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# Haemodynamic Changes in Patients Undergoing Coronary Stenting for Miocardial Infarction

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**Abstract:** Acute Coronary Syndrome or ACS in short has become a bread and butter case scenario seen in every hospital over the whole world with increasing industrialization and people depending more on fast food based diet and less exercise. ACS was previously known as Myocardial Infarction which in technicalities means rupture of plaque formation in the coronary arteries. It is associated with atherosclerosis and hyperlipidemia. As well as increased lipolysis forming Plaque and Emboli (can be septic also). Problems are exacerbated by other co morbidities like hypertension, Diabetes, calcification of the vessels. For management part, thrombolysis is used followed by PCI.

**Keywords:** Acute Coronary Syndrome, Percutaneous Coronary Intervention, Thrombolysis, hypertension, hyperlipidemia, obesity.

**Introduction:** Acute Coronary Syndrome, i.e. ACS which can be further sub classified into ACS with ST elevation seen on Electrocardiogram (STE-ACS/ STEMI) and ACS without any ST elevation visible on Electrocardiogram (NSTE-ACS). So, to simply state it is adequate to state that Ischemic heart disease is improper perfusion of myocardial tissue which can be due to following different causes:

- Plaque formation
- Emboli
- Spasm (Prinzmetal Angina)
- Vasculitis (Takayasu in adults and Kawasaki in children).

To, understand more about the consequences of plaque rupture, we need to understand the coronary anatomy first. So, let us mention about the cardiac dominance.

Cardiac dominance is determined by precursor of Posterior Descending Artery which supplies the posterior inter ventricular sulcus. If the PDA arises from Right Coronary Artery then the person is called as Right cardiac dominant. Vice versa, if the PDA arises from Left Circumflex Artery then the person is referred to be as left cardiac dominant.

Right Coronary Artery:

Originates from Right AV sulcus.

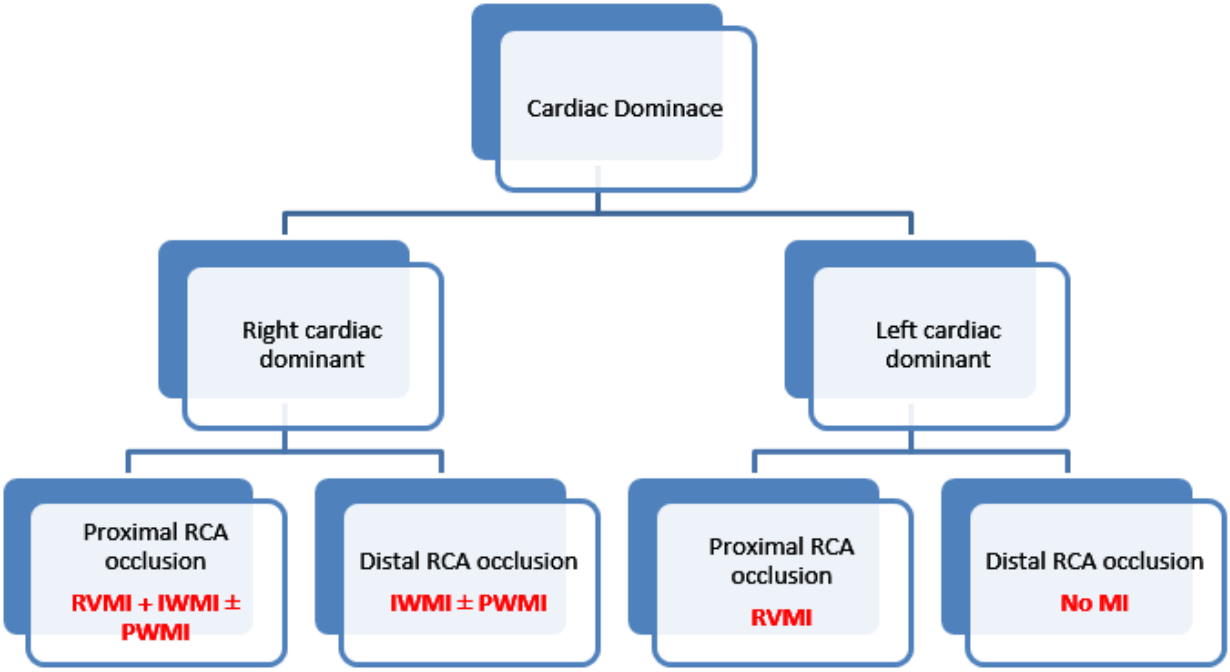
Branches of Right coronary artery:

1) SA nodal artery- Proximal RCA occlusion can produce Sinus Bradycardia, with this it is important to note that bradycardia in right coronary circulation related MI is not always due

to proximal RCA occlusion. Bradycardia can also be due to vagal afferent stimulation or nodal ischemia.

2) Atrial branches: In the RA-RV sulcus, hence proximal RCA occlusion can lead to Atrial Arrhythmias.
3) Acute Marginal Artery: Supplies RV free wall and occlusion of RCA is of two types- Proximal RCA occlusion (proximal to acute marginal artery) leading to RWMI, which is always present in such case. Distal RCA occlusion (distal to acute marginal artery) leading to absence of RWMI.
4) Branch to AV node – posterior AV sulcus.
5) Terminal branches are posteriobasal (15-20% of LV) and posterior descending artery (PDA)

When PDA is involved: IWMI ± PWMI.




Features of RWMI:

Hypotension + Clear lung field + Increased JVP

Features of IWMI:

SA/AV/complete nodal blocks, Atrial tachycardia, RVMI, papillary muscle rupture leading to secondary MR.

Left Circulation: Left coronary artery divides into Left circumflex artery(LCx) and Left anterior descending artery (LAD) further sub branching into:

- D1 = Diagonal 1 ( high lateral wall )
- S1 = Septal 1 (Septal wall )
- D2 = Diagonal 2 (Anterior wall )
- D3 = Diagonal 3 (Lateral wall )

Hence, the occlusion could be present Before D1 or between D1 and S1 or below S1, i.e, either between S1 and D2 or between D2 and D3.

The currently accepted definition of Myocardial Infarction: It is defined as Myocardial injury with clinical evidence of Myocardial ischemia.

Significant Myocardial injury is when Cardiac troponin T or I- at least one value above 99$^{th}$ percentile with acute rise and / or fall of cardiac troponins.

It should be noted that there are various other causes of elevated troponins levels:

Sepsis, Subarachnoid hemorrhage, CKD, Pulmonary embolism, Critically ill patients, Myocarditis, Takotsubo myocarditis, Defibrillator shocks, Tachyarrhythmias.

Clinical evidence of Myocardial ischemia: out of 5 signs is mandatory.

1. Symptoms
2. New onset ECG changes
3. Development of pathological q waves
4. Image evidence
5. Angiography evidence.

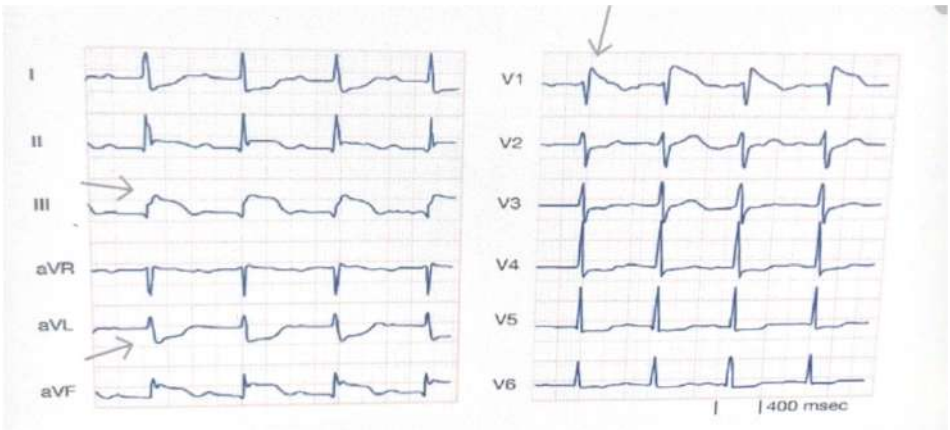
Definition of NSTE- ACS: Rest Angina > 20 minutes, new onset severe angina, Crescendo pattern. ST depression with T wave inversion, Increase in cardiac troponins and TIMI score determines the mortality risk in NSTEMI. Females are more affected with very vague symptoms.

