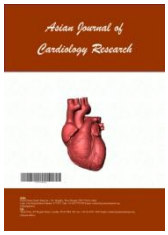

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Causes, Investigations and Treatment of Acute Coronary Syndrome

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

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ABSTRACT

Background: Acute coronary syndrome (ACS) is a general term for the clinical signs and **Symptoms of Myocardial Ischemia:** transient angina, non-ST-segment elevation myocardial infarction, and ST-segment elevation myocardial infarction. This article also describes ACS and the conditions involved; Update your risk factors; Describe its pathology and associated signs and symptoms; Analyze your various diagnostic findings, such as cardiac biomarkers and electrocardiographic changes; And identifies alternative treatments, including recurring treatments and therapies.
Conclusion: This condition results in a sudden decrease in blood flow (and oxygen) to the heart. It is important to understand the cause and to identify its symptoms and signs.

Keywords: Acute coronary syndromes; antiplatelet; antithrombotic; management; non–st elevation mi; revascularization; unstable angina.

1. INTRODUCTION

Acute coronary syndrome (ACS) refers to a variety of presentations found in people with ST-segment elevation myocardial infarction (STEMI) to non-ST-segment elevation myocardial infarction (NSTEMI) or unstable angina. With regard to pathology, ACS is almost always

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associated with atherosclerotic plaque rupture and partial or complete myocardial infarction thrombosis. In some cases, however, stable coronary artery disease (CAD) can lead to ACS in the absence of plaque rupture and thrombosis, in which case the heart needs depression (eg, stroke, blood loss , anemia, infection, tachycardia). The diagnosis of acute myocardial infarction in this setting requires detection of the normal increase and deterioration of biochemical signs of myocardial necrosis in addition to at least 1 of the following: ischemic symptoms, development of pathological Q waves on the electrocardiogram (ECG), significant changes in the ST wave (ST-T-segment-T) or new left bundle branch block (LBBB), visual evidence of new functional myocardial loss or abnormal movement of the regional wall, intracoronary thrombus identified by angiography or autopsy [1].

2. CAUSES AND RISK FACTORS

Acute coronary syndrome (ACS) is mainly caused by atherosclerosis. Many cases of ACS occur as a result of previous malignant lesions (previous atherosclerotic lesions that were not hemodynamically significant but were at risk of rupture). Dangerous plaque is characterized by a large lipid pool, many swollen cells, and a thin, fibrous cap. Increased myocardial oxygen and nutritional requirements, such as strenuous activity, emotional stress, or physical stress (e.g., infection, thyrotoxicosis, or surgery) increase myocardial oxygen and nutritional requirements. Can Without high demand, ACS requires new delivery defects, usually due to thrombosis and / or plaque bleeding. The primary cause of coronary thrombosis is thought to be a rupture of the plaque due to a lack of fibrous cap, a rupture that results in the release of metalloproteinase (collagenase) into the active inflammatory cells. This phenomenon is followed by platelet activation and integration, activation of the clotting pathways, and vasoconstriction. This process results in variable degrees of coronary intracellular thrombosis and vascular obstruction. Distal embolization is possible. The severity and duration of cardiac obstruction, the size of the affected myocardium, the degree of cardiac need, and the ability to compensate for the whole heart are important factors in a patient's medical presentation and outcome. (Anemia and hypoxemia can cause myocardial ischemia if there is no significant reduction in blood flow to the coronary arteries) [2].

2.1 Classification of Acute Coronary Syndrome

Acute coronary syndrome is the name given to three types of coronary artery disease associated with sudden plaque rupture in the coronary artery: chronic angina, non-ST segment elevation myocardial infarction, or heart disease (NSTEMI) and myocardial infarction or heart- attack (STEMI). The location of the obstruction, how long the blood flow is blocked, and the amount of damage that occurs determine the type of acute coronary syndrome. These potentially life-threatening conditions require immediate medical attention (Fig. 1) [3].

2.2 Unstable Angina

Chronic angina is a new symptom or change from stable angina. Angina can occur more frequently, occur more easily during rest, become heavier, or last longer. Although this angina can usually be treated with oral medications, it is unstable and can cause heart attacks. A more aggressive treatment or procedure is usually needed. Chronic angina is a severe coronary syndrome and should be considered a medical emergency [4].

2.2.1 Heart attack: Non-ST segment elevation myocardial infarction (NSTEMI)

This heart attack, or MI, may not cause changes in the electrocardiogram (ECG). However, chemical signals in the blood indicate that damage has been done to the heart muscle. In NSTEMI, prevention can be partial or temporary, and the level of damage is relatively low [4].

2.2.2 Heart attack: ST segment elevation myocardial infarction (STEMI)

This heart attack, or MI, is caused by a sudden and prolonged blood supply. It affects a large area of the heart muscle, and thus causes changes in the ECG and blood levels of vital chemical signals [4].

2.3 Other Terms Associated with a Heart Attack

2.3.1 Stunned myocardium

If blood flow is restored to the heart muscle after a period of ischemia (lack of blood supply), the heart muscle may not pump normally for some

time. This is called the "scared" heart muscle or myocardium [4].

2.3.2 Hibernating myocardium

When they are deprived of enough blood for a long time, some areas of the heart muscle stop working the way they should. Some areas will be permanently damaged. Some areas are able to return to normal function when blood flow is restored to that area (through medication or procedure). Hibernating myocardium heart muscle is “rested” or “asleep” and may return to normal functioning if treated properly [4].

2.3.3 Physiopathology of acute coronary syndrome

Myocardial ischemic symptoms are usually caused by severe degeneration of atherosclerotic plaques at risk. Acute coronary syndrome is caused by an occlusive thrombus in more than 90% of cases. Atherosclerosis is a chronic condition that begins in the arteries and progresses to vascular blockage. This process affects the entire vasculature. Intima layers and media make up the plaque. Plaque formation requires the breakdown or erosion of plaque. Severe ischemic events begin as a result of plaque breakdown, endothelial damage, inflammation and the release of other mediators. Poor integrity in the artery wall restricts flow into the lumen of the vessel, leading to ischemia and bruising. The prevalence of necrosis varies depending on the severity of the lesion, the length of the ischemia, and the size of the area involved [5].

A thrombus, which blocks the lumen of a vessel, is a combination of platelets containing platelet-rich fibrin and erythrocytes. Although several factors are associated with the development of acute coronary syndrome, decreased fibrin degradation due to increased platelet aggregation and a plasminogen activator inhibitor more frequently play a role in the development of cracks and fractures of the lung. plate in the early hours of the morning. USAP, NSTEMI, or STEMI occur due to blockage of blood flow to the vessel lumen due to plaque growth or degree of blockage in the vessel lumen due to a thrombus due to plaque rupture. Pathological studies in patients with NSTEMI acute coronary syndrome have found areas of myocardial injury in locations associated with the corresponding vascular structure (Fig. 2) [5].

