"CORRELATION OF FIBRINOGEN-ALBUMIN RATIO WITH GENSINI SCORE IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION"

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DISSERTATION SUBMITTED TO BLDE (DEEMED TO BE UNIVERSITY), VIJAYAPURA, KARNATAKA.

In partial Fulfilment of the requirements for the award of the degree of

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IN

GENERAL MEDICINE

Under the guidance of

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LIST OF ABBREVIATIONS

- ACC – American college of cardiology.
- ACD – Atherosclerotic cardiovascular disease.
- ACS – Acute coronary syndrome.
- ADP – Adenosine diphosphate.
- AHA – American Heart Association.
- AMI – Acute myocardial infarction.
- BMI – Body mass index.
- BNP – Brain natriuretic peptide.
- BP – Blood pressure.
- CABG – Coronary artery bypass grafting.
- CAD – Coronary artery disease.
- CAG – Coronary angiography.
- CKD – Chronic kidney disease.
- CRP – C reactive protein.
- cTn – cardiac Troponin.
- cTnI – Cardiac Troponin-I.
- cTnT – Cardiac Troponin-T.
- CVD – Cardiovascular disease.
- DES – Drug-eluting stent.
- DVD – Double vessel disease.
- ECG – Electrocardiography.
- ECHO – Echocardiography.

- ESC – European society of cardiology.
- FAR – Fibrinogen Albumin ratio.
- GS - Gensini score.
- HBA1C – Glycated hemoglobin.
- HDL – High Density Lipoprotein
- HIV – Human immunodeficiency virus.
- HR – Heart rate.
- ICAM – Intercellular adhesion molecule.
- ICCU – Intensive coronary care unit
- IHD – Ischemic heart disease.
- IWMI – Inferior wall myocardial infarction.
- JVP – Jugular venous pressure.
- LAD – Left anterior descending artery.
- LBBB – Left bundle branch block.
- LCX – Left circumflex artery.
- LDH – Lactate dehydrogenase.
- LDL – Low Density Lipoprotein
- LMWH – Low molecular weight heparin.
- LV – Left ventricle.
- LVEF – Left ventricular ejection fraction.
- LVF – Left ventricular failure.
- MI – Myocardial Infarction.
- NO – Nitrogen monoxide.

- NSTEMI – Non-ST segment elevation myocardial infarction.
- OM – Obtuse marginal branch.
- PCI – Percutaneous coronary intervention.
- PDA – Posterior descending artery.
- PI – Pharmaco-invasive.
- PL – Postero-lateral branch.
- POBA – Plain old balloon angioplasty.
- RAAS – Renin angiotensin aldosterone system.
- RCA – Right coronary artery.
- RV – Right ventricle.
- RWMA – Regional wall motion abnormality.
- SCD – Sudden cardiac death.
- STEMI – ST-segment elevation myocardial infarction.
- SVD – Single vessel disease.
- TC – Total cholesterol
- TG – Triglycerides.
- tPA – tissue Plasminogen activator.
- TVD – Triple vessel disease.
- UA – Unstable angina.
- UFH – Unfractionated heparin.
- URL – Upper Reference Limit.
- WHF – World heart federation.
- WHO – World health organization.

ABSTRACT

Background & Objectives:

Coronary Artery Disease (CAD) is one of the leading causes of morbidity and death in the globe. The most dangerous kind of CAD is acute ST-segment elevation myocardial infarction (STEMI). In reaction to systemic inflammatory and hemorheology changes, fibrinogen is a crucial component. A key mediator of platelet-induced coronary artery constriction is albumin. Hypoalbuminemia can change endothelial function and raise blood viscosity. Additional information may be provided by the fibrinogen-albumin ratio (FAR), which addresses the pathophysiology of STEMI.

The aim of this study is to calculate FAR in STEMI patients and compare FAR to the Gensini score (GS) derived from coronary angiography (CAG).

Materials & Methods:

70 STEMI patients were participated in this cross-sectional study that lasted for one and a half years, from May 2023 to December 2024. In accordance with the structured proforma, eligible patients for percutaneous coronary intervention had a clinical history, physical examination, and investigations. Frequencies and proportions have been used to represent categorical data. For qualitative data, the chi-square test, also known as Fischer's exact test, was employed as a significance test. The Pearson Correlation coefficient was used to perform correlations. Using the Gensini scoring method, we assessed the degree of coronary artery disease. There was a correlation between the Gensini score and FAR readings. Statistical significance was defined as a P-value of less than 0.05.

There were 23 (32.9%) females and 47 (67.1%) males in this study. 20 (28.6%) had triple vascular disease (TVD), 24 (34.3%) had double vessel disease (DVD), and about 24 (34.3%) had single vessel disease (SVD) and 2 (2.9%) were recanalized . FAR and Gensini score had a negative correlation (Pearson correlation; r = -0.02). This correlation was not statistically significant. Gensini score had a positive correlation with severity of coronary artery disease.

Interpretation & Conclusion:

Fibrinogen and albumin have negative relationships with GS, although FAR may not be a replacement for coronary angiography in assessing the severity of CAD. It is a practical, non-invasive test that can be performed in a primary or secondary healthcare facility. This is done in order to determine which patients are more likely to experience cardiovascular events and to start taking the necessary precautions.

Keywords:

Fibrinogen-Albumin Ratio (FAR); STEMI; Coronary angiography; Gensini score.

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INTRODUCTION

Coronary Artery Disease (CAD) is a significant cause of morbidity and death worldwide. The most deadly kind of CAD is acute ST-segment elevation myocardial infarction (STEMI), which is a heart attack with a particular ECG pattern. In every age group, men are more likely than women to experience myocardial infarction (MI). MI rates are rising in developing countries, despite the fact that the incidence has decreased in industrialised countries due to better health systems and the use of successful public health initiatives. The mortality rate within a year is approximately 10%, despite significant advancements in revascularisation techniques including coronary artery bypass grafting, percutaneous coronary intervention, and effective coronary critical care. [1]

An important component that is crucial in the body's reaction to systemic inflammatory and hemorheology changes is fibrinogen. Fibrinogen is an independent predictor of CAD and MI, raises the risk of thrombosis, and enhances the expression of pro-inflammatory cytokines. [1]

On the other hand, albumin plays a crucial role in mediating the constriction of coronary arteries caused by platelets. Blood viscosity and endothelial function may be affected by hypo-albuminemia due to elevated levels of free lysophosphatidylcholine. As a result, there is a negative relationship between albumin levels and CAD severity. [2, 4]

The morphology, architecture, and degree of stenosis of the coronary arteries form the basis of the Gensini Scoring system, a method for determining the severity of CAD. It offers a thorough and uniform method of assessing the degree of coronary artery disease, which is essential for prognostication and therapy planning. [3]

Serum biomarkers, such as the fibrinogen-albumin ratio (FAR), may be utilized as an early indicator of the pathophysiological processes involved in STEMI, which includes inflammation, hemorheology, and coronary artery lesion. The purpose of this study was to determine if a high fibrinogen-albumin ratio (FAR) is linked to the degree and severity of coronary artery disease (CAD) in STEMI patients who had percutaneous coronary intervention (PCI). Our findings have the potential to have a substantial influence on patient treatment and outcomes in the cardiology area.

OBJECTIVES

- To calculate the fibrinogen-albumin ratio (FAR) in ST-segment elevation myocardial infarction (STEMI)
- To correlate the fibrinogen-albumin ratio (FAR) with the Gensini Score based on coronary angiography.

