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MYOCARDIAL INFARCTION

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ABSTRACT

Myocardial infarction (MI) occurs due to the slow buildup of plaque in arteries, leading to decreased blood flow, which subsequently injures the heart muscles, causing them oxygen starvation. The most common symptoms are pain and discomfort in the chest, dyspenea, perspiring, nausea, vomiting, irregular heartbeat patterns, anxiety, extreme tiredness, weakness, mental pressure, subdued feelings and other underlying reasons. Emergency Medical Responders can offer oxygen, give nitro-glycerine tablets, or treat the muscle tissue injury and blood clot with aspirin. The best way to cope with MI is to work on keeping track of body weight, diet, fat, cholesterol, and salt intake, monitoring blood pressure, as well as exercise and refraining from smoking, excessive drinking, or drug use. MIs can be treated in further detail using thrombolytic or clot-dissolving drugs, anti-hypertensives, painkillers, and nitro-glycerine. Suppressed pacing of life over the last fifty years recorded an outcome of patients' deaths being lower alongside new tech improvement garnering novel treatment. Methods of medical treatment improved dramatically but still had some gaps due to the three- and 0-day death rates of patients suffering with cardiogenic shock and undergoing palliative care. These methods starting from the 70s paved the way into the streamlined abdomen infractive method imprinted interfaces and eradicated the entire boundary of the heart muscle without recognition, while the counterpart had a boundary.

KEYWORDS: Myocardial infarction (MI), dyspenea, anti-hypertensives, painkillers, heart muscle.

INTRODUCTION

An acute myocardial infarction, or heart attack, occurs when a part of the heart suffers necrosis due to a decrease or stoppage in blood supply being delivered to it. The primary cause over here is generally a blood clot within an epicardial artery that supplies that region of the heart muscle. However, it is now understood that not every case requires a blood clot as a cause. A living tissue, such as a heart muscle, requires the blood supply to be equivalent to the

oxygen the muscle is demanding. If there is an imbalance in this supply-demand ratio it is possible that myocardial damage occurs, but a blood clot is not involved at all. To assist clinicians with the AMI diagnosis, a universal definition has been available over the past decade.[1] The particular definition states that there must be evidence for two of the following; Is there one value above the 99th reference limit? And is there at least one rise or fall in a fast-track blood test sensitive to heart muscle damage, which is troponin I or T? Alongside these requirements, clinical proof of having experienced an AMI must also be provided. It includes clinical evidence of ischemia-like symptoms, including changes in the ST segment or an appearance of new left bundle branch block, development of abnormal pathological Q waves on the electrocardiogram (ECG), new wall motion abnormalities on cardiac testing, or some combination of these phenomenon. The majority of heart attacks seen are secondary to angiographic coronary artery disease, which is the most common cause of mortality in the US. Combinations of time and lack of oxygen are devastating to the myocardium. Ischemic heart disease causes patients to feel some form of chest pain or sensation of pressure that has the potential to spread to the neck, jaw, shoulder, or arm. Electrophysiologic changes alongside evidence of myocardial injury such as release of cardiac troponins are also likely to indicate myocardial ischemia.[2]

TYPES OF MYOCARDIAL INFARCTION

TABLE-1

Table 1 Clinical classification of different types of myocardial infarction

Type 1	Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection
Type 2	Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension
Type 3	Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood
Type 4a	Myocardial infarction associated with PCI
Type 4b	Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy
Type 5	Myocardial infarction associated with CABG

Source: https://www.ahajournals.org/cms/10.1161/CIRCULATIONAHA.107.187397/asset/f869ed25-532d-4572-a925-fdd99a6c87c3/assets/graphic/23ff2.jpeg

