Acute Severe Mitral Regurgitation from Papillary Muscle Dysfunction in Acute Myocardial Infarction

Successful Early Surgical Treatment by Combined Mitral Valve Replacement and Aortocoronary Saphenous Vein Bypass Graft

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SUMMARY
A patient with acute severe mitral regurgitation resulting from papillary muscle dysfunction which developed on the third day of acute myocardial infarction underwent early successful mitral valve prosthetic replacement with concomitant aortocoronary saphenous vein bypass. The concept of acute severe mitral regurgitation due to an infarcted papillary muscle which has not actually ruptured and an aggressive approach to such a catastrophic event early in the course of acute myocardial infarction are emphasized. The risk is well worth the gratifying result obtained in our patient who successfully underwent such a combined operative procedure for one of the early complications of acute myocardial infarction.

Additional Indexing Words:
Coronary artery disease  Ruptured papillary muscle  Acute pulmonary edema
Gas endarterectomy  Coronary arteriography  Left ventriculography

THE SYNDROME of acute mitral regurgitation due to ruptured papillary muscle complicating myocardial infarction has been well recognized, but the significance of severe mitral insufficiency resulting from a dysfunctional, but intact, papillary muscle has not been well appreciated until very recently. The characteristics of the syndrome of papillary muscle dysfunction have been recently reviewed. Usually considered a medical problem, papillary muscle dysfunction, if severe, may cause marked mitral regurgitation with such severe hemodynamic derangement that surgical intervention may be necessary as a lifesaving procedure. Following the first report in November, 1966 by Fluck et al. of a successful operation upon one patient with mitral insufficiency from papillary muscle dysfunction, Spencer, Rossi, and Schimert et al. have reported equally successful results with surgical treatment of mitral regurgitation which developed in patients with coronary artery disease. In most instances mitral valve surgery was carried out weeks to months after an acute myocardial infarct. Although Spencer et al. performed concomitant coronary endarterectomy in two of four patients with myocardial infarction secondary to chronic
coronary artery disease, no instance of combined aortocoronary saphenous vein bypass and mitral valve replacement for severe papillary muscle dysfunction, which developed early during the course of an acute myocardial infarction, has been reported to our knowledge. We wish to report such a case with acute severe mitral regurgitation due to papillary muscle dysfunction following acute inferior wall myocardial infarction who underwent successful mitral valve replacement in combination with aortocoronary saphenous vein bypass of the occluded right coronary artery on the fifth day of his illness.

**Case Report**

Patient R.T., a 46-year-old man, was transferred to The George Washington University Hospital from another hospital 5 days after suffering an acute myocardial infarction and 2 days after the discovery of an apical systolic murmur. His hospital course before transfer was complicated by hemoptysis, progressive dyspnea, orthopnea, and persistent tachycardia. The diagnosis of acute inferior myocardial infarction was confirmed by serial ECG changes and enzyme studies.

On admission to The George Washington University Hospital the patient was in frank congestive heart failure with diffuse rales in both lungs (blood pressure 120/80 mm Hg; pulse 128 beats/min; temperature 38°C). A left parasternal lift was noted. No thrill was felt. The first heart sound was accentuated at apex. At the apex there was a systolic murmur which varied in intensity from grade II/VI to IV/VI, in timing from mid-to pansystolic, and in configuration from crescendo-decrescendo to plateau (fig. 1). Both diastolic and presystolic gallops were present.

Cheest roentgenogram, on admission, showed acute pulmonary edema with a cardiac silhouette which was only mildly enlarged (fig. 2A). Serial electrocardiograms confirmed the presence of a recent inferior myocardial infarct.

Emergency right and left cardiac catheterization including selective coronary arteriography was carried out under local anesthesia via the percutaneous transfemoral route. Pulmonary artery pressure was 70/25 mm Hg. Pulmonary capillary tracing showed a larger V wave measuring 48–56 mm Hg and a mean pressure of 30 mm Hg (fig. 3). There was no evidence of a left-to-right shunt. Left ventricular pressure was 95/28 mm Hg. Aortic pressure was 95/60 (mean, 70) mm Hg.

Left ventriculogram showed severe mitral regurgitation predominantly through the posterior commissure and an intact interventricular septum. The appearance of the mitral valve was normal. Neither the left ventricle nor the left atrium was significantly enlarged. The inferior wall of the left ventricle was hypokinetic, whereas the rest of the left ventricle contracted well.

Selective coronary arteriograms showed total occlusion of the right coronary artery just beyond the origin of the acute marginal branch with retrograde filling of the posterior descending branch from the left anterior descending artery which showed only mild disease (fig. 4).

