European Institute of Knowledge & Innovation

Annals of Innovation in Medicine (AIM) ISSN: 2977-0335

Narrative review

A Comprehensive Review of Acute Coronary Syndrome

Dr. Fatima Khurshid [1,*], Dr. Zaib Farooq[2] Dr. Ume Anum Ayesha[3], Dr. Shahzeb Chaudhry[4], Dr. Khadija Malik[5]

[1] Medical Doctor, Shifa International Hospital Ltd. Islamabad; [2] Senior Registrar; Mohtramma Benazir Bhutto Shaheed Medical College, Mirpur AJK; [3] Medical Doctor, DHQ Hospital Mirpur AJK; [4] Medical Doctor, Sargodha Medical College; [5] Medical Doctor, Abrar Surgery and Diagnostic Centre, Islamabad;

* Correspondence: fatimakhurshid61@yahoo.com

https://doi.org/10.59652/aim.v1i2.54

Abstract: A significant sign of coronary artery disease is the acute coronary syndrome (ACS). Angina pectoris, ST-elevation myocardial infarction, and non-ST-elevation myocardial infarction (NSTEMI) are just a few of the conditions that are grouped together under the general term "ACS." The main cause of death worldwide is cardiovascular disease, which puts ACS under a heavy financial strain. Reduced blood flow to the heart, primarily as a result of plaque rupture and thrombus development, is a pathogenesis of ACS. Numerous risk factors, both modifiable (such as smoking, hypertension, diabetes, hypercholesterolemia, obesity, and inactivity) and non-modifiable (like age, gender, and inheritance), can lead to the development of ACS. Electrocardiography (ECG), clinical evaluation, history-taking, and cardiac biomarkers are all used in the diagnosis process. For the best management, prompt diagnosis and risk classification are essential. Reperfusion therapy, anti-anginal therapy, and renin-angiotensin blocking are a few of the suggested procedures in treatment techniques that aim to minimise myocardial ischemia and restore coronary blood flow. Long-term management also strongly depends on modifying one's lifestyle, including giving up smoking, eating a healthy diet, getting regular exercise, and obtaining rehabilitation. Acute cardiac failure, ventricular septum or papillary muscle rupture, arrhythmias, recurrent angina, and other consequences of ACS are also highlighted in the paper. The prognosis varies depending on variables, including persistent myocardial injury and the existence of ventricular arrhythmias; an unfavourable prognosis is frequently related to arrhythmias and is associated with poor left ventricular function. . This article offers a thorough review of ACS and gives readers important information about its pathophysiology, risk factors, diagnosis, therapy, and prognosis.

Keywords: Acute Coronary Syndrome, Myocardial Infarction, Angina, Cardiovascular Disease.

Received: 17 Sep 2023

Accepted: 21 Sep 2023

Published: 22 Sep 2023

INTRODUCTION:

Acute coronary syndrome (ACS), a particularly risky form of coronary artery disease, is common, which has a large cost and health impact. ACS is primarily responsible for CAD-related deaths, which are currently the world's leading cause of mortality.[1] Acute coronary syndromes (ACS) can appear in a variety of clinical ways and are a major cause of mortality. Electrocardiographic signs, particularly ST-segment elevation, are used to make the diagnosis. In emergency care, adherence to scientific recommendations is essential for ensuring optimal treatment based on the likelihood of ACS. In order to select the best treatments and time for revascularization, it is critical to assess ischemia and hemorrhagic risks. Key elements of emergency treatment, such as ACS diagnosis and risk stratification, are summarised in this article.[2] With regard to morbidity and death, acute coronary syndrome (ACS) imposes a heavy burden. In this review, the pathophysiology, diagnosis, treatment, and management of complications of ACS are discussed. It seeks to enhance information in order to improve ACS patients' results.[3] STEMI, NSTEMI, and unstable angina are all included in ACS. Because it encapsulates the similarities in presentation and management across these conditions, the phrase is useful.[4] The three classic forms of acute coronary syndrome (ACS) are unstable angina, NSTEMI, and STEMI. Due to high-sensitivity troponin tests, which can identify myocardial cell death brought on by ischemia even in the absence of a STEMI pattern on the ECG, the diagnosis now favours NSTEMI. With this modification, myocardial ischemia patients are

accurately identified, allowing for proper management and treatment techniques.[5] The 2018 joint task force of the European Society of Cardiology (ESC), American College of Cardiology Foundation (ACCF), American Heart Association (AHA), and World Health Federation (WHF) defined acute myocardial injury, whether STEMI or NSTEMI, as the existence of abnormal cardiac biomarkers in the presence of evidence of acute myocardial ischemia.[6] Acute coronary syndrome, which includes unstable angina and myocardial infarction, is a group of illnesses brought on by an abrupt stoppage of the blood supply to the heart. It causes serious issues for young adults and is quickly rising to the top of the global list of killers, with high rates of morbidity and mortality being reported in Western European hospitals. The prevalence of cardiovascular disease is rising in South Asian nations as well, and this burden is expected to grow significantly in the coming years.[20]

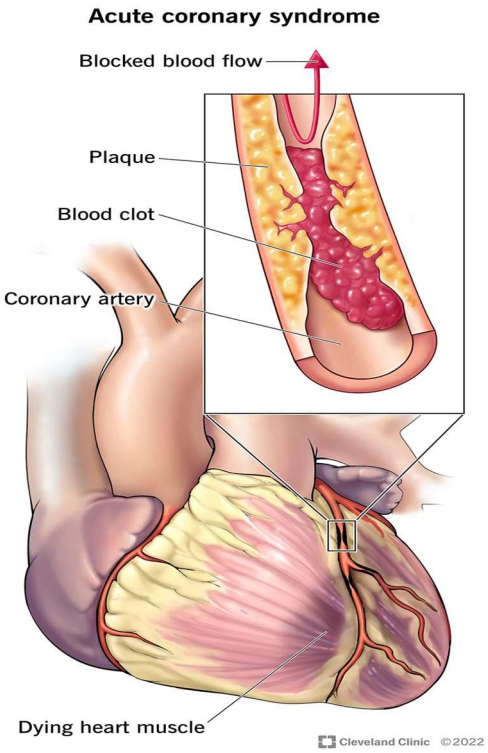

Figure 1: An obstruction causes blood flow to be sluggish or cease in acute coronary syndrome.[17]

