International Journal of Medical and Pharmaceutical Sciences
Open access peer-reviewed Journal
Volume 2, Issue 2, Page 74-84.
https://doi.org/10.5281/zenodo.18514357

Review Article

Acute Coronary Syndrome

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Atherosclerosis continues to be a major cause of death worldwide despite advancements in patient care and research. In addition to calcific nodules and coronary spasm, plaque erosion or rupture is the primary cause of acute coronary syndromes (ACS). Because of this complexity, ACS management must change to emphasise precision medicine in order to account for changing risk profiles and sex-specific variations[1]. The review emphasises the various causes of ACS, the importance of novel imaging methods and diagnostic tools, and the function of AI in individualised treatment. The updated 2023 European Society of Cardiology guidelines include evidence-based suggestions for patient management and treatment approaches, highlighting chances for improved technology utilisation and personalisation in ACS care[2].

Keywords: Acute coronary syndrome ,Acute myocardial infarction ,ST- elevated myocardial infarction , non-ST elevated myocardial infarction , Unstable angina ,Plaque rupture , Coronary spasm , Dual anti- platelet therapy , Risk factors.

INTRODUCTION

The term "acute coronary syndrome" (ACS) describes a range of clinical manifestations, from those associated with ST-segment elevation myocardial infarction (STEMI) to those associated with unstable angina or non-ST-segment elevation myocardial infarction (NSTEMI). [3,4] ACS-related MI should be classified as STEMI or NSTEMI since elevation of the ST segment during ACS is associated with coronary occlusion and influences the choice of therapy (urgent reperfusion therapy). Pathologically, ACS is nearly always linked to partial or total thrombosis of the infarct-related artery and rupture of an atherosclerotic plaque. However, when physiologic stress (such as trauma, blood loss, anemia, infection, or tachyarrhythmia) increases the demands on the heart, stable coronary artery disease (CAD) may occasionally lead to ACS without plaque rupture and thrombosis. The typical rise and fall of biochemical markers of myocardial necrosis, along with at least one of the following are necessary for the diagnosis of acute myocardial infarction in this context:

Symptoms of ischemia

- Pathologic Q wave development on an electrocardiogram (ECG)
- New left bundle branch block (LBBB) or notable ST-segment-T wave (ST-T) changes
- Imaging proof of a new regional wall or new loss of viable myocardium abnormal motion

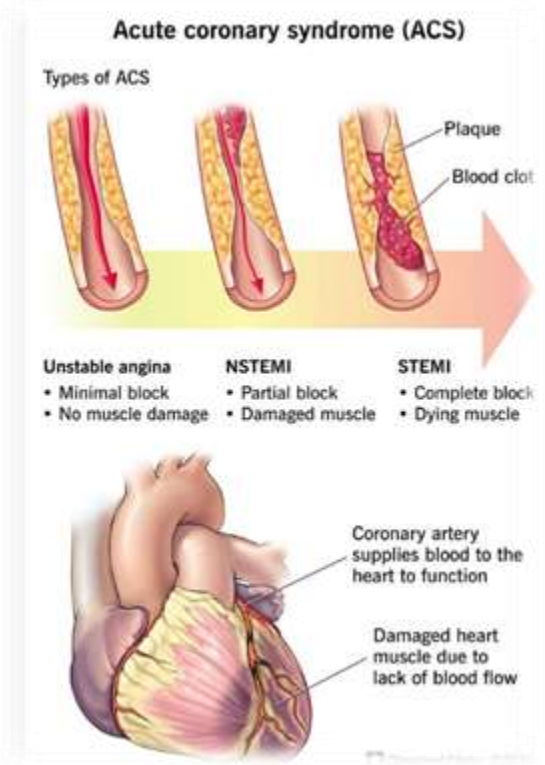
Angiography or autopsy-identified intracoronary thrombus When treating ACS, it's critical to pay attention to the underlying mechanisms of ischemia. Rate-pressure product, which can be reduced by beta blockers (such as metoprolol or atenolol) and pain/stress relievers (such as morphine), is a straightforward indicator of demand. On the other hand, oxygen, sufficient hematocrit, blood thinners (such as heparin, IIb/IIIa agents like abciximab, eptifibatide, tirofiban, or thrombolytics), and/or vasodilators (such as nitrates, amlodipine).