REVIEW OF LITERATURE

Definition of acute myocardial infarction:

The term acute myocardial infarction (AMI) should be used when there is evidence of myocardial injury (an elevation of cardiac troponin values with at least one value above the 99th percentile upper reference limit [URL]) with necrosis in a clinical setting consistent with myocardial ischemia. [5]

Classification of acute myocardial infarction:

Acute myocardial infarction is classified based on the presence or absence of ST-segment elevation on the Electrocardiography (ECG) and is further classified into five types as per the fourth universal definition of myocardial infarction guidelines (2018) [6]

1. Coronary atherothrombosis-induced infarction. (Type 1)
2. Infarction brought on by an imbalance between supply and demand that isn't brought on by acute atherothrombosis. (Type 2)
3. An infarction that results in unexpected death without the chance for biomarker or ECG verification. (Type 3)
4. A percutaneous coronary intervention-related infarction. (Type 4a) and infarction brought on by coronary stent thrombosis. (Type 4b)
5. Coronary artery bypass grafting-related infarction (CABG). (Type 5)

Criteria for Acute Myocardial Infarction (types 1, 2, and 3):

The term "acute myocardial infarction" (AMI) describes a sudden damage to the heart that is accompanied by symptoms of acute myocardial ischaemia. Heart troponin (cTn) levels must increase or decrease, at least one measurement must surpass the 99th percentile upper reference limit (URL), and at least one of the following requirements must be met:

- Myocardial ischaemia symptoms
- Novel ischaemic ECG alterations.
- The emergence of abnormal Q waves.
- fresh regional wall motion abnormalities in a manner compatible with an ischaemic aetiology or imaging indications of a fresh loss of viable myocardium.
- Angiography or autopsy detection of a coronary thrombus (not for types 2 or 3 MIs)

The criteria for type 1 myocardial infarction (MI) are met by postmortem findings that show acute atherothrombosis in the artery feeding the infarcted myocardium. The criteria for type 2 MI are met by an imbalance between the supply and demand of oxygen in the heart that is not caused by acute atherothrombosis. The criteria for type 3 MI are also met by cardiac death in individuals with myocardial ischemia-like symptoms, as well as suspected new ischaemic ECG alterations prior to the availability of cardiac troponin (cTn) results or in the context of abnormal cTn values.

Criteria for Coronary Procedure–Related Myocardial Infarction (Types 4, 5):

Type 4a myocardial infarction (MI) is associated with percutaneous coronary intervention (PCI), but type 5 MI is associated with coronary artery bypass grafting (CABG). As long as the baseline values are normal, a MI that happens within 48 hours after the index operation is defined by an increase in cardiac troponin (cTn) values: >5 times the 99th percentile upper reference limit (URL) for type 4a MI and >10 times for type 5 MI.

Preprocedural cTn readings for patients with elevated levels must either be falling or steady (with less than 20% fluctuation). These patients must change from a baseline of

more than 20% and a >5-fold rise for type 4a MI or a >10-fold increase for type 5 MI. Furthermore, at least one of the requirements listed below needs to be fulfilled:

1. New ischemic changes on the ECG (applicable only to type 4a MI).

2. New pathogenic Q waves are beginning to appear.

3. New imaging evidence suggests a loss of viable myocardium and is compatible with an ischaemic aetiology.

4. Angiographic results show a procedural flow-limiting event, such as distal embolisation, disrupted collateral flow, thrombus in a side branch occlusion, coronary dissection, or blockage of a main epicardial artery or graft.

Suppose cTn levels are elevated and increasing but stay below the thresholds for PCI and CABG. In that case, the isolated emergence of new pathogenic Q waves meets the criteria for either type 4a or type 5 MI in a revascularisation treatment. Type 4B MI, or stent thrombosis, and type 4C MI or restenosis, are additional subtypes of type 4 MI that both fit the criteria for type 1 MI.

Additionally, postmortem detection of a procedure-related thrombus meets type 4a and type 5 MI requirements, primarily when connected to a stent.

Epidemiology:

Approximately one-third of deaths globally are caused by CVD. IHD is the most common of the cardiovascular diseases. [7] IHD is by far the most significant cause of death globally. It typically presents as MI and ischaemic cardiomyopathy and is also known as CAD and atherosclerotic cardiovascular disease (ACD). [8]

IHD is recognised as a critical risk to 21st-century sustainable development. Many people who have non-fatal IHD have a persistent disability and a lower quality of life. [9] Atherosclerosis, an inflammatory artery disease linked to cholesterol buildup

and metabolic changes brought on by several risk factors, is the main pathological process causing IHD. Just 2% to 7% of the general population do not have any risk factors for IHD, but over 70% of those who are at risk have some kind of risk factor. (10)

IHD affects about 126 million people worldwide (1,655 per 100,000), or roughly 1.72% of the global population. IHD was responsible for over nine million fatalities worldwide. The incidence usually began in the fourth decade and rose with age, with men more likely than women to be impacted. IHD is becoming more common worldwide. By the year 2030, the prevalence rate, which is currently 1,655 per 100,000, is predicted to rise to 1,845 per 100,000. Countries in Eastern Europe continue to have the highest incidence. In many areas, age-standardized rates which account for population fluctuations over time have declined. (11)

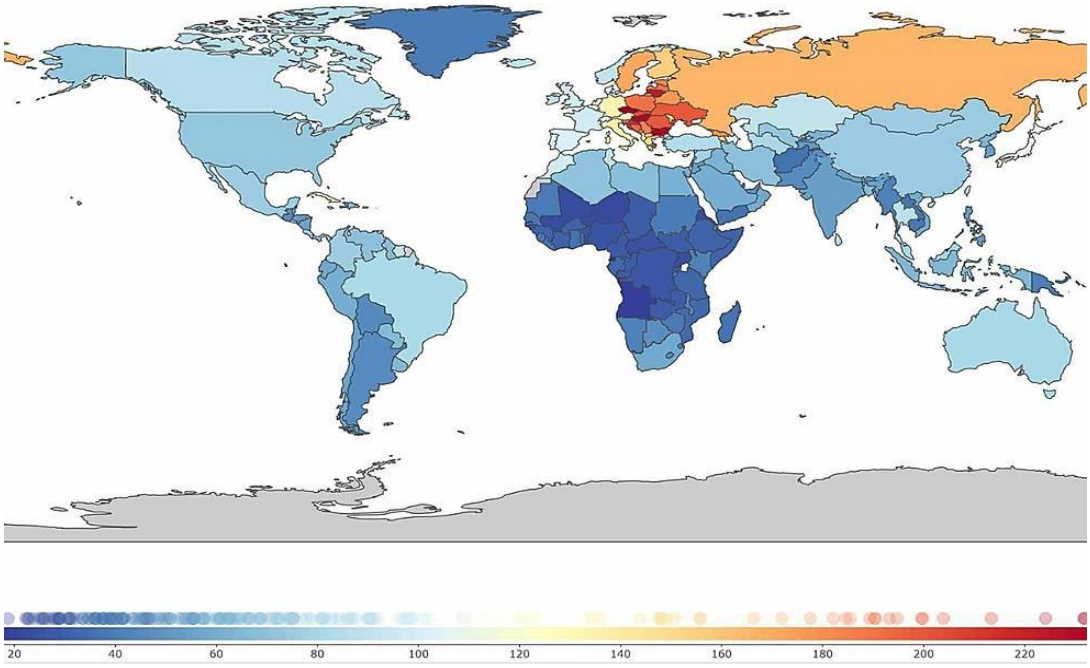


Figure 1: Global distribution of ischemic heart disease. (11) Different Colors in the figure indicate prevalence rates per 100,000 population in 2017.

IHD was the top cause of death globally in 2017, accounting for around nine million fatalities. For over twenty years, IHD has had a prominent role. Regarding regional distribution, central and eastern Europe exhibit the highest prevalence of IHD. [Figure 1]

High-income nations like Germany, Italy, Finland, Denmark, and the United Kingdom fell in comparison. The incidence in Western Europe is still rising and is far higher than in South Asia (the Indian subcontinent) and the rest of the world. [11]

Incidence and Prevalence:

The prevalence of IHD may rise to around 1,845 per 1000,000 by 2030, with an upper confidence estimate of 1,917 per 100,000, according to prevalence indicators derived from specific predictive models. [11] One of the established risk variables is male gender; as a result, men are more likely than women to have IHD (1,786 vs. 1,522 instances per 100,000). This disparity exists across all age groups. Men also seem to have an earlier age of onset. The age distribution displayed the anticipated trends of increasing incidence and prevalence as people age. [Figure 2]

