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Cardiac death by rupture of the right ventricular wall and hemopericardium

Case Report

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Abstract: Right Ventricular (RV) rupture is a rare and dangerous complication of acute myocardial infarction. There are limited reports on RV free wall rupture. In this paper we describe the outcome of an autopsy case of cardiac death by rupture of the free wall of the right ventricle in a 51-year-old man with coronary artery disease and hypertrophic-sclerotic cardiomyopathy.

Keywords: *Cardiac death • Right-ventricular rupture • Hemopericardium*

1. Introduction

Sudden Cardiac Death (SCD) is a sudden and unexpected death due to cardiac etiology that occurs instantly and with absence of apparen symptoms. It can also occur within 1 hour after the onset of acute symptoms or a rapid worsening of clinical conditions, also in the individuals without any potentially known fatal pathologies [1]. Coronary artery disease is the most frequent cause [2,3] and among the mechanical complications of myocardial infarction, by far the most serious one is the ventricular free wall rupture [4].

Free wall rupture occurs in approximately 5% (according to different Authors 4-8%) [5], over 10% in patients who died in the hospital by STEMI [6]. On the other hand, the right ventricular rupture is described as a rare complication after myocardial infarction [7] and only a few cases were reported [8].

The literature describes cases of right ventricular rupture from myocardial infarction [9], accidental [10] or therapeutic [11] mechanical trauma, myocarditis [12] and mediastinitis [13], cardiac [14] and extracardiac [15] surgical complications, and other pathologies [16].

The consequent cardiac tamponade is a life-threatening and time-critical emergency, which often results with death of the patient.

We present the autoptical outcome of a 51-year-old man with coronary artery disease and hypertrophic-sclerotic cardiomyopathy, who died due to rupture of the right ventricular wall and hemopericardium.

2. Case report

A 51-year-old man, 173 cm tall and weighing approximately 80 kg with good general health conditions and good normal constitution was admited to the CCU. He had not known diseases and adipose tissue was with a regular distribution and normal muscle tropism. Skull was without appreciable preternatural motility of the bone surface. Patches of ECG recordings on the chest showed no preternatural motility of the bone surface but slightly globular abdomen for fat.

The examination of the thanatological phenomena showed the following findings: Hypostasis abundantly represented in dorsal surface of the body and up to the

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anterior axillary line. Stiffness present and valid at all joints. There were no putrefactive phenomena.

After dissecting the soft tissue, there was full integrity of the rib cage, diaphragm and bowel loops with the dry abdominal cavity.

Upon removal of the sternal plastron, the lungs were expanded, coating the heart, without adhesions in the pleural cables. The Pericardium was intact and in the opening there was about 300ml of blood, partially coagulated (Figure 1).

Heart was of a globular form (530g weight, 14 cm longitudinal diameter, 13 cm transverse diameter, 5 cm anteroposterior diameter) at its appearance, and

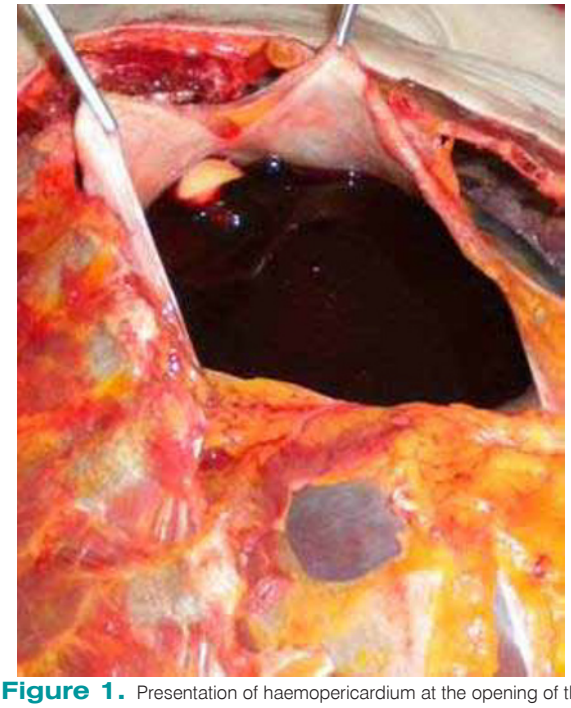

Figure 1. Presentation of haemopericardium at the opening of the pericardial cable.

