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Modern Solutions for The Use of Anticoagulants in Myocardial Infarction and Arrhythmias

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ABSTRACT

Objective: *This study aims to explore the causes, symptoms, diagnostic methods, and treatment strategies for acute myocardial infarction (AMI), focusing on the role of coronary artery obstruction in its development and the available therapeutic options.* ***Methods:*** *Diagnosis of AMI is based on electrocardiography (ECG) and biomarker analysis. Treatment protocols include the administration of antiplatelet agents, anticoagulants, nitrates, beta-blockers, statins, and various reperfusion therapies. The study also examines the use of fibrinolytic drugs, percutaneous interventions, and coronary artery bypass grafting (CABG) in different types of AMI.* ***Results:*** *Acute myocardial infarction presents with symptoms such as shortness of breath, nausea, chest discomfort, and sweating. Diagnosis is confirmed through ECG readings and biomarker testing. Effective treatment varies based on the severity and type of AMI, with reperfusion therapy being critical for ST-segment elevation myocardial infarction (STEMI), while non-ST-segment elevation myocardial infarction (NSTEMI) may require percutaneous intervention or CABG.* ***Novelty:*** *This study contributes to understanding the nuanced diagnostic and therapeutic approaches to AMI, particularly in differentiating treatment strategies for STEMI and NSTEMI, highlighting the importance of timely and appropriate interventions for improving patient outcomes.*

INTRODUCTION

Up to 1.0 million cases of myocardial infarction occur in the United States each year (1). Approximately 300,000 to 400,000 myocardial infarctions (MIs) result in death (see also: Cardiac arrest).

Acute myocardial infarction (MI) is considered an acute coronary syndrome with unstable angina. Acute MI includes non-ST segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI). The distinction between NSTEMI and STEMI is important because the treatment strategies for these two entities are different.

Pathophysiology of acute MI

Myocardial infarction is defined as myocardial necrosis in a clinical setting consistent with myocardial ischemia (1). These conditions can be met by having a cardiac biomarker (primarily troponin [cTn]) level in the blood above the 99th percentile of the upper reference limit (URL) and by at least one of the following:

Signs of ischemia

ECG changes indicate a new focus of ischemia (significant ST/T changes or left bundle branch block)

Development of pathological Q waves

Presence of new areas of myocardial necrosis or abnormal regional wall motion confirmed by imaging Evidence of intracoronary thrombus obtained by angiography or autopsy. Slightly different criteria are used to diagnose myocardial infarction and sudden death during and after percutaneous intervention or coronary artery bypass grafting.

RESEARCH METHOD

Myocardial infarction is divided into 5 types depending on the etiology and circumstances of occurrence (1):

1. Type 1: Spontaneous myocardial infarction resulting from ischemia due to a primary coronary lesion (e.g., plaque rupture, erosion, or fissure; coronary dissection)
2. Type 2: Ischemia due to increased oxygen demand (e.g., hypertension) or decreased oxygen delivery (e.g., coronary artery spasm or embolism, arrhythmia, hypotension).
3. Category 3: associated with sudden unexpected cardiac death
4. Type 4a: associated with percutaneous coronary intervention (with signs and symptoms of myocardial infarction, cardiac troponin values >5 × 99th percentile GRP)
5. Type 4b: associated with confirmed stent thrombosis
6. Category 5: Coronary artery bypass graft-associated (signs and symptoms of myocardial infarction with cardiac troponin value > 10 × 99th percentile GRP).

Location of infarction

Myocardial infarction mainly affects the LV, although the process can spread to the RV and atria. Right ventricular myocardial infarction is usually caused by occlusion of the right coronary artery or dominant circumflex artery. The main manifestations are increased RV filling pressure, often associated with severe tricuspid regurgitation and decreased cardiac output.

