

RESEARCH ARTICLE

EXTENSIVE ANTERIOR WALL ST ELEVATED MYOCARDIAL INFARCTION FOLLOWING STEERING WHEEL IMPACT AND BLUNT CHEST TRAUMA IN A ROAD TRAFFIC ACCIDENT: A RARE CASE REPROT

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Key words:-

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Abstract

Background: Blunt trauma chest may rarely lead to acute myocardial infarction. Shear force generated from trauma causes tearing, laceration of coronary vascular intima and results in intraluminal thrombosis. Left anterior descending (LAD) artery is the most common to be involved secondary to its proximity to anterior chest wall.

Case Presentation: We report a case of 38year old hypertensive male presented in emergency room with complaints of left sided chest pain & diaphoresis for one hour following trauma to his chest from steering wheel following a collision between two four wheeler. During primary survey as per ATLS guideline 12 ECG revealed acute extensive anterior wall STEMI. Other associated injuries were left frontal non hemorrhagic contusion, bilateral minimal pneumothorax, multiple bilateral rib fractures, mild hemoperitoneum with small hematoma in retroperitoneum and serosal surface of stomach. Urgent coronary angiography done by cardiologist on call and patient was diagnosed with single vessel coronary artery disease involving LAD with severe systolic LV dysfunction. Percutaneous transmural coronary angioplasty (PTCA) was done by a drug eluting stent in LAD.

Conclusion: An emergencyphysician should consider cardiac complications in patients with chest trauma including myocardial infarction, early diagnosis of which is critical to save the myocardium. Any delay in diagnosis can be detrimental.

What we already know? There are quite a few case reports that already mentioned about possibility of acute myocardial infarction after blunt chest trauma following road traffic accident secondary to intimal laceration of coronary artery and intraluminal thrombosis. Most coomonly left anterior descending artery is involved.

What this paper adds? This is rare case report of acute & extensive anterior wall myocardial infarction secondary to single vessle coronary artery involvement after blunt chest trauma by steering wheel in a road traffic accident. We specifically points towards the fact that noobvious Clinical guideline to manage these kind of cases mentioned in literature. Hence proper clinical guidelines to manage such kind of cases. Also, in acute myocardial infarction post blunt trauma chest, PCI seems to be a better option than thrombolysis keeping in mind of other injuries.

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Introduction:-

Road traffic accidents are leading cause of death in both developed and developing countries. Approximately 1.35 million people die each year in road traffic crashes. It is also a leading cause of death in young adults and third most common cause of in all age group. Though low and meddle income countries possesses60% approximately of world's vehicle, these countries are accountable for 93% of world's fatalities on roads¹. Thoracic trauma consists of 35% of trauma related deaths and significant mortality morbidity. Thoracic trauma is broadly classified as blunt and penetrating injuries, blunt chest trauma being commoner one, directly comprises 20% - 25% of trauma related deaths. Despite having a higher incident of blunt trauma chest only 10% cases need operative interventions. In penetrating trauma chest almost 15-30% need operative interventions and overall a higher mortality². Blunt cardiac injury (BCI) is a common complication after blunt chest trauma, which can lead to mild arrhythmia, severe chamber or valvular rupture, coronary artery dissection, or even death. Myocardial infarction following blunt chest trauma is a rare but fatal condition^{3, 4}. Multiple case reports have been reported secondary to involvement of left anterior descending artery (LAD), may be due to its proximity to the chest wall. Traumatic shear pressure leads to intimal tear or intraluminal thrombosis. Vascular rupture, embolism to the coronary arteries, fissuring of an atherosclerotic plaque with dislodgment of plaque material, and vascular spasm at the site of the injury are also potential triggers^{5,6}.

Case presentation:

We report a case of 38 year old non obese male patient who had a positive significant history of hypertension on regular medication, non diabetic, no family history of coronary artery disease (CAD) or hyperlipidaemia presented to emergency room (ER) with complaints of left sided moderately severe chest pain associated with diaphoresis following blunt trauma chest by steering wheel while driving a SUV four wheeler which collided with another four wheeler approximately an hour from his presentation in ER. He denied any history of head or neck injury or loss of consciousness or amnesia.