Types Of ACS

1. STEMI (ST elevation myocardial infarction): STEMI is a form of acute coronary syndrome. The total thrombotic blockage of the infarct-related artery causes this life-threatening and urgent emergency. Large myocardial risk areas and intense chest pain are the most common presenting symptoms. In cases of this kind of acute coronary syndrome, quick access to coronary revascularization techniques is advised. Among the risk factors that lead to the development of atherosclerosis and raise the possibility of plaque instability are diabetes, smoking, hypertension, and hyperlipidemia.

2. NSTEMI (Non-ST elevation myocardial infarction): NSTEMI is a pressure-like substernal pain that can happen at rest or during physical activity. This pain can travel to the arm, neck, or jaw and typically lasts longer than ten minutes. Dyspnea, nausea or vomiting, syncope, and fatigue (tiredness) are also linked to it. Male gender, advanced age, a personal or family history of coronary artery disease (CAD), diabetes, and renal insufficiency are risk factors. Atypical symptoms of this kind of acute coronary syndrome include indigestion, isolated dyspnea (shortness of breath), and stabbing or epigastric or abdominal pain.

3. UNSTABLE ANGINA: One of the various acute coronary syndromes that manifests as sudden chest pain while at rest is unstable angina. It should be treated as an emergency because it does not go away with rest and may indicate a heart attack or even cardiac arrest [7].



Epidemiology

According to the American Heart Association (AHA), acute MI or CAD claims the lives of nearly 720,000 Americans each year, with over 335,000 experiencing recurrent events. [5] An estimated 30–40% of all ACS inpatients are 75 years of age or older, making geriatric populations especially vulnerable. ACS primarily affects men. [5] In contrast, ACS rises from roughly 20–25% in women under 50 to roughly 50% in women over 80. Additionally, compared to men, older women have a higher CV burden (such as diabetes and hypertension) and are more likely to have experienced heart failure or heart failure with preserved ejection fraction (HFpEF)

International Data

According to the 2023 EuroHeart Report, out of 40,021 admissions for ACS (STEMI or NSTEMI) in

39,694 patients across 7 countries, nearly half of the admissions were for patients 70 years of age or older, with the most common age group being 70–74 years old (14.4%), and nearly one-third of all hospitalizations were female. With a mean age of 65.6 years, a mean body mass index (BMI) of 27.7 kg/m2, and a history of smoking (current: 4865 of 18,270; previous: 3924 of 18,174), there were 18,644 patients with 18,686 admissions for STEMI. Males made up 70.6% of these patients. The mean BMI was 27.9 kg/m2, the mean age was 69.8 years, and 66.6% of the 21,050 patients with 21,335 admissions for NSTEMI were men [6].

Etiology

CAD is a complex condition. Etiologic factors fall into two general categories: modifiable and nonmodifiable. Gender, age, genetics, and family history are examples of non-modifiable factors. Risk factors that can be changed include lipid levels, smoking, obesity, hypertension, and psychosocial factors. IHD has become more common in the West as a result of people leading faster-paced lives and consuming more fast food and unhealthy meals. Better primary care in the middle and upper socioeconomic groups in the US has caused the incidence to move later in life. The leading cause of cardiovascular disease is still smoking. Adult smoking prevalence in the US was 15.5% in 2016[8]. Compared to women, men are more likely to develop CAD. One significant modifiable risk factor for CAD is still hypercholesterolemia. Elevated high-density lipoproteins (HDL) reduce the incidence of CAD, while elevated low-density lipoproteins (LDL) increase the risk of CAD. The atherosclerotic cardiovascular disease equation on the American Heart Association (AHA) portal can be used to determine a person's 10-year risk of atherosclerotic cardiovascular disease. [9] Inflammatory markers are significant risk factors for CAD. Although its practical applications are debatable, some studies believe that high-sensitivity C-reactive protein (hs-CRP) is the best predictor of CAD. According to research, obesity is associated with a higher prevalence of cardiovascular conditions and an increased risk of cardiovascular disease. Abdominal fat accumulation is more accurately indicated by waist circumference than by body mass index (BMI). People who have a normal BMI but a larger waist circumference are more likely to develop CAD. The waist circumference cut-off values for patients with CAD and a normal BMI are not specifically recommended by the current obesity guidelines. Sharma et al. looked at information from five cohort studies involving 7,057 CAD patients. Despite having a normal BMI, the group discovered that patients with CAD were more likely to die if they had a larger waist circumference or waist-to-hip ratio. [10]

