

Papillary muscle rupture leading to severe mitral regurgitation as a complication of acute myocardial infraction – A case report

Kalyan Munde^{1*}, Gurukirit Singh², Vishal Patil³, Bhanu Duggal⁴

¹Assistant Professor, ^{2,3}Sr. Resident, ⁴Associate Professor and HOD, Department of Cardiology, J. J. Hospital, Byculla, Mumbai– 400008 Maharashtra, INDIA.

Email: kalyanmunde@yahoo.com

Abstract

Among the lesions responsible for acute insufficiency of the mitral valve is Papillary muscle is rupture. The important etiologic factors claimed for rupture of papillary muscle are bacterial endocarditis, rheumatic valvular disease and trauma. Myocardial infarction is a rare cause of mitral Papillary muscle rupture.

Keywords: Papillary muscle rupture, acute myocardial infraction.

*Address for Correspondence:

Dr. Kalyan Munde , Flat no. 18, 4th floor, Swastik building, Doctor quarters, J. J. Hospital Campus, Byculla, Mumbai – 08, Maharashtra, INDIA.

Email: kalyanmunde@yahoo.com

Received Date: 14/12/2015 Revised Date: 28/12/2015 Accepted Date: 01/01/2016

Access this article online

Quick Response Code:



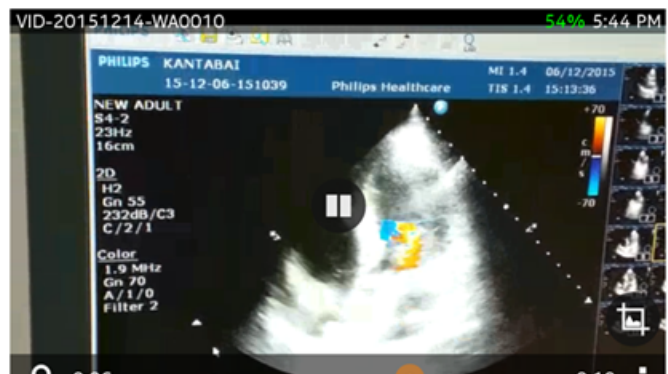
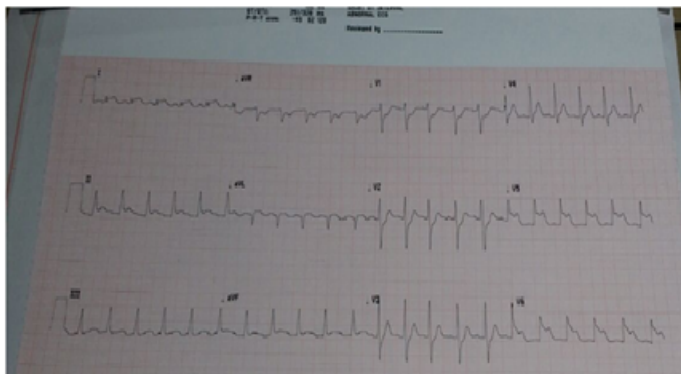
Website:

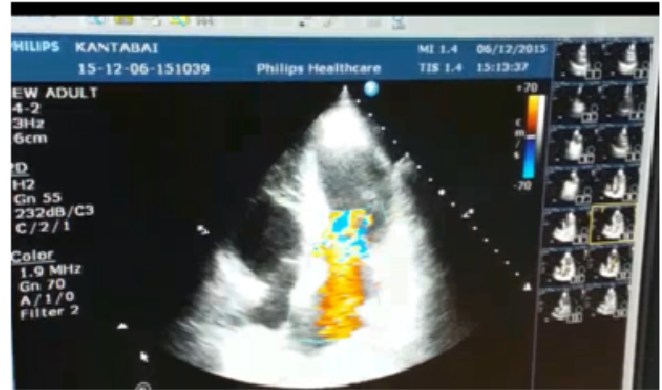
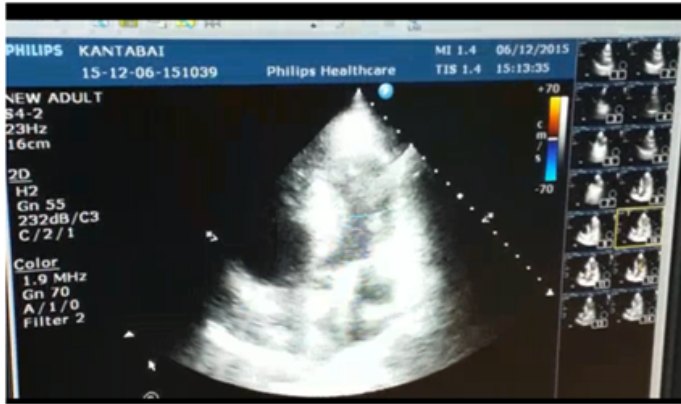
www.medpulse.in