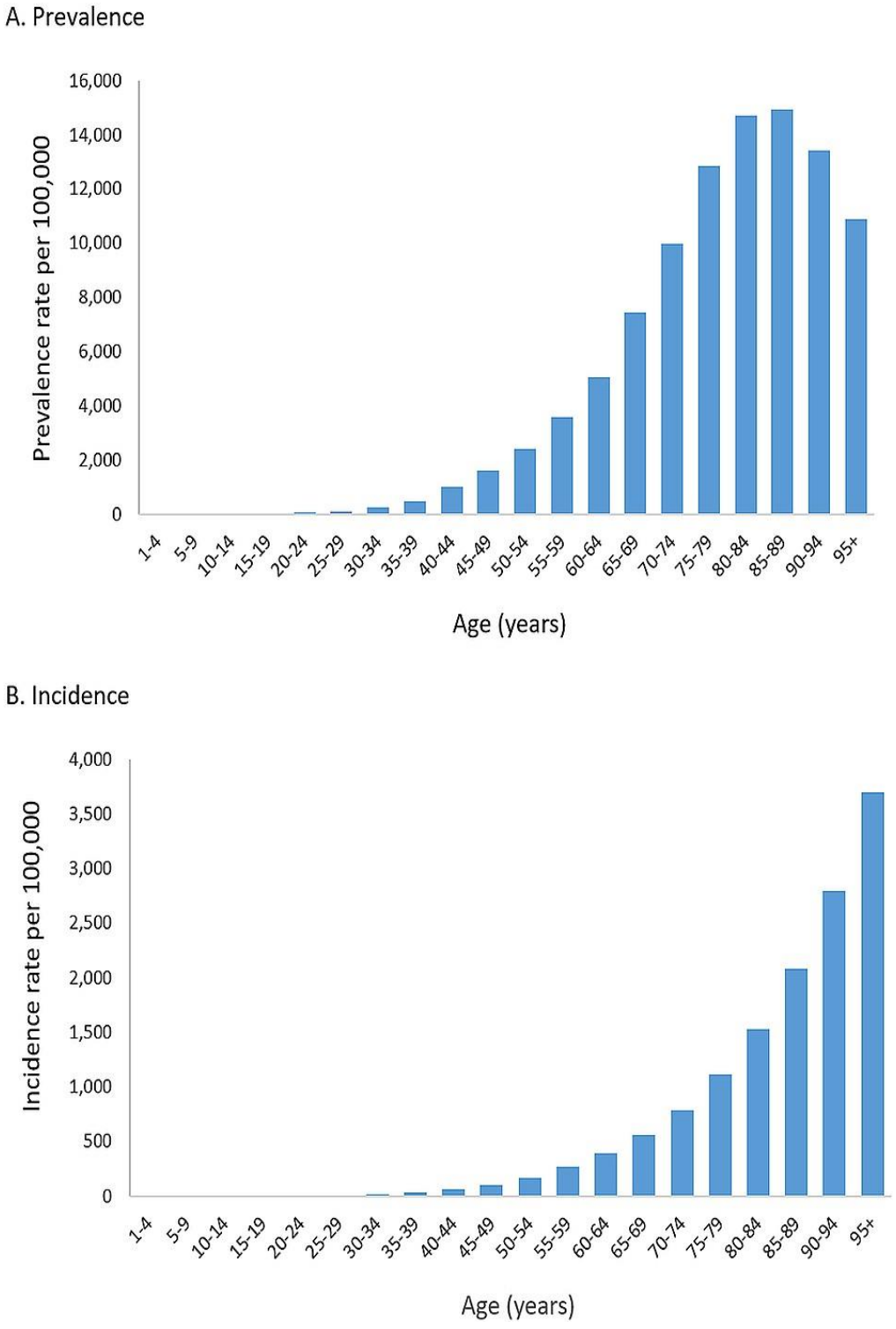


Figure 2: Age distribution of ischemic heart disease (IHD) worldwide based on prevalence (A) and incidence (B), 2017. [(11)]

Pathophysiology:

One must grasp the myocardial supply and demand to comprehend the pathophysiology of myocardial ischaemia. Under normal circumstances, the myocardium will regulate the flow of oxygen-rich blood to prevent myocyte under-perfusion and the following ischaemia and infarction, regardless of the degree of oxygen demand. Heart rate, myocardial contractility, and myocardial wall tension (stress) are the main factors determining myocardial oxygen demand. [12]

A satisfactory level of oxygen-carrying capacity of the blood (as assessed by the inspired level of oxygen, pulmonary function, and hemoglobin concentration and function) and an adequate level of coronary blood flow are necessary for a sufficient supply of oxygen to the myocardium. The majority of blood flow through the coronary arteries happens during diastole. The three groups of arteries that make up approximately 75% of the overall coronary resistance to flow are the major epicardial arteries (Resistance 1 = R1), the pre-arteriolar vessels (R2), and the arteriolar and intramyocardial capillary vessels (R3). R1 is insignificant in the absence of substantial flow-limiting atherosclerotic blockages; R2 and R3 are the main factors influencing coronary resistance. [12]

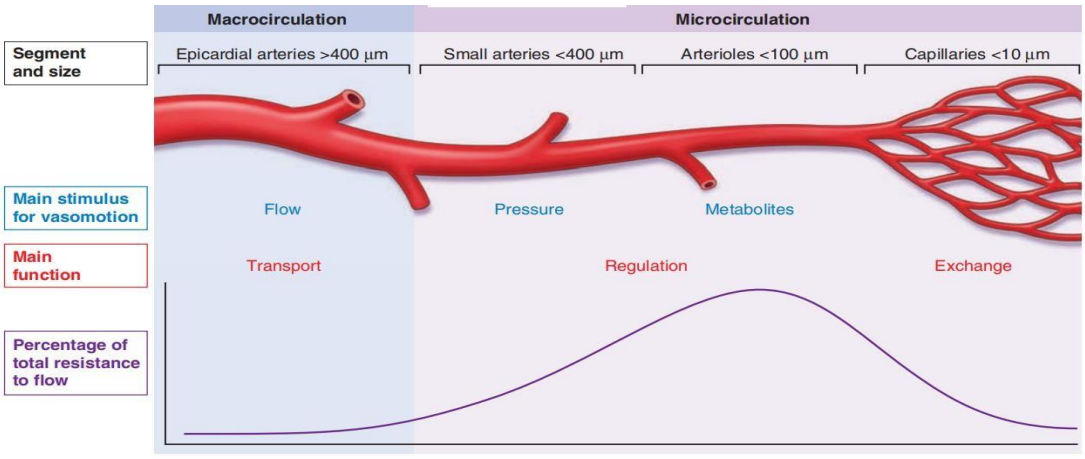


Figure 3: Macro and microcirculation across segments and sizes of arteries. [12]

The heart's need for oxygen dominates and regulates normal coronary circulation. This requirement is satisfied by the coronary vascular bed's capacity to significantly alter its resistance while extracting a high and comparatively constant percentage of oxygen from the myocardium.

Intramyocardial resistance arteries generally have a large dilatation capacity (R2 and R3 drop). For instance, coronary vascular resistance is impacted by the heart's fluctuating oxygen requirements during physical activity and emotional stress, which in turn controls the myocardium's supply of oxygen and substrate (metabolic regulation). In order to keep coronary blood flow at levels suitable for myocardial requirements, the coronary resistance vessels also adjust to physiological changes in blood pressure (autoregulation mechanism). When the demand for flow is increased, as it is during effort or excitement, atherosclerosis restricts suitable increases in perfusion by narrowing the coronary arteries lumen. Myocardial perfusion is decreased in the basal state when the luminal drop is significant. [13]

Vasospasms, arterial thrombi, and infrequently coronary emboli, in addition to ostial narrowing brought on by aortitis, can also restrict coronary blood flow. Myocardial ischaemia and infarction in infancy can result from congenital anomalies, such as the left anterior descending coronary artery's origin from the pulmonary artery. However, adults rarely have this cause. [13]

Coronary Atherosclerosis:

One important location for atherosclerosis disease is the epicardial coronary arteries. High plasma low-density lipoprotein [LDL], low plasma high-density lipoprotein [HDL], cigarette smoking, hypertension, and diabetes mellitus are the main risk factors for atherosclerosis. These factors vary in how they affect the vascular endothelium's ability to function normally. These include maintaining the

antithrombotic surface, controlling diapedesis and inflammatory cell adhesion, and controlling local vascular tone.

Inappropriate constriction, luminal thrombus development, aberrant interactions between blood cells particularly monocytes and platelets and the activated vascular endothelium result from the loss of these defences. The subintimal clusters of fat, smooth muscle cells, fibroblasts, and intercellular matrix that characterise atherosclerotic plaque are the outcome of these alterations. Different parts of the epicardial coronary tree experience uneven atherosclerosis development, which ultimately results in segmental decreases in cross-sectional area or plaque formation.

The ability to increase flow to satisfy increased cardiac demand is hampered when a stenosis reduces an epicardial artery's diameter by 50%. Blood flow may be hindered at rest when the diameter is reduced by about 80%. Even smaller reductions in the stenotic orifice can significantly impair coronary flow, resulting in myocardial ischaemia at rest or under low stress.

Plaque formation is the primary cause of atherosclerotic constriction of the epicardial coronary arteries. The cap that separates the plaque from the bloodstream may burst or erode. Two crucial processes are initiated when the plaque’s contents come into contact with blood:

1. platelets are activated and begin to aggregate, and
2. the coagulation cascade is triggered.