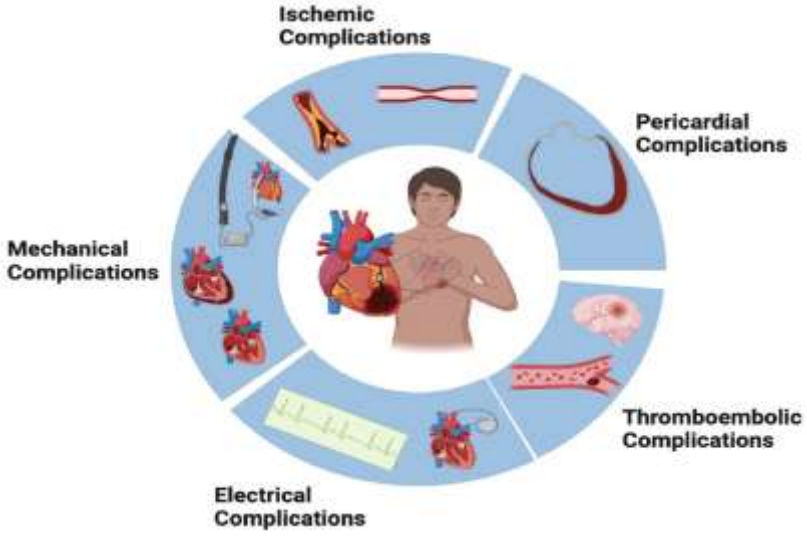

Figure 1. Infero-posterior myocardial infarction related to pancreatic dysfunction and hemodynamic disturbances.

Infero-posterior localization of myocardial infarction often leads to pancreatic dysfunction in about half of patients and is manifested by hemodynamic disturbances in 10-15%. RV dysfunction should be suspected in any patient with inferoposterior MI and increased central venous pressure accompanied by hypotension or shock. The development of RV MI in the setting of LV MI significantly increases the risk of death (2).

Anterior myocardial infarction is usually more common and has a worse prognosis than inferoposterior myocardial infarction. Anterior MI is usually associated with occlusion of the left coronary artery, especially the anterior descending artery; inferoposterior MI is associated with obstruction of the right coronary artery or dominant circumflex artery.

RESULTS AND DISCUSSION

Heart attack prevalence

a. Could be a heart attack.
b. Transmural
c. Non-transmural

Transmural MI involves all layers of the myocardium from the epicardium to the endocardium and is characterized by the appearance of abnormal Q waves on the ECG.

Nontransmural (including subendocardial) infarctions do not extend through the ventricular wall and cause only ST segment and T wave abnormalities (ST-T abnormalities). Subendocardial infarctions usually affect the inner third of the myocardium, where wall stress is highest and myocardial blood flow is most vulnerable to changes in circulatory status. This type of infarction may develop after prolonged hypotension.

Because transmural extension of MI cannot be clinically definitively assessed, MI is usually classified as STEMI or NSTEMI based on ST segment elevation or the presence of Q waves on the ECG. The extent of myocardial injury can be estimated by the magnitude and duration of the elevation of creatine phosphokinase (CPK-CK) or the peak levels of cardiac troponins, which are commonly measured.

Non-ST segment elevation myocardial infarction (NSTEMI, subendocardial MI) is necrosis of the heart muscle (cardiac biomarkers in the blood: troponin T or troponin I and CK-MB levels are elevated) without acute ST segment elevation. NSTEMI is characterized by ECG changes such as ST segment depression, T wave inversion, or a combination of both.

ST-segment elevation myocardial infarction (STEMI, transmural myocardial infarction) is a necrosis of cardiomyocytes accompanied by persistent ST-segment elevation on the ECG that does not disappear after taking nitroglycerin. Troponin I or troponin T and CPK-MB are elevated.

Myocardial infarction in the absence of coronary heart disease (MIHD)

Myocardial infarction without obstructive coronary artery disease (MINOCA) is seen in approximately 5–6% of patients with acute MI on coronary angiography (3). Patients with MINOCA are predominantly young, female, and without dyslipidemia. They typically develop myocardial necrosis without overt coronary atherosclerosis. Plaque disruption and coronary artery vasospasm are common in MINOCA. Causes of MINOCA include coronary thrombosis or embolism and spontaneous coronary artery dissection. Drug therapy should be based on the underlying mechanism of MINOCA in each patient.

