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DRUG THERAPY IN MYOCARDIAL INFARCTION.

1. Pain anxiety and apprehension.

* If pain not relieved by 3 doses of GTN given 5 min apart, an opioid analgesic or diazepam is administered parenterally.

2. Oxygenation by O_2 inhalation & assisted respiration.

3. Maintenance of bld volume, tissue perfusion & microcirculation flow in infusion of saline & dextroses.

4. Correction of acidosis

Acidosis occurs due to lactic acid production corrected by iv sodium bicarbonate infusion.

5. Prevention of & treatment of Arrhythmias.

Prophylactic administration of β blockers as soon as MI patients is seen & its continuation twice daily for a few days & has been shown to reduce the incidence of arrhythmias & mortality.

Tachyarrhythmias treated with IV lidocaine, procainamide / Amiodarone

6. Pump Failure.

to increase CO_2 / $\downarrow$ use filling pressure without paining cardiac work / concerning BP.

a. Furosemide: if pulmonary wedge pressure > 20 mmHg.
 $\downarrow$ $\downarrow$ $\downarrow$ cardiac preload

b. Vasodilators: may be supplemented by IV esmolol or labetalol.

c. Inotropic agents: dopamine / dobutamine IV infusion may be needed to augment the pumping action of heart & tide over the crisis.

7. Prevention of thrombus extension, embolism, venous thrombosis.

• Aspirin should be given for chewing & swallowing as soon as MI is suspected

• Clopidogrel / prasugrel / ticagrelor may be given as alternative or add on antiplatelet drug.

8 Thrombolysis & Reperfusion

: Fibrinolytic agents, i.e. plasminogen activators - alteplase / tenecteplase to achieve reperfusion of infarcted area are indicated only STAMI or high risk NSTEMI

9) Prevention of remodelling of subsequent CHF

- ACE inhibitors are of proven efficacy.

10) Prevention of Future Attacks.

② Platelet inhibitors - aspirin / clopidogrel or similar drugs.

③ β blocker - Reduce risk of reinfarction, CHF & mortality

④ control of hyperlipidaemia - hypolipidemic drugs especially statins are routinely prescribed irrespective of cholesterol level. They have been shown to reduce risk of cardiovascular events.

Pathophysiology of MI

• It is characterised by death of muscle tissue due to lack of cold supply.

• the pathological changes that occur in MI can be divided into gross & microscopic changes. In the 1st week of MI, affected area appears pale, within 6-12 hrs the area may still appear pale or show no change. After 24 hrs a cyanotic red purple area of hemorrhage may be observed. By 48-72 hrs, the affected area becomes pale

& hyperemic. From 3rd - 7th day, centre of infarcted area become yellow and soft, while border remains hyperemic.

- Microscopically coagulative necrosis begins within the 1st week accompanied by neutrophilic infiltration, edema, and hemorrhages.

Coagulative necrosis is complete and a well developed neutrophilic infiltrate is observed. Neutrophils gradually disappear and macrophages start to resorb the necrosed fibres. Fibrovascular response begins.

Clinical features: Sudden, severe, crushing pain in the chest radiating to arms, neck and back. ECG changes includes ST elevation, T wave inversion & appearance of wide deep waves. Serum biomarkers, ex CK-MB which are released into blood from heart muscle. (that are reabsorbed), after MI.