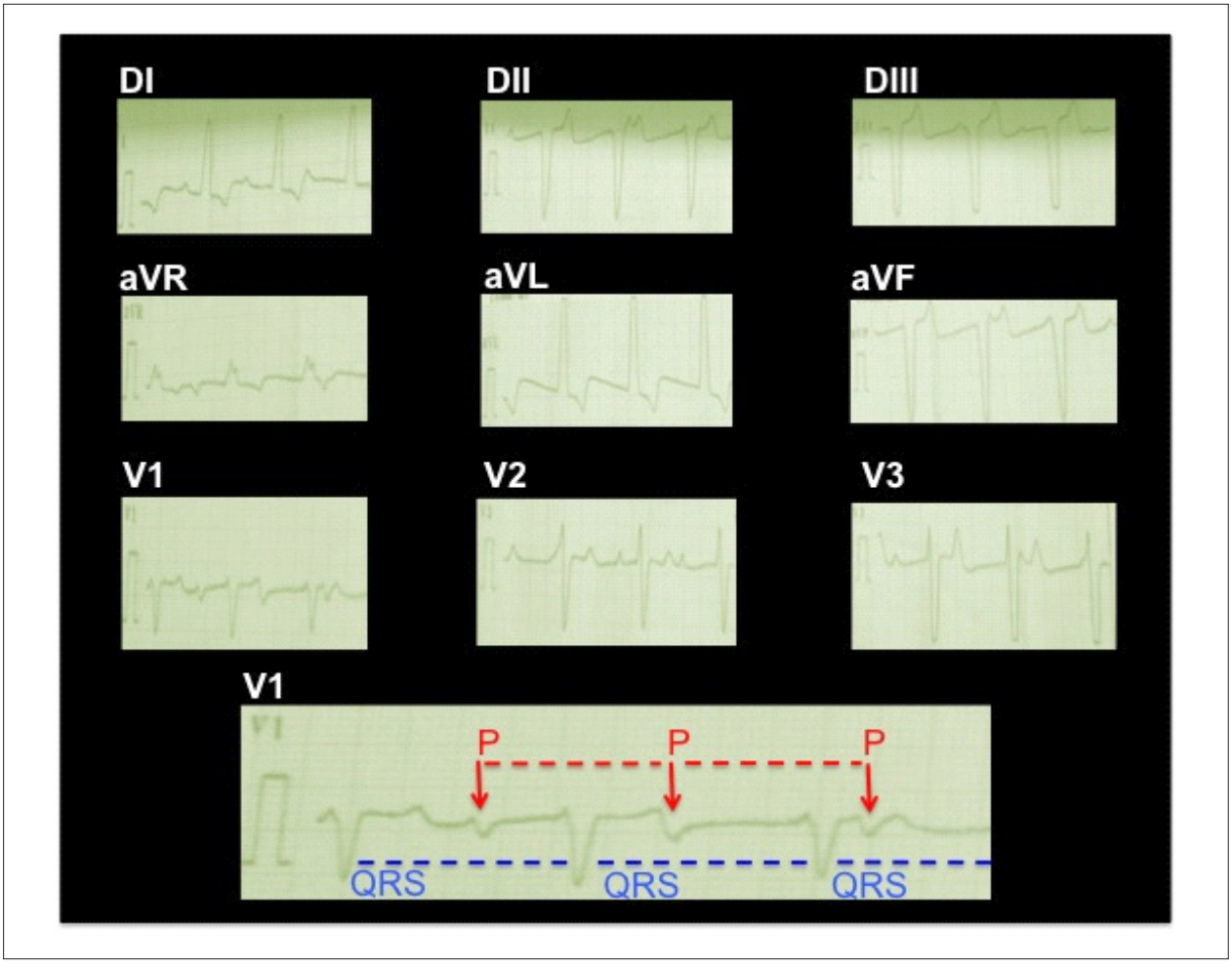

Figure 1 – *Electrocardiogram showing complete atrioventricular block.*

Contusio cordis is a rare condition, being lethal in most cases. It rarely involves only the heart[8]. It is commonly seen in high-impact accidents, such as car accidents, Formula 1 grand prix, in which, despite the existence of cockpit protection, it is unable to absorb all the kinetic energy of a shock at 300 km/h reduced to 0 km/h in a few seconds. The kinetic force usually affects other organs, such as the lungs, trachea, esophagus, and costal arches, leading to pulmonary contusion and costal arch fractures. Thus, *contusio cordis* is more related to lethal cases. However, studies comparing both entities still lack. The situation here reported is unusual, and no similar report has been found in the bases searched. The sum of the force vectors of the train and the stone has contributed to increase the magnitude of the trauma.

The right ventricle and tricuspid valve are most often affected because of their anterior anatomical position in the chest; however, TR is rare, and, when present, is well tolerated[5,9,10]. Usually TR results from the rupture of the papillary muscle or of the anterior cusp[9]. Symptoms can begin immediately after chest trauma or be delayed for years. The diagnosis is usually based on clinical signs and echocardiographic findings. The signs most often present are as follows: pathological jugular venous distension; hepatojugular reflux; holosystolic murmur exacerbated with inspiration; paroxysmal nocturnal dyspnea; hepatomegaly, with palpable and pulsatile liver; lower limb edema; and varicose veins. The deviation of the electrical axis to the right complements the picture. In related case, predominant symptoms and signs of CAVBB - dizziness, wave in the cannon and bradycardia. The electrocardiogram confirmed CAVB and, paradoxically, showed rotation of the electrical axis to the left Right ventricular dysfunction and HF led to drug treatment, which compensated the patient (NYHA functional class I). The need for repair surgery or tricuspid valve change is exceptional.

