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A Comprehensive Review on Myocardial Infarction

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ABSTRACT:
Myocardial infraction(MI) colloquially known as heart attack, is caused by decreased or complete cessation of blood flow to a portion of myocardium. Myocardial infraction may be silent, and go and undetected, or it could be a catastrophic event leading to hemodynamic deterioration and sudden death. Most myocardial infractions are due to underlying coronary artery disease, the leading cause of death in the united states . with coronary artery occlusion, the myocardium is deprived of oxygen prolonged deprivation of oxygen supply to the myocardium can lead to myocardial cell death and necrosis. Patients can present with chest discomfort or pressure that can radiate to the neck, jaw, shoulder, or arm. In addition to the history and physical exam, myocardial ischemia may be associated with ECG changes and elevated biochemical markers such as cardiac troponins .

KEYWORDS
Myocardial infraction, myocardium, coronary Artery, troponins, coronary angiography.

I. INTRODUCTION

Myocardial infraction is defined as sudden ischemic death of myocardial tissue. In the clinical context, myocardial infraction is usually due to thrombotic occlusion of a coronary vessel caused by rupture of a vulnerable plaque. Ischemia induces profound metabolic and ionic perturbations in the affected myocardium and causes rapid depression of systolic function. Prolonged myocardial ischemia activates a wavefront of cardiomyocyte death that extends from the subendocardium to the subepicardium. Myocondrial alterations are prominently involved in apoptosis and necrosis of cardiomyocytes in the infracted heart. The adult mammalian heart has negligible regenerative capacity, does the infracted myocardium heals through formation of a scar. Infract healing is dependent on a inflammatory cascade, triggered by alarmins released by dying cells. Clearance of dead cells and matrix debris by infiltrating phagocytes activates anti-inflammatory pathways leading to suppression of cytokine and chemokine signaling. Activation of the renin-angiotensin-aldosterone system and release of transforming growth factor-β induced conversion of fibroblast into myofibroblasts, promoting deposition of extracellular matrix proteins. Infract healing intertwined with geometric remodeling of the chamber, characterized by dilation, hypertrophy of viable segments, amd progressive dysfunction. This review manuscript describes the molecular sinals and cellular effectors implicated in injury, repair, and remodeling of the infracted heart, the mechanistic basis of the movement common complications associated with myocardial infraction, and the pathophysiologic effects of established treatment stratagies. Moreover, we discuss the implications of pathophysiological insights in design and implementation of new promising therapeutic approaches for patient with myocardial infraction.

ETIOLOGY

Myocardial infraction is closesly associated with coronary artery diseases.The following modifiable risk factors for coronary artery disease:
1.smoking
2.abnormal lipid profile/apolipoprotien [raised ApoB/ApoA1]
3.Hypertension
4.Abdominal obesity [Waist/hip ratio] [greater than 0.90 females and greater than 0.85 for females]
6.Psychosocial factors such as depression, loss of the locus of control, global stress, financial stress,

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and life events including martial separation, job loss, and family conflicts.
7. lack of daily consumption of fruits or vegetables.
8. Lack of physical activity.
9. Alcohol consumption (weaker association, protective).

EPIDEMIOLOGY

The most common cause of death and disability in the western world and worldwide is coronary artery disease. Based on 2015 mortality data from the national health interview survey [NHIS-CDC], MI Mortality was 114,023, and MI any-mention mortality [i.e., MI is mentioned as a contributing factor in the death certificate] was 151,863.

As per the national health and nutrition survey [NHANES]-CDC data from 2011 to 2014, an estimated 16.5 million Americans older than 20 years of age have coronary artery disease, and the prevalence was higher in males than females for all ages. As per the NHANES 2011 through 2014, the overall prevalence of MI is 30% in US adults older than 20 years of age.

Prevalence of MI in the Sub-populations

Non-Hispanic Whites.

- 4.0% [Male].
- 2.4% [Female.]

Non-Hispanic Blacks

- 3.3% [Male]
- 2.2% [Female]

Hispanics

- 2.9% [Male]
- 2.1% [Female]

Non-Hispanic Asians

- 2.6% [Male]
- 0.7% [Female]

Based on the atherosclerosis risk in communities study [ARIC] performed by National Heart, lung, and blood institute [NHLBI] collected between 2005 and 2014, the estimated annual incidence is 605,000 new MIs and new MIs and 200,000 recurrent MIs.

