



A CASE REPORTING OF MANAGEMENT OF INFERIOR WALL ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION: A REVIEW

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ABSTRACT

Inferior ST-Segment myocardial infarction most commonly due thrombotic coronary artery occlusion at the site of ruptured pre-existing atherosclerotic plaques. Primary Percutaneous coronary intervention is the best reperfusion therapy for patient with ST-segment elevation myocardial infarction. Here we report a case of 77 years male patient with diagnosed with inferior ST-Segment elevation myocardial infarction. The patient received emergency coronary angiography which shows occlusion of middle segment of circumflex artery then emergency primary Percutaneous coronary intervention was done in circumflex artery.

Keywords: ST-Segment elevation myocardial infarction, reperfusion therapy, primary percutaneous coronary intervention, Fibrinolytic therapy.

INTRODUCTION

ST-segment elevation myocardial infarction remains a major cause of premature death worldwide. ST-Segment elevation myocardial infarction accounted for 39% of all hospital admission due to myocardial infarction in UK. Most of ST-segment elevation myocardial infarction is caused by acute thrombotic coronary artery occlusion at the site of ruptured pre-existing atherosclerotic plaque with plaque erosion and calcified nodules[1]. Therefore, expeditious restoration of vessel patency represents the cornerstone of treatment of this conditions and although widespread uptake of primary Percutaneous coronary intervention has significantly improved outcomes. Coronary occlusion for more than 20 minutes results in irreversible damage to cardiac myocytes and nearly half of potentially salvageable myocardium is lost within the first hours [2]. The extent of myocardial cell death is dependent on the size of myocardium territory supplied by culprit artery, duration of occlusion and presence of collateral circulations; larger myocardial infarcts are more likely to cause death or cardiac failure.

Case Reporting:

A 77 years old male patient with known case of hypertension and type-2 Diabetes mellitus came in emergency department in our hospital with chief complain of central chest pain for one hour, which radiates to left arm, neck and shoulder which is squeezing in characters. Chest pain is associated with shortness of breath and dizziness. Patient was chronic smokers with no known case of heart disease. His blood pressure and pulse rate was 160/90 mm of hg and 92 beat/ minute respectively. On systematic examination there is a bilateral crepitations and no other added sound is heard. Chest x-ray show cardiomegaly with little pleural effusion on left side and right side is normal. The 12 lead electrocardiogram showing sinus tachycardia with hyperacute T waves in inferior lead and reciprocal ST-segment depression in inferior leads. The second electrocardiogram was taken 40 minutes later show normal sinus rhythm with 2 mm of inferiors ST-segment elevations (Figures.1).

On laboratory investigation total Red blood cell counts was 4.8 million/mm³, hemoglobin was 12 gm/dl, Total white blood cells count was 8 thousand/mm³, Total platelet count was 2.5 lakh/mm³, Random blood sugar was 90 mg/dl, Blood urea was 15 mg/dl, serum creatine was 1.2 mg/dl, sodium was 142 mmol/litre, potassium was 4.2 mmol/litre, CK-MB was 62 U/L, Troponin-I was 5689.40 pg/ml. Thyroid function test was within the normal range. Coagulation profile report was within the normal ranges.