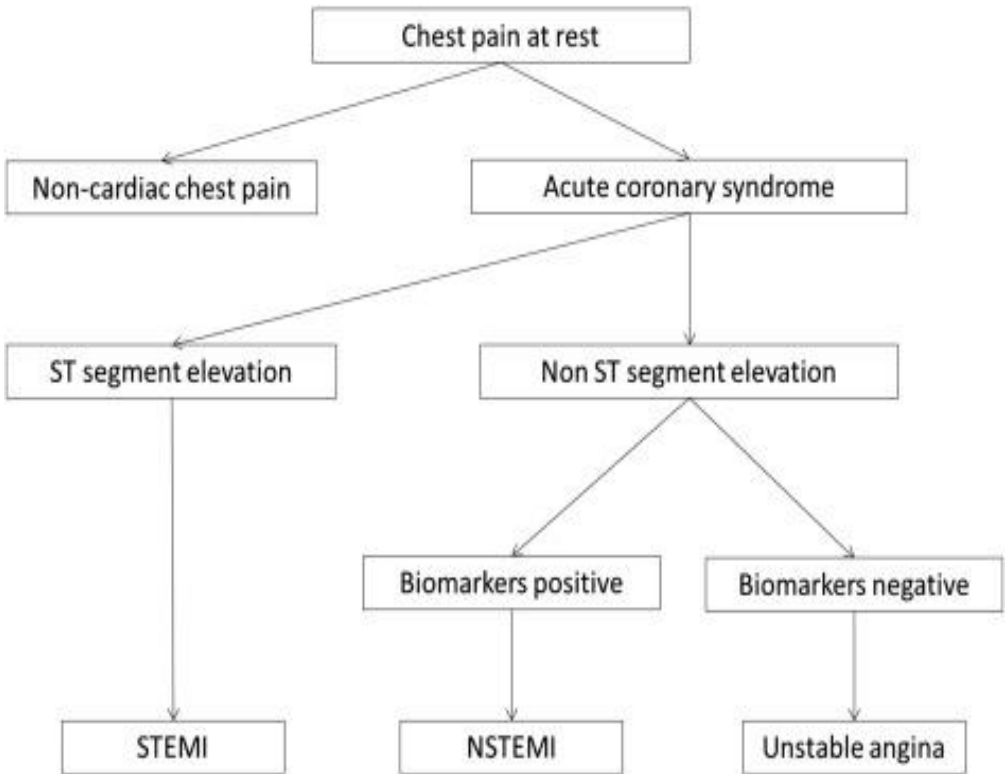

Fig. 1. Classification of acute coronary syndrome [3]

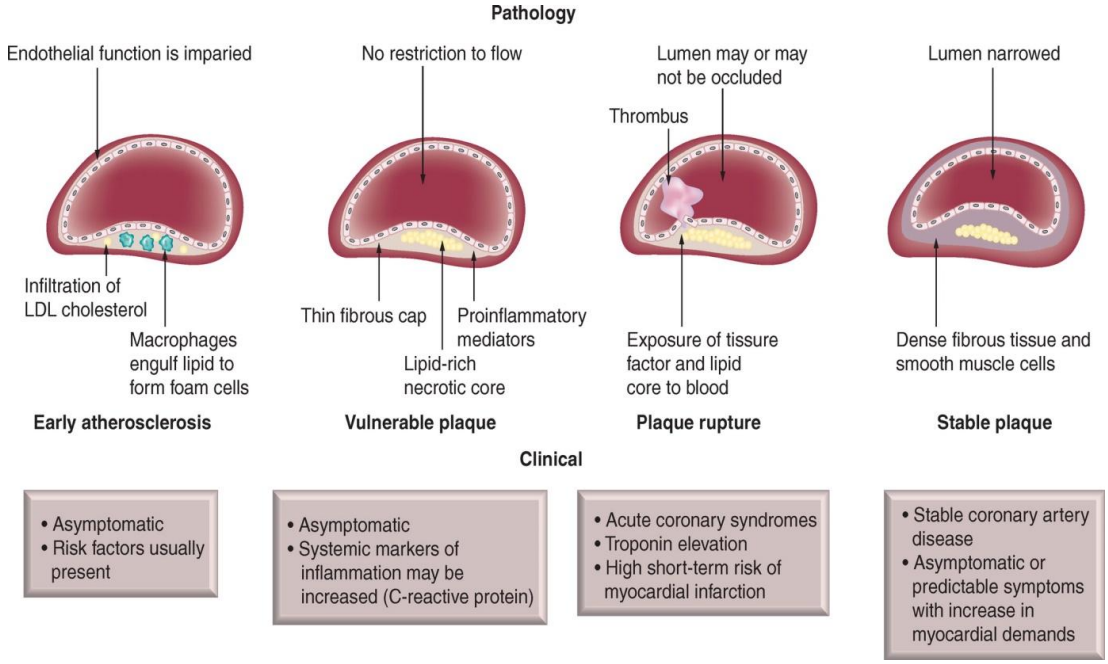

Fig. 2. Physiopathology of acute coronary syndrome [5]

2.3.4 Signs and symptoms

The following is a summary of patient complaints. Heartburn, pain, usually described as a pressure, tension or burning sensation throughout the praecardium, can spread to the neck, shoulders, jaws, back, abdomen, and arms, while dyspnea resolves during laborious rest, Sweating due to microscopic edema, nausea caused by vagal nerve stimulation, impaired exercise tolerance, severe angina, temporary pain lasting 5-15 minutes, actively treated and relaxed or released with nitroglycerin. In transient angina, patients are at increased risk of serious cardiovascular events such as myocardial infarction and death. Angina in the newborn can occur during rest and increases in frequency or duration or is contraindicated for nitroglycerin. Alternative angina (Prinzmetal Angina) is found primarily in rest, caused by smoking and is thought to be caused by coronary spasmodic angina [6].

3. COMPLICATIONS

Acute coronary syndrome can lead to many complications and increase morbidity and mortality. Most of the complications can be classified as follows: electrical dysfunction (driving disorders, arrhythmia), mechanical dysfunction (heart failure, myocardial infarction or aneurysm, papillary tissue dysfunction), thrombotic diseases (ischemia, recurrent coronary artery disease) Electrical dysfunction in patients with infarction (MI) (see also arrhythmia and conduction disorders) Electrical dysfunction usually causing death in the first 72 hours. Existence of complete block (3rd degree) atrioventricular (AV), ventricular tachycardia (VT), and ventricular fibrillation (VF) left-end congestion is rare [6].

3.1 Sinus Node Disturbances

If the artery leading to the sinus node is affected by acute coronary syndrome, sinus node disorder may occur. Most often if there is a sinus node disorder (common in elderly patients). Sinus bradycardia, the most common sinus node disorder, is usually treated as long as there is no hypotension or a heartbeat of less than 50 beats per minute. Low heart rate, if not excessive, means a decrease in heart function and a decrease in infarct size. For hypotension bradycardia (which may reduce myocardial infarction), atropine sulfate 0.5 to 1 mg IV is used. If the answer is not enough, it can be repeated after a while. Some small doses are best because too many doses can cause tachycardia. In some cases, a temporary pacemaker should be installed. Prolonged sinus tachycardia is usually painful, often indicating left ventricular failure and low heart rate. Without heart failure or any other obvious cause, it may respond to arrhythmia beta-blockers, given orally or intravenously, depending on the degree of urgency (Fig. 3) [7].