PATHOPHYSIOLOGY:

Reduced blood flow to a section of the heart muscle is a pathogenesis of acute coronary syndrome (ACS). Vasospasm, plaque rupture, thrombus development, and in some situations, vasospasm can all contribute to this. As a result, infarction and ischemia occur. The location, size, and length of the obstruction are among the variables that affect the severity and outcomes of ACS. There may be some degree of cell necrosis even in milder episodes of ACS, which can vary from transitory ischemia to infarction. Numerous factors, including coronary vasospasm, calcified nodules, plaque rupture, plaque erosion, and myocardial bridging, can cause ACS. For an accurate ACS diagnosis and successful therapy, it is crucial to comprehend the intricate pathophysiology. [7, 8, 13, 15]

Aetherosclerosis and Plaque Rupture:

Nearly all acute myocardial infarctions have thrombosis on a responsible coronary atherosclerotic plaque as their underlying cause. An increased risk of future thrombosis is indicated by vulnerable plaques, which include a big lipid core, a high macrophage density, a low smooth muscle cell density in the plaque cap, a high tissue factor content, and a thin,

disorganised collagen structure in the cap. [11]The likelihood of upcoming acute episodes in people with coronary artery disease depends on the quantity of susceptible plaques. Individual disparities in recurrent occurrences are explained by differences in the quantity of these plaques.[12]

Thrombus formation and coronary artery occlusion:

A plaque rupture causes thrombus to develop, limiting blood flow and resulting in ischemic consequences. Platelets, red blood cells, vasoconstrictors, and fibrin fibres make up the thrombus; the fibrin fibres' properties determine the thrombus' stability and susceptibility to dissolution.[14]Plaque cap disruption or endothelial denudation may lead to thrombosis on a plaque's surface. Major thrombi are more frequently associated with disruption of the plaque than with superficial endothelial denudation.[12] Acute coronary syndromes can result from coronary artery thrombus, which is brought on by plaque rupture or erosion. The patient's prognosis and the results of percutaneous coronary intervention are affected by the existence and length of the thrombus.[16]

Myocardial Dysfunction:

Ischemic tissue has poor relaxation and contractility, which results in hypokinetic or akinetic segments. During systole, these segments could exhibit paradoxical motion. Depending on how much of the affected area is damaged, the implications might range from minor effects to heart failure or cardiogenic shock. Ischemic cardiomyopathy can be brought on by persistent heart failure and poor cardiac output. Mitral valve regurgitation may result from ischemia in the papillary muscle, and mural thrombus development may be facilitated by abnormal wall motion.8

Myocardial Infarction (MI):

Reduced coronary blood flow results in myocardial necrosis and myocardial infarction (MI). Although the right ventricle and atria may also be affected, the left ventricle may be the primary site. Transmural and nontransmural MI are distinguished by whether they affect the entire myocardial thickness and produce aberrant Q waves on the ECG. ST-segment and T-wave abnormalities result from nontransmural infarctions. The distinction between ST-segment elevation MI (STEMI) and non-ST-segment elevation MI (NSTEMI) is based on the presence or absence of Q waves or ST-segment elevation. Large-scale ventricular wall necrosis can result in rupture, ventricular aneurysm, or the development of a pseudoaneurysm.[8]

Electrical dysfunction

Electrical dysfunction is a key factor in acute coronary syndrome. The inability of ischemic and necrotic cells to produce regular electrical activity results in a variety of ECG abnormalities, arrhythmias, and conduction issues. The ST-T abnormalities associated with ischemia include peaked T waves, T-wave inversion, ST-segment elevation, and ST-segment depression. Disturbances in conduction may be a sign of injury to the AV node, sinus node, or specific conduction tissues. While some alterations are transient, others might be long-lasting.[8]

RISK FACTORS:

Diabetes, diet, hypercholesterolemia, hypertension, lack of exercise, obesity, smoking, alcohol use, and stress are all modifiable risk factors for cardiovascular disease. Age, ethnicity, gender, and genetics are all non-modifiable risk factors. People can lower their risk of cardiovascular disease by adopting healthy lifestyle changes like stopping smoking, improving their nutrition, and engaging in more physical activity. [18, 19]

MODIFIABLE RISK FACTORS:

Smoking:

Smoking is a significant contributor to atherosclerosis and myocardial infarction risk. Particularly among women who smoke while using birth control pills, it raises the risk of cardiac

mortality and is a major cause of death in the US. Nicotine has an impact on the cardiovascular system by raising blood pressure, heart rate, and the risk of arrhythmias, as well as vasoconstriction. Additionally, it promotes the growth of smooth muscle cells and platelets in coronary arteries. Peripheral artery disease, which increases the risk of heart attacks and strokes, is associated with smoking. The risk of dying from coronary heart disease is also increased by exposure to secondhand smoke. Health is improved by quitting smoking, and help and medication can increase success. The risk of coronary heart disease is 50% lower after a year of quitting.[21, 22, 23, 24, 25]

Alcohol:

Regularly consuming a small amount of alcohol is frequently viewed as advantageous. However, excessive alcohol use can raise the risk of atherosclerosis progression and be a role in conditions including high triglycerides, high blood pressure, weight gain, and irregular heartbeats. Due to the short epidemiological follow-up in this area, the association between alcohol use and cardiovascular illnesses, particularly their atherogenic and antiatherogenic qualities, is still up for debate.. [26, 27, 28]

Hypertension:

