Dr. Deepak Suman
M.D (K.C) NIA, Jaipur

Myocardial Infarction (MI) : ⇒

⇒ Irreversible necrosis of part of the heart muscle is almost always due to coronary atherosclerosis.

Incidence : ⇒ 5/1000 per year. Fifty percent of death due to MI occur within 1-2 hr. after the onset of symptoms.

Risk Factors :—

Category - I (For which interventions have been Proved to lower CVD[1] risks)

1. Raised LDL[2] cholesterol
2. Reduced HDL[3] cholesterol
3. Cigarette smoking
4. Hypertension
5. LVH[4]
6. Thrombogenic factors

Category - II (For which interventions are likely to lower CVD risks)

1. Diabetes mellitus
2. Increased triglycerides
3. Small dense LDL
4. Obesity

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1. CVD : ⇒ Cardiovascular disease
2. LDL : ⇒ Low-density lipoProtein / Lipid cholesterol
3. HDL : ⇒ High density " — " — " — " —
4. LVH : ⇒ Left Ventricular Hypertrophy

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Category-III (Associated with increased CVD risks that, if modified, might lower risk)

1. Psychosocial factors
2. Increased Lipoprotein a (normal level - 0·3 mg/dl)
3. No alcohol consumption
4. Post-menopausal status

Category-IV (Associated with increased CVD risk which cannot be modified)

1. Age
2. Male gender
3. Low-socio-economic status
4. Family history of early onset CVD

Etiopathogenesis :⇒ The etiologic role of severe coronary atherosclerosis (more than 75% compromise of lumen) is the pathogenesis of about 90% cases of acute MI.

A few notable features in the development of acute MI are as under:-

(i) Myocardial ischaemia :- Myocardial ischaemia is brought about by one or more of the following mechanisms:-

(a) Diminished coronary blood flow eg.- coronary artery disease, Shock.

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(b) Hypertrophy of the heart without simultaneous increase of coronary blood flow e.g.- Hypertension, Valvular heart disease.

(ii) Role of Platelets :⇒ Rupture of an atherosclerotic plaque exposes the subendothelial collagen to platelets which undergo aggregation, activation and release reaction. These events contribute to build up of the platelet mass that may give rise to emboli or initiate thrombosis.

(iii) Acute Plaque rupture :- Coronary atherosclerotic plaque in the form of superimposed coronary thrombosis due to plaque rupture and plaque haemorrhage is frequently encountered in case of acute MI :-

(a) Superimposed coronary thrombosis due to disruption of plaque is seen in about half the cases of acute MI. Infusion of intracoronary fibrinolysins in the first half an hour of development of acute MI in such cases restores blood flow in the blocked vessel in majority of cases.

(b) Intramural haemorrhage is found in about one-third cases of acute MI.

(iv) Non-atherosclerotic Causes — about 10% cases of acute MI are caused by coronary vasospasm, arteritis, embolism, thrombotic diseases & trauma.

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(v) Transmyxal and subendocardial infarcts :- These infarcts play a good role of Pathogenesis of MI by the obstructed blood supply of coronary arterial trunks.

Diagnosis :⇒ The diagnosis of aute MI is made on the observation of 3 types of features — clinical features, ECG changes, and Serum enzyme determinations.

Clinical features :⇒

Symptoms :— Anginal Pain is of greater severity and is associated with nausea, vomiting, Sweating, and extreme distress.

Signs :- Tachycardia, bradycardia, VPCs, gallop
There may be hyper or hypotension
Cold and clammy extremities
Cyanosis may be Present
Mild Pyrexia (< 38.5°c)
Features of complications (LV Failure, Pulmonary oedema, arrhythmias)

Investigations :⇒ ECG-

(i) May be normal initially and hence serial ECG must be taken.

(ii) ST elevation and T-wave inversion with Pathological Q- wave are typically seen in leads adjacent to the infarcted segment of myocardium.

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(iii) Reciprocal ST depression or T-wave inversion in opposite leads.

Chest x-ray :- Signs of heart failure or Pulmonary oedema.

Cardiac Enzymes :-

① CPK-MB (creatine Phosphokinase) :- This cardiac isoenzyme starts rising within 4-6 hr. after development of acute MI, Peaks during the 2nd day (4 fold rise) and disappears in 2-3 days.

② AST :- Starts rising on the 1st day, Peaks in 2-3 days (3 fold rise) and disappears by 3rd day.

③ LDH_1 (Lactate dehydrogenase) :- rising by second day, Peaks around 3 day (3 fold rise) and disappears in 10 day.

④ Troponin T :- It is regulatory contractile Protein and has sensitive and specific marker for myocardial cell damage.

⑤ Myoglobin :- It is increased within 2 hr. of onset of symptoms.
(Normal level is 20-100 ng/L)

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COMPLICATIONS: ⇒

1. Arrhythmias
2. Congestive heart failure
3. Cardiogenic shock
4. Mural thrombosis and thromboembolism
5. Cardiac aneurysm
6. Pericarditis
7. Postmyocardial infarction Syndrome

Management : ⇒

1. Bed-rest
2. Nasal O_2
3. Morphine given in an intravenous form (< 1 mg Per minute) to a maximum dose of 10-15 mg. It acts as a Pulmonary vero dilator.
4. Nitrates may be given Sublingually for rapid action of relief of Pain by coronary vasodilation. If Pain Persist after administration of Sublingual nitrates, IV infusion of nitroglycerine may be given.
5. Aspirin is given orally in the dose ranging from 100 to 300 mg.
6. Thrombolytic therapy may be given.
7. Heparin may be given in a does of 5000U, 12 hourly, Subcutaneously.

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(8) β-Blockers are started immediately. β Blockers can be given even if the ejection fraction as determined by Echo is < 40% but Ca^{+2} channel blockers must be avoided.

(9) ACE inhibitors help in cardiac remodelling.

(10) Advice Patient to stop smoking.

(11) Control of associated risk factors like DM, HTM etc.)

(12) Rehabilitation –
If the Patient is stable and no complications:-
Sit in a chair – 2nd day
Walk to toilet – 3rd day
Return home – 7th day
Back to work – 6th day
Driving – 6 weeks
Air travel – 8 weeks

(13) Surgery – Coronary angioplasty, coronary artery by pass grafting if medical management fails.

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