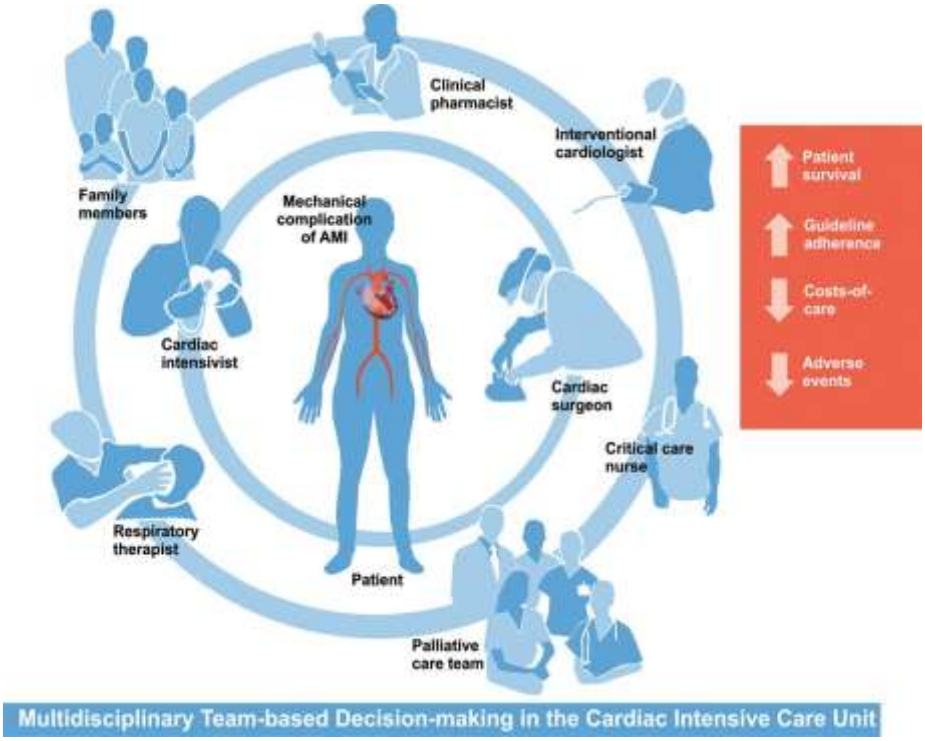

Figure 2. Multidisciplinary team-based.

Signs and symptoms of acute MI

The symptoms of STEMI and NSTEMI are similar. In the days or weeks before the onset of the disease, about two-thirds of patients experience prodromal symptoms, which include unstable or worsening angina, shortness of breath, and fatigue.

Typically, the first symptom of MI is severe chest pain, described by patients as a pressure or aching pain, often radiating to the back, jaw, left arm, right arm, shoulders, or all of these areas. The pain has characteristics similar to angina, but is usually more severe and prolonged, and is often accompanied by shortness of breath, profuse sweating, nausea, and/or vomiting. Taking nitroglycerin and resting have only partial or temporary relief.

However, the discomfort may be mild; approximately 20% of acute infarctions are silent (1). Silent myocardial infarctions are asymptomatic heart attacks or heart attacks that do not cause any symptoms that the patient does not recognize as painful.

Asymptomatic myocardial infarctions are more common in patients with diabetes mellitus or coronary artery disease. Patients often interpret the chest pain as indigestion, which is partially relieved by belching and taking antacids. Silent ischemia sometimes manifests as transient, asymptomatic changes in the ST-T interval, which are recorded during stress testing or 24-hour Holter monitoring. Radionuclide studies can sometimes confirm the presence of asymptomatic myocardial ischemia during physical or emotional stress. Silent ischemia and angina can occur at different times.