**Materials and Methods:** For the following study, 90 patients of MI were closely observed in the Republican hospital of Samarkand, Khavasi, Samarkand, Uzbekistan. Out of these 90 patients, 55 were males and rest 25 were females. All from the age range of 25-55. Patients underwent to record an ECG in 12 standard leads, a KENZCardio 1210 device (Japan) was used. Heart rhythm (sinus or AF) and heart rate (HR) were recorded in the study protocol and database. For sinus rhythm, heart rate gradations were taken into account: less than 60 beats per minute (bpm), 60–80 bpm and more than 80 bpm . In patients with AF, categories of ventricular rate (VRR) were identified: up to 60 beats/min, 60–110 beats/min, and more than 110 beats/min. Transthoracic echocardiography (Echo-CG) was performed on an expert-class GE Vivid S70 device (USA) using a standard method of 2 approaches: parasternal and apical in 2-, 4- and 5-chamber sections. The dimensions of the walls and cavities of the LV were measured: end-diastolic dimension (EDD), end-systolic dimension (ESD), interventricular septum (IVS), posterior wall of the LV (PLW).

**Results:** Following ECGS of three of the patients were received representing the three different occlusions:

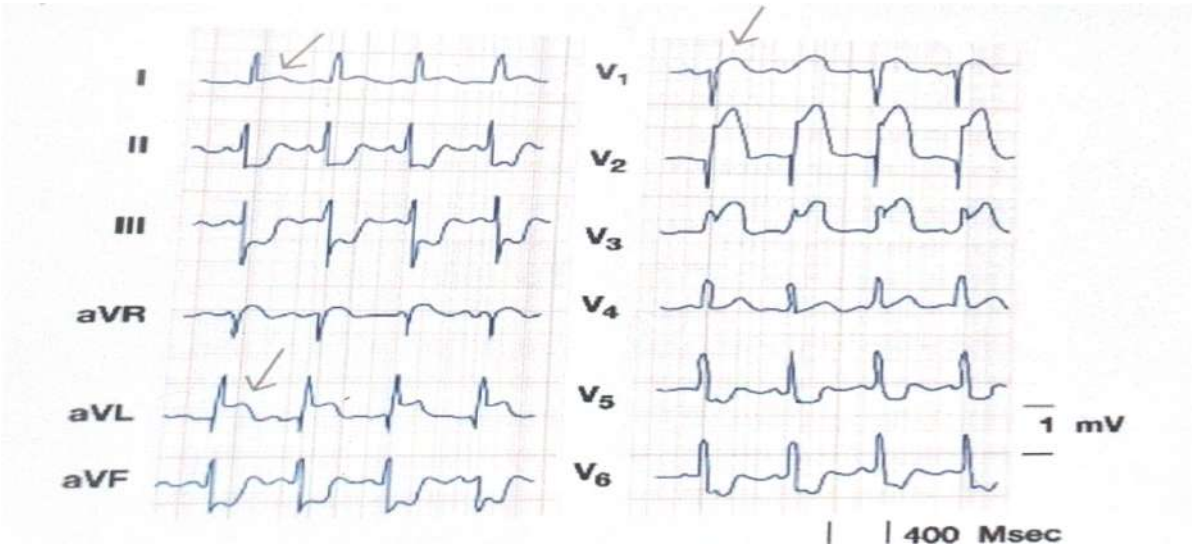
ECG 1

Patient has Proximal RCA occlusion leading to RWMI and IWMI and PWMI.