PATHOPHYSIOLOGY

The acute occlusion of one or multiple large epicardial coronary arteries for more than 20 to 40 minutes can lead to acute myocardial infarction. The occlusion is usually thrombotic and due to the rupture of a plaque formed in the coronary arteries. The occlusion leads to a lack of oxygen in the myocardium, which results in sarcolemmal disruption and myofibril relaxation.These changes are one of the first ultrastructural changes in the process of MI, which are followed by mitochondrial alterations. The prolonged ischemia ultimately results in liquefactive necrosis of myocardial tissue. The necrosis spreads from sub-endocardium to sub-epicardium. The subepicardium is believed to have increased collateral circulation, which delays death. Depending on the territory affected by the infarction, the cardiac function is compromised. Due to the negligible regeneration capacity of the myocardium, the infarcted area heals by scar formation, and often, the heart is remodeled characterized by dilation, segmental hypertrophy of remaining viable tissue, and cardiac dysfunction

DIFFERENTIAL DIAGNOSIS

1. Angina pectoris.
2. Non-ST segment elevation myocardial infarction (NSTEMI).
3. ST-segment elevation myocardial infarction (STEMI).
4. Pulmonary embolism.
5. Pneumothorax.

COMPLICATIONS.

Type and Manifestation.

I: Ischemic.
• Reinfarction.
• Extension of infarction.
• Angina.

II: Arrhythmias.
• Supraventricular or ventricular arrhythmia.
• Sinus bradycardia and atrioventricular block.
• Cardiogenic shock.
• Cardiac rupture (Free wall rupture, ventricular septal rupture, papillary muscle rupture).

III: Mechanical.
• Myocardial dysfunction.
• Cardiac failure.

IV: Embolic.
• Left ventricular mural thrombus.
• Peripheral embolus.

V: Inflammatory,
• Pericarditis (infarct associated pericarditis, late pericarditis, or post- cardiac injury pericarditis).
• Pericardial effusion.

ACUTE MANAGEMENT AND MEDICAL THERAPY OF MYOCARDIAL INFARCTION

Initial Therapy upon Diagnosis: Patients should primarly be managed according to the ALS algorithm. Upon diagnosis, management

Cardiovascular management of MI and unstable angina should simultaneously focus on hemodynamic stabilization, relieving the pain (angina pectoris), decreasing myocardial oxygen consumption (by decreasing heart rate, blood

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pressure, preload or myocardial contractility), increasing myocardial oxygen supply (by administration of oxygen or through coronary vasodilation) and initiating antithrombotic therapy .

Oxygen

Oxygen supplementation should be initiated in patients with arterial oxygen saturation below 94% (or <88% if chronic obstructive pulmonary disease (COPD) is present) to achieve normoxia. Hyperoxia should be avoided since it causes coronary artery vasoconstriction, increases early myocardial injury and infarct size

Nitrates

In the absence of hypotension (systolic blood pressure < 90 mmHg) and signs of right ventricular infarction (up to 40% of inferior STEMI), patients should be given nitrates, namely nitroglycerin 0.4 mg sublingually every 5 minutes up to three times with the intention of increasing coronary blood flow by decreasing preload. This can also alleviate angina. In monitored patients, intravenous nitroglycerin can be used as continuous infusion. Before nitrates are given, one has to make sure the patient did not take phosphodiesterase-5 inhibitors (e.g. sildenafil) in last the 24-48 hours, since combined with nitrates they can cause severe hypotension.