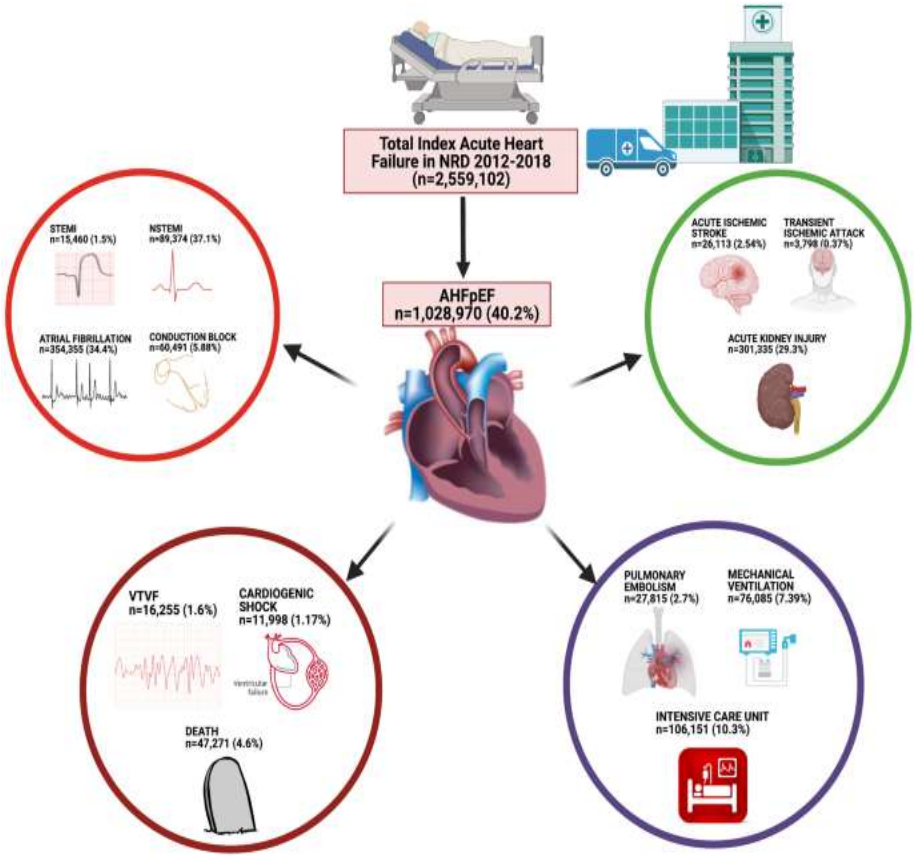

Figure 3. Silent myocardial infarction: heart attack without noticeable symptoms or pain.

In some patients, the disease manifests as loss of consciousness.

Women are more likely to experience atypical chest discomfort; however, even when typical symptoms are present, MI is often unrecognized in them (2). For older patients, complaints of shortness of breath are more common than ischemic chest pain.

In severe cases of ischemia, the patient often has severe pain accompanied by anxiety and fear of death. Nausea and vomiting may occur, especially in inferior myocardial infarction. Dyspnea and weakness are predominant due to left ventricular failure, pulmonary edema, shock, or clinically significant arrhythmia.

The skin may be pale, cold, and clammy with sweat. Peripheral or central cyanosis may occur. A threadlike pulse and blood pressure levels may be variable, although many patients experience arterial hypertension during a painful attack.

Cardiac auscultation findings are characterized by the presence of long murmurs with the 4th heart sound recorded in almost all patients; a soft systolic murmur, maximal at the apex, may be present, reflecting papillary muscle dysfunction. A friction rub or a more pronounced murmur on initial examination may indicate a previous cardiac disorder or another diagnosis. The detection of a pericardial rub within the first few hours after the onset of symptoms of myocardial infarction suggests acute pericarditis rather than myocardial infarction. However, the appearance of a pericardial rub, often intermittent, is common 2–3 days after STEMI. In some patients, the chest wall is tender to palpation.

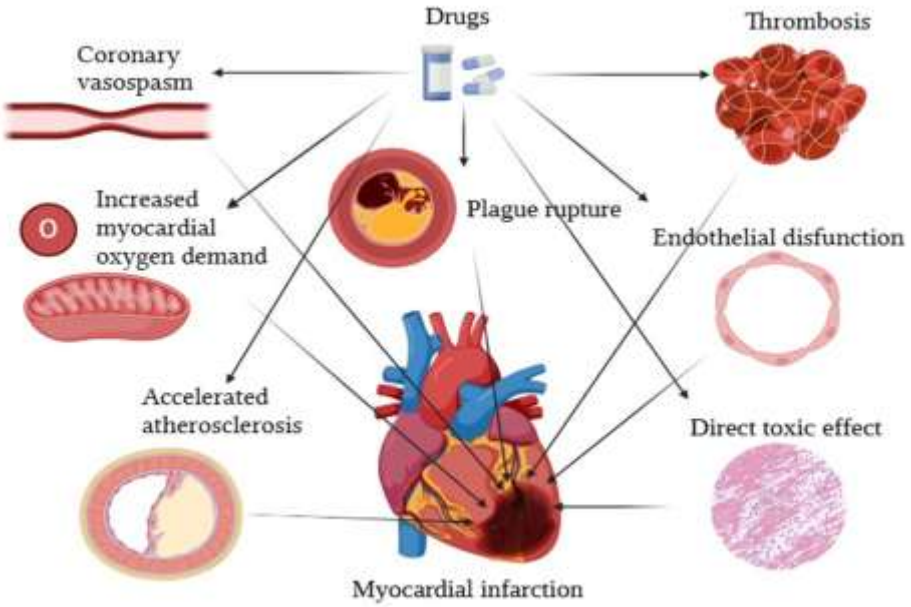

Figure 4. Cardiac auscultation reveals long murmurs and pericardial friction rub.

With the development of right ventricular (RV) MI, there is an increase in RV filling pressure, jugular venous distension (often with Kussmaul's sign), opacification of the lung fields, and hypotension.

