Myocardial infarction in an individual with Wolff-Parkinson-White syndrome

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Abstract: A case of recent myocardial infarction in a 24-year-old man with no known history of Wolff—Parkinson—White syndrome is reported. The patient developed during sport training an exercise tachycardia associated with palpitation and syncope. The electrocardiogram, performed by rescue team, was suggestive of Wolff-Parkinson-White syndrome. He was admitted to Cardiovascular Diseases Institute for this first attack of rapid heart beating which did not readily subside. He died suddenly after admission, with no response to resuscitation maneuvers and before other investigations were performed. Autopsy examination of the heart revealed no accessory atrioventricular connection, but revealed a recent myocardial infarction. The case underlines the potential danger of the Wolff—Parkinson—White syndrome in patients with no or minimal clinical manifestations. The rapid unexpected death can be attributed to atrial fibrillation with rapid ventricular response via the anomalous connection. A meticulous histological study of the atrioventricular function in hearts of young athletes with unexplained death is a necessity.

Key Words: unexpected death, preexcitation, Wolff-Parkinson-White syndrome.

Acute myocardial infarction (AMI) in a patient with Wolff-Parkinson-White (WPW) syndrome was first reported about fifty years ago and was followed by other reports, some of them describing patients with inferior (posterior) infarction developed in a WPW asymptomatic patient [1-2]. When WPW syndrome occurs in an individual with heart disease, the interpretation of electrocardiogram (ECG) may be difficult. This is because WPW syndrome may mask the underlying electrocardiographic abnormalities or may simulate other cardiac disorders. We report the case of a 24-year-old athlete with AMI and WPW who died unexpectedly, during resuscitation maneuvers. All lesions found at necropsy are described and discussed.

CASE REPORT

We report a recent myocardial infarction associated with WPW syndrome occurring in an athlete during sport training. The team colleagues described the patient having palpitations and exertional substernal chest pain lasting minutes and followed by syncope. ECG made by rescue team revealed deep Q waves in leads II, III, and aVF and delta waves in leads V2 and V3. The EKG findings were consistent with WPW syndrome rather than with myocardial infarction. He died shortly after hospital admission with no response to resuscitation efforts undertaken.

Autopsy revealed a heart normal in shape, with no cavity dilatations. The pericardium, cardiac valves, endocardium, and coronary arteries were normal. Histological exam revealed contraction of apparently normal coronary artery and fractures of the contracted cardiomyocytes (Figure 1 a; b). Foci of cardiomyocyte (CM) fracture in apparently normal CMs were also found. The dominant morphologic lesion was represented by CM death of various types, represented by infarct necrosis and contraction band necrosis (CBN).

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Infarct necrosis, which is apparently the result of sudden nutrient flow reduction, was present in the posteroinferior wall, having a transmural extension. (Figure 1 c). At periphery of infarcted area foci of undulated CMs in a fibrotic interstitium (Figure 1 d) were seen.

Coagulative myocytolysis or CBN (Figure 2 a), usually caused by adrenergic stimulation, had a focal distribution being associated in some areas with inflammatory microfoci (Figure 2 b) and in other areas with large zones of hemorrhagic necrosis (Figure 2 c), representing areas of reperfusion injuries (Figure 2 c).

No sign of colliquative myocytolysis, which is generally linked to catecholamine depletion, and no signs of apoptotic-like lesions were found. Focally, the myocardial interstitium was enlarged by replacement of damaged CMs with fibrous tissue of different histological ages, having either a predominant matrixial component with few undulated fibers, or prominent collagen component corresponding to a chronic cardiac ischemia.

In few areas, this progressive connective tissue repair with a neo-angiogenesis component resulted in a CM architectural disturbance (Figure 2 d).

**DISCUSSIONS**

It is known that WPW may either simulate AMI or mask the electrocardiographic abnormalities of AMI [2].

The first possibility is that of WPW mimicking AMI. WPW was confirmed in our patient by the presence of delta waves in leads V2 and V3. Probably, in addition to activation of the ventricles from the normal bundle branches, this patient presented activation of an accessory posteroseptal bypass tract. This resulted in negative delta waves in the inferior leads, which are morphologically identical to infarction Q waves and are present in 16% of patients with WPW [3].

Confusing the negative delta waves of WPW with a myocardial infarction is a common error. It is not possible to reliably differentiate negative delta waves from myocardial infarction based solely on EKG. If there is doubt whether the inferior Q waves represent infarction or negative delta waves, an echocardiogram may be needed to study inferior wall motion. The sensitivity of an echocardiogram is sufficient to rule out old inferior myocardial infarction if no wall motion abnormalities are seen [4].
If the inferior wall is hypokinetic or akinetic, further testing is indicated to determine myocardium at risk. But, in patients with preexcitation, a regular exercise stress test is not indicated [5], as are sport activities. In our patient, chest pain was most likely caused by exercise tachycardia-induced ischemia. He had an exertional angina consistent with coronary artery spasm.

