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LETTER TO THE EDITOR

The revascularization of the atrio-ventricular node artery in a case of right coronary artery occlusion promptly discontinue the atrio-ventricular block

KEYWORDS
ST elevation;
Atrio-ventricular arrhythmias;
Primary percutaneous transluminal coronary angioplasty

1. Introduction

As it is known, ischemia of the specialized myocardial excito-conducting tissue is one of the hypothesized mechanisms in atrio-ventricular (A-V) arrhythmias; unfortunately, in the current context of interventional cardiology, which is mainly focused on ablation and/or pacing, the ischemic hypothesis is scarcely taken into consideration. To discuss this hypothesis, which has a solid pathophysiological basis, we report here a case of ST elevation myocardial infarction (STEMI) with A-V block at onset, in which the revascularization of the A-V artery originating from an occluded right coronary artery (RCA) promptly restores a physiological A-V conduction.

2. Case report

A 44-year-old man was admitted to our hospital for sudden outbreak of chest pain irradiating to the left arm and associated diaphoresis following an emotional stressor. He was reported to be an active smoker (20 packs/year), be previously healthy, have a positive family history for coronary artery disease and be affected by grade I arterial hypertension that was not under pharmacological therapy. He was not taking any medications and had no known allergies. On admission, his blood pressure was 140/70 mmHg, his heart rate was 57 beats per minute, and there were no pathological findings at physical examination; the standard 12-lead ECG showed a sinus rhythm at a ventricular rate of 60 bpm, 2:1 atrioventricular block and ST segment elevation of 0.4 mV measured at the J point in the inferior and lateral leads with reciprocal ST segment depression in lead I, aVL and from V1 to V3, supporting the diagnosis of acute STEMI (Fig 1, top panel, left). He immediately underwent coronary angiography (CA) for primary percutaneous transluminal coronary angioplasty (PTCA) within 90 minutes of onset of symptoms. The antiplatelet and anticoagulant therapies were initiated. The CA demonstrated an occlusion of the RCA at the proximal tract, which was considered the culprit lesion, and was treated with PTCA followed by the implantation of one drug-eluting stent. A 75% stenosis of a small (< 1 mm) diagonal branch and a 50% stenosis of the left circumflex artery were also reported but not treated. The procedure was free of complications, and the flow on the right coronary artery was completely restored (TIMI 3 flow). After revascularization of the RCA, the A-V artery was well detectable and originated from the distal portion of the RCA, as shown in Fig 1 (bottom panels). Immediately after the procedure, in addition to the disappearance of chest pain and reduction of the ST segment elevation, the A-V block resolved without any additional intervention (Fig 1, top panel, right). During the following days, marked elevations of troponine T (up to 6323 pg/ml) and creatine phosphokinase were observed, as expected following a successful reperfusion of the myocardium. The ECG evolved with no more rhythm disturbances. Five days after admission, the patient was discharged without further complications.

3. Discussion

Ischemia is the key pathophysiological mechanism in diseases of complex organisms provided with a circulatory