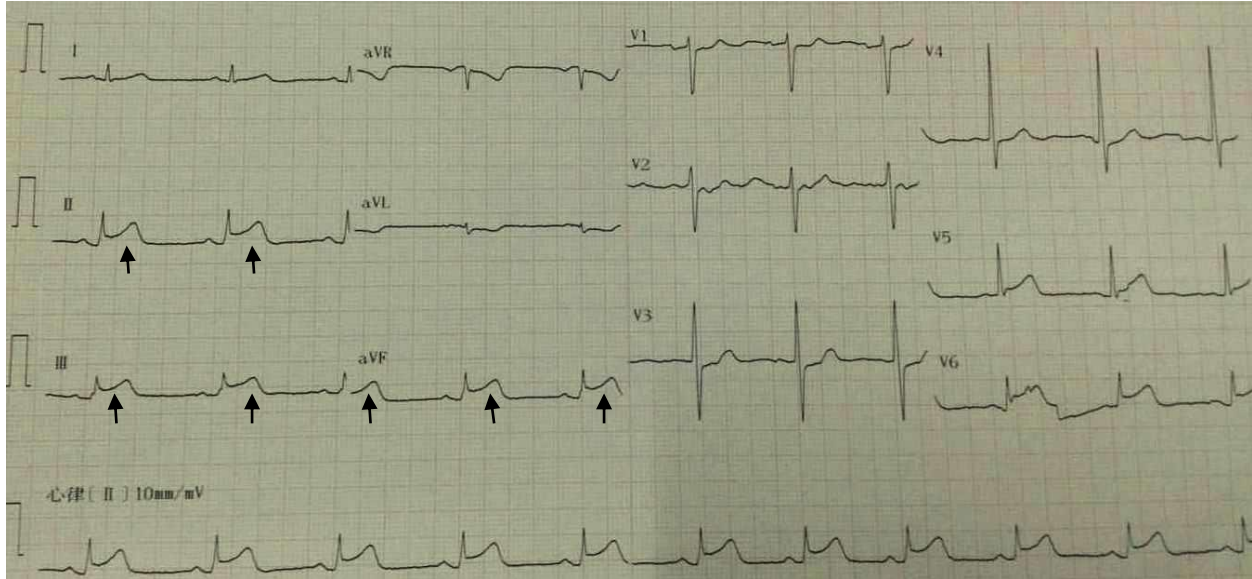


Figure 1: Electrocardiogram show normal axis with normal sinus rhythm with ST-Segment elevation in inferior lead that is in lead II, III and aVF.

With diagnosis of acute inferior wall myocardial infarction, loading dose of Tab Aspirin 300 mg, Tab Clopidogrel 300 mg, Tab metoprolol 12.5 mg and IV morphine sulphate 5-10 mg with IV metoclopramide 10 mg was given with high flow of oxygen. Patient was kept on electrocardiogram monitoring and intravenous access was done. Patient was immediately transferred to catheterization laboratory for emergency angiography through transfemoral approach. Coronary angiography shows severe occlusion in middle portion of Circumflex artery, left anterior descending artery and Right coronary artery was normal and no stenosis was seen(Figure-3). The primary Percutaneous coronary intervention was done on Circumflex artery lesion and successful balloon angioplasty (simpass 2×15 mm, 12 atm AlviMedica, Istanbul, Turkey) with Stenting (bare-metal stent, Ephesus 3×18 mm, 12 atm Medtronic Mineapolis, USA) was performed. The 12 lead electrocardiograms were done after two hours of primary Percutaneous coronary intervention show complete ST-segment resolution (figure-2).

No any PCI related complications occurred during hospitalization and patient was discharged from the hospital after 7 days without any symptom on oral medication, Tab Aspirin 75 mg, Tab Atrovastatin 20 mg, Tab Metoprolol 12.5 mg, Tab Enalapril 10 mg. Echocardiography was done after 5 days of PCI was normal except for concentric left ventricular hypertrophy and grade-1 diastolic dysfunction. He has normal ejection fraction without regional wall motion abnormalities.