3.2 Atrial Arrhythmias

Atrial arrhythmias (atrial ectopic stroke, atrial fibrillation, and gradually atrial flutter) occur in approximately 10% of patients who have had a myocardial infarction and may show left ventricular failure or right atrial infarction. Paroxysmal atrial tachycardia is rare and often occurs in patients who have had previous episodes. Atrial ectopia is usually misdiagnosed, but when the rate increases, the cause is sought, especially for heart failure. A normal ectopic heartbeat can react to a beta blocker (Fig. 4) [8].

3.3 Atrial Fibrillation

Arterial fibrillation is usually temporary if it occurs within the first 24 hours (see picture of atrial fibrillation). Risk factors include> 70 years of age, heart failure, history of myocardial infarction, previous infarction, arterial infarction, pericarditis, hypokalemia, hypomagnesemia, chronic obstructive pulmonary disease and hypoxia. Repeated paroxysmal artery fibrillation is a poorly predicted sign and increases the risk of systemic embolism. With arterial fibrillation, heparin (indigestible or low molecular weight) is commonly used because systemic embolism is dangerous. Intravenous beta-blockers (e.g., atenolol 2.5 to 5.0 mg every 10 to 15 minutes to a full dose of 10 mg every 2 minutes, metroprolol 2 to 5 mg every 2 to 5 minutes to a full dose of 15 mg for 15 minutes immediately to the ventricular heart. This reduces heart rate and is usually given when the heart rate is above 100. Heart rate and blood pressure are closely monitored [9].

3.4 Atrial Flutter

For arterial flutter (see picture atrial flutter), the rate is monitored as arterial fibrillation; Heparin (unchanged or low molecular weight) is necessary because the risk of thromboembolism is similar to that of arterial fibrillation. Control of atrial flutter levels in patients with acute MI is generally unsatisfactory. Low energy synchronized direct current (DC) cardioversion will usually remove the atrial flutter [10].

3.5 Conduction Defects

Mobitz type I block (Wenckebach block, continuous extension of the PR interval and reversible bits) is most common in diaphragmatic infarction (see 2nd degree atrioventricular block according to Mobitz type); it tends to be limited and rarely progresses to the upper block. Mobitz-type 2nd degree atrioventricular block PR interval increases with each rate until atrial pressure and QRS complex decrease (Wenckebach event); Atrioventricular nodal conduction resumes at the next rate and the sequence is repeated. Mobitz type 2nd degree atrioventricular block, Mobitz type 2 block (oblique contractions) usually shows a large pre-myocardial infarction, as well as complete heart block with large QRS; both are rare. The frequency of grade 3 (complete) atrioventricular block depends on the location of the infarction (see Diagram of grade 3 atrioventricular block). Complete AV prophylaxis occurs in 5-10% of patients with mild infarction and is usually temporary (Fig. 5) [11].

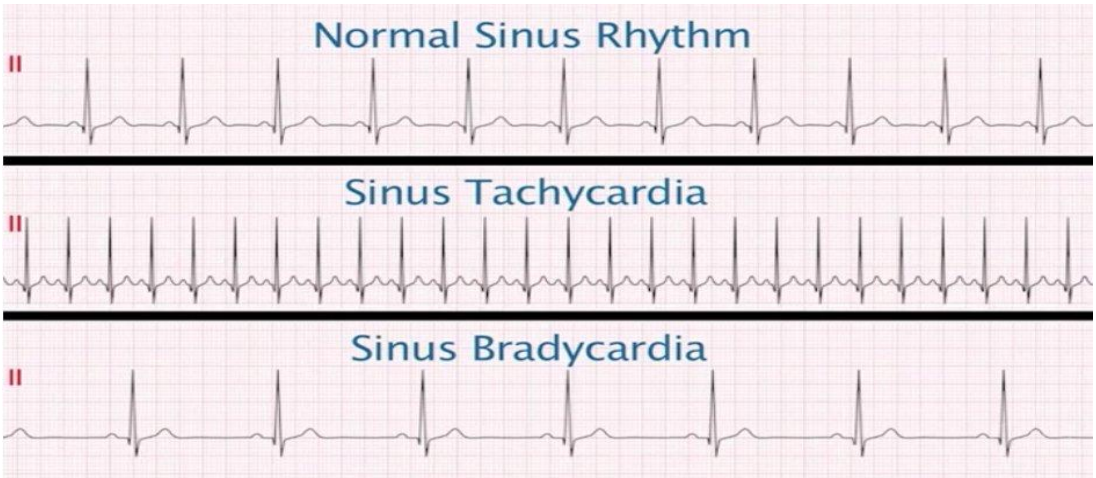

Fig. 3. Sinus node disturbances [7]

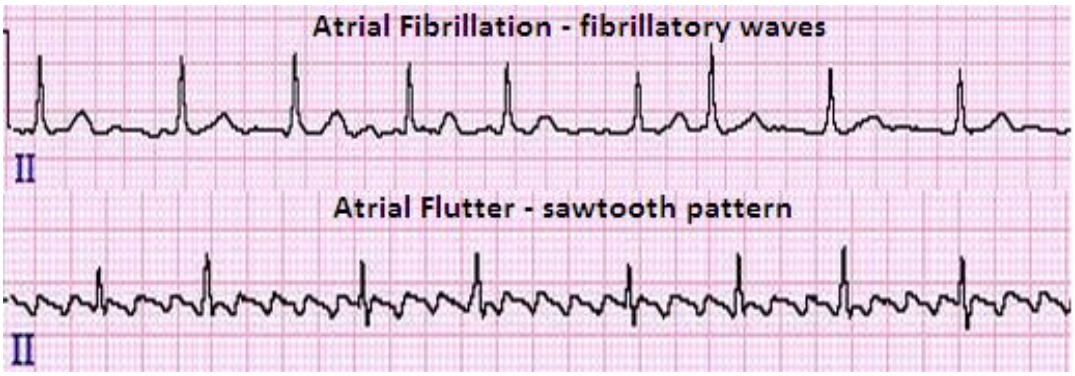

Fig. 4. Atrial arrhythmias [8]