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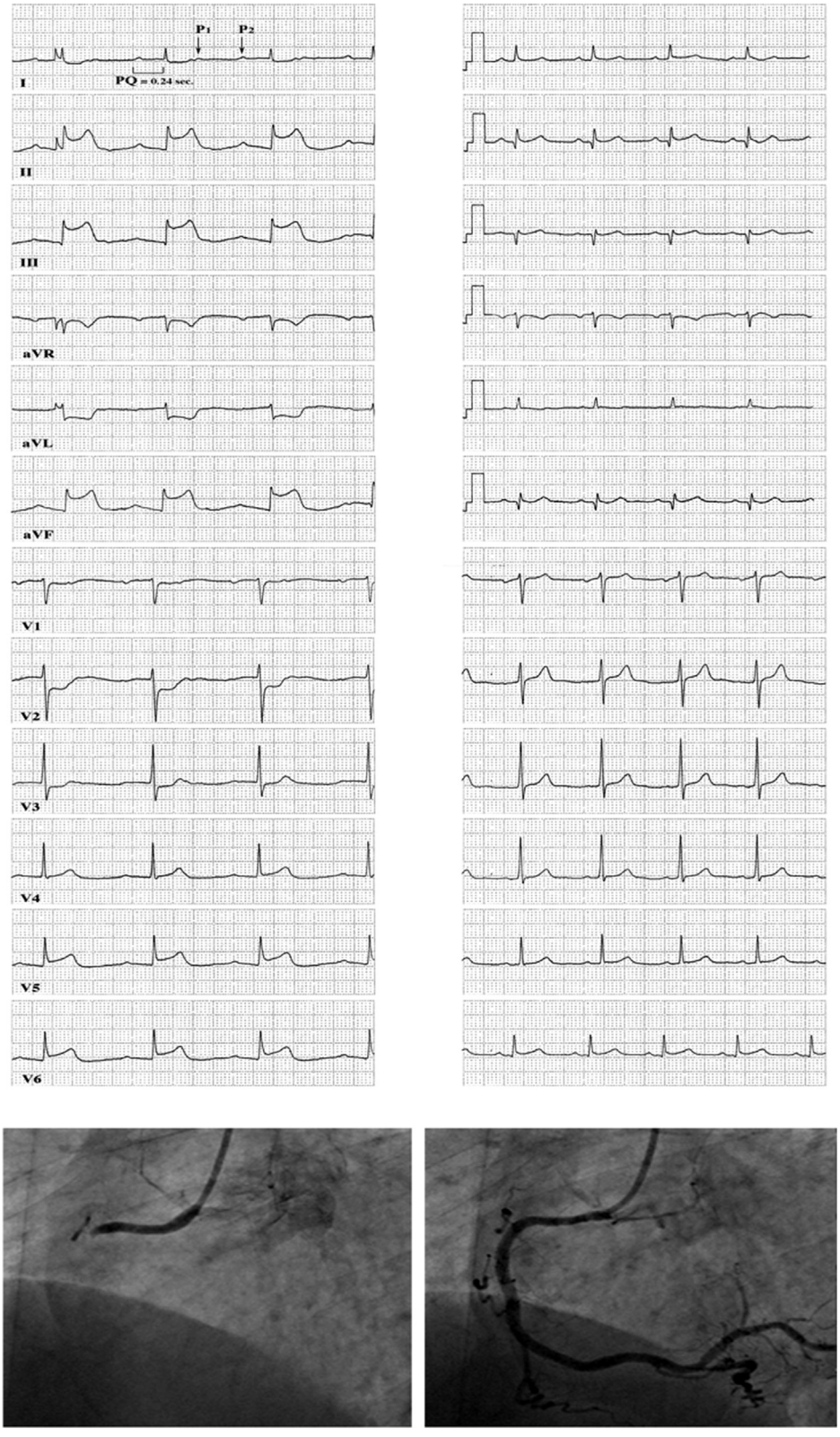

Figure 1 **BEFORE PTCA, top panel, left**: Original 12-lead ECG tracing obtained at admission showing a sinus rhythm with a ventricular rate of 60 bpm conducted with a 2:1 A-V block and an ST elevation in the inferior and lateral leads with a reciprocal ST segment depression in lead I, aVL and from V1 to V3. Arrows indicates evident P waves. (1 mm = 1 mV; paper speed 25 mm/s). **AFTER PTCA, top panel, right**: Original 12-lead ECG tracing obtained immediately after the PTCA showing a regular A-V conduction (PQ 120 ms) and the regression of the acute ischemic alterations. A Q wave is present in the inferior leads. **Bottom panel, left**: Original coronary angiography frame showing the RCA occluded at the proximal tract. No A-V branches can be seen. The asterisk indicates the tip of the catheter = 2 mm. **Bottom panel, right**: Panel B: Original coronary angiography frame showing the RCA after successful revascularization. The arrow indicates the A-V artery.

system and dependent on a predominantly aerobic metabolism. Not surprisingly, ischemia is foreseen by organisms with blood circulation since it is able to act as a powerful stimulus capable of activating complex adaptive responses. In agreement with this statement, since specialized myocardial excitoconducting tissue is also highly vascularized and subject to the same metabolic and energy caveat, there is no reason to believe that the pathogenesis of arrhythmias is not to be understood within adaptation to acute and chronic ischemia. In our case, the prompt resolution of the arrhythmia, concurrently with the angiographic demonstration of the A-V node artery reperfusion and the persistent recovery of A-V conduction in the follow-up (1 month), allows us to support a cause-effect relationship between the A-V node ischemia and arrhythmia. In the literature, the distribution of lesions of the coronary tree that correlate with supraventricular arrhythmias are well documented,[1,2] with most of the studies linking the onset of new arrhythmias with acute ischemia[3]; only a few of them investigate the linkage with the complete or with other A-V blocks.[4] In our case, the prompt percutaneous revascularization of the culprit coronary artery from which the artery of the A-V node originated was decisive for interruption of the arrhythmia and possibly avoided the implantation of a permanent pacemaker, which is what frequently occurs after A-V blocks. The exact time-window during which revascularization will restore the conduction system is not known, but it depends on the ischemic tolerance; in this view, percutaneous angioplasty seems to be more timely than bypass grafting to interrupt an acute A-V block.[5,6] In conclusion, in the presence of the recent onset of A-V arrhythmias, we suggest that the clinical cardiologist should always consider the ischemic hypothesis and the interventional cardiologist should evaluate the patency of the vessels that supply the specialized myocardial excito-conducting tissue.

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