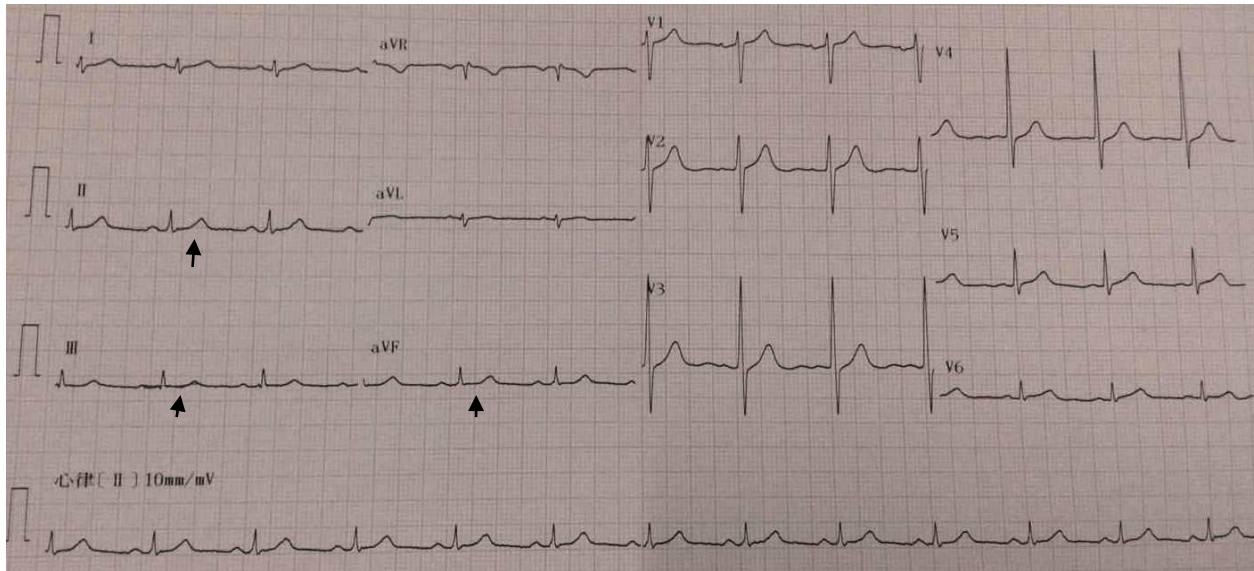


Figure 2: ECG obtained after two hours after PCI showing complete ST-segment resolution.

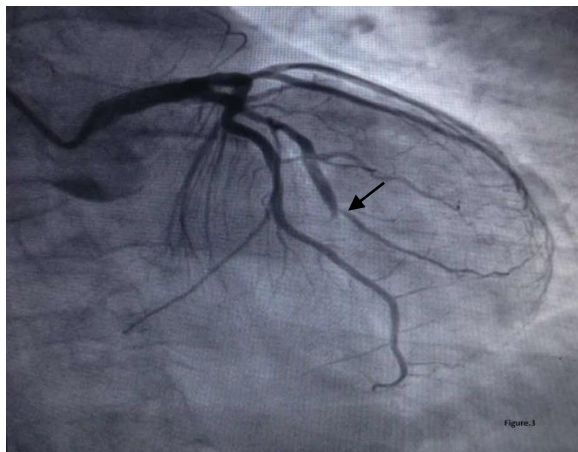


Figure 3: Coronary angiogram showing complete occlusion in middle segment of Circumflex artery

Figure 4: Primary angioplasty-Coronary angiogram showing complete restoration of normal flow following intracoronary stent implantation

DISCUSSION

Diagnosis of ST-Segment elevation myocardial infarction (STEMI) is based on the history, cardiac markers and Electrocardiogram changes. Although STEMI may be silent or may present with sudden cardiac death, majority of patient present with typical ischemic type of chest discomfort along with ST-Segment elevation in Electrocardiogram with reciprocal ST-Segment depression on others leads. Echocardiography may be useful in ruling out acute ST-Segment elevation myocardial infarction by demonstrating absence of regional wall motion abnormalities. Raised cardiac biomarkers confirm the diagnosis of STEMI. Troponin-I measurement are favoured over other biomarkers because of superior sensitivity and specificity.

The key priority of the management of STEMI is rapid restorations of vessel patency in order to maximize myocardial salvage [3]. Reperfusion therapy is recommended in patient who present within 12 hours of symptom onset and have persistent ST-Segment elevation or new left bundle branch block. Beyond 12 hours, reperfusion therapy recommended in patient with ongoing chest pain and persisting ST-Segment elevation or in those with cardiogenic shock [4].primary Percutaneous coronary intervention (PCI) is the best reperfusion therapy for patient with STEMI. The primary PCI is more effective than Fibrinolytic and preferred in experienced centre capable of performing the procedure rapidly specially when diagnosis is doubtful, Cardiogenic shock, risk of bleeding and symptom have been present for > 3 hours. Proceed with IV Fibrinolytic if PCI is not available or if delay in PCI > 1 hour longer than Fibrinolytic could be initiated, those treated within 1-3 hours benefit is most but can be used upto 12 hour if chest pain is persistent or ST-Segment elevated and not developed pathological Q-wave. If chest pain persists or ST-Segment elevation persists > 90 minute after Fibrinolytic consider referral for rescue PCI.

