Autopsy Findings in Ventricular Wall Rupture After Mitral Valve Replacement Case Report

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Abstract: Ventricular wall rupture after mitral valve replacement is an uncommon complication but often lethal. We report the autopsy findings in a case of a 77 year old woman suffering double mitral disease who underwent unsuccessful replacement of the mitral valve using a Saint Jude (ONXM 27/29) mechanical prosthesis. The patient developed consumptive coagulopathy and a type I left ventricular rupture. Her family filed a claim before a court pressing charges against the surgeon. A legal autopsy was ordered and performed. After our forensic report the family dropped the charges.

Keywords: Mitral valve replacement, ventricular wall rupture, surgery complications.

CASE REPORT

A 77 years-old-woman underwent elective mitral replacement of double mitral disease. Her past medical history included arterial hypertension, gastric ulcer, varicose vein surgery, heart failure with bilateral pleural effusion, pericardial effusion and symptomatic mitral disease. Preoperative echocardiography showed severe stenosis and moderate mitral insufficiency. At the time of surgery she was treated with digitalis, diuretics and anticoagulants and was classified as New York Heart Association (NYHA) class III.

During elective valve replacement surgery the calcified valve was excised and a Saint Jude mechanical prosthesis (ONXM 27/29) was implanted in the usual fashion. There were no reported complications during the procedure.

Shortly after discontinuation of cardiopulmonary bypass, massive bleeding occurred and extracorporeal circulation was reinstituted. She showed signs of cardiac and hypovolemic shock with consumptive coagulopathy (1.600 mL in 4 hours from the chest drainage tubes) and received aggressive blood replacement therapy and inotropics. Cardiac monitoring showed sinus rhythm alternating with nodal rhythm (50 beats per minute). Respiratory parameters showed a Tidal Volume (TV) of 450 ml and FiO_2 (0.8), PEEP (Positive end expiratory pressure) 6, Respiration rate 12 rpm. The diagnoses were cardiogenic and hypovolemic shock. Her family was informed about the poor prognosis and they did not allow emergency reintervention. The patient died 4 hours after the end of the elective surgery. Her family filed a claim before a court pressing charges against the surgeon and a legal autopsy was ordered and performed.

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AUTOPSY FINDINGS

Height of 154 cm, weight of 67 kg, and a body surface area of 22.9 Kg/ m^2. Skin and mucosa paleness. Recent surgical median sternotomy. Right hemothorax 250 cc and left 500cc. Pericardial hematic effusion (100 cc). The heart (weight 360 g) showed recent surgical changes: implanted pacing lead, mitral valve prosthesis (Saint Jude) implanted in the usual fashion, surgical sutures without complications and no signs of mitral regurgitation. There was a 9 mm tear under and parallel to the atrioventricular groove with haemorrhage in mitral valve leaflets dissecting adjacent coronary tissues and extending to pericardial that was compressing left coronary arteries. Arterial atherosclerosis (Fig. **1**).

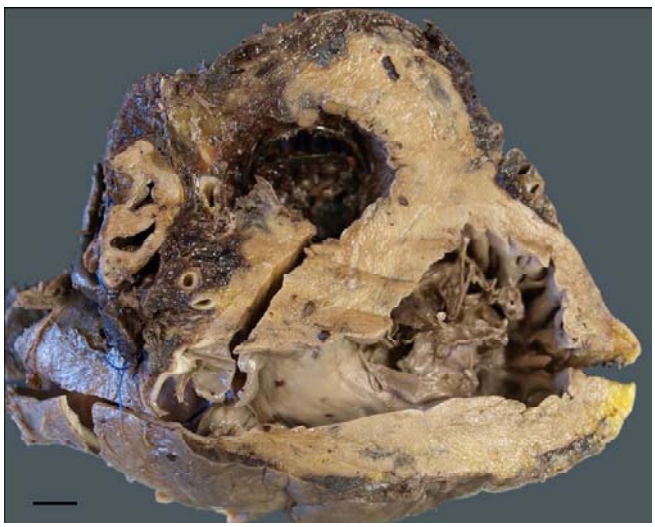

Fig. (1). Zone of dehiscence in left ventricular free wall.

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We describe the presence of erithrocyte infiltration and fibrin inside the tear at the atrio-ventricular groove, spreading through the mitral valve leaflets and extending and compressing the left coronary artery. Besides, we can see some coagulation necrosis with polimorfonuclear infiltration, next to the atroventricular groove, reaching the dislacerated epicardium. We appreciate some autolytic changes as well. The proximal site of both main coronary arteries show atherosclerotic plaques with stenosis about 50% of intraluminal obstruction, with no evidence of acute complications related. The rest of the myocardial tissue shows perivascular and interstitial mild to moderate fibrosis and there weren't any signs of acute myocardial isquemia. Nevertheless we describe some early changes of myocite hypertrophy with slightly increased cytoplasm and hypercromatic nuclei (Fig. **2**).

