
Work in progress - Cardiac general

Papillary muscle elevation: an alternative subvalvular procedure for selective relocation of displaced posterior papillary muscle in posteroinferior infarction

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Abstract

Several subvalvular procedures have been developed for relocating one or both displaced papillary muscles. We describe an original procedure – papillary muscle elevation – in which we relocated the posterior papillary muscle selectively, through a small inferior anteroseptal ventriculotomy, and reduced the coaptation depth from 5 mm to zero. Our procedure can be considered for cases of posteroinferior infarction, which is a frequent cause of ischemic mitral regurgitation.

Keywords: Subvalvular procedure; Papillary muscle relocation; Ischemic mitral regurgitation

1. Introduction

A growing body of evidence supports the hypothesis that ischemic mitral regurgitation (IMR) is not a valvular disease, but a ventricular disease. Displacement of the papillary muscle (PM) due to post-infarction ventricular remodeling is a key issue in the development of IMR. Several subvalvular procedures have been developed to correct displacement of the PM [1–3].

We previously described our procedure for relocating separate heads of the posterior PM through an extended anterolateral left ventriculotomy (LV-tomy), in a case of broad anteroseptal and inferolateral infarction [4]. We used the procedure adjunctively to papillary muscle reapproximation under relatively uncommon cases of separate posterior PM. In the present case of posteroinferior infarction, we used a similar technique, which we call 'papillary muscle elevation', to relocate the entire PPM towards the posterior PM. In the present case of posteroinferior infarction, we used a similar technique, which we call 'papillary muscle elevation', to relocate the entire PPM towards the posteromedial PM, in the presence of an apically dislocated inferolateral PM.

We performed a subvalvular procedure with mitral annuloplasty (MAP), for correction of tethering-induced MR. At operation, transmural infarction extended from the LV apex to the mid-inferior wall. Coronary artery bypass grafting (left internal thoracic artery to left anterior descending artery) was performed first. The mitral annulus was dilated mildly. The posteromedial papillary muscle (PPM) appeared to be dislocated apically and laterally; this was potentially responsible for the retraction of the P2 and P3 segments towards the apex on the water-injection test. It was considered essential to perform a subvalvular procedure with mitral annuloplasty (MAP), for correction of tethering-induced MR.

We selected our original subvalvular procedure, 'papillary muscle elevation', to relocate the entire PPM towards the mitral annulus. A 4-cm ventriculotomy was made from the apex towards the inferior wall (which had become thin with an infarction scar), paying utmost attention to direct-
ed to one side of the PPM base, with a distance of 5 mm from the inside to outside and then from the outside to inside towards the other side of the PPM base. Another suture was placed next to the first one to establish two aligned felts on each side of the PPM. Two 3-0 Nespolene sutures were tied on the other felts (Figs. 2, 3). The inferior LV-tomy was closed by a linear suture method while placing a felt strip outside.

When observed through the mitral valve, the PPM appeared to be closer to the annulus. Furthermore, correction of apical retraction of the P2 and P3 appeared to be considerable. The MAP was performed using a 28-mm Carpentier–Edwards classic ring (Edwards Lifescience, Irvine, CA), which was not undersized. The patient was weaned from the cardiopulmonary bypass, leaving no residual mitral regurgitation. The postoperative course was uneventful. Early postoperative echocardiography showed almost zero coaptation depth (Fig. 1b) and no MR, associated with change of anterior leaflet configuration to convex shape.

Follow-up echo study performed four months later showed no recurrence of MR despite interval enlargement of the LV chamber.
3. Comment

The incidence and severity of IMR are known to be much higher in patients with posteroinferior infarction than in those with anteroseptal infarction. Kumanohoso and coworkers showed that posteroinferior infarction causes localized LV remodeling, but gives rise to greater geometric changes in the mitral valve – with greater PPM displacement – in comparison to broad anteroseptal infarction with global LV remodeling [5].

This new understanding of IMR puts greater emphasis on the significance of surgical correction of displaced PPM caused by localized LV remodeling following posteroinferior infarction. Kron and coworkers relocated the PPM by placing a traction suture between the displaced PPM and the mitral annulus [1]. The concept of our ‘PM elevation’ is similar to theirs in the sense that the displaced PPM is relocated selectively. The difference between the techniques is that we elevated the PPM from its base, via regional LV plication.

There are several advantages to our procedure. First, mechanical tension will be avoided on a limited segment of the PPM, which may partially be involved in infarction. Second, the length of PM elevation can be adjusted roughly by changing the distance between the PM base and the site of passage of the sutures to the LV wall, which should be the same as the coaptation depth determined by preoperative echocardiography. In our case, the PPM was theoretically relocated approximately 5 mm towards the mitral valve, which was verified on postoperative echocardiography.

An apparent limitation of our procedure is that the LV wall underlying the PPM should be sufficiently thin that it may be inverted without excessive wall tension [4]. However, in cases of posteroinferior transmural infarction with localized LV remodeling, the LV wall underlying the PPM could not be kinetic and thick. Small LV-tomy is not thought to increase the operative risk. Because there was only 5% increase in LV ejection fraction at early postoperative echocardiography, contribution of CABG to reduction of MR can be neglected in our case. Although our ‘PM elevation’ technique is available as an etiology-based strategy for relocation of displaced PPM in cases of posteroinferior infarction, more experience with this procedure will be required to determine its practical availability and efficacy.

References