Systolic blood pressure, which is particularly harmful to the coronary arteries and raises the risk of myocardial infarction, is an important predictor of cardiovascular disease risk. Over time, it also puts more strain on the heart, which causes the left ventricle to expand and weaken. The chance of serious cardiovascular events grows as blood pressure raises. Comorbidities raise the risk of heart disease even more. Maintaining a healthy weight, controlling stress, abstaining from smoking, and abstaining from excessive alcohol intake are all necessary to reduce this risk. To control high blood pressure and lower the risk of coronary heart disease, doctors may prescribe medications.29, 30, 31

Diabetes Mellitus:

Diabetes ups the risk of CHD. The most common cause of death for those with diabetes, CVD is a serious consequence of the disease. 65% of diabetic deaths are caused by heart disease and stroke. Diabetes patients have a 2-4 times greater incidence of CHD. [32]Obesity, cholesterol issues, and higher blood pressure rates are all linked to type 2 diabetes and raise the risk of CVD. People with diabetes who smoke have a twofold increased risk of CVD. When cholesterol levels are the same, those with diabetes are more prone to heart disease than people without diabetes.[33]

Blood Cholesterol:

The risk of coronary heart disease (CHD) grows as blood cholesterol levels rise, especially when other risk factors are present. Myocardial ischemia is caused by cholesterol buildup in the coronary arteries, which results in arterial damage and occlusion. Elevated LDL levels contribute to atherosclerosis, whereas elevated HDL levels assist in reducing inflammation and thwarting LDL oxidation. Heart disease is thought to be predicted by low HDL levels. To control and lower the risk of CHD, managing cholesterol levels by diet and medicine is crucial. [34, 35, 36]

Obesity:

In addition to raising the risk of cardiometabolic disorders due to the visceral and subcutaneous fat buildup, obesity is directly linked to myocardial infarction (MI). It has been associated with diabetes, elevated blood lipid levels, and hypertension. Reduced risk of MI can be attained through weight loss brought on by dietary adjustments, improved physical activity, stress reduction, and moderate alcohol consumption.[37,38,39]

Physical Inactivity:

High cardiovascular risk is linked to physical inactivity.[40] Although exercise has a cardioprotective impact, it is not well understood how it helps people with ACS. Resistance training can improve myocardial function, but due to exercise-induced myocardial ischemia, it may hinder the heart's ability to prepare for an ischemic event.[41]The American Heart Association suggests engaging in moderate physical activity for at least 2 ½ hours a week, or 30 to 60 minutes, on most days. The best options include cycling, dancing, swimming, walking, and jogging.[42]

NON MODIFIABLE RISK FACTORS:

Age:

For cardiovascular disease (CVD), age is a well-known, unchangeable risk factor. Progressively, more CVD risk factors are added to an individual as they age.[43] People over 65 account for over 80% of heart disease deaths in humans.[44]

Gender:

Men are more likely to have CHD, although women are more at risk as they age. Cardiovascular risk factors can play different roles depending on a person's gender. Men get heart attacks earlier in life, while women's risk rises after menopause but is still lower than that of men. However, heart disease is the number one killer of both sexes. Men and women both have fair or bad health rates as they age, but women could have more difficulties participating in social and physical activities. Heart disease is a major health problem for both men and women despite the fact that gender influences CVD risk factors.45,46,47

Heredity/Family history:

According to epidemiological research, a major risk factor for coronary heart disease (CHD) is a familial or parental history of myocardial infarction.48 A first-degree blood relative who experienced a coronary heart disease or stroke before the ages of 55 for men and 65 for women is linked to an increased chance of developing heart disease.49

Ethnicity:

People with ancestry from South Asia, Africa, or the Caribbean are more likely to develop cardiovascular disease, possibly as a result of greater prevalence of type 2 diabetes. To prevent heart and circulation disorders, people of all backgrounds are advised to lead healthy lifestyles.50

CLINICAL PRESENTATION OF ACUTE CORONARY SYNDROME:

Three types of ACS exist: unstable angina (UA), non-ST-Elevation myocardial infarction (NSTEMI), and ST-Elevation myocardial infarction (STEMI). Risk categorization and prompt diagnosis are crucial. Restoring blood flow and lessening myocardial ischemia are the main goals of treatment. A increased risk of cardiac mortality and myocardial damage is associated with ACS.[9] Significant ST segment increases on the ECG and increased troponin levels are indicators of STEMI. According to troponin levels, NSTE-ACS refers to situations lacking ST elevations on the ECG and can also be classified as NSTEMI or UA. On the ECG, ST segment depressions and T-wave inversions are frequently seen in NSTE-ACS. [53]

This classification of acute coronary syndromes is illustrated in Figure 3.