Due to progressive deterioration of the patient’s condition and failure to respond to medical treatment which consisted of intravenous furosemide and digitalis administration, rotating tourniquet, and nasal oxygen therapy, open-heart surgery was carried out shortly after cardiac catheterization. Cardiopulmonary bypass was instituted in the usual manner. Before putting the patient on the pump, the surgeon observed considerable mitral regurgitation. There was a fresh myocardial infarct on the inferior wall. There was considerable pleural effusion on both sides. After the patient was placed on total cardiopulmonary bypass, the left atrium was opened and the gross appearance of the mitral valve seemed completely normal with intact chordae tendineae. Both papillary muscles appeared scarred and stretched, the anterior much less so than the posterior papillary muscle which was yellowish, necrotic, but intact. The mitral valve along with its chordae and papillary muscles was excised and replaced by a 3M Starr-Edwards mitral prosthesis. The right coronary artery was replaced by a saphenous vein conduit (fig. 5). After surgery the patient was weaned from bypass slowly and was put back on left ventricular assist pump (LVAD). The patient remained hemodynamically stable and was weaned from the LVAD on the seventh postoperative day. A routine transthoracic echocardiogram on the seventh day showed a mildly dilated left ventricle with an EF of 30% and a normal septum.

**Figure 1**

Phonocardiograms showing the typical triad of papillary muscle dysfunction: accentuated first sound ($S_1$), changing murmur from a soft pansystolic murmur (sm) to a loud crescendo-decrescendo mid-systolic murmur (SM), and diastolic (DG) and presystolic gallops (PSG). MA$_{1f}$ = low-frequency phonocardiogram at mitral area; MA$_{1h}$ = high-frequency phonocardiogram at mitral area; CAR = indirect carotid pulse.
artery was found to be totally occluded beyond its proximal third. A short segment of the occluded right coronary artery was excised for pathologic examination. After carbon dioxide gas endarterectomy of its middle and distal thirds, a saphenous vein bypass graft was inserted between the endarterectomized distal right coronary artery and the ascending aorta. Good flow was noted through the bypass graft.

Microscopic examination confirmed the acute infarction of the posterior papillary muscle and also showed chronic fibrosis of both papillary muscles. The mitral valve itself was normal. The extirpated atheromatous plaque from the right coronary artery showed calcification; there was a recent thrombus with early organization in the excised portion of the middle segment of the right coronary artery.

The postoperative course was entirely uneventful and the patient improved markedly both clinically and radiologically (fig. 2B). He was discharged on the 20th postoperative day and has since returned to work.

Discussion

Systolic murmurs heard early after acute myocardial infarction often disappear with healing of the infarct, such murmurs usually being attributable to papillary muscle dysfunction. Persistent systolic murmur during the course of acute myocardial infarction may be due to rupture of a papillary muscle or chorda, persistent papillary muscle dysfunction, perforation of interventricular septum, development of a ventricular aneurysm, and acute left ventricular dilatation. Although differential diagnosis may sometimes be difficult, when the physical findings and clinical course are considered together it is usually possible to differentiate dysfunction from rupture of the papillary muscle: 1) Murmur of papillary muscle dysfunction usually has an insidious onset whereas onset of murmur of papillary muscle rupture is always abrupt. 2) The systolic murmur of papillary muscle dysfunction has a delayed onset after a loud first sound and is usually crescendo-decrescendo in configuration, whereas murmur of papillary muscle rupture begins immediately with a soft first sound and is plateau in shape. 3) Murmur of papillary muscle dysfunction frequently diminishes in intensity after an extrasystolic pause, whereas murmur of papillary muscle rupture does not. 4) The associated presystolic and diastolic gallops usually become louder during inspiration in papillary muscle dysfunction but during expiration in papillary muscle rupture. 5) The murmur of papillary muscle dysfunction may change in configuration from beat to beat but the murmur of papillary muscle rupture remains constant. 6) The posterior papillary muscle is said to be ruptured more often than the anterior, whereas anterior papillary muscle is as frequently infarcted as the posterior in dysfunction. However, this rule of statistical incidence, which did not apply in our case, is seldom of as much help in differential diagnosis between rupture and dysfunction of the papillary muscle as careful physical examination, with particular attention to the auscultatory characteristics mentioned above. Neither cardiac catheterization nor angiocardiography seems to be of much help either, as large systolic V waves in the pulmonary capillary pressure tracing and massive mitral regurgitation from the left ventricle into a normal-sized or minimally enlarged left atrium are present in both rupture of papillary muscle and severe papillary muscle dysfunction without rupture. Fortunately, when mitral regurgitation is severe and/or congestive heart failure is not controllable by medical treatment, surgical intervention is indicated whether or not the infarcted papillary muscle is actually ruptured and whether or not the posterior or anterior papillary muscle is affected.

