



## **Stunned Myocardium Due with hypoxia Secondary to Severe Pulmonary Artery Hypertension: A Rare case report**

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**Abstract**

*Stunned myocardium refers to a situation in which an acute transient myocardial ischemic event results in a prolonged wall motion abnormality which eventually resolves. 1 If ischemic myocardium is reperfused early before irreversible injury occurs, the affected myocardium will return to normal structure and function. It usually takes days or weeks for the heart muscle in the area to be able to contract again 2. There is, however, usually a long delay before normalcy is reestablished. We are reporting a case of stunned myocardium due to hypoxia secondary to severe pulmonary hypertension. Patient showed a great improvement when oxygen support given via high flow nasal cannula. Stunned myocardium due to severe pulmonary hypertension is a rare entity.*

**Case Report**

A 23 old female who was a known case of bronchiectasis with severe pulmonary hypertension presented to our emergency department with complaints of increase in breathlessness, cough with sputum and high grade fever since 5 days. Patient was on long term oxygen therapy since last 1 year. She had history of recurrent childhood pneumonia in past. She was non diabetic, non hypertensive. There was no history of pulmonary tuberculosis in past. She was giving negative history for head trauma, seizure and headaches. On arrival her vitals were as follow: conscious, dyspneic, oxygen saturation 58% on room air (94% on 5 liters/min oxygen via nasal prong), blood pressure 88/62 mmhg, Pulse 40/min. respiratory rate 36/min, temperature 98.8 F. On inspection there was use of accessory respiratory muscle use. On auscultation there was crepitation in bilaterally infrascapular regions and loud P2 was noticed. Electrocardiogram revealed sinus bradycardia with T inversion V2 to V6 leads. Her cardiac enzymes were within normal limits. Chest radio-graph showed bronchiectatic changes in left lower zone and dilated main pulmonary artery. Arterial blood gases in emergency were as follows: PH 7.42, PCO<sub>2</sub> -58, PO<sub>2</sub>- 41, HCO<sub>3</sub>-37, Sodium 135, Potassium 4.1, lactate 2.1. Other blood investigations were as follows- Hemoglobin 12.9 gm/l, leucocyte- 12990/mm<sup>3</sup>, platelets 2.3 lakh/mm<sup>3</sup>, serum magnesium 2.08 mg/dl, liver and kidney functions were in normal limits. Her random blood sugar was 148 mg/dl and urine examinations were also normal. Patient was in type 1 respiratory failure for which high flow nasal cannula support was given. Repeated 2D electrocardiogram was suggestive of severe pulmonary artery hypertension with stunned myocardium. Dobutamine infusion was started at 3 microgram/kg

which helped in improving pulse rate and blood pressure. Patient's ECG was also improving. Patient's sputum culture was positive for kleibseilla pneumonia, so antibiotics were changed as per sensitivity pattern. With HFNC support and good antimicrobial coverage showed a good response. On 3rd day patients dobutamine support was tapered. Now patient was stable on 2 liters/min oxygen via nasal prong , blood pressure 108/68 mmHg without any vasopressure support and pulse was 76/min. Her latest ECG showed improvement in rate and rhythm. Patient was on long term oxygen therapy for severe pulmonary hypertension and secondary bacterial infection caused exacerbation which lead to increased oxygen demand of body and hypoxia developed. Dobutamine stress Echo confirmed the stunned myocardium. Thus case was diagnosed as stunned myocardium due to hypoxia.