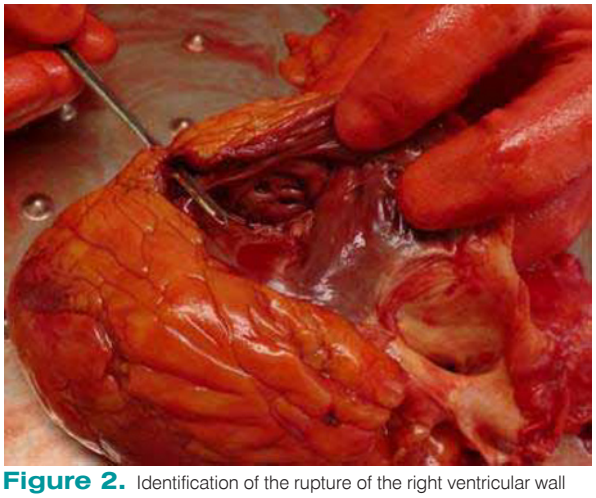

Figure 2. Identification of the rupture of the right ventricular wall

continuous solution of the full thickness of the free wall was in the right ventricle (Figure 2).

The lumen of the right and left coronary were stenotic with the calcified walls. Apical wall of the left ventricle was approximately 2,1 cm, thick while the free wall was 2.4 cm, and the interventricular septum wall was 1.9 cm thick. The thickness of the free wall of the right ventricle was 0.5 cm.

In the longitudinal section of the rest of the heart, the myocardium had marbled features due to the presence of whitish areas alternating with healthy myocardium (widespread sclerotic cardiomyopathy).

Histological examination of the left ventricle confirmed the moderate/severe level of hypertrophy of the left ventricular with increased cell size, squat appearance, and increased volumes of hyperchromatic cell nuclei.

In the area of the right ventricular rupture there was vacuolar degeneration of myocytes, aggregated platelets and a slight fibrin infiltration.

Lungs had increased dimensions and consistency (right lung weight 600g - left 420g) and were crackling. The large, medium and the small bronchi were with mucosal hyperemia and slightly raised in folds. To the full-thickness section of lungs parenchyma was congested, a finding corroborated by squeezing with frothy bleeding.

3. Discussion

The concentric left ventricular hypertrophy caused a systolic overload of the left ventricle resulting in an increase of the volumetric size of the heart and simultaneously in a reduction of the amount of blood received into the ventricular cavity [17].

Stasis of blood in the pulmonary circulation caused the edema and congestion of the pulmonary parenchyma observed at autopsy.

The pulmonary congestion was due to heart failure and not due to cessation of circulation in the central nervous system because the pulmonary congestion would be only in the posterior pulmonary face (visceral hypostasis).

Also, the literature describes pulmonary congestion in the case of acute heart failure [18] and the development of pulmonary congestion during acute myocardial infarction significantly increases the cardiac risk of mortality derived from the left ventricular ejection fraction, with a marked mortality effect in patients with severe pulmonary congestion [19].

However, in our case the pulmonary congestion was not due to cardiac tamponade. The pulmonary

congestion, which contributed to the reduced respiratory function, was due to a chronic heart failure in a patient with ventricular hypertrophy.

The functional overload of the right heart associated with coronary artery disease caused the rupture of the heart and the resulting hemopericardium.

Upon myocardial infarction the necrotic scar tissue undergoes remodeling and with the action of the pressure; increase in the right ventricular filling pressure is a peculiar hemodynamic characteristic in patients with right infarction [20]) that over time results in the formation of a ventricular aneurysm [21-23], which may break and determine a haemopericardium [24].

Yet, the aneurysm is described as a late complication of the infarct [25].

The progression of coronary artery disease in our case, associated with a pressure overload on the right heart, this did not allow to the right ventricle to reshape into the aneurysm, but rapidly led to the rupture of the wall.

Therefore the thinness of the wall, the marked necrosis of the terminal area of the artery, the shortage of the collateral circulation, the effect of delamination of the necrotic part by the muscle contraction and the aging of the myocardium, are all implicated as local factors that lead to rupture [26-31].

The reduced coronary blood flow in patients with cardiac tamponade [32] contributed in compromising the action of the cardiac pump and to the final impairment of cardiac function.

The coagulation of blood collected in the pericardial cavity is indicative of the phenomenon of life remaining (clotting), that exists in the perimortem period and gives the appearance of so-called “flat bread” at the opening of the pericardium.

4. Conclusion

According to the findings of the postmortem examination, death might be related to the circulatory and respiratory failure in a patient with right ventricular wall rupture and hemopericardium.

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