Most patients with ruptured chordae tendineae do not have myocardial infarcts, but suffer from endocarditis or unknown causes. Perforation of infarcted ventricular septum is rather rare, and the systolic murmur is usually louder, rougher, lower pitched, shorter, and more medially located than that of a ruptured papillary muscle. Furthermore, in contradiction to rupture of papillary muscle which leads to left heart failure, septal perforation leads rapidly to right heart failure. Systolic murmur may result from a ventricular aneurysm, although it is rare to encounter such a
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dysfunction should have this combined operative approach should include the following: the extent and severity of the coronary artery disease, the functional status of the left ventricle, and reversibility of left ventricular dysfunction. The presence of segmental coronary artery disease that is demonstrated by coronary arteriography to be amenable to saphenous vein bypass should have a favorable influence on such an operation. The functional status of the left ventricle as estimated by left ventriculography should be a useful guide in deciding for or against operation. If mitral regurgitation is the result of extensive myocardial infarction which happens to also involve papillary muscle, or if the overall left ventricular function is very poor, then it would be futile to operate on such patients just to correct the mitral regurgitation. On the other hand, if acute mitral regurgitation results from severe dysfunction of one or both papillary muscles, but other portions of the left ventricle function well, then repair of the mitral valve may well be worth the while. Thus, to answer the critical question of which patient with acute myocardial infarction who developed a loud systolic murmur and congestive heart failure should or should not be operated on, one need complete hemodynamic and angiographic studies, including selective coronary arteriography, left ventriculography, hemoglobin oxygen-saturation determinations, and indicator-dilution study. The latter would be extremely helpful in detecting a left-to-right shunt and thus diagnosing perforation of an infarcted ventricular septum which may mimic papillary muscle dysfunction. If there is an area of inadequate left ventricular function with occlusive disease of the coronary arteries which are bypassable, aortocoronary saphenous vein bypass operation should be considered in combination with correction of the

Figure 3

Tall V wave (48-56 mm Hg) in pulmonary capillary (PC) pressure tracing.

Factors to consider in deciding whether or not a patient with severe papillary muscle dysfunction should have this combined operative approach should include the following: the extent and severity of the coronary artery disease, the functional status of the left ventricle, and reversibility of left ventricular dysfunction. The presence of segmental coronary artery disease that is demonstrated by coronary arteriography to be amenable to saphenous vein bypass should have a favorable influence on such an operation. The functional status of the left ventricle as estimated by left ventriculography should be a useful guide in deciding for or against operation. If mitral regurgitation is the result of extensive myocardial infarction which happens to also involve papillary muscle, or if the overall left ventricular function is very poor, then it would be futile to operate on such patients just to correct the mitral regurgitation. On the other hand, if acute mitral regurgitation results from severe dysfunction of one or both papillary muscles, but other portions of the left ventricle function well, then repair of the mitral valve may well be worth the while. Thus, to answer the critical question of which patient with acute myocardial infarction who developed a loud systolic murmur and congestive heart failure should or should not be operated on, one need complete hemodynamic and angiographic studies, including selective coronary arteriography, left ventriculography, hemoglobin oxygen-saturation determinations, and indicator-dilution study. The latter would be extremely helpful in detecting a left-to-right shunt and thus diagnosing perforation of an infarcted ventricular septum which may mimic papillary muscle dysfunction. If there is an area of inadequate left ventricular function with occlusive disease of the coronary arteries which are bypassable, aortocoronary saphenous vein bypass operation should be considered in combination with correction of the

Figure 2

Chest roentgenograms. (A) Preoperative: showing marked passive pulmonary congestion with a relatively normal-sized heart. (B) Postoperative: showing clear lung fields and residual right pleural effusion following prosthetic replacement of the mitral valve and aortocoronary saphenous vein bypass.

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acute mitral regurgitation resulting from papillary muscle dysfunction in a patient with an acute myocardial infarct. Recently it was shown by Saltiel et al.\textsuperscript{12} that, even if left ventricular angiography showed left ventricular dysfunction, the latter may be partly or wholly reversible in a good proportion of patients with coronary artery disease by aortocoronary bypass grafting. This extremely important observation, if confirmed by a larger series of studies, would certainly extend the current indications for this combined approach even further.

In conclusion, it is important to stress the marked degree of mitral regurgitation that a patient with coronary artery disease with or without acute myocardial infarction can have by just having papillary muscle dysfunction without actual rupture. An aggressive approach on these patients which consists of combined mitral valve replacement and aortocoronary saphenous vein bypass graft is often very rewarding, even when performed relatively early after myocardial infarction, such as was done in our patient. This concept of massive mitral regurgitation due to infarcted papillary muscles which have not ruptured and have intact chordae tendineae has not been well appreciated and certainly deserves emphasis. It should be further pointed out that cardiopulmonary bypass can be successfully performed upon such critically ill patients with severe coronary artery disease, acute myocardial infarction, severe mitral insufficiency, and congestive heart failure, as long as prompt correction of hemodynamic derangement is combined with immediate restitution of myocardial blood supply, and that the postoperative period can be surprisingly uneventful such as was demonstrated by our patient.
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