A Pathognomonic Case of Isolated Acute Right Ventricular Infarction

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Introduction
Coronary artery atherosclerosis represents the leading cause of disability and death among cardiovascular (CV) diseases [1]. Both the coronary anatomy and the different characteristics of left and right ventricles explain the different prevalence of left and right isolated myocardial infarction. The lower right ventricular myocardial oxygen demand compared to the left ventricle due to the smaller muscle mass of the right one, the coronary perfusion of the right ventricle (RV) occurring both in systole and diastole and the presence of more extensive collateral flow from the left to the right coronary arteries are responsible of the less occurrence of right ventricular impairment in the context of acute myocardial infarction, which can be found in association with the inferior wall infarction of the left ventricle in up to the 50% of cases and rarely with the anterior wall ischemia of the left ventricle [2]. On the other hand, isolated RV infarction occurred only in about 3% of the overall myocardial ischemic episodes [3].

A 72-year-old patient suffering from arterial hypertension went to the emergency room of our hospital for malaise and asthenia for about 12 hours that have increased in 3-4 hours before the arrival.

He was sweating, tachypneic with normal value of the oxygen saturation to the finger (SpO2:96%), with arterial hypotension (pressure values = 85/50 mmHg), turgor of the jugular veins in the absence of peripheral edema. Cardiac examination revealed no pathological noises and lung examination revealed clear lung fields. Electrocardiogram showed sinus tachycardia at a rate of 118 bpm, second degree atrioventricular (AV) block 2:1 type and an AV conduction with a first-degree AV block (P-Q interval: 320-330 msec). A pulmonary P wave was present and the QRS complex showed predominant R waves in V2-V3 with slight repolarization abnormalities (figure, panel A). On the other hand, an ST-elevation in right precordial leads, V3R and V4R was evident. (figure, panel B).

Echocardiographic evaluation showed a hypertrophic left ventricle with normal volume and function, with marked enlarged right sections (figure, panels C-H). Moreover, we can observe the flattening of the interventricular septum with a D shaped left ventricle. Septal flattening occurred only in diastole, expression of RV volume overload (figure, panels F-H) [3].

After taking 180 mg of ticagrelor administrated orally and 150 mg of aspirin intravenously, the patient was immediately transferred to the catheterization laboratory for emergency coronary angiography. A bolus of total of 5000 units unfractionated heparin and abciximab 0.25 mg/kg bolus intravenously were infused during the procedure.
Coronary angiography highlighting an ostial thrombotic obstruction of a co-dominant right coronary artery (RCA) in the absence of significant stenosis of the left coronary circulation (figure, panels I-M).

RCA was cannulated using a Judkins Right 4 guidewire from the right radial artery. After overcoming of the total occlusion with a Whisper ES guidewire (Abbott Vascular, Chicago, Illinois, United States), we performed multiple dilations of ostial and proximal portions of the vessel using 2.0 x 20 mm and 2.5 x 20 mm balloons (Boston Scientific, Marlborough, Massachusetts, United States). Primary percutaneous coronary intervention of RCA was performed by implanting an everolimus eluting coronary stent, Xience Alpine 3.0 x 12 mm (Abbott Vascular, Chicago, Illinois, United States) at 16 atm pressure at the level of the ostial portion of the artery, with post-dilations in the proximal segment at 20 atm pressure and a second Xience Alpine 2.75 x 23 mm (Abbott Vascular, Chicago, Illinois, United States) at 18 atm pressure in the middle portion of the artery with a good postoperative result (figure, panels L).

During the hospital stay, the patient showed a good clinical status without any CV events, with progressively normalization of electrocardiographic abnormalities and persistent sinus rhythm. The patient underwent Normosol-R injection (1.5 liters / day) with blood pressure monitoring, until to normalization of arterial pressure levels. The echocardiographic control shows normal left ventricular ejection fraction, mild RV dilation, and no pericardial effusion after stent implantation.

RV infarction more frequently occurs in association with the postero-inferior transmural ischemia of the left ventricle due to the proximal occlusion of a dominant RCA. In presence of a coronary left dominance, an occlusion of the left circumflex artery may be responsible. Rarely, the occlusion of the left anterior descending artery may determine ischemic RV involvement [4]. Isolated RV infarction is a very rare entity accounting for less than 3% of all myocardial infarctions [2].

In our case, the occlusion of a proximal codominant RCA, with patent collateral vessels protecting the left ventricle coming from the circumflex one, explain the involvement of the RV alone in the ischemic process (figure, panels L-M) [5]. Moreover, the origin of the AV nodal branch from the first portion of the RCA explains the occurrence of atrio-ventricular node conduction disturbances (figure, panel L) [6].

The clinical presentation is often insidious, as chest pain often misses while the triad of hypotension, clear lung fields and elevated jugular venous pressure, traditionally considered as characteristic, has high specificity (96%) but very low sensitivity (25%) [7]. The electrocardiographic pattern characteristic is an ST-segment elevation of >1 mm in the lead V4R, closely related with RV dysfunction [8].

Echocardiography is useful for the comprehensive evaluation of the RV. It can highlight the presence of RV dilation and dyskinesia (the presence of hypokinetic and akinetic segments), the dilation of tricuspid anulus with secondary tricuspid regurgitation and the evidence of right atrial and inferior caval vein dilation, expression of volume overload. [9]. Moreover, through continuous wave Doppler ultrasound, it is possible to non-invasively estimate the systolic pulmonary artery pressure [9].

However, echocardiographic imaging of the RV has technical challenges: the complex shape of the RV together with the mutual interactions with the left one, make it sometimes difficult to completely visualize the right chambers of the heart. Therefore, all available acoustic windows need to be assessed for the complete evaluation of the RV.

The revascularization strategy is the first line therapy in RV infarction, the earliest is the reperfusion of the occluded vessel, the more likely is the recovery of the function of the RV [10].

About prognosis, the RV involvement determines an increased risk of death during hospitalization in subjects with myocardial
infarction [11]. This negative prognosis is related to the major incidence of refractory cardiogenic shock: the impairment of RV is associated with a reduction of left ventricular preload, resulting in reduced systolic output of the left ventricle and systemic hypo perfusion [8].

On the contrary, patients with RV infarction who survive the acute phase have a good long-term prognosis [12].

Declarations of Interest
The authors declare no conflict of interest

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