



Review Article

Mitral Valve Rupture Following CABG and PDA Endarterectomy: A Rare and Potentially Lethal Complication

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Abstract: Mitral valve rupture is a very rare and lethal complication following coronary artery bypass grafting (CABG). We report in this paper a case of mitral valve rupture ninety hours following CABG and coronary endarterectomy and we review the literature for similar cases in order to determine the different etiologies and mechanisms that may precipitate such serious adverse event and to define the measures that will help us diagnosing and treating promptly this complication.

Keywords: Mitral Valve Rupture, CABG, Coronary Endarterectomy, TEE

1. Introduction

Mitral valve rupture is a very rare but fatal complication following coronary artery bypass grafting (CABG). Rupture frequently occurs two to seven days after myocardial infarction and may lead to death within 24 hours unless surgical repair is carried out rapidly.

We report in this paper a case of mitral valve rupture four days after CABG with endarterectomy and we review the literature for similar cases in order to elucidate the mechanism of rupture and the different etiologies that may precipitate such complications. We also discuss the measures that may help diagnosing early this adverse event and treat it in its initial phase.

2. Case Report

A 71 y old male was admitted to our hospital for CABG. He was a known diabetic, hypertensive and dyslipidemic on oral

medications. A cardiac catheterization done after a positive stress test showed 3 vessels disease.

A CT scan of the chest revealed a 4.5 cm ascending aorta and a cardiac echography showed a normal ejection fraction, trace mitral regurgitation (MR) and mild aortic regurgitation (AR).

He underwent a CABG where a left internal mammary artery (LIMA) to left anterior descending artery (LAD) was grafted as well as a reverse savenous vein (RSV) to obtuse marginal (OM) and posterior descending artery (PDA). To note that an endarterectomy to the PDA was done before grafting the vein due to severe calcifications.

He was transferred to the coronary surgery unit (CSU). Two hours post operatively a ST elevation in the inferior leads was noticed without any hemodynamic instability. An infusion of nitroglycerine was started and antiplatelets medications were administered 5 hours post operatively. The blood pressure was maintained above 130/80 mm Hg.

A follow up EKG few hours later showed Q waves in

DII, DIII and aVF. The patient was stable clinically and hemodynamically and was extubated in the morning after. On day two post operatively he was transferred to the floor with a smooth post operative course. On day four (90 hrs post operatively) he developed a sudden and severe dyspnea with desaturation and a drop in blood pressure (80/60 mm Hg). Chest X ray showed severe lung congestion. The patient was transferred back to the CSU and intubated. Inotropes (dopamine 10 to 15 micra) and lasix were started. A central line and a swan ganz were inserted. The pulmonary artery pressure (PAP) ranged between 50 and 60 mm Hg, the wedge showed a v wave and was around 20 mm Hg and the cardiac index was 1.8. The mixed venous saturation was very low (38%). Transthoracic echography (TTE) showed severe mitral regurgitation. Transesophageal echography (TEE) revealed a ruptured chordae to the anterior leaflet of the mitral valve (MV) with flail A2 which was responsible for the severe mitral regurgitation. The papillary muscle appeared intact. The left ventricle (LV) was hyperkinetic with hypokinesis of the basal and mid inferior segments. The pulmonary artery (PA) pressure was estimated to be around 50 mm Hg.

The patient was taken to the operating room (OR) urgently where a mitral valve replacement with a mechanical valve and CABG to the PDA were done. Intraoperatively we found a ruptured chordae tendinae of the anterior leaflet at its insertion at the posteromedial papillary muscle. There was necrosis of the tip of the papillary muscle at the insertion site. On the other hand the graft to the PDA was occluded.

The patient was retransferred to the CSU on inotropes (dopamine 10 micra/kg/mn and neosynephrine 30 micra/mn). The PAP dropped to 28 mm Hg and the cardiac index increased to 2.5. Despite a relative stable cardiac state the patient had a complicated post operative course due to pneumonia and acute

tubular necrosis (ATN) which necessitated prolonged intubation for 10 days, antibiotics and few sessions of dialysis.

He was discharged from the hospital 20 days post operatively after a full respiratory and kidney recovery.

3. Discussion

Mitral valve rupture following CABG is an extremely rare event. Literature is being limited to very few similar cases. Baladi et al reported 3 cases in whom papillary muscle rupture occurred in one to three days after successful CABG. In all three patients the antero papillary muscle was involved. (table 1)

Prompt reoperation was successfully carried out in all patients. Mitral valve replacement was achieved in two cases and mitral valve repair was done in the third patient [1].

Benedetto et al described a fourth case of anteropapillary muscle rupture after a CABG which was diagnosed in its early phase by performing an intra operative TEE following weaning of cardiopulmonary bypass (CPB) (2). In fact shortly after going off bypass the patient became unstable. His blood pressure dropped to 50 mm Hg and the wedge pressure increased from 12 to 20 mmHg. An intra aortic balloon was inserted and high doses of inotropic drugs started (adrenaline 0.1 micra/kg/mn and noradrenaline 0.2 micra /kg/mn). TEE was immediately performed. It showed a new onset of moderate to severe mitral regurgitation with prolapse of both mitral leaflets predominately the posterior one and a partial rupture of the antero lateral papillary muscle. CPB was reestablished and the mitral valve was successfully replaced. To note that the surgical view confirmed the TEE findings. (Table 1)

Table 1. Clinical features and characteristics of 4 patients reported to have developed mitral valve rupture following CABG.