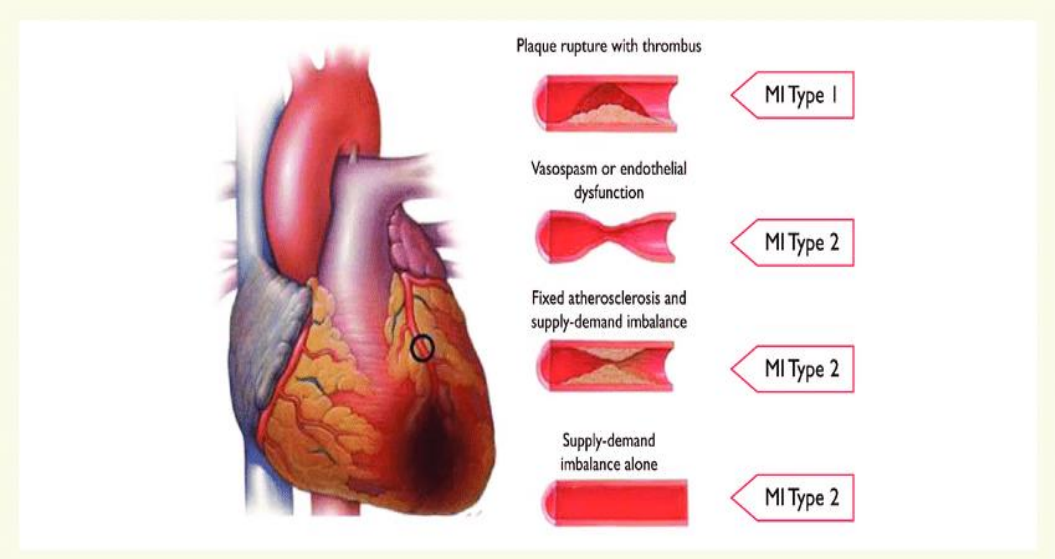

Figure 1.

Source https://www.researchgate.net/figure/Differentiation-between-myocardial-infarction-MI-types-1-and-2-according-to-the_fig1_232713536

CAUSES

Coronary artery spasm: A heart attack may arise from excessive spasm of a coronary artery which interrupts blood flow to the heart muscle. This is far less frequent than blockage caused by plaque or a blood clot. When a spasm happens, the heart muscle goes through ischemia, which contributes to the heart muscle tissue slowly dying or getting irreversibly damaged, leading to an MI. This condition can occur in arteries that do not have substantial plaque buildup or in arteries that do have atherosclerotic plaque, which makes the problem more complex.[3]

Atherosclerosis: Many times, atherosclerosis is responsible that happens with a buildup of plaque in the arteries of the heart, this plaque is made up of cholesterol, fatty substances, cellular waste products, calcium, and fibrin. This buildup can clog arteries and slow blood flow to the heart muscle. Coronary artery disease (CAD) may develop due to narrowing in these arteries, which reduces oxygen-rich blood. A critical event in MI is the rupture of an atherosclerotic plaque, which triggers the formation of a blood clot (thrombus) at the rupture site, blocking the flow of blood through the coronary artery. This blockage can cause damage to heart muscle tissue, leading to a myocardial infarction if blood flow is not restored quickly. The most common cause of MI is the accumulation of plaque within the coronary arteries.[4]

Blood Clots (Thrombus): In atherosclerosis, stable plaques in the coronary arteries become unstable owing to plaque rupture, which can result in the production of a thrombus (blood clot). A heart attack may result from this clot blocking blood flow via the molecule that supplies blood to the coronary artery. The body's clotting mechanism is triggered when plaque bursts, exposing the underlying tissue to blood. Platelets constrict to form a thrombus, or blood clot. Thrombus blockage is a blood artery aggravating factor. This blood thalamic event interrupts the coronary artery blood thalamus, depriving the blood of oxygen and causing damage to critical leaf muscle tissue.[5]

PATHOPHYSIOLOGY

It includes three stages. They are,

Stage-1 Myocardial ischemia: Ischemia of the myocardium is a result of decreased flow to the coronary vessels, increased chest muscular activity, or even the presence of heart enlargement without proportional increase. The muscle's (myocardium) blood supply gets limited and as a result, its oxygenated blood is diminished. Most cases stem from the accumulation of plaque within the coronary arteries, also known as atherosclerosis.[6] A heart attack or a myocardial infarction manifest when one of the coronary arteries is completely obstructed, which frequently happens through a clot forming on the breached plaque. Mildly prolonged ischemia in the myocardium has to do with the precursor condition of a heart attack where tissue of the heart muscle dies. Put differently, ischemia is the 'precautionary message' while infarction embodies the meaning damag.[7]

Stage-2 Role of platelets: The break of an atherosclerotic plaque exposes subendothelial collagen to collagen, which causes platelets to aggregate, activate, and release, resulting in increased platelet mass and risk for embolism or thrombosis. The rupture of an atherosclerotic plaque in a coronary artery leads to thrombi, or blood clots, that can activate platelet adhesion and draw blood away from the heart, which can cause a heart attack. During a heart attack, platelets take part in the inflammatory response, releasing chemical substances that draw other inflammatory cells to the injury placed together.[8]