Table-1.selection criteria for Fibrinolytic therapy:

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| <ol style="list-style-type: none"> 1. Acute chest discomfort characteristic of myocardial infarction. 2. ECG Criteria for ST-segment elevation myocardial infarction. <ol style="list-style-type: none"> a. ST elevation \geq 1mm in at least 2 lead of either: <ul style="list-style-type: none"> -inferior group: II, III, aVF. -Lateral group: I, aVL, V₅, V₆ b. ST elevation \geq 2mm in at least 2 contiguous anterior lead (V₁-V₄). c. New left bundle branch block. 3. Primary PCI not available, or delay to PCI from first medical contact could be >120 min. |
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Current UK recommendations are maximum call to balloon time (time between call for help and balloon inflation) of < 150 minute and door to balloon time (time between arrival of PCI- capable centre and first balloon inflation) of < 90 minute. In situation where is an expected delay > 120 minute before primary PCI can be delivered or when patient present very early (within 2 hours) after symptom onset, Fibrinolytic therapy may have considered in preference to PCI because equivalent outcomes.

Table-2. Contraindication of Fibrinolytic therapy:

- Active internal bleeding
- Previous subarachnoid or intracerebral haemorrhage
- Uncontrolled hypertension
- Recent surgery within one month
- Recent trauma
- High probability of active peptic ulcers
- Pregnancy
- Aortic dissection
- INR \geq 2 on warfarin

In the treatment of STEMI in general, extensive evidences favours use of dual antiplatelet therapy (DAPT) with aspirin and a P2Y₁₂ receptors blockers [5] with additional indications still where primary PCI is performed and stents implanted. The newer P2Y₁₂ agent's ticagrelor and prasugrel have a more rapid onset of antiplatelet activity and more predictable and reliable platelet inhibitions. The OASIS-7 trial of 600 mg vs 300 mg Clopidogrel loading dose in ACS patients suggested a significant reduction in risk of cardiovascular death, myocardial infarction or stroke at 30 days as well as in stent thrombosis with higher dose [6]. A recent meta-analysis has shown that complete revascularization at the time of PPCI result in a reduction in major adverse cardiac event rates, reduction in death or myocardial infarction [7].

Post revascularization care:

All patients should initially be monitored on coronary care unit and should receive secondary preventative therapies including dual antiplatelet therapy, beta blockers, Angiotensin converting enzyme inhibitors (ACE inhibitors) and high dose of statin. Initiations of beta blocker and ACE inhibitor are early within 24 hours, unless there is specific contraindication. In patient with left ventricular ejection fractions is less than 40%, heart failure and diabetes mellitus eplerenone is recommended. All patients should have echocardiography to asses' left ventricular ejection fraction and to detect regional wall motion abnormalities. Smoking cessation advice should be given and all eligible patients should be offered an exercise based cardiac rehabilitation programmed.

The optimal duration of dual antiplatelet therapy in post PPCI is debatable but according to European society of cardiology guidelines recommended 12 month, although 6 month may be safe [8]. The shorter duration of DAPT recommended in high bleeding risk patient and extended DAPT in low bleeding risk patient with high ischemic risk patients. In patient with an indication for oral anticoagulation (OAC) therapy (atrial fibrillation), guidelines recommended triple therapy (DAPT+ OAC) are given for six week followed by OAC and antiplatelet monotherapy for life.

STEMI may be complicated by cardiogenic shock in approx 6% patients [9] and such patient have 50% mortality rate and such patient need early revascularization, inotropic support and mechanical assist device.

Other complications are acute mitral regurgitation, ventricular rupture, tamponade, left ventricular thrombus and embolism, arrhythmias and pericarditis. Patient with impaired left ventricular function need repeat echocardiography at 40 days post STEMI to assess their eligibility for implantable cardioverter defibrillators. Follow up should be arranged with cardiologist for ongoing care in outpatient setting.

CONCLUSION

Primary Percutaneous coronary intervention seems to best perfusion for ST-Segment elevation myocardial infarction within 12 hour of onset of symptom. Thrombolytic therapy is the standard therapy for STEMI who are more than 120 minute away from the hospital that is capable for performing primary percutaneous coronary intervention.

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