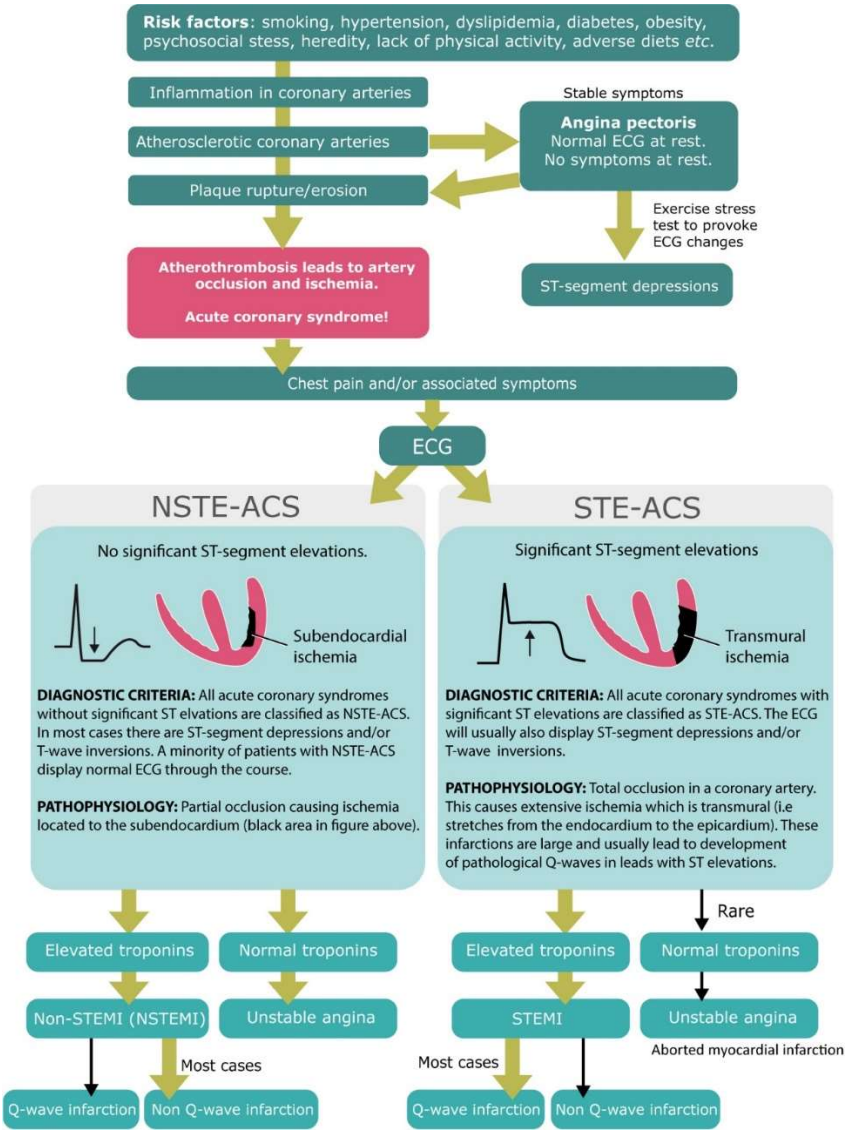

Figure 3: Classification of Acute Coronary Syndromes in STE-ACS (STEMI- ST Elevation Myocardial Infarction) and NSTE-ACS (Non-STEMI and Unstable Angina).[53]

DIAGNOSTIC APPROACHES FOR ACUTE CORONARY SYNDROME:

Clinical Assessment and History Taking

Chest discomfort, breathlessness, sweating, motion sickness, exhaustion, dizziness, and palpitations are classic ACS symptoms. However, in other patient populations, symptoms may be abnormal or even nonexistent. For the best results, early diagnosis and proper management are essential.[51]

The pain associated with acute myocardial infarction (MI) is strong, persistent, and frequently described as a tightness or weight in the chest. There may also be nausea and shortness of breath. It is possible for MI to be silent or painless, especially in the elderly or in people with diabetes. It's critical to get medical attention right once when symptoms appear.[52]

To distinguish between STEMI and NSTEMI/unstable angina, an ECG is performed within 10 minutes after the initial assessment of suspected ACS. The diagnosis is aided by cardiac enzymes, chest X-rays, and blood tests. Considerations should be made for additional problems, such as aortic dissection and pulmonary embolism.[15]

Electrocardiography (ECG)

A bundle branch block or a previous MI block may make it challenging to interpret the normal 12-lead ECG, which is crucial for diagnosing ACS. In roughly one-third of instances, the initial ECG results may be normal or equivocal. Repeated ECGs are essential, especially when there is ambiguity or symptoms that continue.[52]

Cardiac biomarkers

Serial troponin readings are crucial in making an ACS diagnosis. Troponin levels are not significantly elevated in unstable angina, although they are elevated in MI. Troponin levels start to climb in 3 to 6 hours, reach their peak in 36 hours, and then continue to rise for up to 2 weeks. There may be an increase in other cardiac enzymes. Leukocytosis may be seen on a full blood count, and high levels of ESR and CRP are also present. Following presentation, lipid measurements must be done within 24 hours.[52]

Radiography

To determine the size of the heart and detect pulmonary edoema, a chest X-ray is performed. Cardiomegaly may exist as a result of previous cardiac injury.52

Echocardiography

Prior to hospital discharge, echocardiography is conducted to evaluate ventricular function and identify any problems, such as mural thrombus, cardiac rupture, ventricular septal defect, mitral regurgitation, and pericardial effusion.52

Coronary angiography

In high-risk patients who do not react to pharmacological therapy, have significant ECG abnormalities, elevated troponin levels, or have severe stable angina, coronary arteriography is taken into consideration for revascularization. It assists in identifying candidates who would benefit from immediate coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI). [52]

MANAGEMENT STRATEGIES OF ACUTE CORONARY SYNDROME:

Due to the significant risk of death or repeated myocardial ischemia, all suspected ACS patients should be admitted to hospitals immediately. Early medical intervention can at least 60% lessen problems. The in-hospital management should take place in a cardiac unit with the necessary training and resources. Clinical risk factor analysis, such the GRACE score, aids in the identification of patients needing early inpatient coronary angiography and intense therapy. Patients with low risk and no issues may be mobilised on day two and released in two to three days. Low-risk patients without spontaneous angina are advised to do exercise tolerance tests around 4 weeks after an ACS to assess whether further testing is necessary. The principles of long-term management are summarized in Figure 05.[52]