Diagnosis of acute MI

ECG in dynamics

Measuring cardiac biomarker levels over time

Coronary angiography for urgent indications (if fibrinolytics have not been used) in patients with STEMI or its complications (persistent chest pain, hypotension, marked elevation of cardiac biomarkers, unstable arrhythmia)

Delayed coronary angiography (within 24-48 hours) for patients with NSTEMI without complications

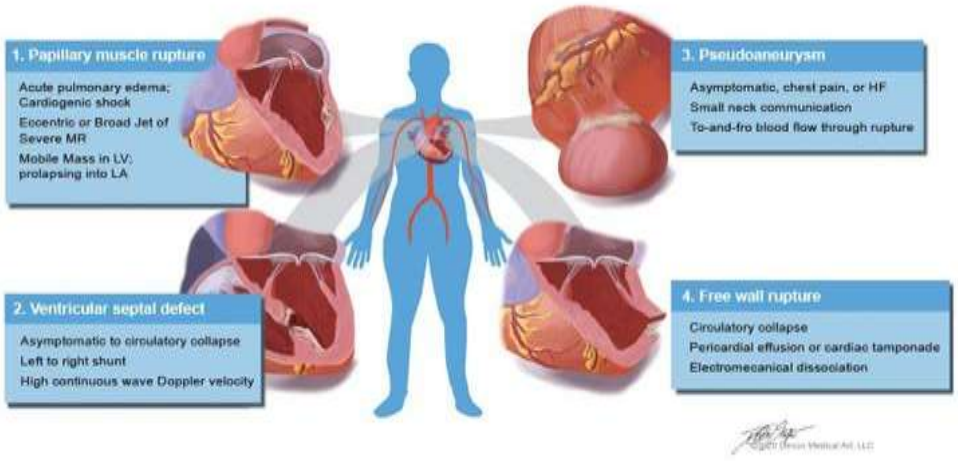

Figure 5. Coronary angiography for STEMI and NSTEMI with complications.

The investigation begins with a baseline ECG and a series of dynamic ECGs, as well as the detection of cardiac biomarkers during dynamics, which help to distinguish between unstable angina, ST-elevation myocardial infarction (STEMI), and non-ST-elevation myocardial infarction (NSTEMI). This distinction is important in the search for solutions, as fibrinolytics are indicated for patients with STEMI, but may increase the risk for patients with NSTEMI and unstable angina. In addition, urgent CG is indicated for patients with acute STEMI, but is usually not performed for patients with NSTEMI.

ECG

An ECG is the most important test and should be done as soon as possible (for example, within 10 minutes of the onset of pain).

The initial ECG for STEMI is usually diagnostic and shows ≥ 1 mm ST segment elevation in 2 or more contiguous leads covering the affected area (see images: Acute Left Ventricular Infarction, Left Ventricular Infarction, Lateral Left Ventricular Infarction (after a few days), Acute Inferior (Diaphragmatic) Left Ventricular Infarction, Inferior (Diaphragmatic) Left Ventricular Infarction, and Inferior (Diaphragmatic) Left Ventricular Infarction (after a few days).

NSTEMI

Acute lateral left ventricular infarction (ECG performed within a few hours of onset of illness). In leads I, aVL, V 4 , V 6, the most acute phase is marked by ST segment elevation and reciprocal depression in leads 4, V6, and other leads. Acute lateral left ventricular infarction (ECG performed within a few hours of onset of illness) MI of the lateral wall of the left ventricle (24 hours after the onset of the disease) In leads I, aVL, V 4 , V 6 4 and V6, there is a decrease in ST elevation, the formation of Q waves and a decrease in the amplitude of the R wave.

MI of the lateral wall of the left ventricle (24 hours after the onset of the disease)

MI of the lateral wall of the left ventricle (a few days after the onset of the disease)

1. Deep Q waves remain and the R wave voltage decreases, returning to ST segment isolation. No significant ECG dynamics are observed over the next few months.
2. MI of the lateral wall of the left ventricle (a few days after the onset of the disease)
3. Left ventricular inferior MI (diaphragmatic) (observation within a few hours of onset of illness)
4. The most acute stage is noted - ST segment elevation in leads II, III, aVF and reciprocal depression in other leads.
5. Left ventricular inferior MI (diaphragmatic) (observation within a few hours of onset of illness)
6. Inferior (diaphragmatic) left ventricular infarction (after the first 24 hours).
7. The formation of Q waves and a parallel decrease in ST segment height in leads II, III, aVF are noted.
8. Inferior (diaphragmatic) left ventricular infarction (after the first 24 hours).
9. Inferior left ventricular infarction (diaphragmatic) (a few days after the onset of the disease)
10. Isoelectric position of the ST segment. Deep Q waves in leads II, III, aVF show the scar formed.
11. Inferior left ventricular infarction (diaphragmatic) (a few days after the onset of the disease)