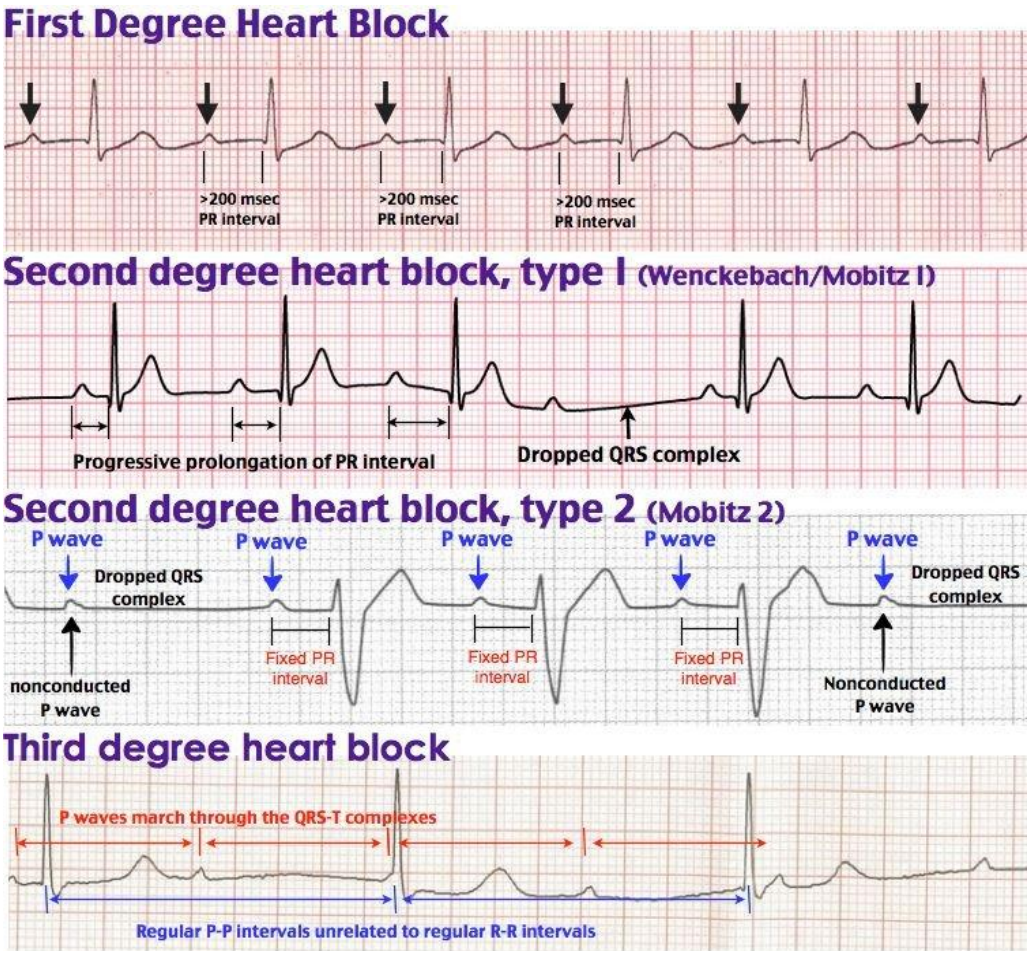

Fig. 5. Conduction defects [11]

3.6 Ventricular Arrhythmias

Ventricular arrhythmias are common and may result from hypoxia, electrolyte imbalances (hypokalaemia, possibly hypomagnesemia), or hypersensitivity to ischemic cells adjacent to the implanted tissue (non-electrical). Investigation and elimination of treatable causes of ventricular arrhythmias. Serum potassium levels should be greater than 4.0 mEq / L (4.0 mmol / L). Intravenous potassium chloride is recommended; usually 10 mEq / hour (10 mmol / h) may be prescribed, but in severe hypokalaemia (potassium level less than 2.5 mEq / L (2.5 mmol / L)) 20 to 40 mEq / hour (20 to 40 mmol / hour) can be fitted with a central venous line.

Ventricular ectopic contractions, which often occur after a myocardial infarction, do not require special treatment. Intravenous beta-blockers at the onset of myocardial infarction followed by continuous oral beta-blockers reduce the incidence of ventricular arrhythmias (including ventricular fibrillation) and mortality in patients without heart failure and hypotension (Fig. 6) [12].

3.7 Ventricular Tachycardia

Chronic ventricular tachycardia (i.e., <30 seconds) and even progressive slow tachycardia (rapid idioventricular rhythm) without hemodynamic instability usually does not require treatment within the first 24 to 48 hours (see figure of Broad QRS ventricular tachycardia). Synchronized cardioversion is designed for: Polymorphic ventricular tachycardia, Sustained (≥ 30 seconds) monomorphic ventricular tachycardia, Any ventricular tachycardia with symptoms of restlessness (e.g., heart failure, blood pressure pain) chest), some doctors also treat complex ventricular arrhythmias with magnesium sulfate 2 g IV for more than 5 minutes whether the serum magnesium level is low or not [13].

3.8 Heart Failure

Heart failure occurs more frequently in four patients: severe myocardial infarction (determined by ECG or heart markers), mechanical problems (eg myocardial infarction or aneurysm, papillary muscle dysfunction), hypertension, dysfunction - diastolic, depending on the size of the clinical findings At the infarct, left upper extremity. Decrease in ventricular filling pressure and cardiac output. Dyspnea, breathlessness and crepitation at the base of the lungs, and hypoxemia are common. Treatment depends on the severity. In severe cases, a diuretic loop (e.g., furosemide 20–40 mg i.v. once or twice a day) is usually sufficient to reduce ventricular filling pressure. In severe cases, vasodilators (e.g., nitroglycerin i.v., nitroprusside) are usually used to reduce pre-stress and post-stress; These drugs are very effective (e.g., for acute pulmonary edema) and can be administered over 24 to 72 hours as needed. During treatment, pulmonary occlusion pressure may be measured by appropriate cardiac catheterization (pulmonary artery), especially if the response to treatment is unfavorable [14].

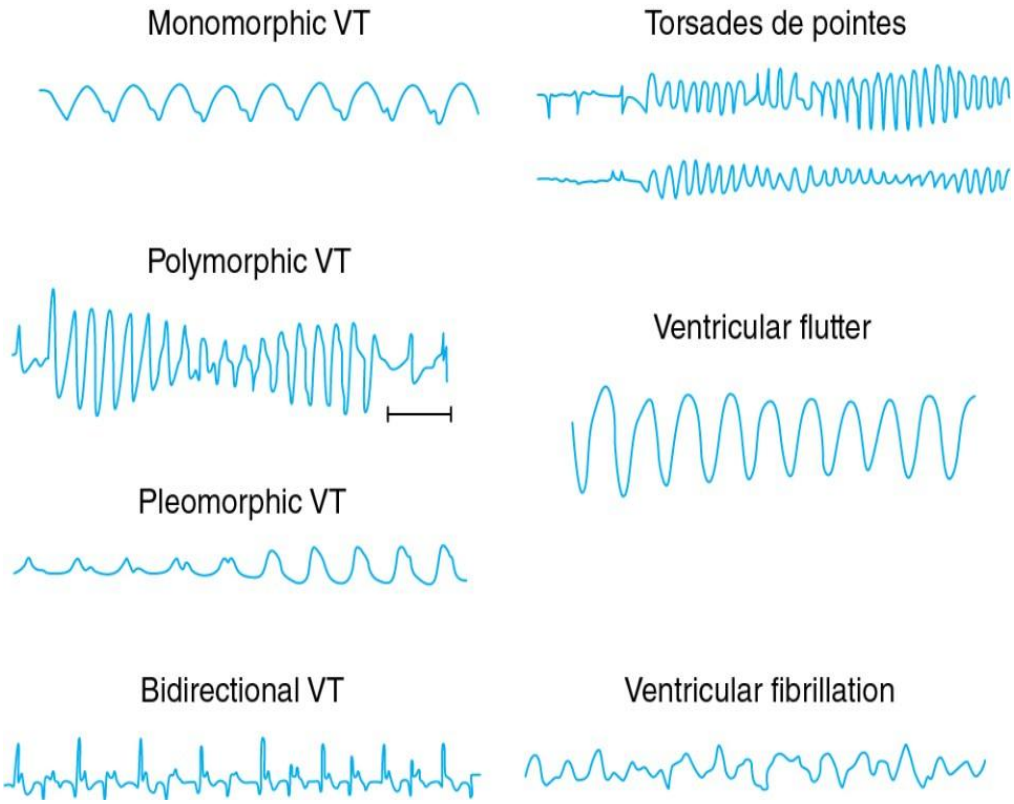

Fig. 6. Ventricular arrhythmias [12]