Consequently, fibrin strands are deposited. Clinical signs of myocardial ischaemia can result from a thrombus made up of platelet aggregates and their fibrin strands trapping red blood cells and lowering coronary blood flow. [13]

Role of Acute Plaque rupture:

After a thrombotic blockage of a coronary artery that had previously been impacted by atherosclerosis, STEMI happens when coronary blood flow suddenly drops. High-grade coronary artery stenosis that develops slowly typically does not cause STEMI because a rich collateral network gradually forms. Rather, STEMI happens when a coronary artery thrombus forms quickly at a vascular damage site. A number of variables, including cigarette smoking, high blood pressure, and cholesterol buildup, cause or contribute to this damage.

At the location of plaque disruption, a mural thrombus develops, obstructing the affected coronary artery. According to histologic investigations, coronary plaques with a thin fibrous top and a rich lipid core are the most likely to be disrupted. Several agonists, including collagen, adenosine diphosphate (ADP), adrenaline, and serotonin, encourage platelet activity following the formation of an initial platelet monolayer at the location of the fragmented plaque. Further platelet activation, the release of thromboxane A2, a potent local vasoconstrictor, and the development of possible resistance to fibrinolysis follow agonist stimulation of platelets. [14]

When agonists activate platelets, they cause a conformational shift in the glycoprotein IIb/IIIa receptor in addition to the production of thromboxane A2. This receptor has a strong affinity for soluble adhesive proteins like fibrinogen (i.e., by integrins) once it has been transformed into its functional state. Platelet aggregation and cross-linking can occur when fibrinogen, a multivalent molecule, binds to two distinct platelets at the same time.

When tissue factor is exposed in injured endothelial cells at the location of the broken plaque, the coagulation cascade is triggered. Activation of Factor VII and Factor X results in the conversion of prothrombin to thrombin, which in turn

transforms fibrinogen into fibrin. An auto-amplification response involving fluid-phase and clot-bound thrombin further activates the coagulation cascade. Eventually, a thrombus made up of fibrin strands and platelet aggregates obstructs the offending coronary artery. [14]

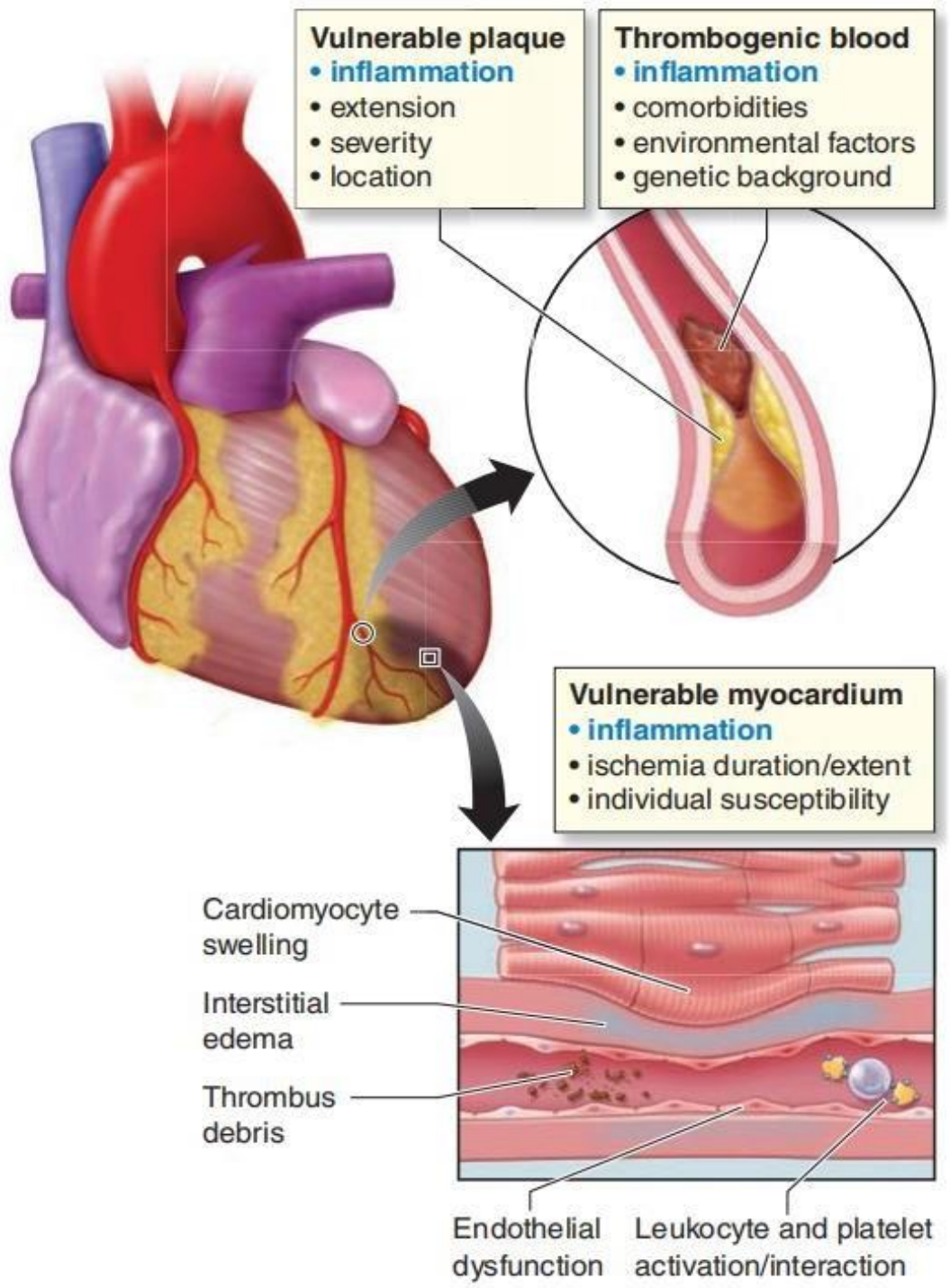


Figure 4: Critical determinants of myocardial infarction injury. [14]

Effects of Ischemia:

Myocardial tissue oxygen tension decreases during episodes of insufficient perfusion brought on by coronary atherosclerosis, which may result in brief disruptions of the myocardium's mechanical, biochemical, and electrical processes.

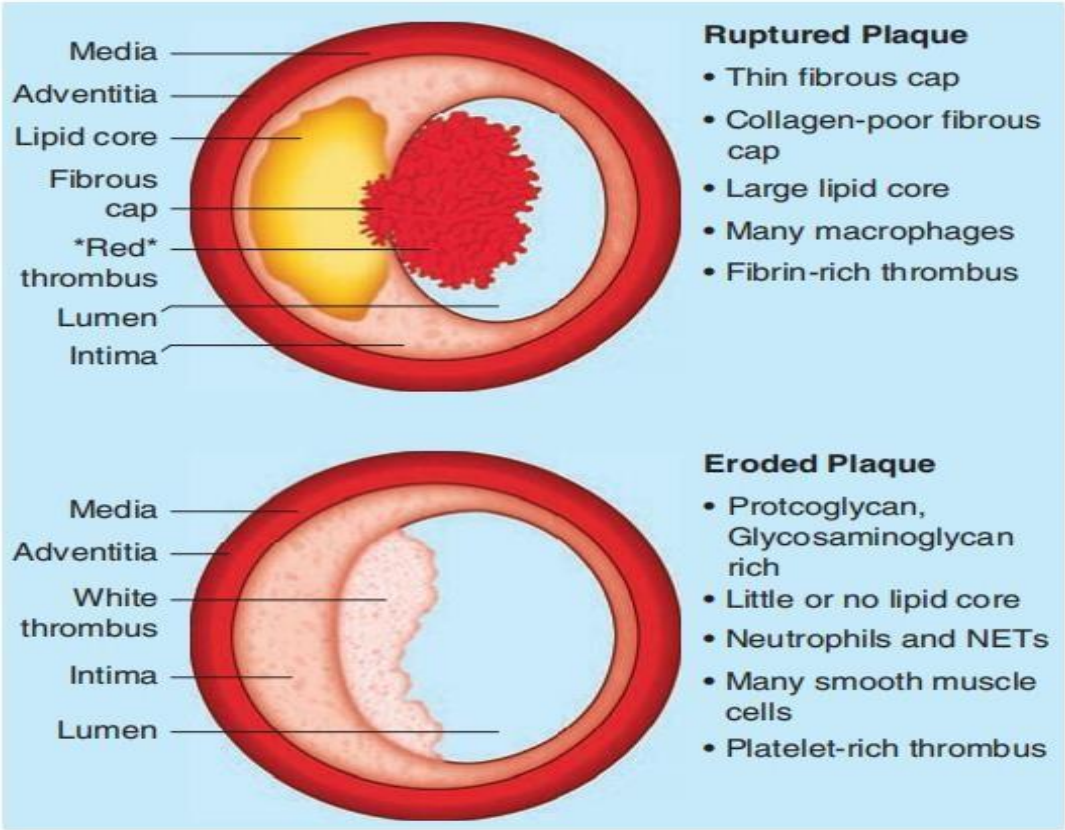


Figure 5: Comparison of the characteristics of human atheroma complicated by thrombosis and causing acute myocardial infarction. [15]

Nonuniform ischaemia is mostly caused by the focused process of coronary atherosclerosis. Segmental hypokinesia, akinesia, or, in extreme situations, bulging (dyskinesia) are caused by localised disruptions of ventricular contractility during ischaemia, which can impair myocardial pump function.