Pathophysiology

Global health is still greatly impacted by ischemic heart disease, especially when it comes to acute coronary syndromes (ACS), which are caused by an abrupt cutoff of blood flow to the heart. Plaque erosion, which is characterized by thrombus formation without fibrous cap destruction; plaque rupture, which involves atherosclerotic plaques and metalloproteinases leading to thrombosis; and non-atherosclerotic causes, such as coronary vasospasm, are the main mechanisms underlying ACS. Furthermore, new ideas in the treatment of ACS highlight the significance of plaque erosion and the identification of "vulnerable patients." The goal of this review is to clarify the pathophysiology and developments in the treatment of ACS [11].

Plaque Rupture

A crucial step in the development of coronary thrombosis is plaque rupture, which happens when the fibrous cap covering the lipid-rich necrotic core cracks, exposing blood to clotting proteins and causing thrombosis. Particularly susceptible and linked to a higher risk of myocardial infarction are thin-capped fibroatheroma (TCFA) plaques. Interstitial collagen degradation, a reduction in vascular smooth muscle cells (VSMCs), and inflammation, which triggers macrophages to produce collagen-degrading enzymes, are factors that lead to TCFA rupture. There are two types of plaque rupture: those with systemic inflammation, where elevated C-reactive protein (CRP) indicates inflammation, and those without systemic inflammation, which is impacted by physical activity and psychological stress. While other mechanisms may cause rupture in the absence of inflammation, anti-inflammatory

therapies may be helpful in cases with systemic inflammation [12].

Plaque Erosion

The idea of "vulnerable plaque" has been reexamined, and the emphasis now is on plaque erosion as a major cause of acute coronary syndrome (ACS). Eroded plaques, in contrast to vulnerable plaques, have intact fibrous caps, are rich in extracellular matrix elements like hyaluronic acid and proteoglycans, and have a notable CD44 localization. As shown in the EROSION study, eroded plaques show less macrophage aggregation and are characterized by the formation of a "white thrombus," which can respond well to antiplatelet therapy without the need for stenting. About one-third of ACS cases are caused by plaque erosion, which primarily manifests as non-ST elevation myocardial infarction (NSTEMI). It has unique epidemiological characteristics, such as being more common in women and younger populations and having fewer typical cardiovascular risk factors. Clinically, compared to plaque rupture, ACS from plaque erosion exhibits fewer adverse events, a favorable lipid profile, and a lower plaque burden [13]. By detecting endothelial cell disruptions and the presence of thrombus without visible fissures in the fibrous cap, optical coherence tomography (OCT) is a helpful diagnostic technique for differentiating between plaque erosion and plaque rupture [14].

1. Innate Immunity and Endothelial Cells Death

Inflammation may be less important in plaque erosion than in plaque rupture, as evidenced by the decreased macrophage presence in eroded plaques. However, certain innate immune responses—such as the desquamation of endothelial cells (ECs) and the basement membranes that underlie them—contribute to erosion and may make an area more susceptible to erosion. Because type IV collagen and laminin are essential for EC adhesion, MMP-2 degradation, which is triggered by MMP-14, may promote desquamation and erosion. The activation of MMP-2 by MMP-14 is essential for erosion mechanisms, in contrast to MMP-1, 8, and 13, which are associated with rupture. The endothelium layer can also be disrupted by endothelial cell apoptosis, which is brought on by damaging stimuli like hypochlorous acid. Increased MPO activity indicates more apoptosis in eroded plaques. Local flow disruptions also play a role in EC damage [15].