Stage -3 Acute plaque rupture: High-grade coronary atherosclerosis may result in ischemic heart disease without precipitating an acute myocardial infarction (MI) and can still prompt episodes of angina pectoris. However, acute coronary atherosclerotic complicating lesions, such as coronary thrombosis on top of plaque rupture and plaque hemorrhage, are mostly seen during an acute MI. In a MI, plaque rupture occurs as the internal core of the plaque is broken due to the severe shear forces associated with strong blood flow. In addition to platelets, other cellular elements within the blood also get activated by the exposed substances and undergo a conformational change that enhances the clotting process. Indeed, the thrombus they form, obstructs the artery and reduces blood flow to the heart muscle and the tissue can become ischemic, later becoming infused with blood or a heart attack.[9] Thus, a thrombus causes damage to coronary arteries at the ruptured plaque**.**

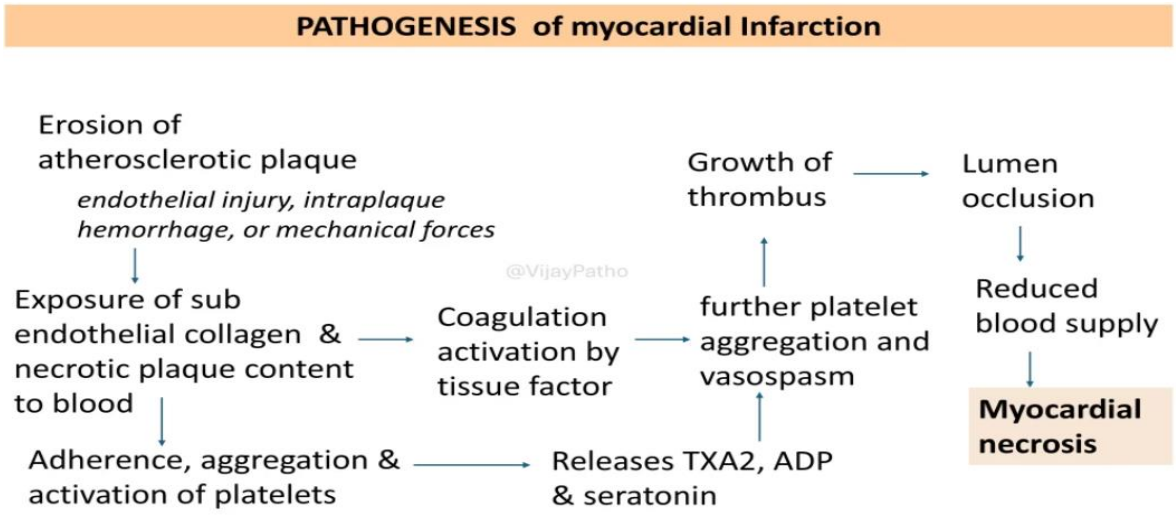

Figure 2.

Source: https://ilovepathology.com/myocardial-infarction-pathogenesis-and-infarct-types/

SIGNS AND SYMPTOMS

- Chest pain
- Shortness of breath
- Heartbeat changes
- Cold sweat
- Nausea and vomiting
- Dizziness
- Fainting
- Stiffness
- Fatigue
- Stomach discomfort
- Chest tightness
- Numbness in the upper body
- Heartburns

RISK FACTORS

- High blood pressure or hypertension
- Family history
- Genetic history
- Obesity or overweight
- Alcohol consumption
- Smoking
- Sedentary lifestyles
- Lack of healthy diet
- High cholesterol
- Lack of physical activity
- Diabetes
- Other any inflammatory diseases

COMPLICATIONS

1. **Congestive heart failure:** Around fifty percent of MI patients experience heart failure (CHF), which can be of the right ventricular type, the left ventricular type, or a combination of both. Heart attacks harm or completely destroy sections of heart muscles, which causes these muscles to become ineffective in pumping blood. As a consequence, the ventricles' pumping becomes less efficient, causing blood and fluids to collect in the lungs and other tissues.[10]
2. **Arrhythmias:** Heart attacks often lead to irregular heartbeats, which called as arrhythmias. These happen when the heart attack damages or irritates the system that controls the heart's rhythm. Other things cause arrhythmias too. When heart muscle cells don't get enough oxygen, they leak potassium. Also, the fluid around the heart cells gets more lactic acid and free fatty acids. These changes can make the heart beat funny. Arrhythmias come in different types. Some people get a fast regular heartbeat, while others have a quivering in their upper heart chambers.