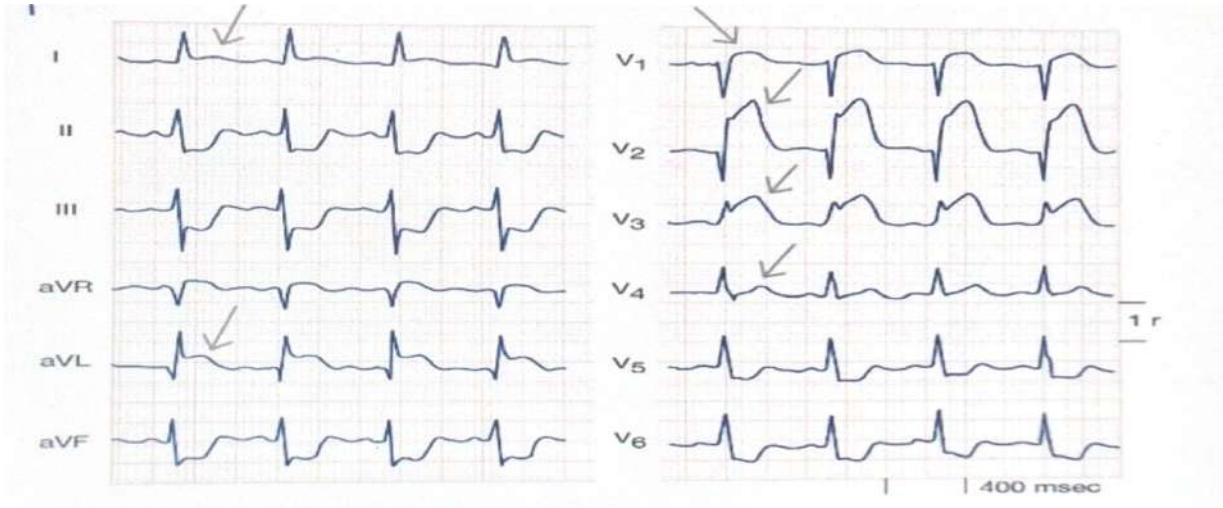
ECG 2

Patient has LAD occlusion before D1 sub branch leading to Antereoseptal Lateral Wall MI.




ECG 3

Patient has LAD occlusion between D1 and S1 sub branches leading to Anteroseptal Wall MI




For management of ACS, first 6 hours are considered as golden hours because Reperfusion via PCI before 6 hours is to salvage the myocardium and after 6 hours of onset of chest pain, only 1/6 th of the myocardium is viable.

Reperfusion (PCI) after 6 hours is to:

1. Reduce the pain.
2. Prevent electrical complications.
3. Prevent complications.

In first 2 hours, efficacy of thrombolysis (Tenecteplase or Reteplase) = efficacy of PCI. It is more important to reopen the artery rather than the mode of reperfusion you choose.

Aim of PCI:

- To establish normal epicardial coronary flow (TIMI 3 flow)
- To attain normal microvascular flow (MPG 3)

Loading dose:

1. Aspirin 325 mg
2. Clopidogrel 300 mg
3. Atorvastatin 40-80 mg
4. S/L Nitrate 5mg up to 3 tablets.

**Conclusion:** At the end of the study, it can be concluded that PCI is necessary for the MI patients and their various kinds of occlusion leading to different types of MI. Ever changing field of medicine has standard definition of MI which covers the all aspects of the injury. Chest pain which usually we underestimate could be Myocardial Infarction, so we should mold our lives to a healthier lifestyle and have frequent body health checkups.

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