These mechanical disruptions during ischaemia are caused by a variety of anomalies in the metabolism, function, and structure of cardiac cells. Fatty acids and glucose are converted to carbon dioxide and water by the typical myocardium. Because of hypoxia, glucose is converted to lactate, preventing fatty acids from being oxidised. Severe oxygen deprivation also lowers intracellular pH and the myocardial stores of high-energy phosphates such creatine phosphate and ADP. Myocyte absorption of sodium, potassium leakage, and a rise in cytosolic calcium are all caused by impaired cell membrane function. [16]

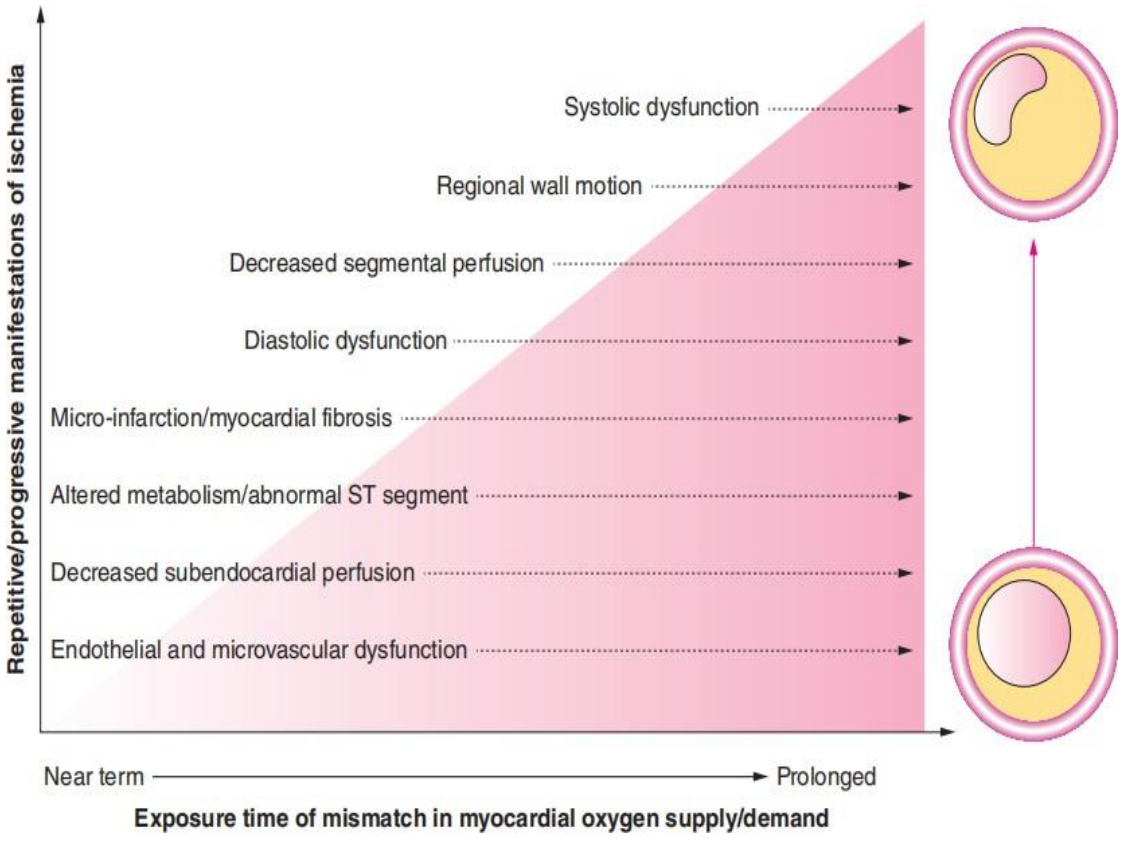


Figure 6: Cascade of mechanisms and manifestations of ischemia. [16]

Determining whether the damage is reversible (≤20 min for total occlusion in the absence of collaterals) or permanent, with eventual myocardial necrosis (>20 min), depends on the severity, length, and imbalance between the myocardial oxygen supply and the demand.

Risk Factors for CAD:

The Framingham Heart Study is presently researching its third generation of participants, having enrolled its first in 1948. This study was among the first to clarify the risk factors linked to cardiovascular disease. Cohort studies have been used ever since to investigate how various risk factors affect cardiovascular disease. Starting in 1972, the FINRISK research is a population-based observational study that is still running strong in Finland. Uppsala University in Sweden was the site of the ULSAM, PIVUS, POEM, EpiHealth, and SCAPIS cohort studies. Another cohort research that was finished in New Zealand was called PREDICT Cardiovascular Disease. These studies divide risk factors for CAD into two main groups:

1. Modifiable
2. Non-modifiable.

Risk variables that cannot be changed include age, sex or gender, ethnicity, and CAD in the family. Stress, poor diet, sedentary lifestyle, smoking, obesity, diabetes, hypertension, and hyperlipidaemia are all modifiable risk factors. Modifiable and non-modifiable risk variables were found to lessen the consequent economic and disease-related burden, given the enormous healthcare impact of CAD. Overall, CAD death rates have significantly decreased as a result of the identification of risk factors. [17]

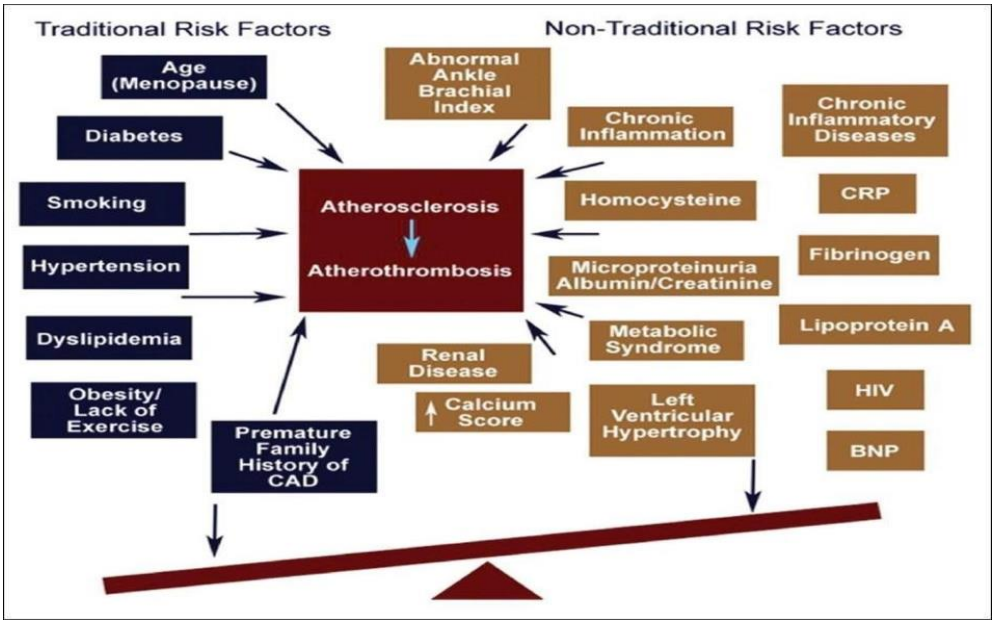


Figure 7: Risk Factors for CAD. [17]

[CAD - Coronary artery disease, HIV - Human immunodeficiency virus, BNP - Brain natriuretic peptide, CRP - C Reactive Protein].