Sometimes the heart throws in extra beats, or the lower chambers quiver. This last type can kill you if not treated. A heart attack can hurt the heart muscle. This can mess up the electrical signals that tell the heart when to beat. As the heart heals, it forms scar tissue. This scar tissue can also cause irregular heartbeats later on. When your body's chemicals get out of balance especially potassium and magnesium, your heart might start to beat.[11] Also, because a heart attack reduces blood flow, it can irritate the heart tissue. This makes the heart more likely to develop an irregular rhythm.

3. **Rupture:** The pericardial cavity's diseased ventricle wall causes the breakdown, which results in hemopericardium and tamponade. Papillary muscle rupture, interventricular septum, and left ventricular infarction are further locations. These ruptures are frequently deadly and typically happen during the first week. A sharp drop in blood pressure is the result of cardiac tamponad, which happens when the heart is compressed by the blood.[12] When blood flow is disrupted by both the ventricles and the papillary muscle, heart failure results, leading to cardiogenic shock. Following a myocardial infarction, breaking is a dangerous consequence that has a high fatality rate.
4. **Pericarditis:** Transmural infarcts frequently result in sterile pericarditis, which is characterized by fibrinous pericarditis and goes away on its own. It frequently has little practical importance and goes away on its own. An immune system reaction brought on by a heart attack may inflame the heart's tissue and maybe spread to the pericardium. A particular kind of pericarditis known as Dressler's Syndrome, which is thought to be an autoimmune reaction, appears weeks to months following a heart attack or surgery.[13]
5. **Mural thrombolysis and thromboembolism:** Wall thrombosis in the heart occurs due to the involvement of endocardial and subsidy in a heart attack and the deceleration of heart rate. These thromboembolic can cause occlusion of pulmonary, kidney, mesenteric, splenic, pancreatic or brain arteries, leading to heart attacks in these organs. The stroke is the most feared complication, as a clot on the left side of the heart can block blood flow to the brain. Peripheral arterial embolism can cause severe pain, coldness, numbness and tissue death, requiring amputation. Pulmonary embolism (PE) is less common, but it is still possible, causing shortness of breath, chest pain, coughing blood and rapid heart rate. Systemic embolism to other organs can cause damage.[14]
6. **Cardiogenic shock:** Acute heart attack often results in cardiogenic shock, which is defined by hypotension and a systolic blood pressure of 80 mmhg or below for many days. This shock can cause low blood pressure, reduced heart production and damage to the heart muscle, all of which can weaken the blood supply to critical organs. Even with the intensive care unit includes cardiogenic shock consequences, arrhythmias, cardiac arrest, lung, liver and kidney failure, as well as secondary infections and high death rates. If the treatment is delayed, the electrical system of the heart can become unstable, resulting in an irregular heart rate and perhaps deadly cardiac arrest.[15] Recovery is made more difficult for the body's vulnerability to infections due to its compromised status.
7. **Post myocardial infarction syndrome:** Mild manifestations of post-myocardial infarction syndrome (PMI) are usually seen between 1 and 6 weeks after an episode of acute MI. The hallmark and chief complaint involve the recurrence of chest pain, which can come as fierce and acute, as that of a heart attack. The pericardial effects, in which there is the buildup of fluid in the pericardial bag, may cause heart compression and cardiac stamping. A situation where the heart is squeezed by such fluids, which already impede normal blood flow, is cardiac tamponade, and this may be fatal and worth immediate medical attention. Pericardial thickening caused by chronic inflammation can thereby impair the function of the heart and may ultimately progress to heart failure.[16] The other effects include inflammation having some repercussion on the lungs as pneumonia or between pleural drivers, and also abnormal heartbeat.