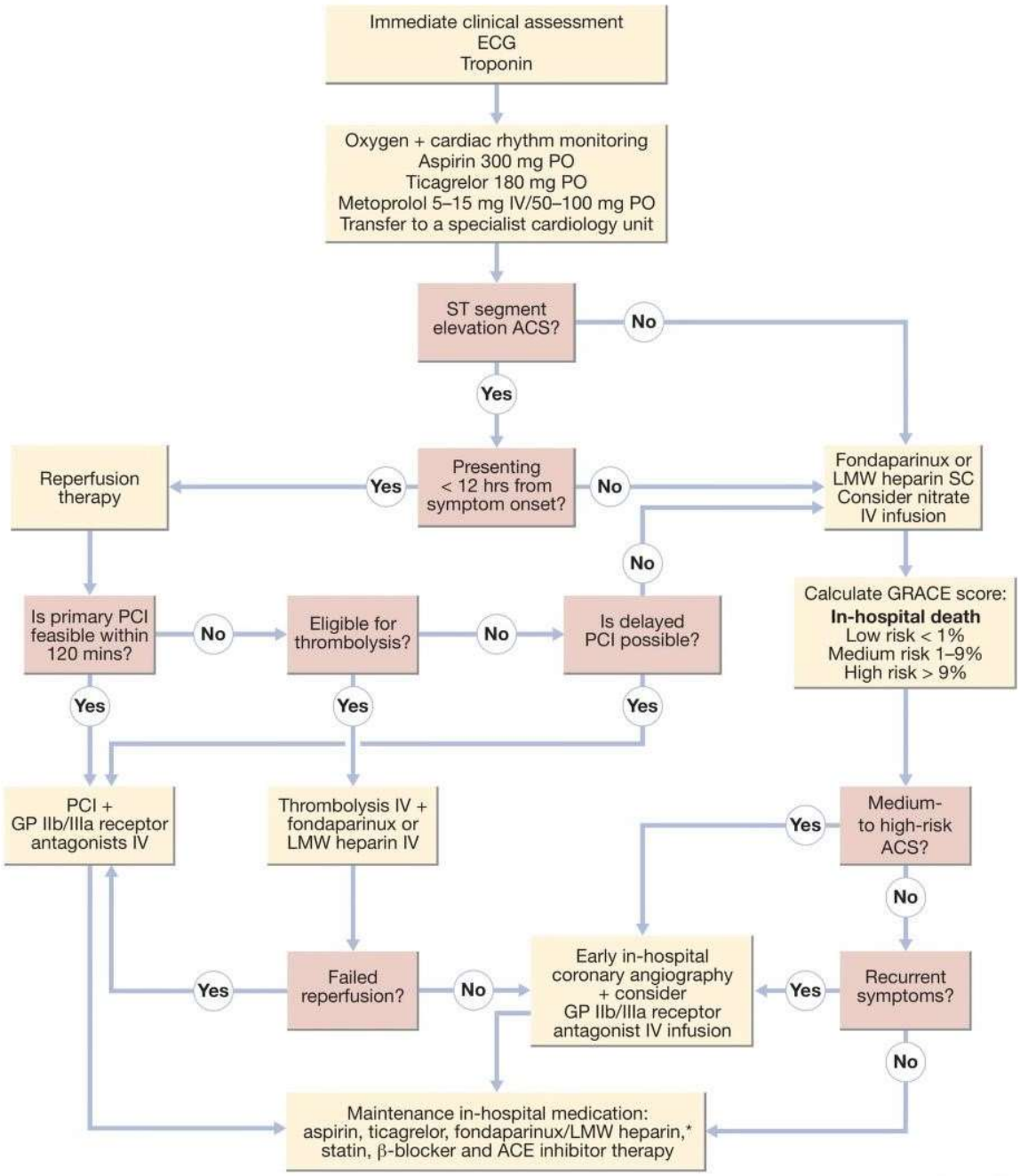

Figure 05. Summary of treatment for acute coronary syndrome (ACS). [52]

Analgesia

To minimise vascular resistance and adrenergic drive, enough analgesia is essential. Intravenous opiates should be administered first, such as morphine sulphate (5–10 mg) or diamorphine (2.5–5 mg). When necessary, take antiemetics such metoclopramide 10 mg.[52]

Reperfusion therapy:

For particular ECG abnormalities in ACS, immediate reperfusion therapy with PCI (percutaneous coronary intervention) is advised. The best time to do PCI is within 12 hours after the onset of symptoms. PCI should still be completed as soon as possible even if timely completion is not achievable within 120 minutes. PCI within the first 24 hours may be taken into consideration, even if spontaneous reperfusion or thrombolytic treatment has already place. Rapid pain alleviation, ST elevation resolution, and decreased mortality are all associated with PCI, which has a high success rate in restoring coronary artery patency. For patients at a medium- to high-risk, coronary angiography and revascularization may be explored.[52]

Thrombolytic therapy:
PCI can be replaced with intravenous thrombolytic treatment (TNK, rPA). It has a risk of cerebral haemorrhage but is most effective in the first 12 hours. If there is a significant danger of major bleeding, thrombolytic therapy should be avoided.[52]

Anti-anginal therapy
In addition to beta-blockers, anti-anginal therapy for ACS also uses sublingual and injectable nitrates. For unstable angina, glyceryl trinitrate is administered sublingually. Nitrates administered intravenously reduce ischemia pain and left ventricular failure. Intravenous beta-blockers decrease arrhythmias, lessen discomfort, and increase short-term mortality.[52]

Renin-angiotensin blockade:
Enalapril, ramipril, valsartan, and candesartan are examples of ACE inhibitors or ARBs that can block the renin-angiotensin system and benefit survival and the prevention of heart failure. These drugs lessen recurrent myocardial infarction, stop heart failure, and avoid ventricular remodelling. Patients with overt heart failure, asymptomatic left ventricular dysfunction, and retained left ventricular function benefit the most from them.[52]

Mineralocorticoid receptor antagonists
For particular patients with acute MI and left ventricular failure (ejection fraction 35%), mineralocorticoid receptor antagonists such as eplerenone or spironolactone, notably those with pulmonary edoema or diabetes mellitus, provide further benefits.[52]

Lipid-lowering therapy
Regardless of their cholesterol levels, patients should receive statin medication after acute coronary syndrome. Patients who need more intense therapy with atorvastatin (80 mg daily) had LDL cholesterol levels above 3.2 mmol/L (about 120 mg/dL). Other lipid-lowering medications such as ezetimibe, fibrates, anion exchange resins, and injectable PCSK9 inhibitors may be tried if statins alone are insufficient.[52]

Smoking cessation
The improvement of long-term results depends on quitting smoking. The 5-year death rate is much lower for patients who stop smoking at the time of developing acute coronary syndrome compared to those who don't. The success of smoking cessation can be increased by supportive advice and medication therapy. [52]

Diet and exercise
Patients with acute coronary syndrome may benefit in the long run by adopting a healthy diet and regularly exercising.