2. Toll-Like Receptor-2 Upregulation

One important innate immune receptor that is noticeably elevated in plaque erosion sites is TLR2. When it is activated, endothelial cells (ECs) are damaged, leukocyte adhesion molecules are expressed more frequently, and more IL-8 is produced, which encourages neutrophil recruitment and EC desquamation. TLR2 agonists impede intimal layer repair, suggesting their involvement in plaque erosion. Endogenous ligands that are linked to plaque erosion, like hyaluronan fragments, can activate TLR2, increasing the expression of VCAM-1 and E-selectin as well as ECs' susceptibility to apoptosis. Additionally, TLR2 activity is modulated by other DAMPs and PAMPs, which contribute to endothelial activation associated with plaque erosion [16].

3. Formation of Neutrophil Extracellular Traps

Thrombosis is thought to result from a two-hit process of plaque erosion. The endothelial layer is compromised and cell death is encouraged by the first hit, which is the innate immune activation of endothelial cells (ECs) brought on by disrupted blood flow. The second hit happens when activated platelets release chemicals that worsen thrombosis and inflammation, while activated ECs release chemokines that draw leukocytes. By encouraging inflammation and thrombosis, neutrophil extracellular traps (NETs), which are created by NETosis, worsen damage and contribute to acute coronary syndrome (ACS). NETs play a crucial role in plaque erosion because studies show that they are more common in erosive plaques than in ruptured ones. Statins reduce the risk of ACS overall, but by increasing NET formation; they may unintentionally increase the risk of thrombosis. Treatment for plaque erosion-related ACS may be improved by targeting NETs with medications like deoxyribonuclease and PAD-4 inhibitors [17].

4. Endothelial-to-Mesenchymal Transition

EndMT is the process by which mature endothelial cells (ECs) undergo significant heterogeneity and transdifferentiate into mesenchymal-like cells in

response to different stimuli. Extracellular matrix (ECM), pro-inflammatory proteins, and EndMT markers, such as mesenchymal and vascular smooth muscle cell (VSMC) markers, are activated during this process, resulting in alterations to EC morphology, polarity, and function. EndMT is associated with endothelial dysfunction and may lead to plaque erosion, particularly when ECs are exposed to low shear stress, oxidative stress, hypoxia, and inflammation through TGF-β and other pathways. EndMT also damages adherens junctions and VE-cadherin, which compromises endothelial integrity and increases thrombosis. However, research on EndMT's role in plaque erosion and acute coronary syndrome (ACS) is still ongoing, and discussions about its possible advantages and disadvantages are still going on [18].

Calcific Nodule

Patients with acute coronary syndrome (ACS) may develop acute coronary thrombosis due to calcified nodules (CN), a rare form of coronary plaque. Cap fissures and thrombosis are caused by the fragmentation of necrotic core calcifications in CN, which is characterized by prominent nodular calcifications and thrombus attached to the luminal surface. Due to mechanical stress, these are especially troublesome in the right coronary artery, increasing the risk of poor cardiac outcomes following PCI and causing plaque formation. The gold standard for CN diagnosis is optical coherence tomography (OCT). Plaque modification procedures like intravascular lithotripsy, excimer laser angioplasty, and coronary rotational atherectomy are effective treatments [19].

Non-Atherosclerotic Causes

Acute myocardial ischemia can result from a number of non-atherosclerotic causes of ACS in addition to plaque rupture, plaque erosion, and CN. Coronary vasospasm, spontaneous coronary artery dissection (SCAD), MB, stress-induced cardiomyopathy (Takotsubo syndrome), and coronary artery embolism due to thrombus from another part of the body causing obstruction are common non-atherosclerotic causes of ACS [20].

1) Coronary Vasospasm

Myocardial ischemia and type 2 myocardial infarction result from temporary coronary artery constriction brought on by coronary vasospasm. Men between the ages of 40 and 70 are more likely to have it, especially Japanese men. Vascular dysfunction and autonomic nervous system disorders are factors that contribute to its pathophysiology. Nitrates and calcium channel blockers are useful treatments, and the main diagnostic technique is the ergonovine test. For this diagnosis, cardiac magnetic resonance imaging is debatable [21].

2) Spontaneous Coronary Artery Dissection

Acute Coronary Syndrome (ACS) is rarely caused by SCAD (Spontaneous Coronary Artery Dissection), especially in younger women without conventional risk factors. Muscle fiber dysplasia, hormonal fluctuations, childbirth, and connective tissue disorders could all be contributing factors to its unclear etiology. A false lumen formation in the coronary intima-media causes SCAD, which may result in intramural hematoma and blood flow obstruction. The formation of a compressive inter-membranous hematoma can be caused by either bleeding from vessels supplying the arterial wall or rupture of the inner membrane with subsequent blood infiltration. Coronary angiography is used for diagnosis; if angiography is not conclusive, Optical Coherence Tomography (OCT) is used as a backup technique [22].