DIAGNOSIS

The important diagnostic criteria are:

ECG changes: Changes in the ECG are very important for the diagnosis of acute myocardial infarction (MI) since the most distinctive feature is the elevation of the ST segment. The initial ECG is typically diagnostic for STEMI, displaying ST-segment elevation to at least 1 mm in two or more contiguous leads that cover the affected region.[17] For diagnosis, the presence of Pathologic Q waves is not required. The specificity for diagnosing myocardial infarction with ST-segment elevation on ECG is 90%, while sensitivity is 45%. From a few days, serial tracings demonstrate a gradual evolution towards a stable pattern of abnormal Q waves. If there is suspicion for right ventricular infarction, a 15-lead ECG is typically recorded, adding leads at V4-6R and V8 and V9. The diagnosis of MI becomes more complicated with the presence of a left bundle branch block configuration because it mimics the changes of STEMI.[18]

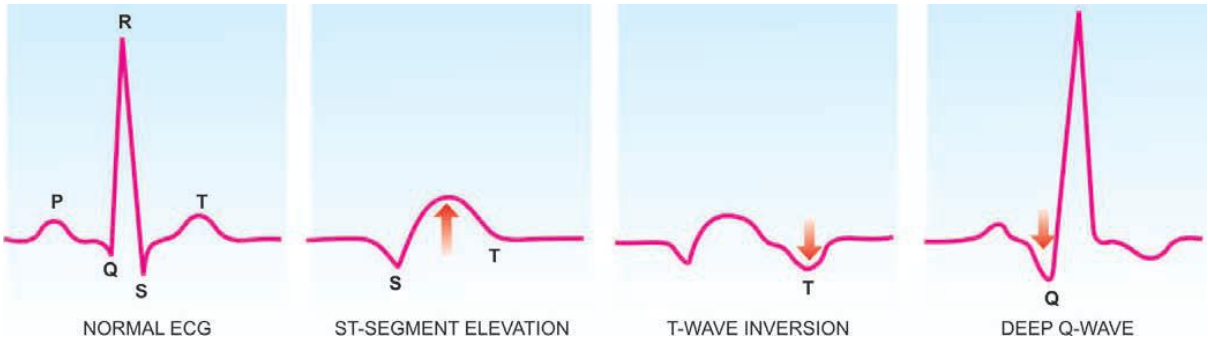

Figure 3.

Source: https://www.shutterstock.com/image-vector/diagnosis-myocardial-ischemia-nstemi-stemi-260nw-1158130930.jpg

Cardiac Biomarkers: After myocardial cell necrosis occurs, cardiac biomarkers, which include cardiac enzymes and cell contents, enter the bloodstream. There is wide variation in the sensitivity and specificity of these biomarkers. High-sensitivity assays of cardiac troponin (hs-cTn) are preferred because their ability to quantify cTn is as low as 0.003 to 0.006 ng/mL (3-6 pg/mL). Cardiac troponins detected with less sensitive cTn tests may not be found in patients unless the patients already have an acute cardiac disorder. With hs-cTn tests, however, small amounts of troponin are detected in many healthy people; therefore, hs-cTn levels must reference the normal range and are designated "elevated" only when exceeding 99% of the reference population.[19] Cardiac troponin levels can be increased by acute coronary syndromes or other cardiac and non-cardiac disorders, but not all elevated levels detected with hs-cTn represent myocardial infarction or myocardial necrosis has resulted from an acute coronary syndrome event. By depicting minute troponin changes, these hs-cTn assays allow for earlier detection of myocardial infarction and replace other cardiac biomarker tests in various hospitals. In conclusion, cardiac biomarkers, together with cardiac enzymes and enzymes, are a crucial component of diagnosis and management in myocardial injury.[20]

Echocardiogram: An echocardiogram is a non-invasive test using ultrasound to create images of the heart, revealing the inside and outside of the heart. It helps to identify areas of heart muscle or valves damaged by heart attack. It uses high-frequency sound waves to create images of a moving heart, thus enabling the doctor to assess the squeezing action and function of the valves of the heart. An echocardiogram comes in very handy to detect any damage sustained by the heart valves [21]

Coronary angiography: Coronary angiography is a less invasive procedure whereby a catheter is inserted into the coronary artery through the leg, arm, or neck, allowing it to take pictures of this conduit. This test involves measuring the diameter of the artery and blood flow rate. With a contrast dye, the opening of the artery becomes much more visible and easier to assess. While this dye is being used in order to show better pictures, in case of finding a blockage during angiography, the procedure is called an angioplasty or stent.[22] Usually, the catheter is inserted into the artery in the leg, and dye is flushed through it to make the pictures clearer.