DOI: 20 January
2016

INTRODUCTION

A 52 year old hypertensive female presented to us in Dec 2015 in a state of pulmonary edema and cardiogenic shock. ECG showed inferoposterior lateral myocardial infarction. On physical examination, patient has cold clammy extremities. Auscultation revealed soft systolic murmur heard at left 3rd intercostal space parasternal area. 2D echo showed rupture of postero medial papillary muscle leading to severe MR with jet directed anteriorly. Patient succumbed on same day before she could be taken for surgery.





DISCUSSION

The overall incidence of rupture of papillary muscle as a complication of acute myocardial infarction is 1 %. Posteromedial papillary muscle being more frequent than anterolateral papillary muscle. The complication has a bimodal peak within 24 hours and 3- 5 days (Range 1 – 14 days). The clinical presentation is in the form of abrupt onset of shortness of breath, Pulmonary edema and hypotension. Physical finding are in the form of a soft systolic murmur in some cases, no thrill, variable signs of the RV overload, severe pulmonary edema and cardiogenic shock. Echo findings are in the form of hypercontractile LV, torn papillary muscle or chordae tendineae, flail leaflet, severe mitral regurgitation on colour Doppler echocardiography. Partial or total rupture of a papillary muscle is a rare but often fatal complication of Transmural MI. Complete transection of a left ventricular papillary muscle is incompatible with life. Because the sudden massive mitral regurgitation that develops cannot be tolerated. Rupture of a portion of a papillary muscle usually the tip or head of the muscle that results in severe although not necessarily overwhelming mitral regurgitation is much more frequent and is not immediately fatal. Inferior wall Myocardial infarction can lead to rupture of the poster medial papillary muscle, which because of its singular blood supply, occurs more commonly than does rupture of anterolateral muscle, a consequence of anterolateral MI. Unlike rupture of ventricular septum, which occurs with large infarcts, papillary muscle rupture occurs with relatively small infarction in approximately half of cases, These patients can sometimes have a modest extent of CAD as well. Rupture of right ventricular papillary muscle is unusual

but can cause massive tricuspid regurgitation and right ventricular failure. The systolic murmur may become softer or disappear as arterial pressure falls.

Management

Invasive monitoring is generally indicated on recognition of a major mechanical complication of STEMI. Right and left ventricular filling pressures [Right atrial pressure and pulmonary capillary wedge pressure] guide fluid administration or the use of diuretics. Whereas measurements of cardiac output and mean arterial pressure permit calculation of SVR to direct vasodilator therapy. Nitroglycerine or Nitroprusside should be instituted unless systolic blood pressure is below 90 mmHg. Inotropes may also be needed to support adequate cardiac output. If pharmacologic therapy fails to achieve hemodynamic stability, IABP should be instituted rapidly.

Operative intervention is most successful. Surgical survival is predicted by early surgery, short duration of shock and mild degrees of right and left ventricular impairment.

CONCLUSIONS

Mechanical complications of acute MI should be meticulously ruled out in every case of STEMI for timely intervention. Prognosis is generally worse compared other STEMI cases.

REFERENCES

1. Textbook of cardiovascular medicine.- Braunwald
2. Textbook of interventional cardiology – Topol
3. Textbook of cardiac catheterisation and angiography- Grossman.

Source of Support: None Declared
Conflict of Interest: None Declared