3) Myocardial Bridging

A congenital coronary artery malformation known as mural coronary artery (MB) occurs when a portion of the coronary artery, usually the left anterior descending artery, pierces the heart's muscular layer. Although the majority of cases are asymptomatic, depending on the thickness and length of the bridge, complications like stable angina, acute coronary syndrome, and sudden cardiac death may result. Depending on the depth of the coronary artery, MB can be divided into superficial and deep types. Myocardial fibers compress the blood vessels during the cardiac cycle, which may result in myocardial infarction and ischemia. By displaying the aberrant coronary segment, coronary CT angiography is useful in the diagnosis of MB. The prognosis for MB is generally good, and treatment options include calcium

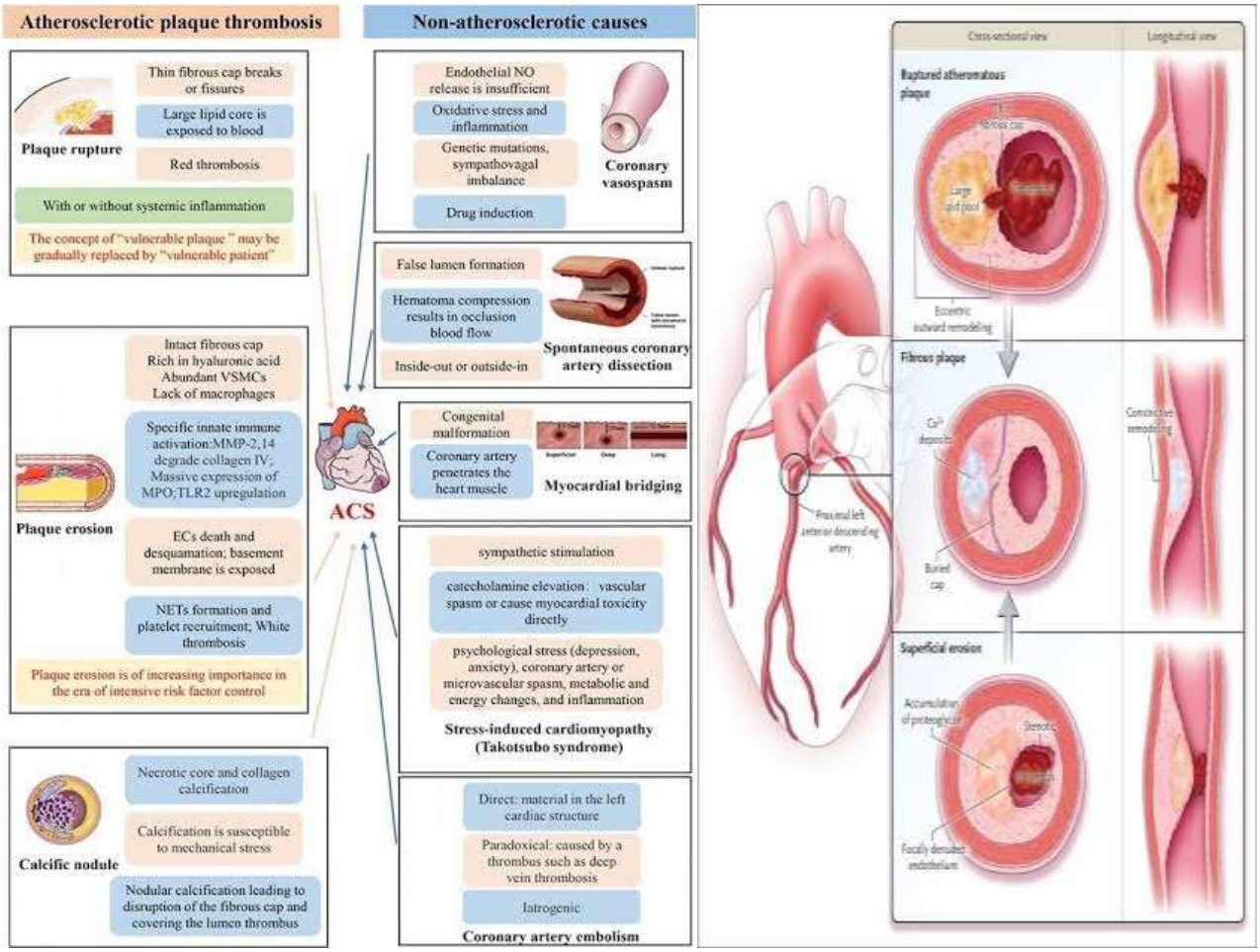
channel blockers and β-blockers, as well as surgery if pharmacological treatment is not working [23].