The most common clinical manifestations of *contusio cordis* are chest pain resulting from local trauma and syncope[8]. The clinical findings might vary, and unspecific symptoms, such as fainting, nauseas and vomiting, can be reported. Clinical assessment, searching for TR, in addition to electrocardiography and echocardiography should be used when damage to the tricuspid valve or conduction system is suspected. In the present case, HF was only detected after decompensation, which might have been

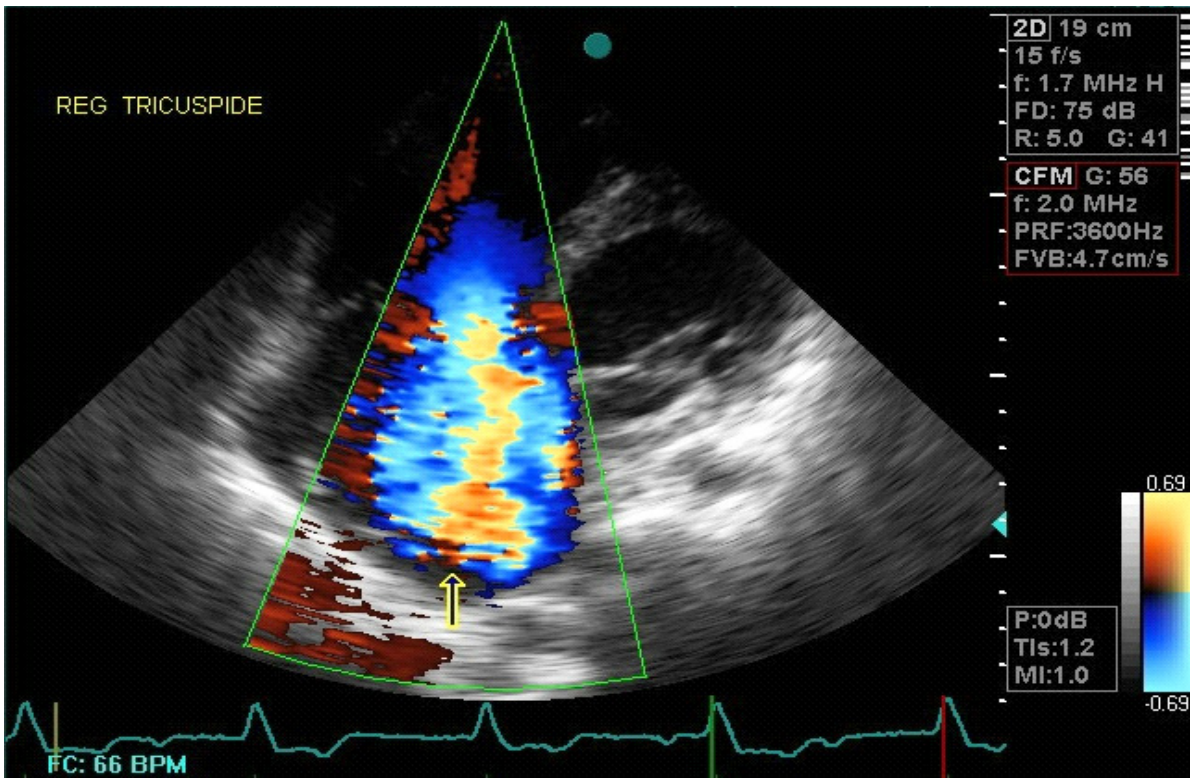

Figure 2 - *Echocardiogram evidencing tricuspid regurgitation.*

aggravated by bradycardia secondary to CAVB. The fact that the patient skipped follow-up for seven years, which means that, having an implanted pacemaker, he should have attended at least two consultations per year, and the aggravation of clinical findings, propitiated the appearance of many classical HF manifestations, which are commonly seen among patients lacking information and access to a health care system.

There are several case reports on cardiac laceration or arrhythmias caused by chest trauma, whose diagnosis is missed on a first inspection. In such cases, some tests, such as baseline electrocardiography, Holter monitoring, echocardiography, and nuclear magnetic resonance imaging, might help the complementary investigation. The early diagnosis can prevent a fatality.

Author contributions

Acquisition of data: Longo Neto GC, Martins WA, Viallacorta H, Silva EN, Haffner PM, Gerardt D; Analysis and interpretation of the data: Longo Neto GC, Martins WA, Viallacorta H, Silva EN, Haffner PM, Gerardt D; Writing of the manuscript: Longo Neto GC and Martins WA; Critical revision of the manuscript for intellectual content: Longo Neto GC, Martins WA, Viallacorta H, Silva EN, Haffner PM, Gerardt D.

Potential Conflict of Interest

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Study Association

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