3.9 Papillary Muscle Disorders

Papillary muscle dysfunction occurs in about 35% of patients within the first few hours after a heart attack. Ischemic dysfunction of the papillary muscles leads to incomplete integration of the mitral valve ducts, which is temporary in most patients. But in some patients, papillary tissue or free wall scars cause irreversible mitral valve recurrence. Papillary muscle dysfunction is characterized by a yellow apical systolic murmur and usually resolves without treatment. Papillary muscle fractures often occur after a lower posterior infarction due to obstruction of the right coronary artery. This causes a severe and severe recurrence of the mitral valve. Papillary muscle fractures are characterized by the sudden onset of apical holosystolic apnea and agitation, usually with pulmonary edema. Sometimes difficult replay is silent. Sudden hemodynamic collapse leads to clinical suspicion of papillary muscle rupture; Echocardiography should be done regularly to make a diagnosis. Emergency mitral valve repair or replacement is required [15].

3.10 Myocardial Rupture

Interventricular septum or free wall rupture occurs in 1% of patients with acute myocardial infarction. It causes 15% of hospital deaths. Interventricular septum rupture, although rare, is 8 to 10 times more common than papillary muscle rupture. Interventricular septum rupture is characterized by the sudden appearance of intermediate fasciculations near the left sternal border in the third or fourth intercostal region with or without severe systolic murmurs and signs of left ventricular failure. The diagnosis can be confirmed using a balloon-shaped catheter and comparing the oxygen saturation or pO2 of the right artery, right ventricular and pulmonary artery [16].

3.11 Ventricular Aneurysm

A local bulge in the ventricular wall, usually the left ventricular wall, can occur in the area of the major infarct. Ventricular aneurysms are common, especially with a large transmural infarction (usually anterior). Aneurysms can develop in days, weeks, or months. They are unlikely to rupture, but can cause ventricular arrhythmia, slow heart rate, and mural thrombosis with systemic embolism. A ventricular aneurysm can be suspected when precordial paradoxical movements are visible or audible, the ECG indicates a progressive exacerbation of the ST segment, and the chest radiograph indicates a rupture of the coronary artery element [17].

3.12 Hypotension and Cardiogenic Shock

Hypotension may be caused by: Decreased ventricular congestion, Loss of secondary contraction in large myocardial infarction, Significant hypotension (e.g., systolic BP <90 mm Hg) with tachycardia and symptoms of hypoperfusion of the last organ (reduced urination, confusion, diaphoresis., cold edges) is called cardiogenic shock. Pulmonary congestion develops rapidly in cardiogenic shock. Decreased left ventricular filling is often caused by a decrease in secondary venous recurrence in hypovolemia, especially in patients receiving intensive loop diuretic therapy, but may indicate the incidence of ventricular infarction. Marked pulmonary congestion suggests loss of left ventricular contractility (left ventricular failure) as a cause. Treatment depends on the cause. In some patients, finding the cause requires the use of a pulmonary artery catheter to measure intracardiac pressure. If the pulmonary occlusion pressure is <18 mm Hg, reduction of filling, usually due to hypovolemia, is possible; if the pressure is above 18 mm Hg, left ventricular failure may occur. With hypotension due to hypovolemia, fluid replacement with 0.9% saline is usually possible without left cardiac output (excessive increase in left atrial pressure) [18].

3.13 Cardiogenic Shock

About 5 to 10% of patients with acute myocardial infarction have a heart attack. In cardiogenic shock, alpha agonist or beta agonist may be temporarily effective. Catecholamine, with dopamine, alpha and beta-1 effects, is given intravenously at a dose of 0.5-1 mcg / kg / min, until the response is satisfactory or increased to about 10 mcg / kg / min. High doses cause vasoconstriction and arterial and ventricular arrhythmias. Dobutamine, a beta agonist, can be given intravenously in doses of 2.5-10 mcg / kg / min or more. Often causes or worsens hypotension; Most effective when hypotension increases low cardiac output and peripheral arterial resistance. Dopamine is more effective than dobutamine when a vasopressor effect is also needed. In cases of cardiogenic shock, a combination of dobutamine and dopamine is possible. A combination of dobutamine and a

drug with a high alpha-adrenergic effect (phenylephrine, norepinephrine) can work without causing multiple arrhythmias [19]..

Right Ventricular Ischemia or Infarction: Right ventricular infarction is rarely uncommon. The first indication may be an increase in hypotension in an already stable patient. The correct ECG can detect changes in the lead ST segment. Loading 1 to 2 L volume of 0.9% saline is generally efficient. Dobutamine or Milrinone (with better diffusion effects) may help. Nitrates and diuretics are not used. They reduce pre-loading (and therefore heart failure), leading to severe hypotension. Increased filling pressure on the right side with IV fluid infusion should be maintained, but high volume filling may interfere with left ventricular filling and cardiac output [20].

Recurrent Ischemia: Chronic or recurrent chest pain 12 to 24 hours after myocardial infection may represent recurrent ischemia. Ischemic pain after MI indicates an increased risk of additional myocardium infarction. In general, recurrent ischemia can be detected by reverse ST-T mutations of the ECG. Blood pressure may rise. Recurrent ischemia can subside in one-third of patients (ECG changes without pain), so regular ECGs are performed daily and every 8 hours. Recurrent ischemia (actually LLC Brownwald's classification of unstable angina, which is classified as a stage of unstable angina) is treated like chronic angina. Sublingual or IV nitroglycerin usually works best. Consideration should be given to coronary angiography to save ischemic myocardium and percutaneous coronary intervention or coronary artery bypass grafting for revascularization [21].

Mural Thrombosis: Before treating the modern age, wall thrombosis occurs in approximately 20% of patients with acute myocardial infarction. Systemic embolism occurs in approximately 10% of patients with left ventricular thrombosis. The risk is highest during the first 10 days, but lasts for at least 3 months. The risk is very high in patients with major anterior infarction (mainly including the distal septum and apex), diffuse hypomotility with an enlarged left ventricle, or chronic atrial fibrillation (about 60%). With modern treatments, the risk of wall thrombosis is very low. Antidepressants are generally not recommended to prevent wall thrombosis after acute coronary artery disease. In patients with STEMI and anterior wall dyskinesia or dyskinesia, anticoagulant therapy may be considered, but if anticoagulation is chosen, the patient's risk of bleeding is assessed by two antiplatelet therapy and three. It is necessary to consider the therapeutic effect. Antipsychotics are recommended for patients at high risk for ACS, atrial fibrillation, thromboembolism (e.g., CHA2DS2-VASc ≥ 2), prosthetic heart valves, venous thromboembolism, and hypercoagulant disease. It also makes sense to prescribe anticoagulants to patients with STEMI, Asymptomatic LV wall thrombus confirmed [21].

