Repair of Hammock Mitral Valve with Hypoplastic Posterior Leaflet in an Adult

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Background and aim of the study: **Congenital hammock mitral valves are rarely discovered in adulthood. The case is presented of a 29-year-old woman in NYHA functional class III and with markedly depressed left ventricular (LV) function, who underwent surgery for severe mitral regurgitation.**
Methods and Results: **Preoperative transthoracic echocardiography showed grade 3+/4 mitral regurgitation due to restricted leaflet motion, and an almost immobile posterior leaflet due to the attachment of fibrotic chordae high on the posterior LV wall. At surgery, limited motion of both leaflets and partial agenesis of the posterior leaflet were found. The valve was repaired by resecting the posterior leaflet defect, mobilizing the papillary muscle, and then widening the posterior leaflet. Postoperative echocardiography revealed no further mitral regurgitation, and the valve remained competent despite a postoperative episode of cardiac decompensation.**
Conclusion: **Hammock mitral valve repair is a highly challenging procedure, particularly in patients presenting with LV dilatation. The greatest degree of leaflet mobility must be restored in order to prevent tethering. Thus, it is strongly recommended that the posterior leaflet is systematically widened, even in the presence of an apparently large leaflet surface.**

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The hammock mitral valve is a congenital anomaly associated with variable degrees of papillary muscle malformation (1,2). A common characteristic is the variable displacement of the papillary muscle attachments to the posterior wall of the left ventricle, resulting in stenosis, regurgitation, or both. Stenosis occurs due to obstruction of the mitral orifice by abnormal papillary muscle tissue, along with a net of intertwined chordae from both leaflets, while regurgitation is due to poor leaflet mobility. Herein, the case is described of a successful repair of a hammock mitral valve and of severe hypoplasia of the posterior leaflet, in an adult who presented with markedly depressed left ventricular (LV) function.

Case report

A 29-year-old woman in NYHA functional class III, who had no history of rheumatic valve disease or bacterial endocarditis, was admitted to the authors' hospital to undergo mitral valve surgery. Transthoracic echocardiography demonstrated grade 3+/4 mitral regurgitation due to restricted leaflet motion. The posterior leaflet was almost immobile due to the attachment of short fibrotic chordae high on the posterior LV wall. A well-developed anterior leaflet was pulled posteriorly by chordae arising from the posterior wall (Fig. 1A). Transesophageal echocardiography revealed that the chordae from both leaflets originated from a muscular mass located beneath the posterior leaflet (Fig. 1B). The patient, who was in sinus rhythm, had a 25% LV ejection fraction, 53 mm left atrial diameter, and 58/68 mm end-systolic/end-diastolic LV diameters.

Surgical repair

At surgery, an almost immobile posterior leaflet with severe hypoplasia of the P2 area was found, while chordae from both leaflets were attached to an irregular muscular mass on the LV posterior wall. The anterior leaflet was well developed. The remnants of P2 were excised, and the remaining posterior leaflet detached from the annulus, uncovering several fibromuscular bands that connected the papillary muscles to the ventricular wall. These bands were divided

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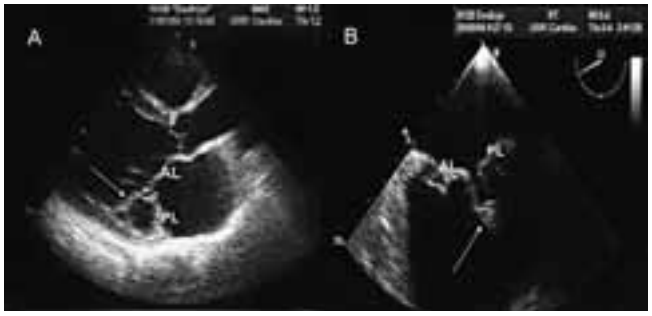

Figure 1: Preoperative echocardiography. A) Transthoracic long-axis, cross-sectional view, showing restricted motion of the posterior leaflet (PL). The fibrotic chordae (arrow) to the anterior leaflet (AL) are attached to the posterior LV wall. B) Transesophageal echocardiography. The chordae to the anterior (AL) and posterior (PL) leaflets are attached to an anomalous papillary muscle mass (arrow).

Figure 2. Intraoperative images. A) Mobilization of the papillary muscles. The posterior leaflet (PL) was detached from the annulus (AN). The fibromuscular bands, which attached the papillary muscles to the ventricular wall, were divided with scissors (arrow). B) Leaflet widening. Two large triangular patches (a) were used to widen the posterior leaflet and close the gap in the P2 area (arrow).

down to the posterior wall, in order to liberate the papillary muscles and mobilize the leaflet (Fig. 2A). Two triangular pericardial patches, prepared in 0.6% glutaraldehyde, were used to enlarge the P1 and P3 leaflet areas, and to supply enough tissue for sliding of the leaflets and closure of the gap in the P2 area (Fig. 2B). A saddle ring (no. 30; St. Jude Medical, Inc., St. Paul, MN, USA) was used for the annuloplasty. Postoperative echocardiography showed no further mitral regurgitation, and a large surface of leaflet coaptation. Following inotrope treatment of a postoperative episode of cardiac decompensation, the patient was discharged from hospital, at 10 days after surgery.

Follow up

At six months postoperatively, no mitral regurgitation could be demonstrated by echocardiography. The patient was still in sinus rhythm, and her NYHA functional status had improved to class I. The LV ejection fraction had remained unchanged, though a slight improvement was identified in the end-systolic/end-diastolic LV diameters, to 56/65 mm.

Discussion

The hammock mitral valve is a congenital malformation rarely found in adulthood. When present, the diagnosis is usually established intraoperatively, as in the present case. Echocardiographic findings, such as a restrictive leaflet motion of unknown origin in particular, an abnormal chordal anatomy or an atypical papillary muscle appearance, call attention to possible hammock mitral valve anomaly.

To the best of the authors' knowledge, this is only the fifth report of the successful repair of hammock mitral valve in an adult (3,4), and the first to describe the concomitant repair of an anomalous posterior leaflet. Although the hammock valve is usually stenotic in children, all adults described thus far have presented with valvular regurgitation. Therefore, it was hypothesized that these children had reached adulthood due to the absence of stenosis rather than because of an anatomically preserved anterior leaflet (3).

The main pathologic manifestation of this malformation is limited leaflet motion. In order to restore leaflet mobility, the papillary muscles were first mobilized by cutting the fibromuscular bands that attached the papillary muscles to the posterior LV wall (Fig. 2A). A technique similar to that applied in Ebstein's anomaly was used to mobilize the anterior leaflet of the tricuspid valve. Additional mobility was obtained by widening the posterior leaflet, using a patch, which also created a large surface of coaptation. This was particularly important to prevent tethering of the leaflet for a patient in whom the LV cavity was markedly dilated. The mitral valve remained competent, despite a postoperative episode of cardiac decompensation. Consequently, this widening maneuver is strongly recommended, even in the presence of an apparently sufficiently large leaflet surface. A larger leaflet also enabled its overlap and a satisfactory closure of the gap in the P2 area.

In conclusion, the diagnosis of hammock mitral valve, a congenital anomaly rarely discovered in adulthood, is usually made intraoperatively. The repair of this malformation is highly challenging, particularly in the presence of LV dilatation, in which case leaflet tethering represents a potential additional restrictive complication. Thus, leaflet enlargement is considered essential to restore the greatest valvular mobility, and to achieve a successful and stable repair in such a com-

plex case. This approach is recommended whenever repairing a severely restrictive mitral valve, even in the presence of a sufficiently large leaflet surface.

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