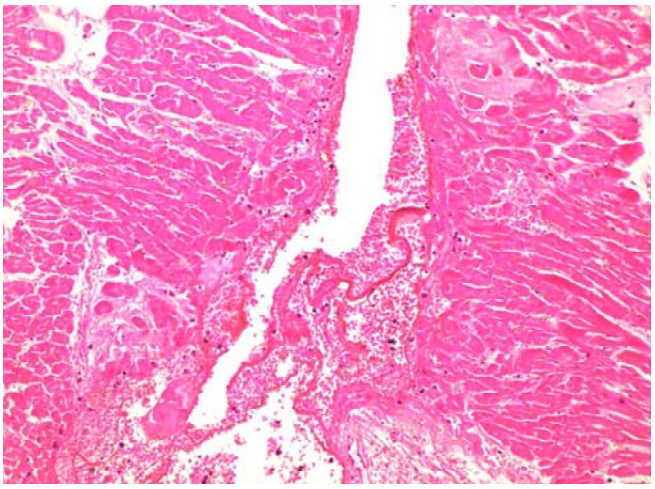

Fig. (2). Region of dehiscence in left ventricular free wall, with isquemic necrosis around the edges and precipitation of fibrin.

AUTOPSY REPORT

We concluded that the patient suffered an early type I left ventricle rupture after mitral replacement and an associated consumptive coagulopathy.

DISCUSSION

Ventricular wall rupture after mitral valve replacement is an uncommon complication that may be lethal. The causes of it are still controversial and the incidence of such complication is 1.6% [1].

There are some methods described to attempt the repair of the ventricular rupture after mitral replacement [1-5] and a growing literature widely reported the results of successful surgical repair with cardiopulmonary by pass [1, 4].

In our case report the indication of surgical replacement of the diseased heart mitral valves was correct and based on the beneficial impact that this replacement would have on the patient's heart function. The preoperative study was accurate and well done. It's well known that an operation to repair or replace a mitral valve usually takes 2 – 3 hours to perform. In this case, the surgical time was 12 hours, but the surgeon did not describe the cause of this extended surgical time on the surgical report. The death was attributed to a hypovolemic and cardiac shock but the rupture of the left ventricular was not reported as a specific diagnoses.

The rupture of the left ventricular wall can be classified according to two criteria:

1) According to the site and location: type I (posterior atrioventricular groove), type II (posterior wall of left ventricle at the base of the papilary muscle) and type III (area between atrioventricular groove and papillary muscles) [4]

2) According to time patterns: early (event occurred during the operation room anytime after discontinuation of cardiopulmonary bypass), delayed (in the recovery room) and late rupture (days to years after valve replacement and presented as false aneurysm of the left ventricle) [5].

In some of the previously published cases the autopsies were unobtainable and the discussion was based on the medical history. In our case it was not difficult to define the site of rupture by the spreading hematoma beneath the epicardical tissue. The reported case is, according to the location and time patterns, an early rupture and type I.

The following causes have been considered by medical literature as predisposing risk factors: female sex, advanced age, intrinsic myocardial disease, mitral stenosis, small body size, and a small left ventricle [2, 3] or in cases with osteogenesis imperfecta tarda [6]. Taking into account the risks of surgery and the autopsy findings we concluded that in our case report there were several predisposing risk factors that may lead to unfavourable operative outcomes: female sex, advanced age, mitral stenosis, small body size, and a small left ventricle but they are, all of them, common risk factors in heart surgery.

We also must consider the reported intraoperative factors:

- Resection of excessive tissue during removal of the mitral valve and consequent injury of the annulus.
- Inaccurate sizing of the annulus and insertion of an oversized prosthesis.
- Entrance of deeply placed sutures into the ventricular myocardium.
- Forceful traction on the mitral annulus.
- Apical venting of the left ventricle with dislocation of the heart and consequent distortion of the left ventricular posterior wall when a rigid mitral prosthesis is in place.
- Mechanical injury to the ventricular endocardium caused by such devices as metal vents and cardiotomy suckers, scissors used during valve excision and retractors used to expose the cordae tendineae.
- Forceful compression of the ventricle against the prosthesis during manual massage.

Positioning the strut of a bioprosthesis against the posterior left ventricular wall [4, 7, 8]. Although the precise cause of the rupture in our case was unclear we considered that one of the contributing factors was an excessive removal

of the calcified mitral annulus because it is a well known cause of dissecting hematoma of the atrioventricular groove with hemorrhage and possibly external perforation.

The autopsy report concluded that the patient suffered a type I left ventricle rupture after mitral replacement and according to the clinical report a consumptive coagulopathy was associated and with this early rupture subsequently died. After our forensic report and without having carried out any special report on the medical conduct, the family withdrew the charges without any explanation.

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CONFLICT OF INTEREST

The authors declare that we have no conflict of interest.

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