4) Stress-Induced Cardiomyopathy

Takotsubo syndrome (TTS), also known as stress-induced cardiomyopathy, is a transient condition that primarily affects postmenopausal women and is characterized by transient left ventricular motion abnormalities. It frequently resembles acute coronary syndrome (ACS). Because of the shape of the left ventricle, it was discovered in the early 1990s and is named after a Japanese octopus pot. Although the precise mechanisms are still unknown, increased sympathetic activity and elevated catecholamine levels are thought to be important, possibly leading to myocardial toxicity and vascular spasms. Inflammation, metabolic alterations, coronary spasms, and psychological stress are additional contributing factors. The main goals of treatment are to manage symptoms, address underlying causes, and provide supportive care [24].

5) Coronary Artery Embolism

Myocardial ischemia and possible myocardial damage result from coronary embolism, a condition in which emboli obstruct blood flow in the coronary arteries. Blood thrombus detachment, tumors, fat, air, and foreign materials are the causes of 3–5% of acute coronary syndromes (ACS). Common causes include valve replacement and infectious endocarditis. Coronary embolisms can be classified as either direct (caused by materials from left cardiac structures), paradoxical (caused by thrombus detachment outside the coronary arteries), or iatrogenic (caused by medical interventions). Diagnosis requires patient history and imaging studies, while treatment options depend on the embolism's cause, including thrombus aspiration, stent implantation, balloon angioplasty, anticoagulation, and anti-infective therapy [25].



Symptoms

The most frequently reported symptom of acute coronary syndrome (ACS) is chest pain. This sudden chest pain gets worse when you move or are at rest. Chest pain is frequently described by patients as burning, aching, stabbing, pressure, tearing, gas with the urge to eructate, indigestion, and occasionally sharp, needle-like pain [26]. The following are signs and symptoms of acute coronary syndrome:

- Shoulder, arm, neck, jaw, back, or abdomen pain Tightness, squeezing, crushing, burning, choking, or aching pain or discomfort that doesn't go away when you're at rest
- Breathlessness (SOB)
- Fear
- Feeling queasy
- Perspiration
- Lightheadedness or dizziness
- irregular heartbeat
- Nausea or vomiting
- Pain or burning in the upper belly, called indigestion
- Fainting

Risk Factors

Uncontrollable risk factors for coronary artery disease include:

- **Age:** The risk of artery damage and narrowing rises with age.

- **Birth sex:** In general, coronary artery disease is more common in men. But after menopause, women are more at risk.

- **Ancestry:** You are more likely to develop coronary artery disease if you have a family history of heart disease. This is particularly true if a child, brother, sister, or parent had heart disease at a young age. If your mother or sister had heart disease before the age of 65, or if your father or brother had it before the age of 55, you are at the highest risk.

Coronary artery disease risk factors you can control are [27]:

- **Smoking:** Give up smoking if you do. Heart health is negatively impacted by smoking. Smokers are far more likely to develop heart disease. The risk is also increased by breathing in secondhand smoke.

- **Elevated blood pressure:** Arteries can become hard and stiff due to uncontrolled high blood pressure. Atherosclerosis, which results in coronary artery disease, may result from this.

- **Cholesterol:** An elevated blood level of "bad" cholesterol can raise the risk of atherosclerosis. Low-density lipoprotein (LDL) cholesterol is referred to as "bad" cholesterol. Atherosclerosis is also caused by low levels of high-density lipoprotein (HDL) cholesterol, or "good" cholesterol.