Non-modifiable risk factors:

1. Age: Both men and women are more likely to have CAD after the age of 35. After the age of 40, the lifetime risk of getting CAD is 32% for women and 49% for men.[18]
2. Gender: Compared to women, men are more vulnerable.
3. Ethnicity: Southeast Asians, Blacks, Hispanics, and Latinos are among the ethnic groups most at risk for CAD morbidity and mortality.
4. Family history: Another important risk factor is family history. Younger people under 50 who have a family history of early heart disease are more likely to die from CAD.

Modifiable risk factors:

The role of the modifiable risk factors is reduced but still important. Significantly fewer CAD incidents occurred as a result of controlling modifiable risk variables.

1. **Hypertension:** One risk factor that is becoming more prevalent in the Indian population is hypertension. The prevalence of hypertension is 15% higher in women than in men. In both sexes, the prevalence of hypertension rises with age. Women between the ages of 45 and 54 have greater rates of hypertension than males do. Men are more likely than women to have hypertension in those under 35. At least 70% of heart disease is caused by hypertension, which is even worse for the heart as people age. The elevated risk of coronary heart disease in hypertensive people can be explained by a number of causes. Hypertension has detrimental functional effects on the coronary circulation, accelerates the effects on atheroma, increases shear stress on plaques, and compromises endothelial function and sympathetic tone regulation. [18]

2. **Hyperlipidemia/Dyslipidemia:** The second most prevalent risk factor for ischaemic heart disease is thought to be hyperlipidaemia. The World Health Organisation (WHO) estimates that 2.6 million deaths were caused by elevated cholesterol. Total cholesterol (TC), low-density lipoprotein (LDL), triglycerides (TG), apo B, or lipoprotein (a) levels above the 90th percentile or HDL and apo A levels below the 10th percentile of the general population are considered to be indicators of dyslipidaemia, a significant risk factor for CVD. Predisposing risk factors for MI include tiny and dense LDL particles and elevated TG levels. Particularly when the TC level is similarly elevated, the non-fasting TG level seems to be a reliable and independent indicator of future risk for AMI.

The cause of this is that elevated TG and lower HDL-C levels lead to metabolic disruptions, which have negative effects. Thus, hypertriglyceridemia, followed by hypercholesterolaemia, elevated LDL, and low HDL, is associated with a greater risk of AMI. [18]

3. **Diabetes Mellitus:** In India, type 2 diabetes is in danger of spreading like wildfire. Diabetes has an equivalent risk to ageing fifteen years, which is comparable with, if not greater than, that of smoking. According to a 2017 meta-analysis, those with diabetes who had a Glycated Haemoglobin (HBA1C) > 7.0 were 85% more likely to die from cardiovascular disease than those whose HBA1C was less than 7.0%. [19] By itself, insulin resistance increases the risk of congestive heart failure and likely explains why obesity is associated with this common vascular complication. However, long before clinical diabetes manifests, the risk of CVD begins to rise. Despite the fact that microvascular disease is linked to hyperglycemia, there is evidence that insulin resistance is an independent risk factor for atherothrombosis and that it promotes atherosclerosis even before it produces frank diabetes.

4. **Metabolic Syndrome/Obesity:** Metabolic syndrome, also known as syndrome X or insulin resistance syndrome, is a group of metabolic changes and cardiovascular disease risk factors linked to excess body fat. In addition to hypertriglyceridemia, low HDL levels, weak fibrinolysis, hypertension, microalbuminuria, a predominance of small dense LDL particles, and central obesity, it consists of a cluster of glucose intolerance and hyperinsulinemia. The incidence of MI is directly correlated with an elevated body mass index (BMI).

Being a known risk factor for myocardial infarction, excessive obesity significantly increases it. It has been established that insulin resistance and the hyperinsulinemia that goes along with it are key factors in the pathophysiology of the syndrome, which has a causal link to hypertension. This could be due to both a central action leading to stimulation of sympathetic activity and, in turn, the renin-angiotensin-aldosterone system (RAAS) and insulin in the peripheral vasculature. [17]

5. **Smoking:** Beyond acute, highly unfavourable effects on blood pressure and sympathetic tone and an unpredictable reduction in myocardial oxygen supply, smoking contributes to the pathogenesis of atherothrombosis by several mechanisms. Long-term smoking may enhance oxidation of LDL cholesterol & impair endothelium-dependent coronary artery vasodilation. This latter effect has been linked to 'dysfunctional endothelial nitric oxide' biosynthesis with chronic as well as acute cigarette consumption. In addition, smoking has adverse hemostatic and inflammatory effects, including increases in levels of C-reactive protein (CRP), soluble intercellular adhesion molecule-1 (ICAM-1), fibrinogen, and homocysteine.

   Additionally, smoking is associated with spontaneous platelet aggregation, increased monocyte adhesion to endothelial cells, and adverse alterations in endothelium-derived fibrinolytic and antithrombotic factors, including tissue-type plasminogen activator and tissue pathway factor inhibitor.

   Compared with non-smokers, smokers have an increased prevalence of coronary spasms and a reduced threshold for ventricular arrhythmias. More recent evidence suggests that insulin resistance represents an additional 'mechanistic link' between smoking and premature atherosclerosis.

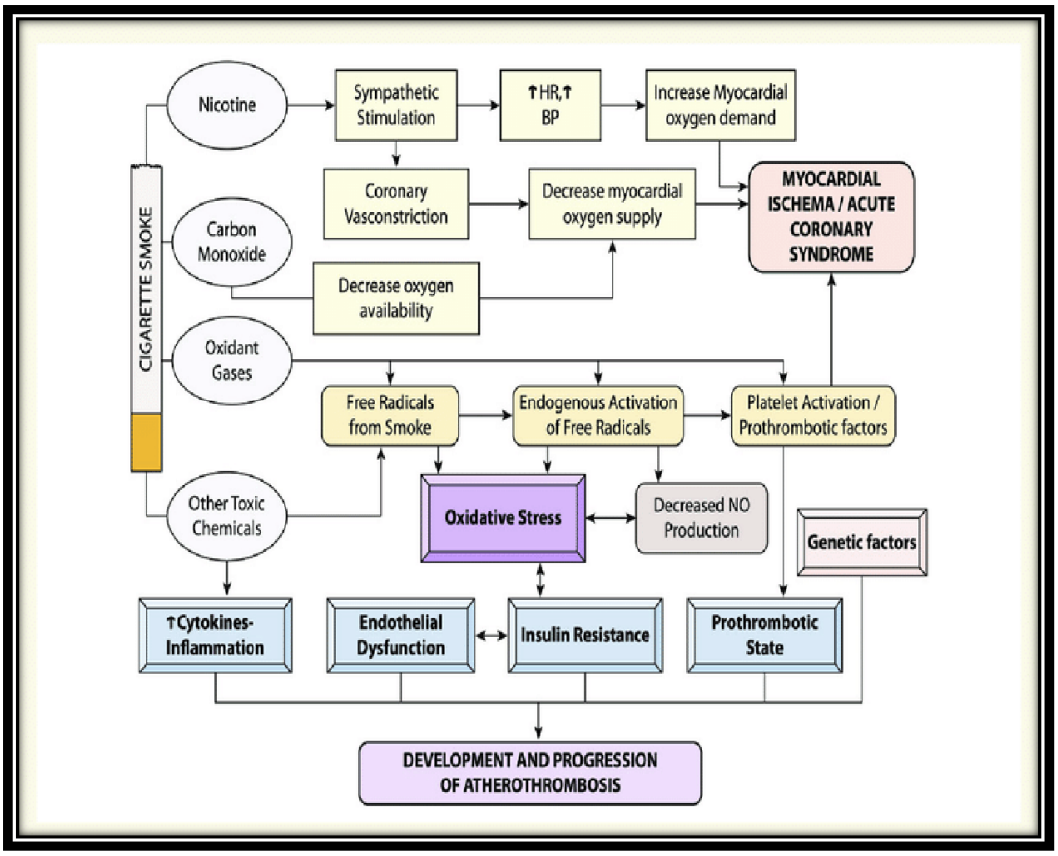


Figure 8: Pathophysiological effect of cigarette smoking and nicotine on CVD. (HR- heart rate; BP- blood pressure; NO- nitrogen monoxide) [20]

6. **Physical activity:** Inactive people with multiple cardiac risk factors are more likely to develop AMI. Physical activity may contribute up to 20%-30% reduced risk of coronary heart disease. However, there are studies which have shown that different types of physical activity may have different effects on the risk of cardiovascular disease (CVD) and may interact together. For example, some leisure time activities such as walking, stair climbing, and cycling provide protection against CVD. There are some interactive effects between lack of exercise and sitting at work, and also between demanding household work and sitting at work, which has increased the risk of acute myocardial infarction (AMI). [18]

7. **Stress:** Chronic life stress, social isolation and anxiety increase the risk of heart attack and stroke. Acute psychological stress is often associated with increased risk for coronary artery disease, and it's been reported that intense grief in the period after the death of a significant person may trigger the onset of myocardial infarction. The pathophysiological mechanism involved in this still remains unclear, but it is assumed to be related to some hemodynamic alteration in the coronary arteries and rupture of an atherosclerotic plaque, with consequent thrombosis.