Blood tests: Damage done to the heart because of a heart attack allows certain heart proteins to leak into the bloodstream. These proteins can be detected by blood tests for cardiac markers. One such cardiac marker is the cardiac troponin, which leaks into the bloodstream because of damage to heart muscle cells and forms a highly reliable diagnostic tool.[23]

TREATMENT

Nitroglycerin: Nitro-glycerine is a drug used to treat sudden onset of chest pain (angina) by relaxing and widening blood vessels, which increases blood flow to the heart. It functions through vasodilation, which relieves and enlarges blood vessels within arteries and veins. This dilation increases blood flow to the heart muscle, which is essential during an MI since blood supply is impaired. And during an MI when the heart is working so hard, it helps lower the heart's workload by decreasing preload and afterload. A potent vasodilator, nitro-glycerine is administered to relieve angina, to enhance blood flow to the ischemic heart muscle, and to reduce blood pressure, often contributing to MI.[24] It can be delivered in multiple forms, such as sublingual tablets or spray, intravenous infusion, and transdermal patches or ointment. However, this should not be combined with certain drugs, especially phosphodiesterase-5 inhibitors, as it can lead to a severe drop in blood pressure.[25]

Aspirin: Aspirin is an antiplatelet medication that decreases blood coagulation in MI. It allows blood to flow through narrowed arteries and prevents additional clotting. It is primarily given in the immediate setting of a suspected MI, typically as a chewable dose to increase drug absorption. Aspirin is also used for long-term prevention of cardiocerebrovascular events after MI. For an acute myocardial infarction, the first prescription is usually at a higher dose; however, for long term therapy the dose is lower.[26] One of the principal dangers salient to the use of aspirin is the possibility of internal bleeding, particularly in the stomach, so there has to be a good reasoning in caretaking the consequences and advantages of therapy with aspirin, especially for those patients with a previous history of disorders of bleeding.[27]

Statins: The examples for statins which are used in the treatment for myocardial infarction are Atorvastatin, Rosuvastatin and Pravastatin. Statins are drugs that lower non-healthy cholesterol that can obstruct arteries and lead to heart attacks. Statins lower the body’s cholesterol levels by blocking a liver enzyme that produces cholesterol, which lowers low-density lipoprotein (LDL) cholesterol. This helps existing plaques in coronary arteries from further increasing along with preventing new ones from forming. Statins also mitigate the risk of plaque rupturing, the major cause of heart attacks by stabilizing atherosclerotic plaques. Additionally, statins have other advantages such as lowering inflammation in the vessel walls. Statins help mitigate further cardiovascular events after an MI, which makes them one of the most important cornerstone medications for secondary prevention. They are brought into use soon after an MI and taken chronically. After an MI, having undergone statin therapy improves overall health regardless of the reason, which is why the dosage is generally customized depending on the individual’s risk factors along with the LDL

cholesterol. Although there is a possibility of muscle pain and raised liver enzyme levels after an MI, the benefits of statins after an MI outshines the drawbacks.

Angiotensin converting enzyme (ACE) inhibitors: The examples for ACE inhibitors which are used in the treatment for myocardial infarction are Ramipril, Captopril, Trandopril and Enalapril. Drugs used for relaxing blood pressure and easing the heart's strain by disabling the production of angiotensin II, a vessel constricting hormone is known as ACE inhibitors. This eases blood vessels, that helps in decreasing blood pressure and relaxing the heart's workload. After an MI, heart failure is common due to cardiac remodelling, and these inhibitors help slow or even completely prevent that [28]. They also relax the blood vessels thereby reducing the afterload that the heart has to pump against. After an MI, these inhibitors are great for patients with left ventricular dysfunction of heart failure, high blood pressure, or diabetes. They aid a lot in improving the chances of surviving and decreasing probability of suffering from cardiovascular problems in the future. Early usage is common, as long as the patient is hemodynamically stable. The use of ACE comes with other risks like a dry cough, dizziness or low blood pressure. Patients with kidney problems have to be careful while using them during pregnancy even much more.[29]

