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Acute Severe Mitral Regurgitation Secondary to Papillary Muscle Rupture: A Rare Mechanical Complication of Acute Myocardial Infarction

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ABSTRACT
Acute myocardial infarction in current era is detected promptly and emergent percutaneous intervention is initiated in most part of the world as a standard of care which has led to significant reduction in all the complications associated with acute myocardial ischemia. A 46-year-old man presented with acute onset of chest pain and shortness of breathand was found to have a non ST-segment elevation myocardial infarction and acute mitral regurgitation due to papillary muscle rupture. This case describes a rare and potentially fatal mechanical complication of acute myocardial infarction. Use of transesophageal echo guides for the understanding the mechanism and guide the management.

Keywords: acute myocardial infarction; acute mitral regurgitation; papillary muscle rupture.

INTRODUCTION
Acute myocardial infarction in current era is detected promptly and emergent percutaneous intervention is initiated in most part of the world as a standard of care which has led to significant reduction in all the complications associated with acute myocardial ischemia. However rarely papillary muscle rupture can occur which leads to acute severe mitral regurgitation, acute pulmonary edema and cardiogenic shock in various proportions. It has a high mortality rate unless promptly detected and treated. We describe a classic case of acute mitral regurgitation and pulmonary edema due to papillary muscle rupture.[1]

Case Report
A 46 years old male was presented to a primary hospital with complaints of sudden onset breathing difficulty with burning sensation over the central area of the chest and upper abdomen for two days. He also noted difficulty in lying flat and loss of sleep for the same duration. He had cough for last few days. He was hemodynamically stable there and treatment with diuretics and bronchodilators, he has significant relief of orthopnea but dyspnea on minimal activities remained. Patient actually left against medical advice there before further investigations and presented to our emergency after few hours from then in a state of acute pulmonary edema. Vital signs revealed normal body temperature, oxygen saturation of 60% at room air that increased to 92% on 10 L/min O_2 supplementation, pulse rate of 114 per min, BP of 144/88 mmHg and respiratory rate of 32 breaths per minute. Physical examination showed preserved mentation, dyspnea, distended jugular veins, generalized crackles in the lung auscultation and harsh pansystolic murmur at the apex and along the left sternal border. Acute pulmonary edema was diagnosed by the attending physician and intravenous infusion of furosemide and nitroglycerine was started and admitted in intensive care unit in view of possible need of mechanical ventilation for respiratory support. The cardiology consultation was sought then and there were ischemic ECG changes. Bedside echocardiography revealed hypokinesia of inferior wall with preserved ejection fraction with severe eccentric mitral regurgitation. The cardiac chambers were of normal dimensions suggesting the acuity of the regurgitation process. He had no known medical conditions prior to this admission. He was an active smoker with 90 pack years of smoking. At admission, his renal parameters were normal. Cardiac Troponin I was elevated (3.5 ng/ml) and N-terminal B-type natriuretic period level was elevated as well (9000 pg/ml). His glycated hemoglobin was 7.2% rest of the blood parameters were in normal range. He was shifted to coronary care units for standard of care and his cardiac biomarkers were elevated to suggest acute ischemia for the possible cause of regurgitation. Treatment for acute coronary syndrome was started, diuretics

and vasodilators were continued. Urgent coronary angiography was performed that showed right dominant circulation with 90% diffuse lesion in proximal LAD, 80% stenosis in mid LAD, 70% stenosis in right posterior descending artery and 80% stenosis in major posterolateral branch from right coronary artery. Left main coronary artery and left circumflex artery were free of atherosclerosis angiographically. He was subjected to transesophageal echocardiography under general anesthesia for further clarification of mechanism of mitral regurgitation. There was flail P3 segment of posterior mitral leaflet causing anteriorly directed severe mitral regurgitation with loss of support due to rupture of papillary muscle head. During period of extubation, he developed another episode of frank pulmonary edema with pink frothy sputum. He was continued with endotracheal intubation and referred to cardiothoracic surgeon for emergency surgical revascularization and mitral valve management. Surgical findings were consistent with TEE findings with posteromedial papillary muscle rupture with severance of chordae tendonae of medial scallop of posterior mitral leaflet. Attempt to repair the valve was not possible due to friable tissue, and a mechanical prosthesis (ONX #25/33) was implanted with preservation of subvavlular apparatus on next day. Concomitant coronary artery bypass was done with LIMA-LAD, SVG to PDA and SVG to PL branches. He was extubated after 7 hours postoperatively. Postoperative period was complicated by development of delirium and left sided pleural effusion. Effusion was treated by tube thoracostomy for 2 days. Then his general condition was improved and discharged home in a good condition on postoperative day 20.

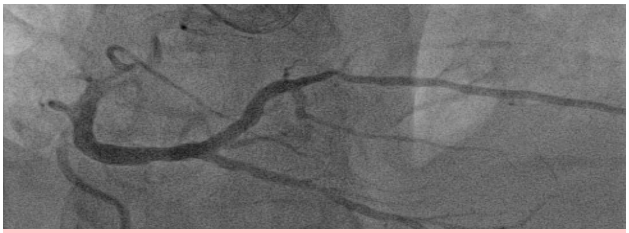

Figure 1. Right coronary angiogram showing significant lesions in Right posterior descending artery and right poserolateral branch.

