ACUTE CORONARY SYNDROME

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Acute coronary syndrome (ACS) refers to a group of conditions that include ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina. It is a type of coronary heart disease (CHD), which is responsible for one-third of total deaths in people older than 35. Some forms of CHD can be asymptomatic, but ACS is always symptomatic.

ACS is a manifestation of CHD (coronary heart disease) and usually a result of plaque disruption in coronary arteries (atherosclerosis). The common risk factors for the disease are smoking, hypertension, diabetes, hyperlipidemia, male sex, physical inactivity, family obesity, and poor nutritional practices. Cocaine abuse can also lead to vasospasm. A family history of early myocardial infarction (55 years of age) is also a high-risk factor.

CHD affects about 15.5 million in the United States. The American Heart Association estimates a person has a heart attack every 41 seconds. Heart disease is the leading cause of death in the United States. Chest pain is among the top reasons for emergency department visits. Evaluation: The first step of evaluation is an ECG, which helps differentiate between STEMI and NSTEMI unstable angina. American Heart Association guidelines maintain that any patient with complaints suspicious of ACS should get an ECG within 10 minutes of arrival. Cath lab should be activated as soon as STEMI is confirmed in a percutaneous coronary intervention (PCI) center. Cardiac enzymes especially troponin, CK-MB/CK ratio is important in assessing the NSTEMI versus myocardial ischemia without tissue destruction. A chest x-ray is useful in diagnosing causes other than MI presenting with chest pain like pneumonia and pneumothorax. The same applies for blood work like complete blood count (CBC), chemistry, liver function test, and lipase which can help differentiate intraabdominal pathology presenting with chest pain. Aortic dissection and pulmonary emboli should be kept in differential and investigated when the situation warrants. Myocardial infarction is defined as myocardial necrosis in a clinical situation consistent with myocardial ischemia (1). These conditions can be met by increasing blood levels of cardiac markers (primarily troponin [cTn]) above the 99th percentile of the upper reference limit (URL) plus at least one of the following: Symptoms of ischemia ECG changes indicating 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 as demonstrated by imaging Evidence of intracoronary thrombus by angiography or autopsy Slightly different

criteria are used in the diagnosis of myocardial infarction during and after percutaneous intervention or coronary artery bypass grafting, and as a cause of sudden death . Myocardial infarction can be divided into 5 types based on its etiology and circumstances of occurrence:

Type 1: spontaneous myocardial infarction due to ischemia due to primary coronary injury (eg, plaque rupture, erosion, or fissure; coronary dissection)

Type 2: ischemia due to increased oxygen demand (eg, hypertension) or decreased oxygen delivery (eg, coronary artery spasm or embolism, arrhythmia, hypotension)

Type 3: related to sudden unexpected cardiac death

Type 4a: Associated with percutaneous coronary intervention (signs and symptoms of myocardial infarction with cardiac troponin values >5 × 99th percentile GRP)

Type 4b: associated with confirmed thrombosis cType 5: associated with coronary artery bypass grafting (signs and symptoms of myocardial infarction with cardiac troponin values > 10 × 99 percentile GRP)

Location of infarction

Myocardial infarction predominantly affects the LV, with the process possibly spreading to the RV and atria. Right ventricular MI usually occurs due to occlusion of the right coronary artery or the dominant circumflex artery. The main manifestations are increased RV filling pressure, often associated with severe tricuspid regurgitation and decreased cardiac output. Infero-posterior location of myocardial infarction often leads to RV dysfunction in approximately half of patients and manifests as hemodynamic compromise in 10-15%. RV dysfunction should be suspected in any patient with inferoposterior MI and elevated central venous pressure with hypotension or shock. The development of RV MI on the background of LV MI significantly increases the risk of death. Anterior myocardial infarction is usually more common and has a worse prognosis than inferoposterior myocardial infarction. The development of anterior MI is usually associated with occlusion in the territory 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. Prevalence of heart attack There may be a heart attack Transmural Nontransmural 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) infarcts do not pass through the ventricular wall and cause only ST segment and T wave abnormalities (ST-T abnormalities). Subendocardial infarctions typically affect the inner third of the myocardium, where wall stress is highest and myoardial blood flow is most vulnerable to changes in circulation. The development of this type of infarction may follow long periods of hypotension. Because transmural extension of MI cannot be accurately verified clinically, MI is usually classified as STEMI or NSTEMI depending on the presence of ST segment

elevation or Q waves on the ECG. The extent of myocardial damage can be approximated by the magnitude and duration of the increase in creatine phosphokinase or, more commonly, the peak level of cardiac troponins. Non-ST segment elevation myocardial infarction (non-ST segment elevation myocardial infarction, subendocardial myocardial infarction) is necrosis of the heart muscle (confirmed by analysis of cardiac markers in the blood: troponin T or troponin I levels and creatine kinase are elevated) that is not accompanied by acute ST segment elevation on the ECG. NSTEMI is characterized by ECG changes such as ST segment depression, T wave inversion, or a combination of both. Myocardial infarction with ST segment elevation (STEMI, transmural myocardial infarction) is necrosis of cardiomyocytes, accompanied by persistent ST segment elevation on the ECG, which does not disappear after taking nitroglycerin. Troponin I or troponin T and creatine kinase are elevated. Myocardial infarction in the absence of coronary heart disease (IMIHD) Myocardial infarction in the absence of obstructive coronary artery disease (MINOCA) is found in approximately 5-6% of patients with acute MI who undergo coronary angiography. (2). Patients with MINOCA are predominantly younger, female, and do not have dyslipidemia.

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