Pericarditis: Pericarditis is caused by an increase in the wall myocardial necrosis of the epicardium; It begins in about one-third of patients with severe transmural myocardial infarction, although the rate is much lower in patients with premature relapse. Frequent fractures usually begin 24 to 96 hours after the onset of myocardial infarction. Premature onset of friction is uncommon, although bleeding pericarditis sometimes complicates the first stage of myocardial infarction. Acute tamponade is rare. Pericarditis is diagnosed by ECG, which indicates elevated ST segment and sometimes PR-intermittent depression. Echocardiography is frequently performed, but the results are usually general. Occasionally a small pericardial fusion occurs with an unexpected tamponade. Aspirin or another anti-inflammatory drug (NSAID) usually relieves symptoms. Colchicine 0.5 to 1 mg once daily is added orally, alone and especially in conventional therapies, to speed up recovery and help prevent relapse. High doses or prolonged use of NSAIDs or corticosteroids may impair the treatment of infections and should be avoided; Corticosteroids may increase the likelihood of relapse. Antibody is not contraindicated in early peri-infarction pericarditis but in the most recent post-MI (Dressler) syndrome [22].

Post-MI Syndrome (Dressler Syndrome): Post-MI syndrome begins days, weeks, and even months after an acute myocardial infarction in some patients. Incidents also seem to have decreased in recent years. It is characterized by fever, rubbing pericarditis, pericardial effusion, pleurisy, pleural effusion, lung infection, and arthralgia. The disease is caused by an autoimmune response to substances derived from necrotic muscle cells. You can repeat it. It can be difficult to distinguish between Dressler syndrome and enlarged or recurrent infarction. However, in post-MI syndrome, cardiac symptoms do not increase significantly and changes in ECG are less direct. NSAIDs usually work well, but the syndrome can recur several

times. Colchicine is effective in treating and preventing recurrence. In severe cases, a short and intensive course of another NSAID or corticosteroid may be required. High doses of NSAIDs or corticosteroids may interfere with early ventricular healing after acute myocardial infarction and should not be used for more than a few days [23].

Prevention: There are ways to reduce the chances of having an NSTEMI and improving overall heart health. Steps people can take to reduce their risk of an NSTEMI include: having a healthy, nutritious, and balanced diet, including fruits, vegetables, healthy fats, and whole grains, reducing and limiting foods that are high in saturated and trans fats, exercising regularly, recommended as at least 30 minutes five times a week, managing stress levels, quitting a smoking habit, remaining at a healthy weight, If a person has diabetes, high cholesterol, or high blood pressure, it is essential to manage these conditions well. Not doing so could increase the risk of a heart attack. If a person is deemed to be at risk of having a heart attack, it is advisable for them to take precautions so that they are prepared. They should ensure emergency contact numbers, a list of current medications and any allergens are to hand whenever they are heading out or going away, in case a heart attack occurs [23].

Investigations: If you have symptoms or signs related to coronary syndrome, the emergency room doctor will probably order several tests. Other tests can be done when your doctor asks you questions about your symptoms or medical history. Testing includes [24].

Electrocardiogram (ECG): The electrodes attached to your skin measure the electrical activity of your heart. Abnormal or abnormal emotions can indicate that your heart is not working properly due to lack of oxygen. Some patterns in electrical signals may indicate a common obstruction area. The test can be repeated several times (Fig. 7) [24].

Blood tests: Certain enzymes can be found in the blood when cell death has caused damage to heart tissue. A positive result indicates a heart condition. Information from these two tests and your signs and symptoms are used to diagnose acute coronary syndrome. Your doctor can use this information to determine whether your condition can be classified as a heart attack or angina (Fig. 8) [25].

Coronary angiogram: This procedure uses X-ray images to identify the blood vessels in your heart. A long, narrow tube (catheter) is attached to the arteries of your heart through a vein, usually in your arm or uterus. The dye flows through a tube into your vein. A series of X-rays will show how the dye moves through your arteries, revealing any blockages or thinning. Catheters can be used for treatment (Fig. 9) [26].

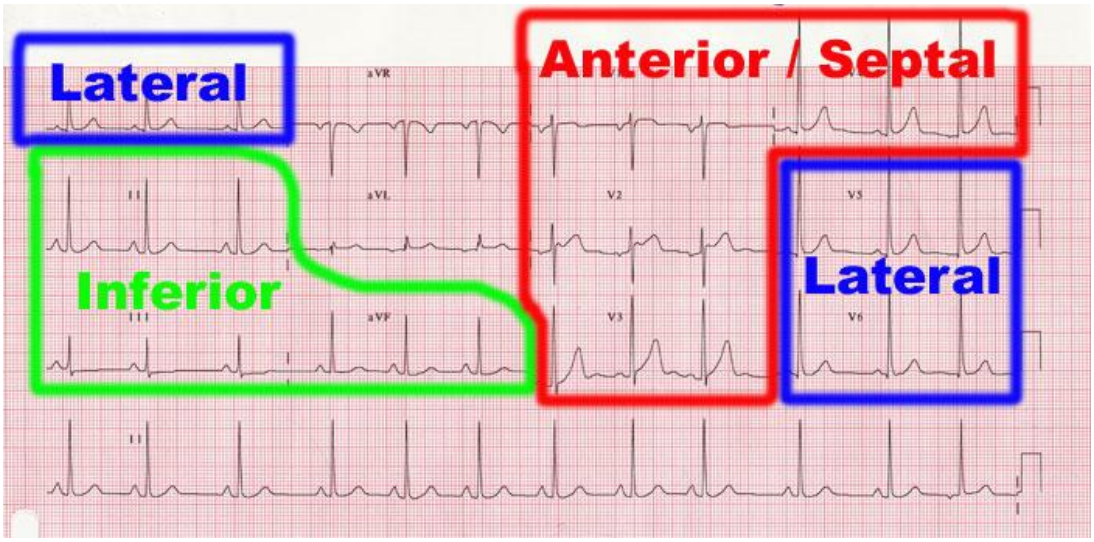

Fig. 7. Electrocardiogram (ECG) for acute coronary syndrome [24]

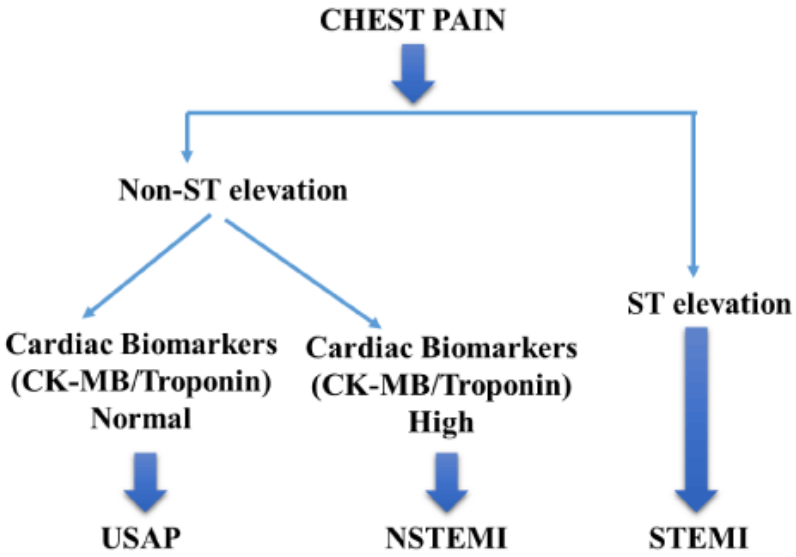

Fig. 8. Blood tests for acute coronary syndrome [25]

