Constrictive pericarditis following a pyopericardium due to *Staphylococcus aureus*

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Abstract

We report the case of a 58-year-old man who presented 3 weeks after a purulent pericarditis due to *Staphylococcus aureus* with a constrictive pericarditis confirmed by computed tomography-scan and haemodynamic findings. Pericardiectomy and epicardiectomy were performed with an excellent immediate and mid-term result. Constrictive pericarditis due to *S. aureus* is rare and pericardiectomy is associated with a high mortality risk.

Keywords: Purulent constrictive pericarditis; *Staphylococcus aureus*

1. Case report

A 58-year-old man was referred to our hospital for septic shock and right heart failure. Past medical history was marked by diabetes mellitus, alcohol addiction, and hypertension. He presented 3 weeks before with a urinary tract infection (UTI) due to methicillin-sensitive *Staphylococcus aureus* treated by specific antibiotics. No local cause was found to explain the UTI (urinary obstruction, instrumentation or risky behaviour).

After 5 days of oral antibiotic treatment, the patient was admitted into a local hospital for septic shock and tamponade, confirmed by trans-thoracic echography (TTE). Renal echography was normal. A pericardial drainage via a subxiphoid incision was performed, and 700 ml of pus were extracted. A chest tube was placed into the pericardium. Examination of the pericardial liquid revealed the same *S. aureus* that was identified in the urine examination. Antibiotics were changed into intravenous route. Nevertheless, the relief of the tamponade did not completely correct the haemodynamics and the patient remained cyanotic and oligouric. Central venous pressure was between 18 and 20 mmHg and systolic arterial pressure low despite introduction of inotropic support (dobutamine and norepinephrine).

A TTE was performed and showed a conserved left ventricular function, but an altered right ventricular motion without major signs of compression (normal pulmonary and hepatic venous waveforms and transmitral flow, no respiratory variation of ventricular size or compression of left ventricle during diastole), associated to a thick pericardium (12 mm) and a moderate pericardial effusion. Haemodynamic evolution was progressively favourable allowing the weaning of inotropic drugs, but the pleural effusions and the lower limbs oedemas remained notable, and the weaning from respiratory support was impossible. A CT-scan performed 3 weeks after the surgical drainage revealed a 22-mm-thick pericardium, a 11-mm-thick epicardium, a moderate pericardial effusion and bilateral pleural effusions (Fig. 1). Two chest tubes were inserted and the patient was referred to cardiac intensive care for further management.
At arrival, the patient was intubated. A Swann Ganz catheter was inserted into the right internal jugular vein. The haemodynamic parameters showed a low cardiac output (2.5 l/min) and index (1.2 l/min per m²), low systemic vascular resistances (802 dn-s/cm⁵) and venous oxygen saturation (SVO₂, 50%). Intervenous inotropic drugs (epinephrine and norepinephrine) were reintroduced but with no real efficacy upon the patient’s haemodynamics. Even if there was no good evidence that the patient’s conditions was only due to ventricular compression, a pericardiectomy was decided.

Surgical approach was performed via total sternotomy. The pericardium was removed completely from phrenic to phrenic nerve, and posterior to the left phrenic to the level of the left pulmonary veins. Epicardium was then dissected gently and removed from the right ventricular free wall and diaphragmatic surface, from the right atrial surface, then from the left ventricular wall. Removal revealed to be easy with moderate ventricular aggression, and was possible without the need of a cardiopulmonary bypass. Haemodynamics remained stable during dissection. After total epicardiectomy, cardiac output raised up to 8.9 l/min and SVO₂ to 82% allowing the weaning of inotropic support peroperative. Eight redon drains were inserted into the pericardial cavity, and the sternum was closed [1].

The patient was extubated at postoperative day 5. Cultures of pericardial biopsies remained sterile. Pathologic examination showed diffuse leukotic infiltration. The redon drains were maintained for 10 days in postoperative with regular bacteriological control. Antibiotherapy was maintained for 45 days (including 21 days in intravenous way). Further evolution was uneventful.

After 3 months, the patient was free from dyspnoea and chest pain. Sternal wound was perfect. Biological findings (leukocytes, C-reactive protein) were beyond normality. A TEE did not show residual pericardial effusion or ventricular compression.

2. Discussion

Purulent pericarditis is diagnosed when pus is drained from the pericardial space or when bacteria are cultured from the pericardial fluid [2]. This rare disease is often diagnosed late, when severe haemodynamic compromise develops due to pericardial tamponade. It is usually a complication of pneumonia [3], especially if there is empyema as well, and often follows chest surgery or chest wall infections. It sometimes appears in patients with septicemia, especially when they are debilitated or immunocompromised. Diagnosis is aided by echocardiography or computed tomography (CT)-scan. Pericardiocentesis and drainage of the pus, as well as prolonged antibiotic treatment, are mandatory [4]. Delay in diagnosis and treatment often results in death. Some surviving patients may develop constrictive pericarditis and require pericardiectomy. The pericardial extension following a renal infection is rare and reveals a hematogenous contamination [5] rather than a local propagation. The pericardial drainage in addition to specific antibiotics constituted an adapted attitude and was performed quickly in this case, but secondary evolution was unfavourable. Moreover, the echographic findings did not affirm the ventricular compression and the surgical reintervention decision was more or less speculative upon the CT-scan images. The early extended pericardiectomy and epicardiectomy probably allowed a complete treatment of the epicardial lesions and the consequent favourable evolution of the patient, contrarily to what is usually feasible in tuberculosis lesions. Some authors have described cross-hatching ‘waffle’ incisions on the left ventricular epicardium [6] when pericardiectomy revealed to be too demanding. The peroperative drainage with multiple redon drains seems to be an important factor, because of stronger intrapericardial suction (≈ 700 mmHg) [7]. Therefore, we believe that it constitutes an important part in the surgical management of a pyopericardium.

References


Fig. 1. CT-scan showing an important thickening of the parietal pericardium and of the epicardium. A moderate pericardial effusion and severe bilateral pleural effusions are also noted.