On presentation, his airway was patent. Bilateral air entry of chest was equal with bilateral equal chest rise. Left side of chest was diffusely tender on palpation. His blood pressure (BP) was on lower side 100/70 mmHg, Heart Rate(HR)=108bpm, SpO2= 98% on room air, Respiratory Rate(RR)=24/min, temperature(T)= normal, Capillary blood glucose(CBG)=119mg/dl. He was conscious, alert and oriented, GCS: 15/15.Denied any neck pain. His pupils were bilaterally equal and reacting to light. No apparent neurodeficit was noticed.

During primary survey his 12 leads ECG revealed something which shocked me, though as an emergency physician I always anticipate few and far between. What I witnessed was significant ST segment elevations in lead I, AVL, V2-V6 with reciprocal depression in lead III, aVF, aVR suggestive of an extensive ST elevated myocardial infarction.

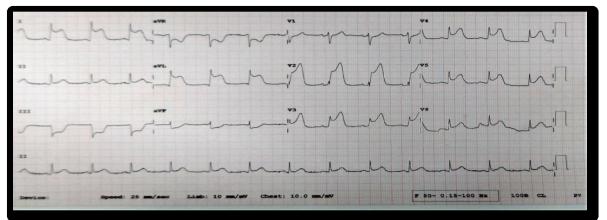


Figure 1:- Extensive Anterior Wall MI - ST Elevation in lead 1, AVL, V2-V6 with reciprocal ST depression lead III, AVF, AVR.

Patient's Non Contrast CT scan brain: small non hemorrhagic contusion right frontal lobe.

Non Contrast CT thorax revealed:minimal bilateral pneumothorax (right>left). No evidence of hemothorax. Bilateral lung contusions involving bilateral upper lobes, right middle lobe, lingual, bilateral lower lobes. Mildly displaced fracture of left anterior $3^{rd} - 8^{th}$ ribs, left posterior 6^{th} & 7^{th} ribs, right anterior 5^{th} , 6^{th} , 7^{th} ribs.Small subcutaneous hematoma over left lower ribs. Cardiac shape appeared withing normal limits.

Non Contrast CT Abdomen revealed: mild hemoperitoneum with small hematoma in retroperitoneum and serosal surface of stomach. Tiny left renal calculus (2mm).

Cardiologist was involved earliest. Bedside echocardiography impression was a moderate left ventricular systolic dysfunction with left ventricular ejection fraction of 38%. After ruling out any great vessel dissection, loading dose of aspirin (325mg), clopidogrel (300mg), atorvastation (80mg) orally and intravenous heparin 5000 IUgiven preceding a detailed discussion with cardiologist and general surgery team about the risk of secondary increase of bleed to benefit ratio. It was initially difficult to convince patient's family how the patient in this age had a heart attack after motor vehicle collision and blunt trauma chest. But fortunately after persuasion and immediate involvement of cardiologist on call, with family's consent a primary coronary angiography done. It revealed proximal acute thrombotic occlusion of left anterior descending (LAD) artery. As a revascularisation strategy primary percutaneous transmural coronary angioplasty (PTCA) was done by a drug eluting stent in LAD.

Post procedure patient remained in a state of cardiogenic shock with sinus tachycardia. He was kept in ICCU for next 2 days and managed with abciximab (antiplatelet, a glycoprotein IIb/IIIa) iv infusion for 6 hours, prasugrel, aspirin, statin, ionotrop& vasopressor infusion (Noradrenaline), antibiotic and other supportive medications.

Eventually patient recovered from cardiogenic shock and discharged on prasugrel, aspirin, statin, proton pump inhibitor on day 4.