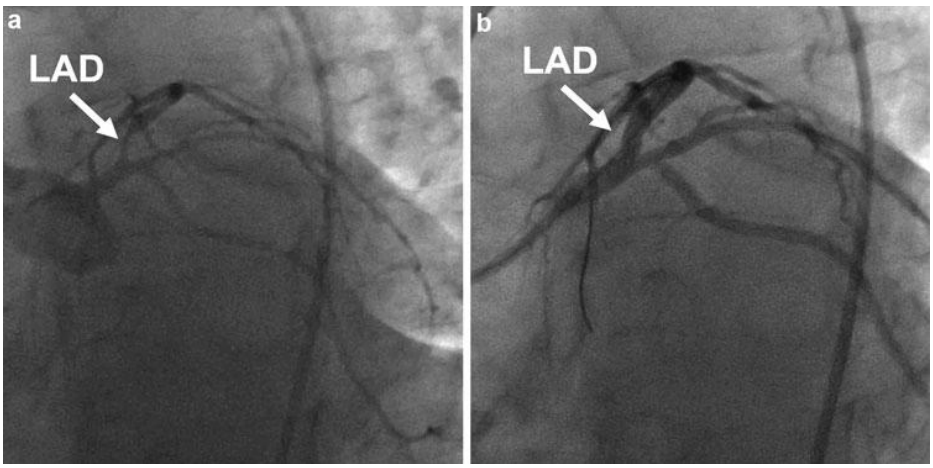

Fig. 9. Coronary angiogram for acute coronary syndrome [26]

Echocardiogram: An echocardiogram uses sound waves, directed at your heart from a wand-like object, to produce a live image of your heart. An echocardiogram can help determine if the heart is pumping properly (Fig. 10) [27].

Myocardial perfusion imaging: These tests show how well the blood flows to your heart muscle. A small, safe amount of radiation is injected into your bloodstream. A special camera captures route images of your heart. They show your doctor if adequate blood flows to the heart muscle and blood flow is reduced (Fig. 11) [28].

Computerized tomography (CT) angiogram: A CT angiogram uses a special X-ray technique that can create two separate 2D parts of your heart. These images can detect small or blocked coronary arteries (Fig. 12) [29].

Stress test: Stress tests will tell you how well your heart works when you exercise. In some cases, you may take medication to increase your heart rate instead of exercising. This test is performed only if there are no symptoms of acute coronary syndrome or other fatal heart conditions. During stress testing, ECG, echocardiogram or myocardial perfusion imaging can be used to see how your heart is working [30].

Treatment: Depending on your diagnosis, emergency medicine or ongoing care (or both) may include the following: Thrombolytics (clot buster) helps to dissolve the blood clot that

blocks the artery. Nitroglycerin improves blood flow by temporarily dilating blood vessels. Antiplatelet drugs help prevent blood clots and form aspirin, clopidogrel (Plavix), prasugrel (Effient) and others. Beta blockers help to relax your heart muscle and slow down your heart rate. They lower your heart rate and lower your blood pressure. Examples include metoprolol (Lopressor, Toprol-XL) and nadolol (Corgard). Angiotensin-converting enzyme (ACE) inhibitors increase blood vessels and improve blood flow, allowing the heart to function better. They include lisinopril (Prinivil, Zestril), benazepril (Lotensin) and others. Angiotensin receptor blockers (ARBs) help control blood pressure and include irbesartan (Avapro), losartan (Cozaar) and a few others. Statins reduce the amount of cholesterol in the blood and may stabilize plaque deposits, making them less likely to explode. Statins include atorvastatin (Lipitor), simvastatin (Zocor, Flolipid) and several others [31].

Surgery and other procedures: Your doctor may recommend one of these procedures to restore blood flow to your heart muscle [32].

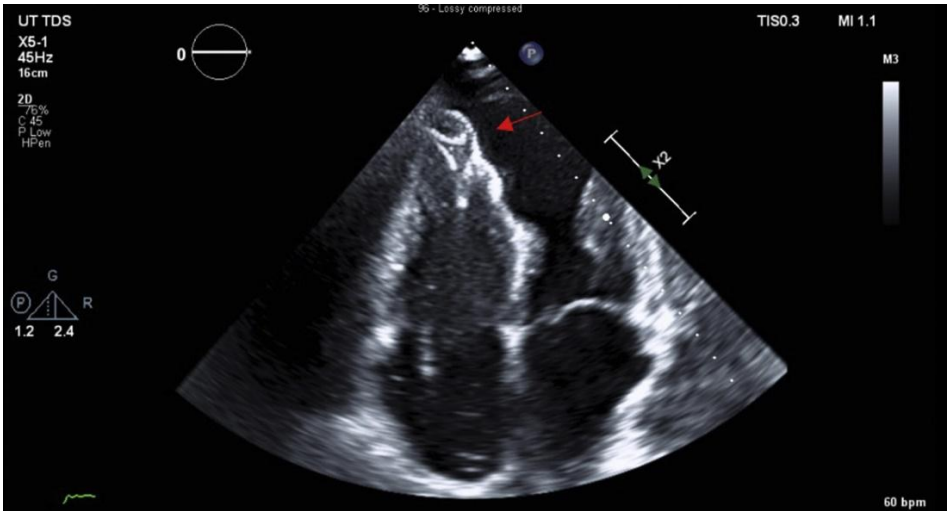

Fig. 10. Echocardiogram for acute coronary syndrome [27]

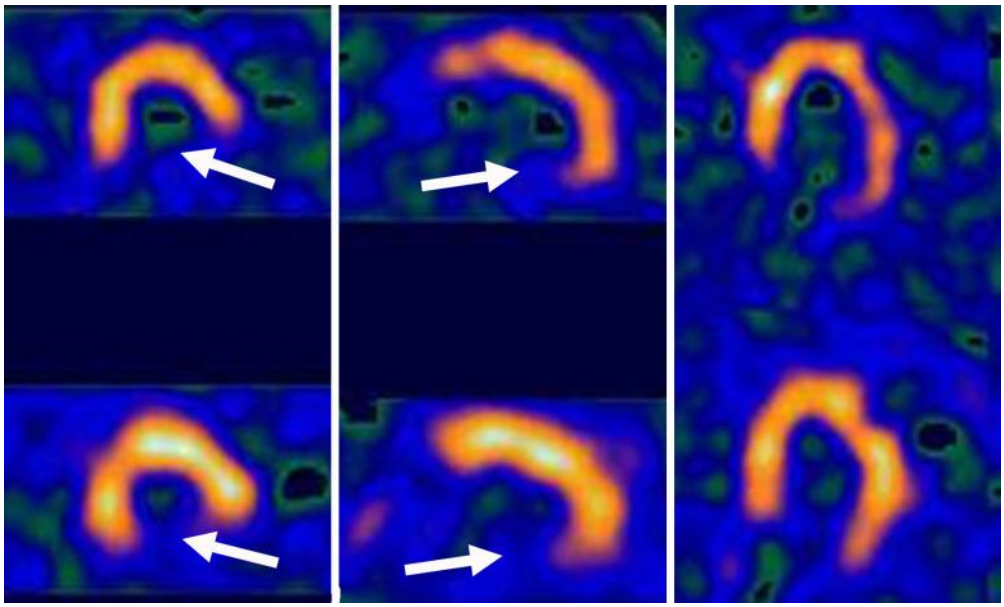

Fig. 11. Myocardial perfusion imaging for acute coronary syndrome [28]