- **Diabetes:** Coronary artery disease is more likely in people with diabetes. Certain risk factors, like obesity and high blood pressure, are shared by type 2 diabetes and coronary artery disease.

- **Obesity:** Excess body fat is detrimental to general health. High blood pressure and type 2 diabetes can result from obesity. Find out from your medical team what a healthy weight is for you.

- **Chronic renal illness:** Coronary artery disease is more likely in people with chronic kidney disease.

- **Not exercising enough:** Being physically active is essential for maintaining good health. Coronary artery disease and some of its risk factors are associated with inactivity.

- **A great deal of stress:** Emotional stress can exacerbate other coronary artery disease risk factors and harm the arteries.

- **Unhealthy diet:** Consuming foods high in sugar, salt, trans fat, and saturated fat can raise the risk of coronary artery disease.

- **Consumption of alcohol:** Damage to the heart muscle can result from heavy alcohol consumption. It may also exacerbate additional coronary artery disease risk factors.

- **Quantity of sleep:** An elevated risk of heart disease has been associated with both excessive and insufficient sleep.

Other possible risk factors for coronary artery disease may include [27]:

- **Obstructive sleep apnea:** It is a term for breathing pauses that occur during sleep. Breathing stops and starts while you sleep as a result of this condition. Abrupt drops in blood oxygen levels may result from it. To pump blood, the heart has to work harder. The blood pressure rises.

- **Elevated levels of high-sensitivity C-reactive protein (hs-CRP):** When the body is experiencing inflammation, this protein is more prevalent than usual. Heart disease risk may be associated with elevated hs-CRP levels. It is believed that the blood level of hs-CRP increases as coronary arteries constrict.

- **Elevated triglycerides:** This is one kind of blood fat. Particularly for women, high levels may increase the risk of coronary artery disease.

- **Elevated homocysteine levels:** The body uses homocysteine to build and maintain tissue as well as to produce protein. However, elevated homocysteine levels may increase the risk of coronary artery disease.

- **Preeclampsia:** High blood pressure and elevated urine protein are the results of this pregnancy complication. Later in life, it may result in an increased risk of heart disease. Additional difficulties during pregnancy: Pregnancy-related high blood pressure and diabetes are recognized risk factors for coronary artery disease.

- **Some autoimmune conditions:** Atherosclerosis is more common in people with diseases like lupus and rheumatoid arthritis.

Diagnosis

Presentation in clinical setting: Every patient who exhibits ischemic symptoms should be evaluated for an ACS diagnosis. Chest pain, upper limb, mandibular or epigastric discomfort, dyspnea, diaphoresis, nausea, exhaustion, or syncope are some of the clinical signs and symptoms of ischemia. An ACS event can cause diffuse rather than localized pain and discomfort, which can happen at rest or during physical activity. One Both diaphoresis-related pain and pain radiating to the left arm, right shoulder, or both arms are more likely to be linked to MI. 5 Not every patient having an ACS event experiences these symptoms, and they are not unique to MI. Certain patient populations, including women, the elderly, diabetics, and those recovering from surgery, may experience atypical ACS symptoms [29].

Previous Health History:

For patients with suspected ACS, getting a complete medical history is crucial to ensuring the right diagnosis and treatment. The type of angina symptoms a patient is experiencing, their age, sex, history of coronary artery disease (CAD), and the existence of risk factors for ACS are all important considerations. An alternative disease process should be taken into consideration for patients who do not have these characteristics [28].

Differential Diagnosis:

It's crucial to keep in mind that MI stands for myocardial necrosis brought on by myocardial ischemia. ACS does not apply to other clinical conditions that are nonischemic, cardiac causes of myocardial injury, such as pericarditis, dissecting aortic aneurysm, and mitral valve prolapse. Additionally, a number of non-cardiac conditions, such as musculoskeletal pain, esophageal discomfort, pulmonary embolism, or anxiety, can present with symptoms similar to those of ACS. To choose the best course of treatment, it is crucial to identify the true cause of a patient's symptoms [28,29].