It is advised to keep your weight in check, eat like the Mediterranean Diet, and control your blood pressure and diabetes well.[52]

Rehabilitation
Recovery from ACS depends greatly on rehabilitation. For the first 4-6 weeks, limit physical activity to promote healing. On the second day, get the patient moving and progressively ramp up the activities. A four-week return to work target has been set. Long-term results are improved by emotional support, counselling, and formal rehabilitation programmes with graded exercise and counselling.[52]

Implantable defibrillators
In patients with significant left ventricular dysfunction (ejection fraction 30%) following myocardial infarction, implantable defibrillators (ICDs) are helpful in averting sudden cardiac death. ICDs shock the heart to treat potentially fatal arrhythmias. Patients who are at a high risk of sudden cardiac death should use them.[52]

COMPLICATIONS OF ACUTE CORONARY SYNDROME:

Arrhythmias: Arrhythmias are common in ACS, but they're frequently brief and have no real clinical impact. Arrhythmia risk can be reduced by providing appropriate pain management, getting enough rest, and treating hypokalemia. Vulnerable ventricular function and a higher

risk of sudden death, however, may be indicated by the occurrence of ventricular arrhythmias during the recovery period.[52]

Recurrent Angina: Patients who experience recurrent angina following an acute coronary syndrome should get immediate coronary angiography to see if they can get revascularized. Treatment options include emergency coronary revascularization, intra-aortic balloon counter-pulsation, and intravenous glycoprotein llb/llla receptor antagonists.[52]

Acute Heart Failure: It is characterised by severe myocardial injury as well as a dismal prognosis. When acute heart failure is present, managing additional consequences is crucial.[52]

Pericarditis: Develops after an infarction and is characterised by positional pain and audible pericardial rub. While NSAIDs and steroidal anti-inflammatory medications should be avoided, opiate-based analgesia is advised.[52]

Dressler's Syndrome: Usually develops weeks or months after a myocardial infarction and is characterised by a lingering fever, pericarditis, and pleurisy. High-dose aspirin, NSAIDs, or glucocorticoids may be used as a form of treatment.[52]

Papillary Muscle Rupture: Presents with severe mitral regurgitation, acute pulmonary edoema, and shock. Echocardiography confirms the diagnosis, and it could be required to replace the valve immediately.[52]

Ventricular Septum Rupture: Causes abrupt right heart failure by causing left-to-right shunting through a ventricular septal defect. There needs to be an urgent surgical correction.[52]

Ventricular Rupture: Typically, deadly condition that causes cardiac tamponade. Some situations might require emergency surgery.[52]

Embolism: The development of thrombus on the endocardial surface might result in systemic embolism, which can result in consequences like strokes or ischemic limbs. Early mobilisation and preventative anticoagulants help lower the danger.[52]

Ventricular Remodeling: Acute transmural myocardial infarction may have the side effect of thinned and stretched ventricular walls. Heart failure can be avoided with the aid of ACE inhibitors and mineralocorticoid receptor antagonists.[52]

Ventricular Aneurysm: Develops in around 10% of myocardial infarction patients, especially when the infarct-related artery is persistently blocked.[52]

PROGNOSIS:

Following acute coronary syndrome survival, the prognosis is influenced by variables such persistent ischemia, the degree of damage, and the existence of arrhythmias. Given that a major portion of fatalities happen within minutes or during the first 24 hours, immediate medical attention is essential. Compared to myocardial infarction, unstable angina normally has a lower fatality rate. Poor left ventricular performance, an AV block, and persistent arrhythmias are a few factors that have an impact on long-term outcomes. In comparison to inferior infarcts, the prognosis for anterior infarcts is typically worse. Death rates are higher when a person is older, depressed, and socially isolated. After surviving an attack, about 80% of patients live for at least one year, with survival statistics eroding over time. Mortality and recurrent cardiovascular events are rather common in young patients with ACS, and poor prognostic indicators include hypertension, LAD disease, and coronary intervention without stenting. Early risk factor-targeting and compliance-improving therapies, especially in hypertensive patients, may improve prognosis.[52, 54]

CONCLUSION:

In conclusion, acute coronary syndrome (ACS) is a serious symptom of coronary artery disease (CAD) and a major global health concern. This article has given a thorough review of ACS, including information on its categorization, risk factors, diagnosis, and management techniques. It emphasises the significance of lifestyle changes including quitting smoking,

adopting a nutritious diet, exercising frequently, and managing risk factors like high blood pressure and high cholesterol. For rapid risk stratification and effective care, an early and accurate diagnosis using a combination of clinical assessment, electrocardiography (ECG), and cardiac biomarkers is essential. Reperfusion therapy, anti-anginal drugs, and dietary changes are available as forms of treatment. To further enhance results and lessen the burden of ACS, further research and innovations are required. For early diagnosis and prevention of this potentially fatal disorder, increasing awareness of ACS among medical professionals and the general public is crucial. With perseverance, we can enhance care, lessen the effects of ACS, and save lives.

References:

1. Valentin Fuster, Jason C. Kovacic, 13,May 2014, https://doi.org/10.1161/CIRCRESAHA.114.302806, Circulation Research. 2014;114:1847–1851

2. Gach O, El HZ, Lancellotti P. [Acute coronary syndrome]. Revue Medicale de Liege. 2018 May;73(5-6):243-250. PMID: 29926562.

3. Makki N, Brennan TM, Girotra S. Acute Coronary Syndrome. Journal of Intensive Care Medicine. 2015;30(4):186-200. doi:10.1177/0885066613503294.

4. Achar SA, Kundu S, Norcross WA. Diagnosis of acute coronary syndrome. Am Fam Physician. 2005 Jul 1;72(1):119-26. PMID: 16035692.

5. Braunwald E, Morrow DA. Unstable angina: is it time for a requiem? Circulation. 2013 Jun 18;127(24):2452-7. doi: 10.1161/CIRCULATIONAHA.113.001258. PMID: 23775194.

6. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, White HD; Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol. 2018 Oct 30;72(18):2231-2264. doi: 10.1016/j.jacc.2018.08.1038. Epub 2018 Aug 25. PMID: 30153967.