Morphine

If pain persists, intravenous (i.v.) morphine of 3 to 5 mg may be given and repeated every few minutes until the patient is pain-free. Morphine diminishes sympathetic stimulation caused by pain and anxiety, and therefore reduces myocardial oxygen consumption. Generally, morphine should be used only in patients with extreme pain as it was shown to reduce the antiplatelet effect of P2Y 12 receptor blockers, presumably through slowing intestinal absorption

Antithrombotic Therapy

Antiplatelet Therapy

Patients should chew non-enteric-coated aspirin (150 to 300 mg) which blocks further platelet aggregation as a central pathophysiologic mechanism of MI after plaque disruption. Also, i.v. preparation of 150 mg can be given if the patient is unable to take medications orally. Additional inhibition of platelet aggregation is achieved by P2Y12 receptor antagonists, normally stimulated by ADP. Clopidogrel and prasugrel are irreversible, whereas ticagrelor is a reversible P2Y12 inhibitor. Prasugrel and ticagrelor have a more rapid and consistent action, while clopidogrel is known to have hypo- and hyper-responders. Dual antiplatelet therapy (DAPT) with clopidogrel therapy has been shown to reduce ischemic events compared to aspirin alone. Studies comparing clopidogrel to prasugrel or ticagrelor found a sig- nificant MACE reduction (due to reduction in MD) with patients patients over 75 years of age, below 60 kg or history of TIA/stroke should not receive prasugrel, whereas on the other hand, ticagrelor is contraindicated in patients with history of intracranial hemorrhages, and caution is advised in patients with bradycardia (increased incidence of asymptomatic ventricular pauses) .

In STEMI patients with planed PCI, pretreatment with a loading dose of 180 mg of ticagrelor or 60 mg of prasugrel or 600 mg of clopidogrel should be given, bearing in mind the contraindications. The choice of P2Y12 antagonists should always be guided by local protocols or by consultation with a PCI center. When thrombolysis is planned, newer P2Y12 antagonists should be avoided as there no data in this setting, and clopidogrel should be used instead (300 mg loading dose up to an age of 75 and 75 mg without loading dose if >75 years of age) .

In NSTE-ACS with planed PCI, pretreatment with a loading dose of 180 mg of ticagrelor or 300 mg of clopidogrel should be given taking into account their contraindications . Before coronary anatomy is known and patients are not proceeding to PCI, prasugrel should not be given in NSTE-ACS situations, since it was shown to increase major bleeding events without reducing thrombotic events. Ticagrelor and clopidogrel were not investigated in regard to the timing of the treatment, nonetheless they are recommended soon after diagnosis, regardless of the management strategy .

If conservative management is preferred, prompt initiation of DAPT is warranted, preferably with ticagrelor in the absence of contraindications in STEMI or NSTE- ACS patients . Anticoagulation in adjunct to DAPT was shown to reduce ischemic events. Unfractionated heparin (UFH) (initial bolus 70-100 U/kg when no glycoprotein (GP) IIb/IIIa inhibitor is planned or 50-60 U/kg when the use of GP IIb/IIIa inhibitors is expected is mainly used in patients re- quiring immediate PCI . Recent studies suggest that enoxaparin (0.5 mg/kg i.v. followed by s.c. treatment) may be preferred over UFH since it was shown to reduce

Anticoagulation Therapy

After initiation of DAPT, anticoagulants should also be given Anticoagulation in adjunct to DAPT

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Reperfusion Strategy

The timing of invasive strategy depends on the risk of complications. In STEMI and very high risk NSTE-ACS patients, reperfusion should be initiated as soon as possible in less than 120 min if the time from symptom onset is less than 12 hours Beperfusion can be achieved by percutane

IMMEDIATE TREATMENT OF MYOCARDIAL INFRACTION

- Morphine.
- Oxygen.
- Nitroglycerin.
- Aspirin.
- Thrombolytics.
- Anticoagulants
- Stool softeners.
- Sedatives.

Long-Term Management

Lipid-lowering treatment: It is recommended to start high-intensity statins that reduce low-density lipoproteins (LDLs) and stabilize atherosclerotic plaques. High-density lipoproteins are found to be protective.

Antithrombotic therapy: Aspirin is recommended lifelong, and the addition of another agent depends on the therapeutic procedure done, such as PCI with stent placement

ACE inhibitors are recommended in patients with systolic left ventricular dysfunction, or heart failure, hypertension, or diabetes.

Beta-blockers are recommended in patients with LVEF less than 40% if no other contraindications are present.

Antihypertensive therapy can maintain a blood pressure goal of less than 140/90 mm Hg

Mineralocorticoid receptor antagonist therapy is recommended in a patient with left ventricular dysfunction (LVEF less than 40%).

Glucose lowering therapy in people with diabetes to achieve current blood sugar goals.

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