The second possibility is that WPW may also mask the EKG abnormalities of acute myocardial infarction. It is difficult to recognize acute myocardial infarction in patients with WPW because preexcitation masks the Q waves of transmural infarction [6]. Histologically, in our case the lesions at any level of the coronary system were absent, even in the presence of a myocardial infarction. This silent or almost-silent infarct occurred approximately 10 hours before the terminal episode, not impeding the athlete from habitual, vigorous physical training.

Referring to the relation between myocardial damage and myocell function, Baroldi [7] described 3 types of cell deaths, because the damaged myocardial cell may stop functioning in irreversible relaxation or contraction, or may progressively lose its force and velocity.

The first damage, known as infarct necrosis, which is an atonic cardiomyocyte death in irreversible relaxation, was associated in our study with others lesions. Focally, we saw an area of hemorrhagic infarct necrosis, usually seen after fibrinolytic therapy [8], representing the reperfusion hallmark of ischemic myocardium. Rarely, a myocardial infarct may be hemorrhagic, such as in the case when associated with wall rupture [9], or therapeutic procedures. Another observation concerns “wavy fibers”, which are undulated myocardial fibers representing an early sign of myocardial ischemia. Due to their lack of specificity, they do not allow a diagnosis of ischemia. In practice, wavyness of normal myocells is usually observed around hypercontracted myocardial fibers [10].

Secondly, we revealed contraction band necrosis (CBN) lesions, representing a form of tetanic death in irreversible contraction. This form of myocardial necrosis has a morphofunctional pattern opposite to infarct necrosis. Here, the myocell is unable to relax and its function arrests in hypercontraction because of extreme reduction in sarcomere length.

We observed several different CBN morphologies corresponding to different CBN histological ages.
The lesion was either paradigmal, when limited to sarcomeres adjacent to the disc, or pancellular, with involvement of the entire myocardium. The lesion healed by the replacement of damaged cells with interstitial fibrosis [11]. This injury has also been defined as coagulative myocytolysis. Coagulative is added to the term myocytolysis for emphasizing the coagulation of discal contractile proteins seen in a pancellular vacuolated CMs. Generally, CBN is associated with catecholamine infusion, detected in many other pathologic conditions, such as ischemic heart disease, electric shock, psychological stress, etc. [12]. Indeed, our patient was resuscitated with no success.

Coagulative myocytolysis must be differentiated from colliquative myocytolysis, which defines failing death of myocytes by progressive function loss, which is a reversible lesion unlike CBN [7]. No colliquative myocytolysis lesions were found. No CM apoptosis was found, as well.

Instead, interstitial fibrotic foci were noticed. Spotty fibrotic myocardium raised the question of ischemic lesion age. Are the ischemic events recent or repetitive? We believe that many other previous silent events developed before this last one and are demonstrated by different degrees of fibrotic interstitium. Indeed, localized myocardial fibrosis may be interpreted as a consequence of repetitive acute, nonfatal, events which, associated with chronic ischemia, lead to progressive myocardial fibrosis. The myocardial disarray might be included among the adrenergic-related effects. Its linkage with catecholamine was already demonstrated in the literature [13]. In our experience “pathologic” disarray occurred in conditions of adrenergic stress.

At present, we have few human morphological evidence of adrenergic stress associated with unexpected death. Nevertheless, it is clear that CBN is observed in cases of sudden death [14]. CBN was described in young subjects dying suddenly and unexpectedly with no clinical history of any disease. They had normal coronary arteries; the unique findings were CBN and myofiber breakup. The latter may be also associated with cardiac arrhythmia. Like Gayaz [14], we consider most of these lesions related to adrenergic storm producing acute structural changes in cardiac tissue. Indeed, cardiopulmonary resuscitation per se, including noradrenaline infusion, electrical defibrillation, could explain many found lesions.

Comment
This case is an important reminder that EKG changes caused by WPW syndrome may mask myocardial infarction EKG changes. In conclusion, a more thorough pathologic study with strict clinical case correlation of acute myocardial infarctions and WPW syndrome is needed. In patients with WPW syndrome atrial fibrillation with a very rapid ventricular rate may be life threatening. Early recognition and correct treatment allows rapid restoration of normal sinus rhythm and may decrease morbidity and mortality.

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