7. Singh A, Museedi AS, Grossman SA. Acute Coronary Syndrome. [Updated 2023 Jul 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459157/

8. Ranya N. Sweis , Arif Jivan, Overview of Acute Coronary Syndromes (ACS), MSD Manual Professional version, Reviewed/Revised Jun 2022 | Modified Sep 2022. https://www.msdmanuals.com/professional/cardiovascular-disorders/coronary-artery-disease/overview-of-acute-coronary-syndromes-acs

9. Lal C Daga, Upendra Kaul, Aijaz Mansoor Approach to STEMI and NSTEMI. Journal of the Association of the Physicians in India. Volume: 59. December 2011. ISSN 0004-5772.

10. Deqiang Yuan, Jiapeng Chu, Jun Qian, Hao Lin, Guoqi Zhu, Fei Chen, Xuebo Liu, Rev. Cardiovasc. Med. 2023, 24(4), 112; https://doi.org/10.31083/j.rcm2404112.

11. Davies MJ. The pathophysiology of acute coronary syndromes Heart 2000;83:361-366.

12. Michael J. Davies. Stability and Instability: Two Faces of Coronary Atherosclerosis.15 Oct 1996 https://doi.org/10.1161/01.CIR.94.8.2013Circulation. 1996;94:2013–2020.

13. Deqiang Yuan, Jiapeng Chu, Jun Qian, Hao Lin, Guoqi Zhu, Fei Chen, Xuebo Liu. New Concepts on the Pathophysiology of Acute Coronary Syndrome. Rev. Cardiovasc. Med. 2023, 24(4), 112. https://doi.org/10.31083/j.rcm2404112

14. Adnan G, Singh DP, Mahajan K. Coronary Artery Thrombus. 2022 Oct 3. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan–. PMID: 30521229.

15. Singh A, Museedi AS, Grossman SA. Acute Coronary Syndrome. [Updated 2023 Jul 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459157/.

16. Adnan G, Singh DP, Mahajan K. Coronary Artery Thrombus. 2022 Oct 3. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan–. PMID: 30521229.

17. Acute Coronary Syndrome. Cleveland Clinic. August 22, 2023. 866.320.4573. https://my.clevelandclinic.org/health/diseases/22910-acute-coronary-syndrome.

18. Norton C. Acute coronary syndrome: assessment and management. Nurs Stand. 2017 Mar 15;31(29):61-71. doi: 10.7748/ns.2017.e10754. PMID: 28294020.

19. Ada's Medical Knowledge Team, Cardiovascular Disease Risk Factors. September 7, 2022, https://ada.com/cardiovascular-disease-risk-factors/.

20. Cheema FM, Cheema HM, Akram Z. Identification of risk factors of acute coronary syndrome in young patients between 18-40 years of age at a teaching hospital. Pak J Med Sci. 2020 May-Jun;36(4):821-824. doi: 10.12669/pjms.36.4.2302. PMID: 32494281; PMCID: PMC7260892.

21. Braunwaldet .al: Harrisons principles of internal medicine, 15 edition .McGraw Hill; 2001:2629.

22. Inoue T. Cigarette Smoking as a Risk Factor of Coronary Artery Disease and its Effects on Platelet Function. Tob Induc Dis. 2004 Mar 15;2(1):2. doi: 10.1186/1617-9625-2-2. PMCID: PMC2669461.

23: Allen RH, Cwiak CA, Kaunitz AM. Contraception in women over 40 years of age. CMAJ. 2013 Apr 16;185(7):565-73. doi: 10.1503/cmaj.121280. Epub 2013 Mar 4. PMID: 23460635; PMCID: PMC3626808.

24. George Papathanasiou1 , Anastasia Mamali2 , Spyridon Papafloratos3 , Efthimia Zerva. HEALTH SCIENCE JOURNAL. VOLUME 8 (2014),ISSUE 2. E-ISSN:1791-809x:274.

25. A. Verma A, A. Prasad, G.H. Elkadi, Y.-W.Chi. Peripheral Arterial Disease: Evaluation, Risk Factor Modification, and Medical Management. JCOM. 2011/18(2): 34 – 47.

26. Camargo CA Jr, Hennekens CH, Gaziano JM, Glynn RJ, Manson JE, Stampfer MJ. Prospective study of moderate alcohol consumption and mortality in US male physicians. Arch Intern Med. 1997 Jan 13;157(1):79-85. PMID: 8996044.

27. Doll R, Peto R, Hall E, Wheatley K, Gray R. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. BMJ. 1994 Oct 8;309(6959):911-8. doi: 10.1136/bmj.309.6959.911. PMID: 7950661; PMCID: PMC2541157.

28. Foppa M, Fuchs FD, Duncan BB. Alcohol and atherosclerosis. Arq Bras Cardiol. 2001 Feb;76(2):165-76. English, Portuguese. doi: 10.1590/s0066-782x2001000200009. PMID: 11270318.

29. Mourad JJ. The evolution of systolic blood pressure as a strong predictor of cardiovascular risk and the effectiveness of fixed-dose ARB/CCB combinations in lowering levels of this preferential target. Vasc Health Risk Manag. 2008;4(6):1315-25. doi: 10.2147/vhrm.s4073. PMID: 19337545; PMCID: PMC2663439.

30. Dunn FG. Hypertension and myocardial infarction. J Am Coll Cardiol. 1983 Feb;1(2 Pt 1):528-32. doi: 10.1016/s0735-1097(83)80084-9. PMID: 6338085.

31. Oparil S, Zaman MA, Calhoun DA. Pathogenesis of hypertension. Ann Intern Med. 2003 Nov 4;139(9):761-76. doi: 10.7326/0003-4819-139-9-200311040-00011. PMID: 14597461.

32. Kannel WB, McGee DL. Diabetes and cardiovascular disease. The Framingham study. JAMA. 1979 May 11;241(19):2035-8. doi: 10.1001/jama.241.19.2035. PMID: 430798.