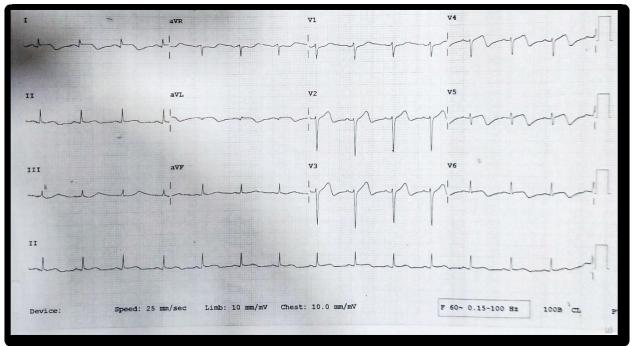
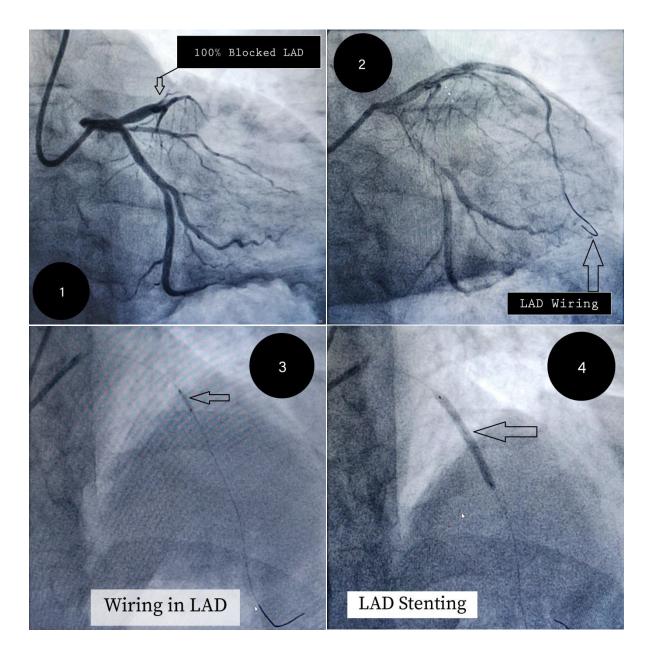


Figure 2:- Post PTCA 12 LEAD ECG : Resolved ST segment elevation in lead I, AVL, V2 - V6.



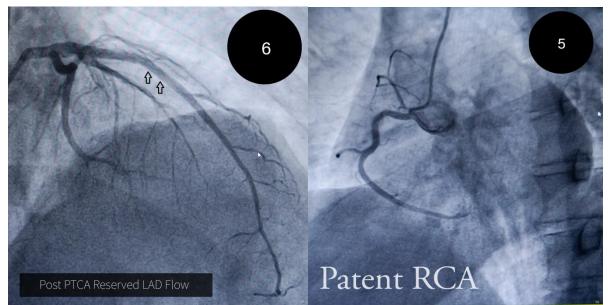


Figure 3:- CAG showing Single Vessel Disease. Primary PTCA with DES to LAD.

Discussion and Conclusion:-

Among the non-atherosclerotic aetiologies of acute myocardial infarction (AMI) in young adults, blunt chest trauma is one of the extremely rare mechanisms. Also, AMI can be a rare but catastrophic complication of blunt trauma chest. Christensen et al. described 77 cases of blunt trauma chest causing AMI, among which 64% resulting from road traffic accident and 4% related to assault. Literature review suggests the mechanism leading to an AMI in cases of blunt trauma chest is shearing force to the coronary artery resulting in intimal tear, precipitating platelet aggregation and intraluminal thrombosis in coronary artery. It has been noticed from previous case reports that LAD is the most commonly involved, because of its proximity to the chest wall. The second most commonly affected artery is the right coronary artery, which is most vulnerable at its origin, perhaps because of acceleration/deceleration injury. The left main artery and left circumflex artery are seldom involved⁸.

If missed or delay in diagnosing AMI in blunt trauma chest it may result in catastrophic consequences. However, these are not easy as many of these cases are often unrecognized and treated late because traumatic chest pain sometimes masks angina pain and is interpreted as being secondary to chest contusion or is masked by combined injuries. According to the practice guidelines of the Eastern Association for the Surgery of Trauma, all patients suspected of having BCI should undergo ECG during admission and then serial ECGs and cardiac troponins-I (cTnI)^{9, 10}. Echocardiography is necessary in patients with compromised hemodynamic to rule out mechanical complications, such as cardiac tamponed, ventricle rupture, or valve injury¹¹. The combination of norm ECG and negative cTnI value 4-6 hours post injury almost excludes any cardiac involvement, with negative predictive value ranging from 98% to 100%. These patients can be safely discharged home¹².

The management of AMI after blunt trauma chest remains controversial and lacks proper guideline. Emergency coronary angiography should be performed as soon as possible. Emergency percutaneous intervention (PCI) is the best treatment of choice as it is minimally invasive in comparison to emergency surgical coronary artery bypass graft (CABG) and avoid the high risk of bleeding from associated injuries with thrombolytic therapy^{13, 14}.

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