Clinical manifestations of AMI:

1. **Chest pain.**

The pain of AMI is variable in intensity. In most patients, it is severe, and in some instances, it is intolerable. The pain is prolonged often lasting more than 30 minutes, and frequently for hours. The discomfort is described as constricting, crushing, or compressing; often, the patients complain of a sensation of a heavy weight or squeezing in the chest, and sometimes, it may be characterized as stabbing.

The pain is usually retrosternal in location, frequently spreading to both sides of the anterior chest, with a prediction for the anterior chest. Often, pain radiates down the ulnar aspect of the left arm, producing a tingling sensation in the left wrist, hand, and finger.In some, pain of AMI, may begin in the epigastrium and simulate a variety of acute abdominal disorders. In other patients' discomfort of AMI radiates to the shoulder, upper extremities, neck, jaw, and interscapular region. In some patients, particularly the elderly, it is manifested clinically not by chest pain but rather by symptoms of acute left ventricular failure, chest tightness, extreme weakness, and fatigue or frank syncope. [21] These symptoms may be accompanied by diaphoresis, nausea, and vomiting. The pain of myocardial infarction represents ongoing ischemia.

Other Modes of Presentation:

1. **Nausea and vomiting**

It occurs in more than 50% of patients with transmural MI and severe chest pain, due to activation of vagal reflex or to stimulation of left ventricular receptors. These symptoms occur more commonly in the inferior wall rather than anterior wall MI. [22]

2. **Breathlessness**

Severe attacks of dyspnea on exertion or at rest, and nocturnal orthopnea, often with peripheral oedema in a person who has no complaints of pain in the chest, maybe the only indication that MI has occurred. [23, 24]

3. **Palpitations**

Patients with acute MI may have just about any arrhythmia, particularly ventricular arrhythmia. Some persons with acute MI have no other symptoms or signs, except irregularities of the heart. It may or may not be described as palpitation, fluttering of the heart or a heart racing. [23,24]

4. **Atypical locus of pain.**

Atypical locus of pain or pain only in the area to which it's usually referred is another masquerade. Example: pain in the elbow, left shoulder, upper arm, jaw, etc.

5. **Syncope.**

Acute Myocardial Infarction may present as a syncopal attack.

6. **GIT Symptoms.**

A crushing type of pain in the epigastrium, vomiting, sweating and collapse do indeed have features suggesting the alimentary canal rather than the heart as a source of trouble. Flatulence, pressure, belching, a sense of fullness constriction, compressing or crushing pain may characterize the clinical pattern.

7. **Silent infarction.**

It is now widely recognized that spontaneous episodes of MI may often occur unaccompanied by pain. The reason for this symptomless ischemia is quite unclear.

8. **Stroke.**

CNS Manifestation includes an important, though not large group of masquerades. A person may have a stroke, classical in all its features with hemiparesis, convulsion, or bizarre mental aberrations with varying degrees of confusion, mania, or delirium. [25]

9. **Apprehension and nervousness.**

An occasional patient who describes no pain, whatever, with acute MI may present clinically with nothing but an overwhelming sense of apprehension, nervousness, and a weird all-pervading anxiety.

10. **Peripheral embolism.**

Peripheral embolism may be the signal of MI. Another clue, particularly in postoperative patients, is the sudden appearance of coldness in the foot, a clammy, pulseless extremity. [26]

Physical examination:

Patients with AMI often appear anxious and in considerable distress. In patients with Left ventricular failure (LVF) and sympathetic stimulation, excess perspiration and skin pallor may be evident.

1. **Pulse rate.**

Although many patients have normal pulse rates, about 1/4th of patients with anterior wall myocardial infarction, have tachycardia due to sympathetic nervous system hyperactivity, and half of the patients with inferior wall myocardial infarction (IWMI) have bradycardia due to parasympathetic overactivity.

2. Blood pressure (BP).

The majority of patients are normotensive. A slight hypertensive response is seen in the first few hours, secondary to a sympathetic response to pain. Patients with cardiogenic shock have systolic pressure below 90mmHg and evidence of end-organ hypoperfusion. More than half of the patients with IWMI have excessive parasympathetic stimulation with hypotension, bradycardia, or both. About ¼ of the patients with anterior wall MI showing sympathetic stimulation have hypertension, tachycardia, or both. [27]

3. Jugular venous pressure (JVP)

It is not surprising that usually, the JVP fails to show any abnormality in AMI. However, associated right ventricular infarction often results in a rise in JVP and in patients with cardiogenic shock.

Cardiac examination.

Despite severe symptoms and extensive myocardial damage, the findings on examination of the heart may be unremarkable. The precordium is usually quiet, apical impulse may be difficult to palpate.

On auscultation, the first heart sounds are muffled or inaudible immediately after the infarction, and their intensity increases during convalescence. Soft S1 may also indicate prolonged PR intervals.

In the presence of the left bundle branch block (LBBB), there will be a paradoxical splitting of the second heart sound. S3 is usually heard in patients with large infarction, more common in the transmural anterior wall than in the inferior wall or non-transmural MI. An S4 is almost universally heard in patients with acute MI in sinus rhythm.

Systolic murmur, transient or persistent can be heard due to papillary muscle dysfunction or left ventricular dilatation or tricuspid regurgitation, with prominent C-V wave in jugular venous pulse in tricuspid regurgitation.

Most rales are audible in patients who develop LVF or a reduction in left ventricular compliance in AMI. In 1967, Killip and Kimball proposed a prognostic classification based on the presence and severity of rales. [28]

Killip Classification:

Class I - Free from Rales and S3.

Class II - Rales in <50% of lung fields with or without S3.

Class III - Rales>50% of lung fields with S3 frequently have pulmonary oedema.

Class IV - Cardiogenic shock with systolic pressure < 90 mm Hg and evidence of peripheral vasoconstriction, cyanosis, mental confusion, and oliguria.

Laboratory Findings:

STEMI progresses through the following temporal stages:

1. Acute (first few hours–7 days),
2. Healing (7–28 days),
3. Healed (≥29 days).

The myocardium undergoes a series of cellular responses in the infarct zone, beginning with the recruitment of polymorphonuclear leukocytes (for removal of dead cells and clearance of extracellular macromolecules), followed by proinflammatory monocytes (that recruit fibroblasts) and ultimately reparative monocytes (that promote angiogenesis and interstitial collagen production). When evaluating the diagnostic tests for STEMI, the temporal phase of the infarction must be considered and prioritized. The laboratory tests of value in confirming the diagnosis may be divided into four.

