Clinical vignette

A sixty-nine-year-old woman was admitted to the emergency department of a peripheral hospital because of chest pain and dyspnea at rest. Electrocardiography showed ST segment depression at the precordial lead and cardiac enzymes were elevated. An emergency coronary angiogram was performed. This showed a subtotal stenosis of the left circumflex artery (LCX) and a severe (80%) stenosis of the right coronary artery (RCA) (Video 1). Percutaneous stenting of the LCX as culprit lesion was successfully performed. On the following day, a transesophageal echocardiogram (TOE) was accomplished because of hemodynamic instability and pulmonary oedema. The TOE showed severe mitral regurgitation (MR) secondary to the rupture of the anterolateral papillary muscle (PM). The TOE was interrupted because of worsening of the cardiogenic shock; the patient was intubated and referred to our institution for urgent cardiac surgery.

Surgical techniques

At admission in our department, the patient underwent an emergency operation through a median sternotomy. Cardiopulmonary bypass (CPB) was instituted with ascending aorta and bicalve cannulations. Cardioplegic arrest was achieved through Histidine-Tryptophan-Ketoglutarate solution. After performing a distal anastomosis with a saphenous vein graft on the RCA, the mitral valve was exposed through an incision in the Waterstone's groove. The anterolateral PM was partially ruptured, whilst the surrounding tissue was fragile and inflamed. Due to hemodynamic instability, the surgeon decided for a replacement. After partial resection of the posterior and anterior leaflets, and preservation of the remaining subvalvular apparatus, a biological prosthesis (Perimount Mitral 27 mm, Edwards Lifescience, Irvine, CA, USA) was implanted. The weaning from the CPB was uneventful and the patient was transferred to the intensive care unit.

Comments

The surgical management of the mitral valve regurgitation due to ischemic rupture of a PM is one of the most challenging situations for a cardiac surgeon. The hemodynamic instability, but most importantly, the uncertain stability of the myocardial tissue, are important factors the surgeon must face.

As a modern Hamlet, the cardiac surgeon should decide to repair or not repair. The main reasons for not repair are: (I) questionable durability of a repair that inevitably requires suturing onto friable and infarcted myocardial tissues, (II) longer operative ischemic time, and (III) potential of recurrent MR after ventricular remodeling progression (1).

The technique to repair depends on the anatomical presentation. Whatever repair is performed, there are two key concepts that should always be followed: the first is to use the non-involved PM as reference for the coaptation height, and the second is to perform an annuloplasty using a prosthetic ring, in order to stabilize the annulus and reduce the tension on the leaflets (2).

In case of total rupture, there will be two possibilities. If the rupture occurs in the middle or at the basis of the PM,
the necrotic tissue will be removed from the chordae, and the fibrous cuff will be implanted in the subvalvular muscle in a position that restores the tension on the chordae. 4-0 mattress sutures will fix the chordae as deep as possible. This technique aims to restore a quite normal anatomy: however, the risk to manage a yet ischemic myocardial tissue should not be underestimated. An interesting alternative technique has been recently described by Lee and colleagues, who reattached the anterior PM to the posterior, completing it with an undersized annuloplasty (1). If the rupture occurs at the edge of the PM, the chordae could be fixed directly to the fibrous tip of the PM (if the latter maintained a normal length). An alternative consists in the placement of artificial chordae. This is an elegant solution, which has the advantage to be simple, and reproducible, with a high probability of success as this technique belongs to the normal armamentarium of each cardiac surgeon. However, the risk to anchor the artificial chordae to a fragile (because ischemic) myocardium should always be considered.

In case of partial/incomplete rupture, which also includes an elongation, the PM can be directly sutured and eventually shortened. The limitations of this latter solution are the same of others, which means risk of repeated rupture (due to the fragility of tissues) or risk of restriction (due to shortening of the subvalvular apparat).

Theoretically advantages of the valve repair over its replacement (in this particular scenario) have been not yet established so far. In many statistics the in-hospital mortality ranges from 0% (3) up to 11% (4). The question “repair or not repair?” would need more studies with larger sample size to be answered. However, the attention surrounding Hamlet’s dilemma should not distract the operator from other important aspects of the surgical strategy. For example, the need for a concomitant revascularization, which significantly influences the survival. Prior studies found the operative mortality declining from 67% (before 1990 without coronary artery bypass grafting) down to 8.7% (after 1990, in combination with coronary artery bypass grafting) (5).

To conclude, the repair of a mitral valve regurgitation in case of ischemic PM rupture is possible, but should be performed as a sniper: precise, parsimonious and effective (but not lethal!).

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**Footnote**

**Conflicts of Interest:** TF is consultant for LivaNova. The other author has no conflict of interest.

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**References**


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