Beta-blockers: The examples for beta blockers which are used in the treatment for myocardial infarction are Metoprolol, Carvedilol and Bisoprolol. Beta blockers are used to decrease heart contractility and lower blood pressure and these are decreases cardiac work load upon heart, which lessens damage to the heart muscle and averts future heart attacks. They are administered to nearly all patients who sustain a heart attack and used in the short and long-term. Beta blockers decrease heart rate and myocardial oxygen consumption, along with blood pressure, arrhythmias, and myocardial injury. In MI patients, beta-blocker therapy is often started early in the hospital admission unless contraindicated, beta blockers are used for secondary cardiac event prevention in patients with or without ventricular dysfunction following an MI. Several studies confirm the notion that beta blockers, especially in post-MI patients, are associated with reduced mortality and improved survival.[30] In spite of these facts, they can potentially cause some unwanted adverse effects like tiredness, fainting, and slow heart rate, and can intensify asthma for some patients. Certain patients may not respond to beta blockers because of other coexisting medical issues. Their selection and the amount are based on each individual patient's needs and medical history.[31]

Fibrinolytics and Thrombolytics: Thrombolytic drugs are used to dissolve blood clots that block blood flow to the heart in order to restore perfusion to the heart muscle. They stimulate the body to dissolve existing clots by converting plasminogen to plasmin. The fibrin formed during clotting is then acted on by plasmin and broken down. For this reason, thrombolytic therapy is used to achieve reperfusion in patients with ST-elevation myocardial infarction (STEMI), a kind of heart attack.[32] It is most efficiently conducted within a few hours after the onset of symptoms and is often used when a PCI capable facility is not readily available. Time is of the essence when treating MI; the sooner blood flow is restored, the less the heart muscles are damaged. Important considerations include bleeding as a risk and active bleeding, history of haemorrhagic stroke or recent surgery as limitations. The preferred procedure for the recanalization of the blocked artery may be PCI in case thrombolytics cannot be administered during a timely manner.[33]

Morphine: In treating myocardial infarction, it is typical for a physician to use morphine as a simple painkiller in order to reduce chest pain and anxiety. Morphine has the potential for causing vasodilation, meaning a reduced workload on the heart itself by reducing preload and sympathetic activity. However, there has been a growing concern in research

related to potential negative effects of morphine use in cases of myocardial infarction, including delayed absorption of antiplatelet medications, the tendency for more adverse outcomes to occur, the risk of respiratory depression, and increased risk of hypertension and bradycardia.[34] Morphine is usually kept for very severe cases of pain where other forms of treatment cannot adequately deal with it, and is usually used in a controlled setting in the hospital. Introduced first in cases of myocardial infarction, nitro-glycerine is much preferred for chest pain among specialists, while morphine is reserved for the extreme form of pain which does not yield to any treatment. It is important to include morphine in a controlled hospital setting because of safety, as well as effectiveness.[35]

SURGICAL PROCEDURES

Coronary angioplasty with stenting: Angioplasty or coronary angioplasty, a type of coronary heart disease treatment, is a procedure utilized for unblocking the blocked coronary arteries. Unlike open heart surgery, modern invasive surgeries, such as angioplasty, restores blood flow to the muscle of the heart by inserting a catheter / tube through the skin. During the process, the physician guides a thought flexible tube which is connected to a heart catheter to the point where the artery of the heart narrows and puts a balloon inside it that expands and restores the flow of blood. In order to reduce the chances of the artery getting narrow again, a small wire mesh tube (stent) is placed in the artery. In some *drug eluting* stents, medication is added to lower the formation of scar tissue in the stents.[36] Potential complications of earning stent placement, angioplasty, catheter thrombectomy, and other interventional procedures may include: haemorrhage at the catheter access site, thrombosis, local or systemic infection, arrhythmias, myocardial infarction, cerebrovascular accident, angina pectoris, coronary artery dissection, contrast media allergy, and lethargy from contrast media. It is helpful to talk about your issues with the medical team prior to the procedure.[37]

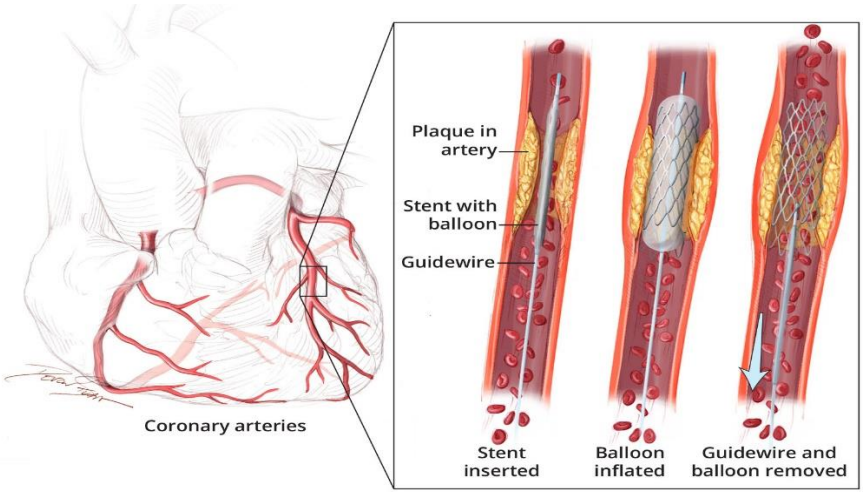

Figure 4:

Source: https://www.nhlbi.nih.gov/sites/default/files/inline-images/NHLBI_Fig02b_final03.jpg

Coronary artery bypass surgery: Coronary artery bypass surgery (CABG) involves relocating a blood vessel from the chest or leg region to connect with a blocked artery in the heart.[38] This aids in bypassing the blocked artery so that blood can flow unobstructed. Although it doesn't resolve heart disease entirely, it can help to alleviate chest pain and decreased breathing while also potentially decreasing heart disease associated mortality. Possible CABG complications include bleeding, heart infarction, and long-term use of ventilators, even leading to irregular heartbeats, renal failure, amnesia, and strokes too.[39] Performing CABG as a last resort increases the chance of suffering from these

complications during and after the surgery. Patients are often given medication prior to the surgery in order to manage potential bleeding and infections as well as control blood pressure. One area that modern CABG surgery lacks in is finding more effective and protective strategies associated with the heart that aid in decreasing morbidity and mortality.[40]

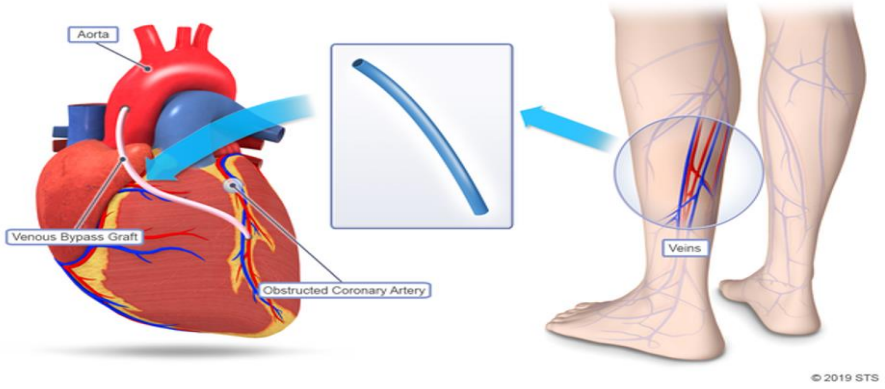

Figure 5.

Source: https://newportcts.com/wp-content/uploads/2020/02/during-cabg-graft.png

CONCLUSION

Cardial infarction (MI) is a heart attack caused by plaque buildup in arteries, resulting in decreased blood flow and damage to the heart muscles. Common symptoms include chest pain, dyspenea, nausea, vomiting, irregular heartbeat patterns, anxiety, fatigue, weakness, and mental pressure. Emergency medical responders can offer oxygen, nitro-glycerine tablets, or treat muscle tissue injury and blood clot with aspirin. Coping with MI involves tracking body weight, diet, fat, cholesterol, salt intake, blood pressure, exercise, and refraining from smoking, excessive drinking, or drug use. MIs can cause complications such as congestive heart failure, arrhythmias, rupture, pericarditis, mural thrombolysis and thromboembolism, cardiogenic shock, and post-myocardial infarction syndrome (PMI). Diagnosis of MI involves ECG changes, cardiac biomarkers, and cardiac enzymes. High-sensitivity assays of cardiac troponin (hs-cTn) are preferred for early detection. Myocardial infarction treatment involves various medications, including nitro-glycerine, aspirin, statins, ACE inhibitors, beta-blockers, fibrinolytics, and morphine. Nitro-glycerine is used to treat angina, aspirin decreases blood coagulation, statins lower non-healthy cholesterol, ACE inhibitors relax blood vessels, beta blockers decrease heart rate, myocardial oxygen consumption, blood pressure, arrhythmias, and myocardial injury, fibrinolytics and thrombolytics dissolve blood clots, and morphine is used for severe cases of pain. Thrombolytic drugs dissolve blood clots that block blood flow to the heart, and morphine is used for pain relief.

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