Angioplasty and stenting: In this procedure, your doctor inserts a long, narrow tube (catheter) into a closed or narrow portion of your artery. A wire with a melted balloon is transferred from a catheter to a narrow space. The balloon is then inflated, opening the artery by pressing plaques on the walls of your arteries. A mesh tube (stent) is usually left in the vein to help keep the vein open (Fig. 13) [32].

Coronary bypass surgery: With this procedure, the surgeon takes a piece of blood vessel (connective tissue) from one part of your body and creates a new blood vessel that circulates (passes through) the blocked blood vessel (Fig. 14) [32].

4. DISCUSSION

Acute coronary syndrome is caused by severe blockage of a coronary artery. Outcomes depend on the severity and extent of angina to non-ST segment elevation myocardial infarction (NSTEMI), ST segment elevation myocardial infarction (STEMI), and original sudden cardiac death. Symptoms are similar in each of these syndromes (except for sudden death) and include chest pain with or without shortness of breath, nausea, and sweating. Diagnosis is made by the presence or absence of ECG and serological symptoms. Treatment with antiplatelet agents, anticoagulants, nitrates, beta-blockers and, in STEMI, emergency replacement

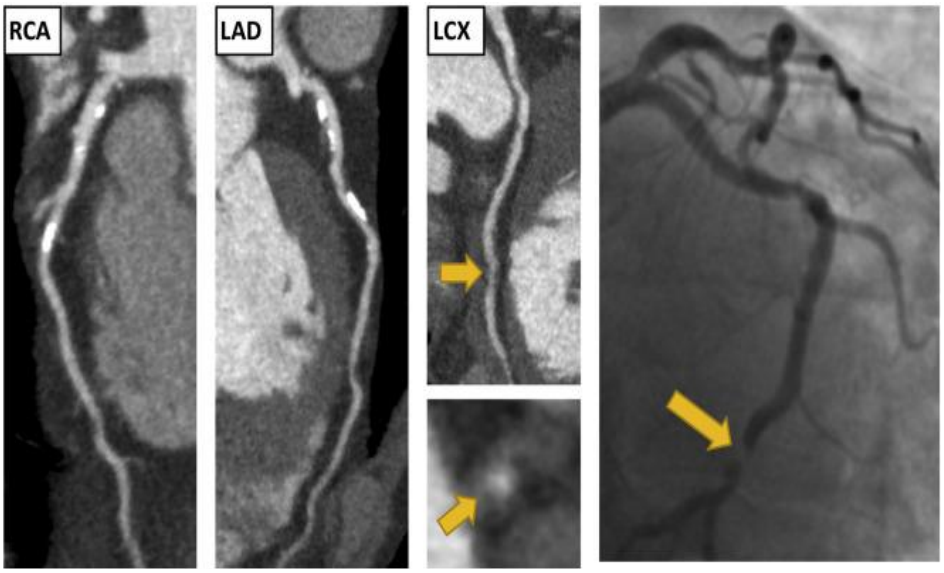

Fig. 12. Computerized tomography (CT) angiogram for acute coronary syndrome [29]

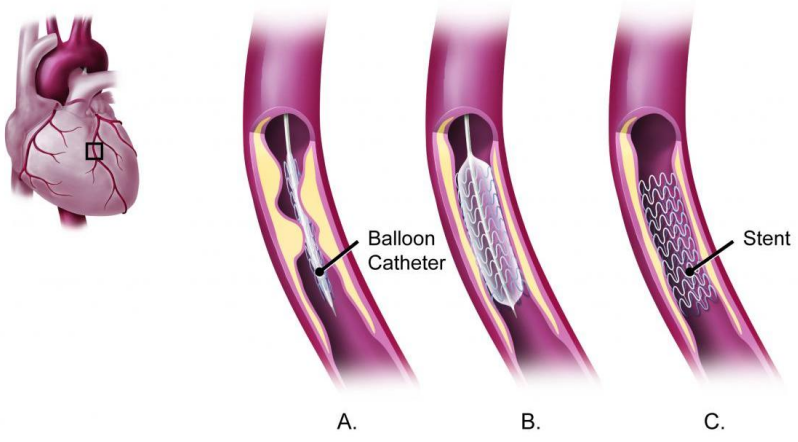

Fig. 13. Angioplasty and stenting for acute coronary syndrome [32]

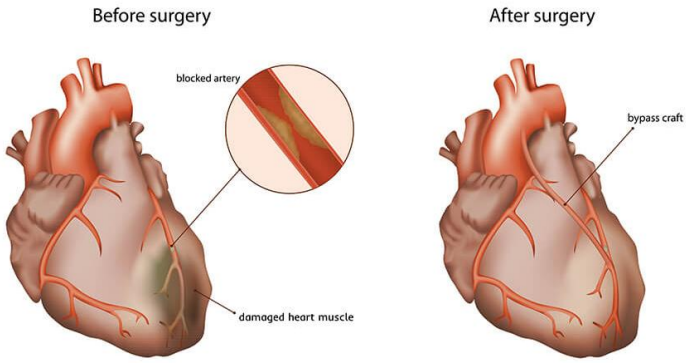

Fig. 14. Coronary bypass surgery for acute coronary syndrome [32]

with fibrinolytic drugs, percutaneous intervention or, intermittently, graft surgery for grafts - coronary bypass surgery [32].

5. CONCLUSION

Acute coronary syndrome is one of the leading causes of illness and death in the United States. Family physicians should identify and mitigate risk factors early and diagnose and respond to coronary syndrome cases immediately in any medical setting. Diagnosis can be made based on the patient's history, symptoms, electrocardiographic findings, and biomarker cardiac symptoms, which distinguish between ST-elevation myocardial infection and non-ST-elevation acute coronary syndrome. Rapid recovery with primary percutaneous coronary intervention is a medically advanced goal. With proper medical management, percutaneous coronary interventions can improve short- and long-term outcomes after myocardial infarction. If percutaneous coronary intervention is not performed immediately, patients with ST elevation myocardial infection can be treated with fibrinolytic therapy. Fibrinolysis is not recommended for patients with non-ST elevation acute coronary syndrome. Therefore, these patients should seek medical treatment if they are at low risk for coronary events or if percutaneous coronary intervention is not possible. Post-myocardial infarction care should be closely monitored by the patient's cardiologist and supported through a comprehensive secondary prevention strategy to prevent recurrence, disease and death.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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