Cardiac Biomarkers

Elevated levels of cardiac biomarkers, especially troponins, indicate possible myocardial injury. Nevertheless, myocardial infarction (MI) cannot be diagnosed solely by these elevations, which do not identify the cause. These biomarkers can also rise in

conditions like heart failure and pulmonary embolism. Because of its sensitivity and specificity, troponin is recommended; elevations are defined as values that are higher than the 99th percentile of normal. To distinguish between acute and chronic elevations, troponin levels must be monitored as soon as possible—within six hours of the onset of pain and again within six to twelve hours. CKMB serves as a backup in the event that troponin is unavailable. In order to accurately assess acute coronary syndrome (ACS), both should ideally be evaluated sequentially [30].

Changes in the ECG

Changes in the PR segment, the QRS complex, and the ST-segment are ECG abnormalities that may indicate myocardial ischemia. An accurate assessment of ECG changes can help determine the patient's prognosis, the amount of myocardium at risk, the time of the event, and the best course of treatment. The classic indicator of a STEMI is ST-segment elevation on an ECG. One like cardiac biomarkers, the ECG by itself is frequently insufficient to diagnose an acute MI, and serial evaluations improve the ECG's sensitivity and specificity. 9 Other conditions like left ventricular hypertrophy, left bundle branch block, or acute pericarditis can cause ECG abnormalities like ST deviation [31].

Management / Treatment

Aspirin (300 mg), a heparin bolus, and, assuming there are no contraindications, an intravenous (IV) heparin infusion are the first treatments for all ACS. Additionally, ticagrelor or clopidogrel antiplatelet treatment is advised. The decision is based on the preferences of the local cardiologist. Patients undergoing thrombolysis do not receive ticagrelor. [32] As needed, supportive measures are given, such as oxygen in the event of hypoxia and morphine or fentanyl for pain management. Pain relief can also be achieved by sublingual or infusion nitroglycerin. Nitroglycerine should be administered very carefully, if at all, in situations of inferior wall ischaemia since it can result in severe hypotension. It is necessary to continuously check the heart for arrhythmias. Whether ACS is a STEMI, NSTEMI, or unstable angina will determine how it is treated going forward. For STEMI, the American Heart Association (AHA) advises immediate catheterisation and percutaneous intervention (PCI) with a door-to-procedure start time of less than ninety minutes. If PCI is not accessible and the patient cannot be moved to the catheterisation lab in less than 120 minutes, a thrombolytic (tenecteplase or another thrombolytic) is advised. The door-to-needle (TNK/other thrombolytics) time must be fewer than thirty minutes, according to AHA guidelines. Aspirin and heparin are used as part of the initial treatment for NSTEMI/Unstable Angina-Symptom control [33]. Urgent catheterisation is advised if the patient's pain persists. The timing of catheterisation and other diagnostic methods, such as myocardial perfusion studies, might be decided on a case-by-case basis based on comorbidities if symptoms are well managed. Admission and an emergency cardiology examination are always warranted with ACS. Depending on availability and the cardiologist's desire, computerised tomography angiography may potentially be used for additional workup. Unless there are contraindications, beta-blockers, statins, and ACE inhibitors should be started as soon as possible in all ACS cases. Cases that cannot be treated with PCI are either treated medically, depending on the patient's preferences and comorbidities, or undergo CABG (coronary artery bypass graft) [33].

Surgery and other procedures Stenting and angioplasty:

This procedure opens clogged heart arteries using a tiny balloon and a thin, flexible tube. The tube is inserted by a surgeon into a blood vessel, typically in the wrist or groin, and guided to the constricted heart artery. The tube is filled with a wire that has a deflated balloon on its tip. The artery widens when the balloon is inflated. After being deflated, the balloon is taken out. To help keep the artery open, a mesh tube known as a stent is typically inserted.

CABG, or coronary artery bypass grafting:

A healthy blood vessel from the leg or chest is removed during this major procedure. We refer to this section of healthy tissue as a graft. The graft's ends are affixed beneath a blocked heart artery by a surgeon. This opens up a new channel for blood to reach the heart [34].

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Cite: Dr. Padige Srivarsha*, Ramanathi Bhargav, Mounika Nenavath, Peram Navya, Acute Coronary Syndrome, Int. J. Med. Pharm. Sci., 2026, 2 (2), 74-84. https://doi.org/10.5281/zenodo.18514357