CARDIAC DEATH IN AORTIC VALVE SCLEROSIS AND CORONARY ARTERY DISEASE. AN AUTO-
PSY REPORT

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ABSTRACT

The Calcific Aortic Valve Disease (CAVD), already in early stage of Aortic Valve Sclerosis (AVS), may be associated with a major cardiac event. The authors present a report of cardiac death by AVS explaining the possible pathogenetic mechanism involved in exitus. In the literature several studies reports that, already in the early stage of the disease, a reduction of ventricular function could be enlightened. When the condition persists over time, the resulting concentric ventricular hypertrophy determines increase in perfusion requirement, and, if it is associated with a Coronary Artery Disease (CAD), the myocardium undergoes a process of sclerosis that further depresses the function of cardiac pump. The reduced cardiac effectiveness leads to an overload of the lungs with edema and congestion in the terminal phase that characterize the cardiac and respiratory failure.

Key words: aortic valve sclerosis, left ventricular hypertrophy, coronary artery disease.

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Introduction

The Calcific Aortic Valve Disease (CAVD) is characterized by a progressive increase in the levels of fibrosis and calcification of the aortic valve leaflets. The etiology, according to the most recent references, could be attributable to disorders of mineral metabolism as: abnormalities of phosphate metabolism in kidney disease, hyperparathyroidism and vitamin D receptor polymorphisms1-6. The initial stage is the Aortic Valve Sclerosis (AVS) in which the changes that occur, such as thickening of the leaflets or microcalcification deposits, do not preclude the outflow of the left ventricle7.

However, many studies have shown that patients with AVS already have a degree of left ventricular function depression if compared to those without AVS8-10. Also the atherogenic risk is greater in patients with AVS8, 11-13, but this association is widely discussed. Both diseases have possible common etiologies, such as diabetes, hypertension, smoking, dyslipidaemia10, 14-17, and also some pathological aspects are similar18. Thus, even in the initial stage, AVS, especially when associated with Coronary Artery Disease (CAD), leads to a significant increase in the risk of myocardial infarction or cardiovascular death10, 11, 16, 19.

In this manuscript we describe an autopsy outcome of cardiac death by AVS and CAD explaining the possible mechanism of cardiac and respiratory failure.

Case report

The case concerns a 74-year-old man, with the length of 170 cm and weighing approximately 85 kg. Good were the general hygiene conditions and normal were the constitution. The distribution of adipose tissue were regular and normal were the muscle tropism.
After the examination of the clothes the examination of the thanatological phenomena showed hypostasis abundantly represented in dorsal surface of the body and up to the anterior axillary line and face, the stiffness resolved and present only at legs, and there were no putrefactive phenomena.

We then continued with the autopsy. By the dissection of the soft parts we founded the integrity of the rib cage, diaphragm and bowel loops with the abdominal cavity dry.

Removed the sternal plastron lungs were expanded, without adhesions, with the exception of the left pleural cavity with adherence on the lateral surface of lung.

Pericardium was intact and to the opening of which were found few cc of serous fluid.

After removal from the pericardial cavity heart had a globular form (450 g weight, 10.5 cm longitudinal diameter, 12 cm transverse diameter, 4.5 cm anteroposterior diameter). To the section of the cardiac apex there was an evident whitish discolored area extending along the entire circumference of the section (see figure 1).

The thickness of the apical and free wall of the left ventricle were approximately 2.5 cm. The thickness of the interventricular septum were 2 cm.

To the opening of the outflow way of the left ventricular there was opacification of the endocardium and severe stiffness of the aortic annulus with calcific deposits in the context of the semilunars and fusion and shortening of the chordae (see figure 2).

By the inspection of the coronary we found calcification wall and stenotic lumen, accentuated in the lower third of the right coronary artery, the lower third of the anterior descending coronary artery and the middle third of the circumflex coronary.

We then proceeded to the evisceration of both lungs that were with increased dimensions and consistency (weight 660 gr right and 520 gr left) and crackling to palpation. Large, medium and small bronchi had mucosal hyperaemic and to the full-thickness section of lungs parenchyma was congested, a finding corroborated by squeezing with frothy bleeding.

Figure 1: Appearance of the myocardium to the apical transverse section.

Figure 2: Left ventricular with aortic sclerosis.

Discussion

The ventricular hypertrophy can be consequent to the systolic overload of the chamber (concentric hypertrophy) and it manifest with an increase in the thickness of the ventricular walls, or to an diastolic overload (eccentric hypertrophy) characterized by an increase of the volume of the ventricular chamber and reduction of the thicknesses\(^20\).

In our case it was evident the increase of thickness of the left ventricular wall resulting to AVS that, with an increase in systolic pressure, has resulted in a functional overload of the left ventricle and, therefore, in a concentric hypertrophy with important increase of the thickness of the walls and reduction of the volume.

In the literature AVS is associated with left ventricular hypertrophy by an endothelial dysfunction, which may provide stimuli for development of left ventricular hypertrophy independent of afterload\(^21\).

The increase in oxygen demand not satisfied by a severe CAD resulted in a severe ischemia of the heart, inevitably degenerated in myocardial sclerosis.

The discoloured area appreciable to the cross-sectional area of heart (Fig. 1) gives evidence of a three-vessel perfusion suffering. In fact, the regional infarction (which extends along the entire circumference) is distinguished from lacunar infarction (isolated) where the discoloration is located.

The reduced blood flow, highlighted by the presence of plaques in the coronary arteries, has led
to an insufficient blood supply to the myocardium\(^{(22)}\). The CAD characterized by the accumulation of calcified plaques in the anterior descending, circumflex and right coronary arteries, has induced the activation of immune cells, cytokines and chemokines with fibroblast proliferation\(^{(23,24)}\). Hypoperfused myocardial areas are going to meet coagulative necrosis, resulting in inflammatory reaction, removal of necrotic myocytes by macrophages, and granulation tissue genesis, followed by degenerative changes of the fibres and proliferation of the interstitial connective tissue, for the activation of fibroblasts’ growth factors, such as TGF (tissue growth factor)\(^{(25)}\). Then the CAD caused the myocardial sclerosis, through the activation of fibroblasts and the replacement of myocardial muscle fibres with fibrous connective tissue\(^{(26)}\).

The reduced myocardial perfusion, the reduced supply of oxygen to the hypertrophic heart muscle, resulted in inadequate pumping action of the heart, with volume overload and pressure increase in ventricle\(^{(27)}\).

Edema and pulmonary congestion are associated with hypertrophic increase of the size of the left ventricle\(^{(28)}\), and, therefore, with the reduction in the volume of the ventricular chamber, as supported by Hagger et al.\(^{(29,30)}\) (Ventricular Mass Index [VMI] correlates strongly with mean Pulmonary Artery Pressure-[mPAP]).

The appearance of sclerosis and the coexistence of edema and pulmonary congestion suggest that the mechanism of heart failure has developed in a progressive manner and not acute.

**Conclusion**

According to the findings of postmortem examination, death may be related to chronic cardiac and respiratory failure by left ventricular hypertrophy caused by AVS and not supported by CAD.

**References**


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