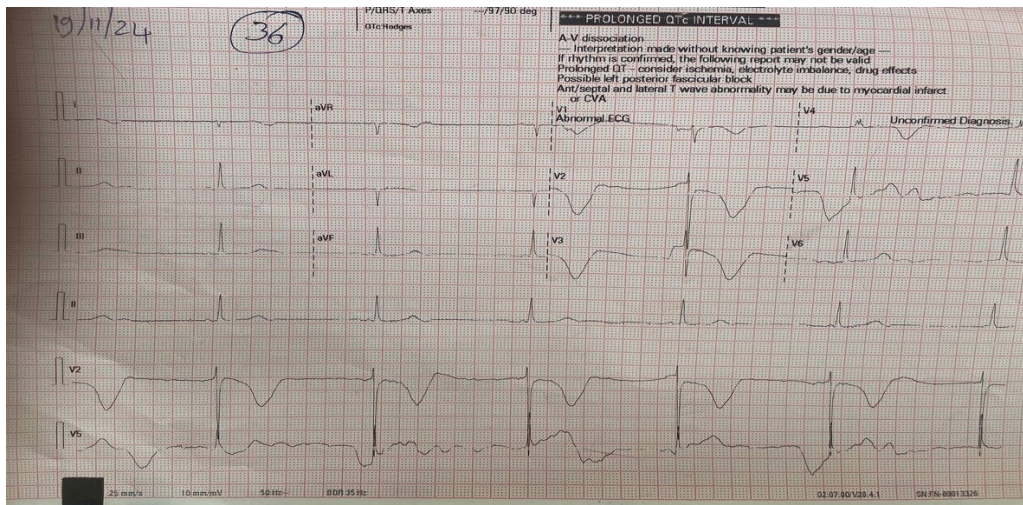


Figure 1- ECG on day of presentation

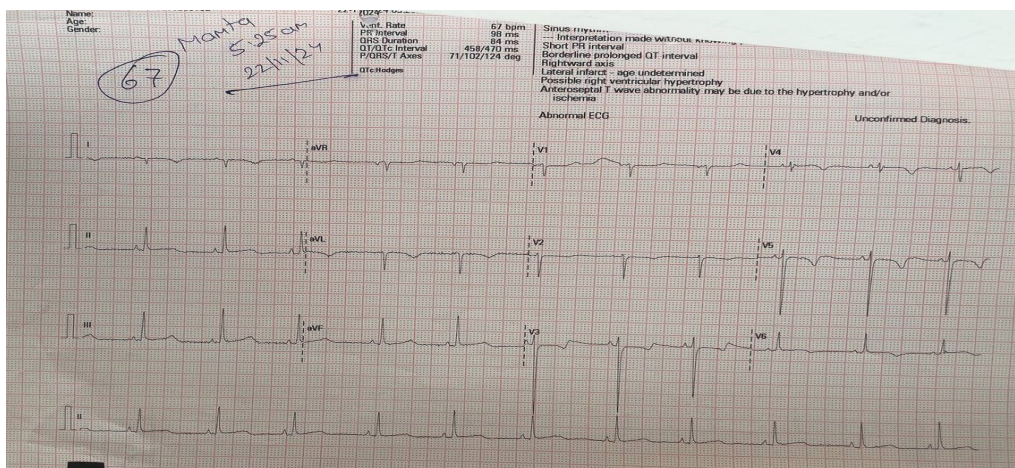


Figure 2- ECG after management of hypoxia

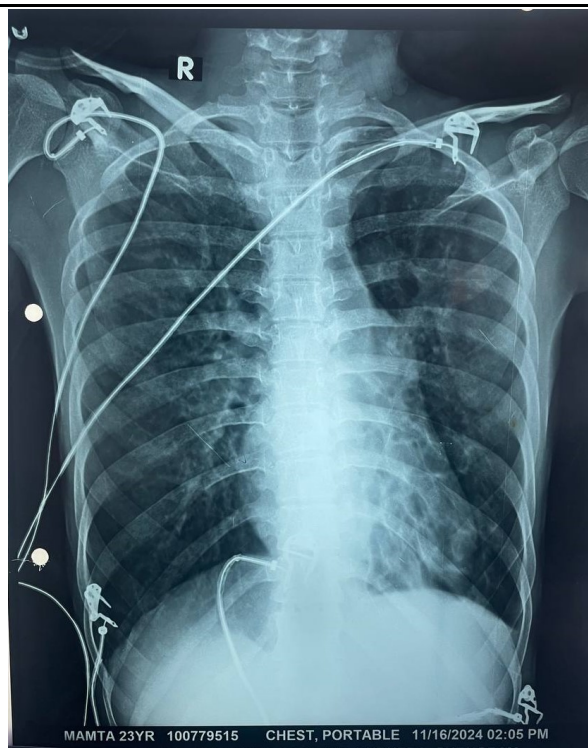


Figure 3- Chest Xray PA view

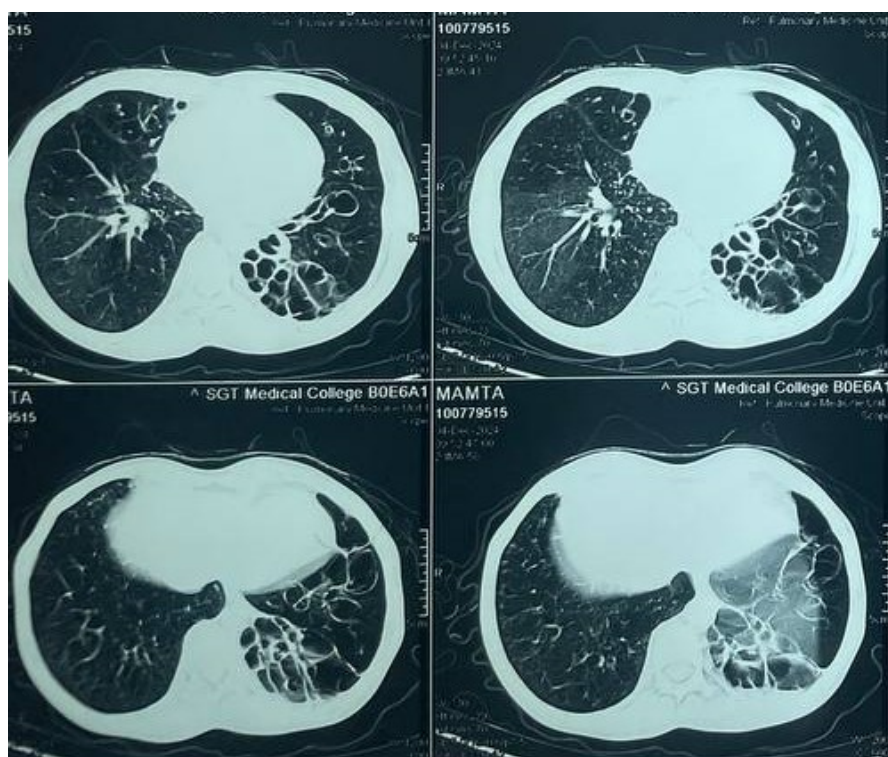
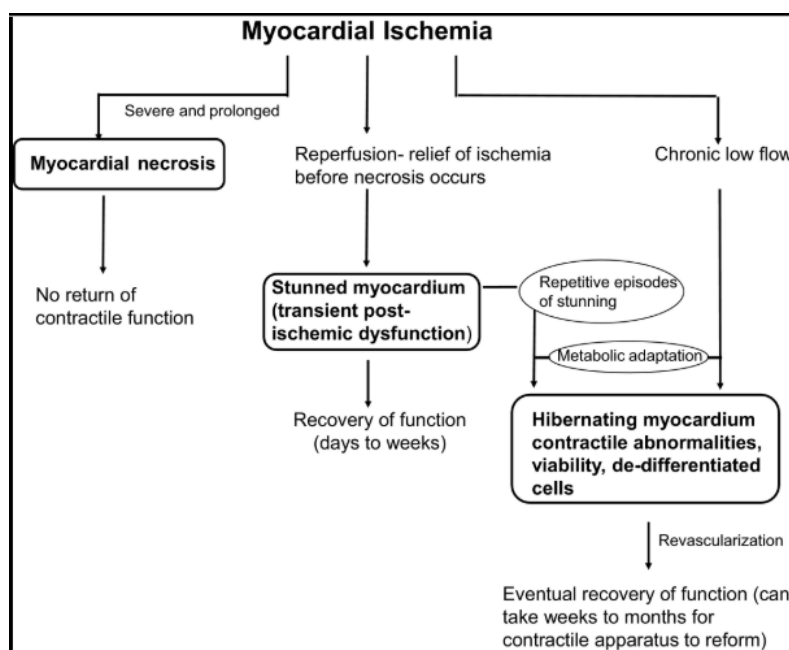


Figure 4- Computer Tomography of chest

## Discussion

Reperfusion after myocardial ischemia of brief duration does not induce necrosis, but results in prolonged contractile dysfunction 3-5. This phenomenon, which has come to be known as myocardial stunning 6, is manifested clinically in the sluggish recovery of pump function after coronary revascularization after brief periods of ischemia. Stunned myocardium refers to a situation in which an acute transient myocardial ischemic event results in a prolonged wall motion abnormality which eventually resolves. This condition is more rapid and occurs over minutes or hours when the supply of blood or nutrients to your heart muscle suddenly drops or stops altogether. Your heart muscle goes into a “stunned” state where it stops contracting. If ischemic myocardium is reperfused early before irreversible injury occurs, the affected myocardium will return to normal structure and function. It usually takes days or weeks for the heart muscle in the area to be able to contract again. There is, however, usually a long delay before normalcy is reestablished. Systolic and diastolic dysfunction and metabolic changes may persist for 1 week. Hence, the myocardium is “stunned.”



Biochemical changes implicated in myocardial stunning include (1) depression of ATP content by as much as 50%; (2) abnormal calcium transport; (3) interruption of the creatine phosphate shuttle; and (4) abnormalities of cardiac sympathetic innervation. 7-8

