

Case Report

Acute Coronary Syndrome Presenting with Severe Pulmonary Edema due to Papillary Muscle Rupture Necessitating Urgent MVR and CABG

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ABSTRACT

Papillary muscle rupture (PMR) leading to acute severe mitral regurgitation is usually seen with acute transmural myocardial infarction (MI). Here, we report a case of acute coronary syndrome (ACS) with minor ST-T changes consistent with ischemia (and without any rise in the serial cardiac enzymes suggesting acute MI), who developed severe acute pulmonary edema secondary to papillary muscle rupture. This case emphasizes the point that even without obvious changes of acute transmural

MI patients might develop this serious complication. Hence, a high index of suspicion is needed to diagnose papillary muscle rupture and mitral regurgitation (MR) in a patient who presents with ACS and seemingly minor ST-T changes but suddenly deteriorates and passes into acute pulmonary edema. The physical findings suggesting this complication might be quite subtle and proper diagnosis with timely action can save the patient's life.

KEYWORDS: coronary atherosclerosis, mitral regurgitation, mitral valve replacement

INTRODUCTION

Papillary muscle rupture is usually associated with acute transmural myocardial infarction (MI)^[1]. However, it can rarely be seen with minor ST-T changes in the electrocardiogram (ECG). At times, physical findings may be very subtle requiring a high index of suspicion to diagnose this condition^[2].

Here, we report a case, which did not have electrocardiographic ST-T changes consistent with a transmural MI but showed those suggestive of infero-lateral ischemia. Furthermore, there was no rise in serial cardiac enzymes indicating acute MI and physical findings of acute severe MR were very subtle. However, per operatively, rupture of papillary muscle was found.

CASE REPORT

A 60-year-old Kuwaiti gentleman, a known case of hypertension and diabetes mellitus, with history of chronic smoking was admitted with a complaint of chest pain on and off for the past one week. The pain was retrosternal, compressive with radiation to both shoulders and was brought on by minimal exertion and relieved by rest or sublingual glyceryl trinitrates within a few minutes. He did not have any dyspnoea on admission.

General physical examination was unremarkable. Cardiovascular examination revealed a normal first and second heart sound without any added sound or murmur.

Examination of respiratory system revealed normal breath sounds with no rhonchi or crepitations. ECG on admission showed evidence on inferolateral ischemia without any serial evolutionary changes suggesting acute MI (Fig. 1).

He was started on nitroglycerine and heparin infusions along with other anti-ischemic medications such as betablockers and aspirin. Despite intensive medical management, the patient continued to experience occasional attacks of chest discomfort and started complaining of sudden shortness of breath along with chest pain on the next day.

Physical examination at this time showed that the patient was anxious, dyspneic and restless. Careful cardiovascular examination showed the appearance of a short systolic murmur (grade II-III/VI) at the apex, which was difficult to perceive because of adventitious respiratory sounds. Examination of the chest showed bilateral basal and mid zonal crepitations. In view of the above developments, an urgent echocardiogram was done which showed an almost flail posterior mitral leaflet (Fig. 2). ECG did not show any evidence of acute transmural MI but only changes of inferolateral ischaemia. Serial cardiac enzymes were not elevated.

As the patient's condition continued to deteriorate in spite of adequate diuretic and after-load reduction therapy, the patient was



Fig. 1: Admission ECG showing ST-T wave changes in inferolateral leads



Fig. 2: Apical four chamber transthoracic view showing flail posterior mitral valve leaflet

immediately referred for urgent cardiac catheterization and coronary angiography. However, the situation became very critical requiring urgent intubation and ventilation even before cardiac catheterization could be undertaken. Urgent cardiac catheterization showed significant lesions of circumflex and posterior descending coronary arteries.

Circumflex lesion was particularly critical. Hence, an urgent percutaneous transluminal coronary angioplasty (PTCA) of the circumflex artery was done under intra-aortic balloon pump (IABP) support hoping that alleviation of ischemia might improve and stabilize the patient's condition. But the patient did not show any improvement over next several hours. Hence, an urgent mitral valve replacement and coronary artery bypass graft (CABG) was successfully performed. During the operation, rupture of posteromedial papillary muscle was detected. The patient had a smooth postoperative recovery and he was later discharged from the hospital in a good condition. Since then, he has been keeping good health and is able to perform all his daily activities without any limitations.

Table 1
Serial cardiac enzyme values

Enzymes	0 hr after admission	12 hrs after admission	24 hrs after admission	48 hrs after admission
AST	33 I.U./L	48 I.U./L	18 I.U./L	15 I.U./L
CK	43 I.U./L	48 I.U./L	37 I.U./L	31 I.U./L
LDH	138 I.U./L	200 I.U./L	149 I.U./L	145 I.U./L

hr(s) = hour(s).

All values are within normal limits

DISCUSSION

Partial or total rupture of a papillary muscle is a rare but often fatal complication of transmural MI^[1]. The electrocardiogram often shows evidence of inferior or posterior infarction, which in many cases, may be limited to seemingly minor ST - T changes. This deceptively benign appearance despite the catastrophic clinical deterioration, was seen in our patient^[2]. Hence, in a case presenting with acute coronary syndrome and seemingly minor ST-T changes with severe pulmonary edema not responding to adequate medical management, a high index of suspicion is needed to diagnose PMR. Even the murmur of resultant MR may not be very impressive and could easily be missed if not specifically looked for, especially when the patient is in severe respiratory distress with widespread adventitious sounds due to acute pulmonary edema. In acute severe MR, because of rapid equilibration of left atrial and left ventricular pressures in late systole the systolic murmur may be brief, non descript, or even completely absent^[2].

Papillary muscle rupture usually involves the posteromedial muscle, because its blood supply is derived only from the posterior descending artery, whereas the anterolateral papillary muscle has a dual blood supply from left anterior descending and circumflex branches^[3]. In our case, rupture of the posteromedial papillary muscle was seen and coronary angiogram showed significant lesions of the posterior descending and circumflex arteries. Anterolateral muscle escaped any damage probably because of its dual blood supply.

CONCLUSION

The purpose of this case report is to emphasize the fact that even in a patient presenting with minor electrocardiographic ST-T changes (without transmural MI), if sudden hemodynamic instability develops, one should suspect mechanical complications of MI and carefully look for them. The physical findings could be very subtle. Our case had a very soft and short systolic murmur

suggestive of MR. Timely diagnosis and intervention could be life saving even in a seemingly very sick patient.

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