The presence of pathological Q waves is not necessary for the diagnosis of MI, since ST segment elevation may be palpable, especially in the lower wall of the LV (II, III, aVF) and sometimes the physician's attention is drawn more to leads with depression in the ST segment. In the presence of a typical clinical picture, ST segment elevation on the ECG is characterized by 90% specificity and 45% sensitivity in the diagnosis of MI. A dynamic ECG study (registration every 8 hours during the first day, then daily) allows you to confirm the diagnosis in the event of the development of pathological Q waves within a few days from the onset of the disease.

If right ventricular (RV) MI is suspected, a 15-lead ECG should be recorded: additional electrodes are placed in positions V4-6R (see figure for right ventricular (RV) VR1 to VR6), and for the diagnosis of posterior MI - in positions V8-V9.

Right ventricle (RV) leads from VR1 to VR6

Right ventricle (RV) leads from VR1 to VR6

The ECG diagnosis of myocardial infarction is difficult in patients with complete left bundle branch block because the QRS complex pattern is similar to that seen in myocardial infarction. ST segment elevation corresponding to the QRS complex, if ST elevation > 5 mm in at least two precordial leads, is a clear sign of myocardial infarction. In any case, a patient with an appropriate clinical presentation and new onset of left bundle branch block (or no block) is considered to have STEMI.

Cardiac biomarkers

Cardiac biomarkers (serum markers of myocardial cell damage) include

1. Cardiac enzymes (e.g., CK-MB [creatine kinase-MB])
2. Intracellular content (e.g., troponin I, troponin T, myoglobin)

These biomarkers are released into the bloodstream after myocardial cell necrosis. Biomarkers appear at different times after injury and their levels decline at different rates. The sensitivity and specificity for myocardial cell damage among these biomarkers vary considerably. Tests that measure cardiac troponin (cTn) levels, which have been used for many years, are sensitive and specific. Newer high-sensitivity cardiac troponin (hs-cTn) tests are preferred, which also have high accuracy. These methods can reliably detect cardiac troponin (T or I) levels as low as 0.003-0.006 ng/mL (3-6 pg/mL); with some research methods, as low as 0.001 ng/mL (1 pg/mL) is recognized.

Less sensitive cTn tests cannot detect cardiac troponins except in patients with acute heart disease. Thus, a "positive" cardiotropin test (i.e., above the detection limit) was highly specific. However, hs-cTn tests can detect small amounts of troponin in many healthy individuals. Thus, troponin levels determined using a high-sensitivity test should be compared with normal values and should be considered elevated if they exceed 99% of the reference population.

CONCLUSION

Fundamental Finding : The study highlighted the prevalence and classification of myocardial infarction (MI), which is primarily categorized into STEMI, NSTEMI, and MINOCA. It outlined that the pathophysiology of MI involves myocardial necrosis due to ischemia, with biomarkers like troponin being critical in diagnosis. A significant aspect of MI is the differentiation between transmural and non-transmural infarctions, influencing treatment strategies. The location of infarction, whether in the left or right ventricle, has notable implications for prognosis, particularly with right ventricular involvement increasing mortality risk. **Implication :** This study emphasizes the importance of early diagnosis through ECG and cardiac biomarkers in managing MI. The distinction between STEMI and NSTEMI guides treatment choices, particularly in using fibrinolytics and coronary interventions. Understanding MI's pathophysiology and risk factors, like hypertension or coronary artery dissection, can lead to more targeted therapeutic approaches. Additionally, recognizing the unique presentation of MI in women and elderly patients could improve early detection and outcomes. **Limitation :** One limitation of the study is its reliance on conventional diagnostic tools like ECG and biomarkers, which may not always accurately reflect the full extent of myocardial damage. Additionally, while the study mentions MINOCA, it does not delve deeply into the underlying mechanisms or potential advancements in its diagnosis. The data primarily focuses on established methods and may not fully address emerging diagnostic technologies. **Future Research :** Future research should explore the advancements in high-sensitivity cardiac troponin tests and their potential to detect MI at earlier stages.

Investigating the pathophysiology of MINOCA in greater depth, particularly through genetic and molecular studies, could provide new insights for personalized treatment. Moreover, research into non-invasive imaging techniques to assess infarction in real-time could further improve diagnostic accuracy and management of MI patients.

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