The morphological changes observed in stunned myocardium range from none, to those observed in early reversible ischemic injury: nuclear chromatin clumping and margination, glycogen depletion, edema, evidence of I bands, and only minimal mitochondrial swelling. Interestingly, scanning electron microscopic studies of stunned myocardium has shown marked disruption of the extracellular collagen matrix, with destruction and loss of extracellular matrix components associated with an increase in myocardial compliance. 9 These findings in the extracellular matrix early after ischemic injury provide a structural explanation for the abnormal contractile function of stunned myocardium. but some areas of heart muscle don't recover right away. Stunned myocardium can also result from other conditions, including Stroke, Stress, Cardiomyopathy, Subarachnoid hemorrhage, Seizure 10

These assessments distinguish between a stunned or hibernating myocardium that can be revived and scar tissue that can't. . Dobutamine stress echo cardiogram and Cardiac MRI can help in diagnosing stunned myocardium.11

Nuclear imaging techniques have documented the presence of stunned myocardium following episodes of unstable angina.9 Recent studies suggest that adding a measure of wall thickening in addition to EF measure on single-photon emission computed tomography imaging, improved the ability to diagnose stunned myocardium.11-12 There is ongoing controversy regarding the meaning of biomarker release of creatine kinase–myocardial band and cardiac troponin I and T associated with stunned myocardium. Release of these markers, typically associated with MI, into the blood stream have been well documented in experimental models of brief ischemia and reperfusion, not usually associated with MI.13-14 Cardiac troponin has also been detected in the circulation after exercise or pacing-induced ischemia,15 as well as after vigorous bouts of exercise not necessarily associated with known ischemia.

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