33. Moss SE, Klein R, Klein BE. Cause-specific mortality in a population-based study of diabetes. Am J Public Health. 1991 Sep;81(9):1158-62. doi: 10.2105/ajph.81.9.1158. PMID: 1951827; PMCID: PMC1405646.

34. Nelson RH. Hyperlipidemia as a risk factor for cardiovascular disease. Prim Care. 2013 Mar;40(1):195-211. doi: 10.1016/j.pop.2012.11.003. Epub 2012 Dec 4. PMID: 23402469; PMCID: PMC3572442.

35. Parthasarathy S, Raghavamenon A, Garelnabi MO, Santanam N. Oxidized low-density lipoprotein. Methods Mol Biol. 2010;610:403-17. doi: 10.1007/978-1-60327-029-8_24. PMID: 20013192; PMCID: PMC3315351.

36. Philip Barter. The role of HDL-cholesterol in preventing atherosclerotic disease. European Heart Journal Supplements (2005) 7 (Supplement F), F4–F8 doi:10.1093/eurheartj/sui036.

37. Lovren F, Teoh H, Verma S. Obesity and atherosclerosis: mechanistic insights. Can J Cardiol. 2015 Feb;31(2):177-83. doi: 10.1016/j.cjca.2014.11.031. Epub 2014 Dec 6. PMID: 25661552.

38. Manna P, Jain SK. Obesity, Oxidative Stress, Adipose Tissue Dysfunction, and the Associated Health Risks: Causes and Therapeutic Strategies. Metab Syndr Relat Disord. 2015 Dec;13(10):423-44. doi: 10.1089/met.2015.0095. PMID: 26569333; PMCID: PMC4808277.

39. Wing RR, Lang W, Wadden TA, Safford M, Knowler WC, Bertoni AG, Hill JO, Brancati FL, Peters A, Wagenknecht L; Look AHEAD Research Group. Benefits of modest weight loss in improving cardiovascular risk factors in overweight and obese individuals with type 2 diabetes. Diabetes Care. 2011 Jul;34(7):1481-6. doi: 10.2337/dc10-2415. Epub 2011 May 18. PMID: 21593294; PMCID: PMC3120182.

40. Matthias AT, de Silva DKN, Indrakumar J, Gunatilake SB. Physical activity levels of patients prior to acute coronary syndrome - Experience at a tertiary care hospital in Sri Lanka. Indian Heart J. 2018 May-Jun;70(3):350-352. doi: 10.1016/j.ihj.2017.08.020. Epub 2017 Aug 25. PMID: 29961449; PMCID: PMC6034015.

41. Jorge Jde G, Santos MA, Barreto Filho JA, Oliveira JL, de Melo EV, de Oliveira NA, Faro GB, Sousa AC. Level of Physical Activity and In-Hospital Course of Patients with Acute Coronary Syndrome. Arq Bras Cardiol. 2016 Jan;106(1):33-40. doi: 10.5935/abc.20160006. Epub 2015 Dec 22. PMID: 26690692; PMCID: PMC4728593.

42. American Heart Association Recommendations for Physical Activity in Adults. Updated:Jul 27,2016.

43. Ravi Dhingra, Ramachandran S. Vasan. Age As a Risk Factor. Medical Clinics of North America,Volume 96, Issue 1, 2012: 87-91. ISSN 0025-7125. https://doi.org/10.1016/j.mcna.2011.11.003.

44. Michael W. Rich, MD, FACC, AGSF. HealthinAging.org. https://www.healthinaging.org/tools-and-tips/ask-expert-heart-disease.

45. Christina Chrysohoou, Demosthenes B. Panagiotakos, Christos Pitsavos. Gender Differences on the Risk Evaluation of Acute Coronary Syndromes: The CARDIO2000 Study. Official Journal of the American Society of Preventive Cardiology. Volume6, Issue2 Spring 2003. Pages 71-77. https://doi.org/10.1111/j.1520-037X.2003.01609.x

46. Sadia H, Rabia T, Fatima A, Khawaja T M. Modifiable and Non-modifiable predisposing Risk Factors of Myocardial Infarction -A Review. Journal of Pharmaceutical Sciences and Research. Vol.4(1), 2012,1649-1653. ISSN: 0975-1459.

47. Nassis GP, Geladas ND. Age-related pattern in body composition changes for 18-69 year old women. J Sports Med Phys Fitness. 2003 Sep;43(3):327-33. PMID: 14625514.

48. Sesso HD, Lee IM, Gaziano JM, Rexrode KM, Glynn RJ, Buring JE. Maternal and paternal history of myocardial infarction and risk of cardiovascular disease in men and women. Circulation. 2001 Jul 24;104(4):393-8. doi: 10.1161/hc2901.093115. PMID: 11468199.

49. Shea S, Ottman R, Gabrieli C, Stein Z, Nichols A. Family history as an independent risk factor for coronary artery disease. J Am Coll Cardiol. 1984 Oct;4(4):793-801. doi: 10.1016/s0735-1097(84)80408-8. PMID: 6481018.

50. Ada's Medical Knowledge Team. Cardiovascular Disease Risk Factors. September 7, 2022. https://ada.com/cardiovascular-disease-risk-factors/.

51. American Heart Association (www.heart.org).

52. Davidson Principles of Medicine. Cardiology. Coronary artery disease. Page: 495-496. https://archive.org/details/davidson23/page/494/mode/2up?view=theater.

53. ECG and Echo Learning. Non-STEMI (non ST elevation myocardial infarction) & Unstable angina: Criteria, ECG, Diagnosis, Management. Section 3, Chapter 21. https://ecgwaves.com/topic/nstemi-non-st-elevation-myocardial-infarction-unstable-angina-criteria-ecg-diagnosis-management.

54. Dovepress. Yagel O, Shadafny N, Eliaz R, Dagan G.Long-Term Prognosis in Young Patients with Acute Coronary Syndrome Treated with Percutaneous Coronary Intervention. Dovepress. Volume 2021:17, 153-159.