Acute Myocardial Infarction in a 20 year Pregnant Female with Prosthetic Mitral Valve

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Abstract
Myocardial infarction is very uncommon in young females. We are reporting a case of acute myocardial infarction in a 20 year pregnant female with prosthetic mitral valve. Embolism from mitral valve prosthesis, in-situ thrombosis due to hypercoagulable state of pregnancy or coronary spasm could be responsible.

Case Report
A 20 year female was referred for echocardiographic evaluation. She was pregnant (seven months) and complained of chest pain and increased breathlessness for three days. She had mitral valve replacement seven years back (Medronic Hall 25 mm). She was taking Tab. Frusemide (40 mg) once daily and Tab. Warfarin 3 mg/day. Examination revealed severe orthopnoea, pulse-124/minute regular, respiratory rate 50/minute, BP-100/50 mmHg, Temp. 98.4°F and bilateral basal crepts. Jugular venous pulsation could not be interpreted properly due to tachypnoea and use of accessory muscles of respiration. All arterial pulsation were normal. There was no bruit on any artery. There were no clinical signs of infective endocarditis, rheumatic activity or deep vein thrombosis.

Blood investigations showed Hb-10.3 gm%, TLC-16500/ cu mm, Polymorphs-80%, Lymphocytes 20%, International normalised ratio of 1.4, platelet counts 2.5 lakhs/cu mm, ESR-24 mm/1 hr, Urine- NAD. Electrocardiogram showed ST segment elevation with T wave inversion in leads III, aVF and right sided chest leads (V5 to V1) with ST depression in leads I, aVL and V1 to V6 (Figure 1). Transthoracic echocardiography revealed enlarged left atrium (LA volume index 59.2 ml/m2), normal left ventricular dimensions, (end-diastolic-46.3 mm, end systolic-24.6 mm) with mild hypokinesia of inferior wall of left ventricle and right ventricular anterior wall (Figures 2a and b). Prosthetic valve disc was moving normally in both parasternal long axis (Figures 3a and b) and apical four chamber views (Figures 4a and b). Mitral valve area calculated by pressure half time was 1.65 sqcm. No thrombus or vegetation was visible. There was no mitral regurgitation or aortic dissection. Transoesophageal echocardiography could not be performed because of marked orthopnoea.

After obtaining well-informed risk to foetus, patient was given slow iV infusion of inj. Frusemide and 20 lakh units of Urokinase. Her blood pressure rapidly fell inspite of inj. Dobutamine support. She did not tolerate saline infusion. She died of cardiogenic shock.

Discussion
Shock is an established complication of right ventricular infarction. Right ventricular infarction also produces breathlessness.
due to ventricular interaction. Marked breathlessness in this case could be partly due to patient-prosthesis mismatch with low effective mitral valve area.

Patient did not have any coronary risk factors and there was no family history of premature coronary artery disease or sudden cardiac death. There is high probability of non-atheromatous occlusion of coronary artery. Congenital anomalies of coronary arteries usually involve left system and cause effort angina, syncope and anterior myocardial infarction. Myocardial bridging also affects left anterior descending territory. Connective tissue diseases like Hurler syndrome, Homocystinuria, Ehler-Danlos syndrome, Pseudoxanthoma elasticum and Kawasaki disease cause ischaemic heart disease in children and have classical systemic features. Spontaneous coronary dissection is common in post-mortem period and mostly involves left anterior descending coronary. There were no clinical features of Polyarteritis nodosa, Giant cell arteritis, Scleroderma or Takayasu arteritis. It is possible that she had embolisation from mitral valve prosthesis. In-situ thrombosis due to hypercoagulable state of pregnancy could be another possibility. Coronary spasm can also produce myocardial infarction in young persons.

References