Patients	Paper reference	Sexe	Age	Per op MI	Per op MI	Post op MI	PM rupture	Days pos op
1	Baladi et al	M	65y	Lateral wall MI			AL	2
2	Baladi et al	M	72y	Inferior MI			AL	1
3	Baladi et al	M	73y	-	Inferior MI		AL	3
4	Benedetto et al	F	70y	Antero-septal MI			AL	Per op

M: Male, F: Female, MI: Myocardial infarction, PM: Papillary muscle, AL: Anterolateral, op: operatively.

In our case, the patient developed an inferior myocardial infarction few hours post CABG and PDA endarterectomy without any hemodynamic changes. Ninety hours post operatively he became unstable and developed pulmonary edema. A TEE performed revealed a ruptured anterior chordae to the anterior leaflet with flail A2 which was responsible for severe mitral regurgitation surprisingly the papillary muscles appeared intact. The patient was reoperated for mitral valve replacement and a CABG to the PDA since the vein that was grafted on the PDA earlier was occluded by a thrombus.

The cause of this uncommon event is speculative. In order to understand the cause and mechanism of mitral rupture it is very important to understand the anatomy of the mitral valve apparatus. The valve itself comprises an anterior and a posterior leaflet. Valvular competence is maintained by the two papillary muscles (antero lateral (AL) and posteromedial

(PM)) that are attached to the leaflets via the chordae tendinae. Typically the AL muscle is a single large structure whereas the PM muscle can have one to three heads. To note that the two papillary muscles send chordae to both leaflets of the mitral valve. As a result either leaflet can be affected by rupture of either papillary muscle [3]. On the other hand, blood supply to the AL muscle is usually supplied by the LAD and the circumflex arteries whereas the PM is supplied by the RCA. Rupture of the PM muscle is more common because of its single blood supply [3]. However many cases of unusual blood supply to the mitral valve papillary muscles have been reported. Hattori et al described a case of unusual perfusion of the LAD to posterior papillary muscle while Stefanovski et al reported a rupture of the AL papillary muscle following isolated distal RCA occlusion [4-5]. On the other hand, Okamoto et al described two cases in which occlusion of

diagonal coronary artery was found to be the culprit lesion of an acute myocardial infarction which lead to complete rupture of the AL papillary muscle. [6]

In all reported cases, we surprisingly noticed that the mitral regurgitation was due to rupture of the antero lateral papillary muscle which is usually less common than the posteromedial papillary muscle.

Baladi et al suggested that this rupture may be due to shear forces generated at areas with a discordant level of recovery from ischemia and enhanced by an overall improvement of myocardial contractility. A similar mechanism was proposed in cases of complete rupture of papillary muscle after successful percutaneous transluminal coronary angioplasty [1].

However benedetto et al think that these uncommon cases of ruptured papillary muscle were simply the completion of recent acute myocardial infarction and the hemodynamic consequences became evident after or during surgery. In fact all the cases reported had acute myocardial infarction before, during or after surgery [2].

Our patient developed an inferior myocardial infarction post CABG with PDA endarterectomy. This infarction resulted in rupture of the chordae tendinae of the anterior leaflet at its insertion site at the posteromedial papillary muscle leading to a severe mitral insufficiency (with a flail A2) that became evident few days after.

Mitral rupture is a lethal complication especially if not diagnosed early. It can be fatal if not treated in the following 24 hours. This explains the importance of monitoring tools that can be used to diagnose such adverse events. TEE is very reliable in diagnosing mitral valve rupture in its early phase with a sensitivity ranging between 65 to 85%. It can identify prolapsed, flail leaflets and the direction of the regurgitant jet. However direct visualization of the rupture is necessary for definite diagnosis especially that the findings do not reliably predict the affected papillary muscle [3].

To note that in the case reported by benedetto et al, the peroperative TEE allowed an early diagnosis of a partial papillary muscle rupture which may not appear in all standardized imaging planes, in contrast to complete rupture that is easily visualized [2]. Thus, intraoperative echo, done by experienced and trained operators will help in diagnosing and treating this serious complication in its early phase.

The mitral valve rupture occurred in all cases after ischemic insults that occurred before, during or after surgery. Most of the cases reported were CABG to both left and right systems. In our patient, grafts were achieved to the LAD, OM and PDA on which an endarterectomy was necessary due to severe calcifications and disease.

Most of the studies showed that CABG with coronary endarterectomy is followed by a high incidence of morbidity and mortality. The rate of myocardial infarction post endarterectomy ranges from 1.5 to 19%. [7-8-9] and is believed to be due to removal of the endothelial lining after performing endarterectomy resulting in thrombosis [10]. This high incidence explains why most of the authors prefer direct bypass to endarterectomy. However in desperate cases with

diffuse CAD, it can be done without bringing additional risks to the patient [7-11].

4. Conclusion

We report in this paper a very rare case of mitral valve rupture following coronary bypass grafting and PDA endarterectomy. We review the literature for similar cases in order to understand the etiology and mechanisms responsible of such rare complication. We emphasize also on the role of Transesophageal echography preoperatively in diagnosing this adverse event in its early phase and treat it especially that the outcome will be very poor if the rupture is not managed in the following 24 hours.

On the other hand it seems beneficial to keep a high index of suspicion whenever we notice any clinical, electrical or hemodynamic changes especially in patients at high risk of ischemia perioperatively i.e. patients undergoing coronary artery bypass grafting with severe coronary disease and in whom a coronary endarterectomy is performed.

Finally, additional cases of mitral valve rupture following coronary artery bypass grafting are needed in order to clarify issues that are still debatable concerning etiology and mechanisms so that we can improve our practice and management.

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