DISCUSSION

Owing to prompt medical therapy and, timely revascularization in particular, has resulted in fewer mechanical complications today than before (about 6% to less than 1%).[1] A small proportion of patients however present with life-threatening complications due to increasing age, renal impairments, uncontrolled hypertension among others. In an

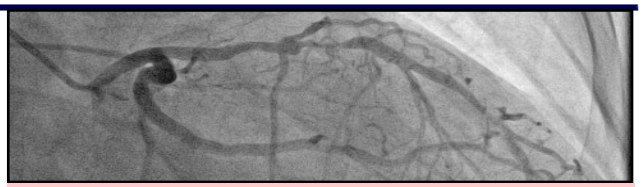

Figure 2. Left coronary angiogram showing critical diffuse lesion in proximal and mid left anterior descending artery.

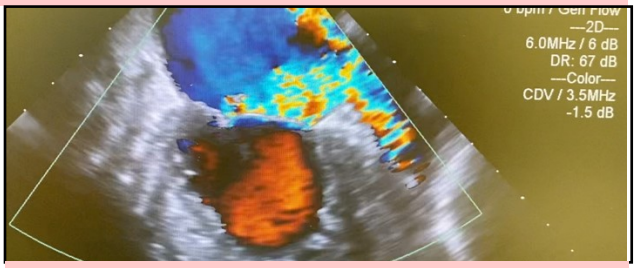

Figure 3. TEE: eccentric severe MR directed to the anteriorly towards LAA.

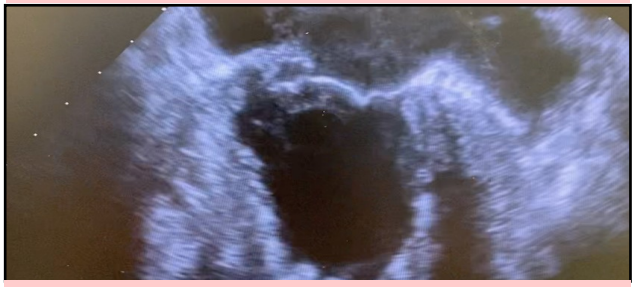

Figure 4. Flail posterior mitral valve leaflet (arrow).

Figure 5. Surgical specimen of mitral valve leaflets with ruptured chordae at the tip of PPM.

analysis of 4 million patients admitted with STEMI diagnosis between 2003 to 2015, the incidence of papillary muscle rupture/chordal rupture was only 0.05%.[2] However, the mortality rate is still very high at around 40%.[3] In cases of acute myocardial infarction, coagulative necrosis occurs within 24 hours and progresses to the softening of the infarcted myocardium. Granulation tissue and early collagen deposition helps to hold the tissues in these periods, but if the strength is not enough, it can lead to rupture either at the head or tip of the papillary muscle. So, it usually occurs in 2-7 days after the onset of chest pain.[4] Posterior papillary muscle rupture occurs much more frequently than anterior muscle because posterior muscle has singular supply from right posterior descending artery from the

dominant artery branch (75% vs 25%). About half of the patients has small myocardial infarction with preserved ejection fraction which is usually not the case with intraventricular muscle rupture or free wall rupture which occurs in extensive myocardial necrosis.[5] Complete transaction of the whole papillary muscle is usually fatal as heart is not prepared for such a torrential regurgitation. Rupture of a portion of it leads to flail of part of mitral valve leaflet causing moderate to severe mitral regurgitation that is not immediately fatal.[5] Anatomically anterolateral muscle usually has a large trunk whereas posteromedial muscle has one to three subheads. Each papillary muscle gives chordae attachment to both of the leaflets, so there may be prolapse of either of leaflet with either of the muscle rupture. Our case has a flail posterior mitral leaflet at its medial most (P3 segment) scallop. Transthoracic echo (TTE) is often the first modality to diagnosis for PMR, but owing to obesity, difficulty in positioning in acute condition, ongoing respiratory distress, among others for suboptimal acoustic windows, it is often very hard to delineate the exact anatomical defect. Transesophageal echo offers much superior quality and has diagnostic sensitivity of 95-100%. Use of color flow doppler and spectral doppler will necessary for the assessment of presence and severity. Once diagnosis is established, it is prudent to understand pathophysiology to stabilize the clinical condition and offer emergency surgical intervention. If necrosis is limited, it may be possible to repair the valve but often mitral valve replacement is necessary because of extensive myocardial necrosis as is our case. Of course, the surgical bypass of the coronary artery lesion is to be performed concomitantly. Preoperative management is dependent upon hemodynamic status. In unstable patients, its best advised for invasive monitoring with right heart catheter to guide the fluid therapy or diuretics and to direct the vasodilatory therapy. Inotropes support with or without intraaortic balloon counterpulsation is often required in those unstable cases.[4] Our case was hemodynamically stable and did not require for so. Surgery should not be delayed except for preparation and only if the multidisciplinary heart team along with the patient and proxies considers a high degree of futility. Low cardiac output state may be stabilized with support from Tandem heart and extracorporeal membrane oxygenation support in selected cases based on availability and expertise.[6] In modern era of percutaneous structural intervention, there are few reports of transcatheter mitral valve repair but this is in infancy at the current stage.[7]

CONCLUSION

Early revascularization and improved technique of intervention has drastically reduced all complications rates in acute myocardial infarction including mechanical complications, it should be strongly suspected in those with hemodynamic instability. Papillary muscle rupture is such a rare but potentially fatal complication that can be lifesaving if it is detected and managed in a timely fashion. Use of transesophageal echocardiography is often necessary to completely understand the nature of the anatomic derangement and the specific therapy can be instituted.

Conflict of Interest: None.

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