1. ECG,
2. Serum cardiac biomarkers,
3. Cardiac imaging,
4. Non-specific indices of tissue necrosis and inflammation.

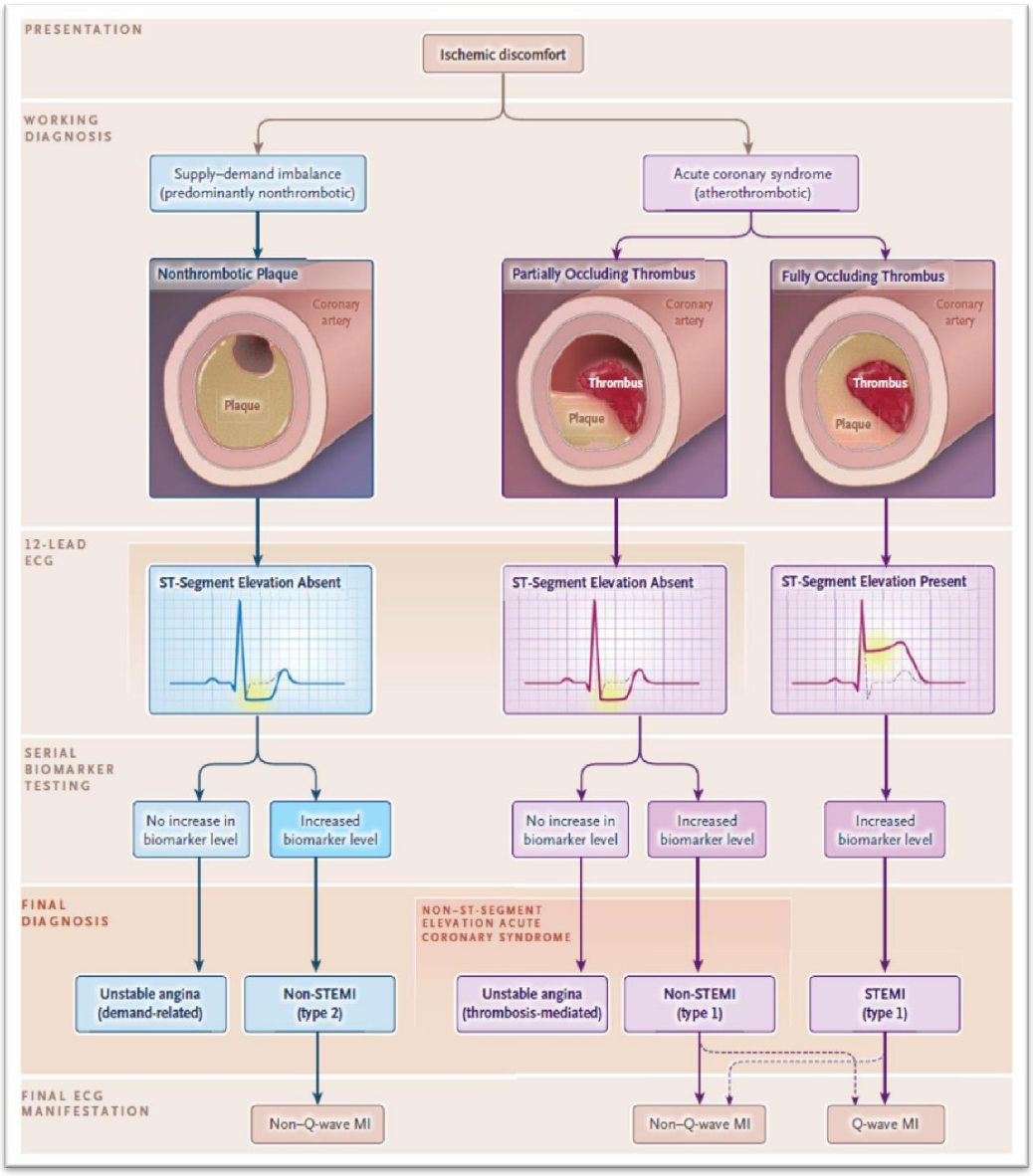


Figure 9: Spectrum of Pathologic and Clinical STEMI and Non-STEMI Acute Coronary Syndromes. [29]

ELECTROCARDIOGRAM (ECG):

During the initial stage of the acute phase of MI, total occlusion of the infarct artery produces ST-segment elevation. Most patients initially presenting with ST Elevation evolve Q waves on the ECG and are ultimately diagnosed as having sustained Q wave MI.

Among patients presenting with ST-segment elevation, if a serum marker is detected and no Q wave develops, the diagnosis of non-Q wave MI is ultimately made.

A small proportion of patients may sustain a non-Q wave MI. When the obstructing thrombus is not completely occlusive, i.e., obstruction is transient, or if a rich collateral network is present, then no ST elevation is seen. Such patients are considered as either unstable angina or an NSTEMI depending on Cardiac Enzymes. Serial standard 12-lead ECGs remain a potent and extremely clinically helpful method for the detection and localization of MI. [30]

ECG Changes at presentation in STEMI:

Typical:

- ST Elevation (at least 2 contiguous leads)
- 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2–V3.
- 1 mm (0.1 mV) in another contiguous chest leads or the limb leads.
- New or presumably new LBBB.

Atypical:

- STEMI in Evolution - Hyperacute T-wave changes.
- Evolving/Evolved STEMI - Q waves with T wave inversion with variable ST elevation
- Posterior wall MI - ST depression in 2 precordial leads (V1–V3) + positive T waves in V1.

- Left main or proximal left anterior descending (LAD) artery occlusion - ST depression in multiple leads with ST elevation in lead aVR and V1(aVR > V1: left main, V1 > aVR: proximal LAD) [30]

Acute anterior (including apical and lateral) wall ischemia is reflected by ST elevations or increased T-wave positivity (hyperacute) in one or more of the precordial leads (V1–V6) and leads I and aVL.

Inferior wall ischemia produces changes in leads II, III, and aVF.

Posterior wall ischemia (usually associated with lateral or inferior involvement) may be indirectly recognized by reciprocal ST depressions in leads V1 to V3 (thus constituting an ST elevation "equivalent" acute coronary syndrome).

Right ventricular ischemia usually produces ST elevations in right-sided chest leads (RV4).

Recommendations:

1. A 12-lead ECG must be performed in all patients with suspected STEMI.
2. Presentation to ECG diagnosis of STEMI < 10 min.
3. A low threshold for performance of ECG in patients who do not present with classical anginal pain likely to present with atypical symptoms. Hence ECG must never be missed.
4. Continuous ECG monitoring should be done in cases with suspected MI.
5. Right precordial leads (V3R, V4R, V5R) must be recorded in patients with IWMI.
6. True posterior wall MI may be diagnosed as ST elevation (>1 mm) in additional lateral chest leads V7 – V9.

Localization:

ECG might provide more specific information about the location of the occlusion within the coronary system.

In patients with an IWMI, the presence of ST-segment elevation in the lead III exceeding that in the lead II, particularly when combined with ST-segment elevation in lead V1, is a useful predictor of occlusion in the proximal to the midportion of the right coronary artery.

In contrast, the presence of ST-segment elevation in lead II which is equal to or exceeding that in lead III, especially in accordance with ST-segment depression in leads V1 to V3 or ST-segment elevation in leads I and aVL, suggests occlusion of the left circumflex artery (LCX) or it can be a distal occlusion of a dominant right coronary artery (RCA).

Right-sided ST-segment elevation is indicative of acute right ventricular myocardial injury and usually indicates occlusion of the proximal RCA. Of note is the finding that acute right ventricular infarction can project an injury current pattern in leads V1 through V3 or even V4, thereby simulating anterior infarction.

In other cases, there can be simultaneous ST-segment elevation in V1 (V2R) and ST-segment depression in V2 (V1R). Lead aVR may also provide important clues to artery occlusion in myocardial infarction.

Left main disease should be considered when leads aVR and V1 show ST-segment elevation, especially in concert with diffuse prominent ST depression in other leads.[31,32]

SERUM CARDIAC BIOMARKERS:

These are certain proteins, referred to as serum cardiac biomarkers, released from necrotic heart muscle after STEMI. The rate of liberation of specific proteins

differs depending on their intracellular location, their molecular weight, and the local blood and lymphatic flow.

Cardiac biomarkers are detectable in the peripheral blood once the capacity of the cardiac lymphatics to clear the interstitium of the infarct zone is exceeded, and spillover into the venous circulation occurs.

1. **Cardiac-specific troponin T (cTnT) and cardiac-specific troponin I (cTnI):** These proteins have amino-acid sequences that differ from those of the skeletal muscle forms of these proteins. These differences permitted the development of quantitative assays for cTnT and cTnI using highly specific monoclonal antibodies. cTnT and cTnI may increase after STEMI to levels many times higher than the upper reference limit (the highest value seen in 99% of a reference population not suffering from MI), the measurement of cTnT or cTnI is of considerable diagnostic usefulness, and they are now the preferred biochemical markers for MI. With improvements in the assays for the cardiac-specific troponins, it is now possible to detect concentrations <1 ng/L in patients without ischemic-type chest discomfort. Levels of cTnI and cTnT may remain elevated for 7–10 days after STEMI.
2. **Creatine Kinase – Muscle brain (CK-MB):** CK rises within 4–8 h and generally returns to normal by 48–72 h. An essential drawback of total CK measurement is its lack of specificity for STEMI, as CK may be elevated with skeletal muscle disease or trauma, including intramuscular injection. The MB isoenzyme of CK has the advantage over total CK in that it is not present in significant concentrations in extracardiac tissue and, therefore, is considerably more specific. However, cardiac surgery, myocarditis, and electrical cardioversion often result in elevated serum levels of the MB isoenzyme. A ratio

of CK-MB mass to CK activity ≥2.5 suggests but is not diagnostic of a myocardial rather than a skeletal muscle source for the CK-MB elevation. Markers such as myoglobin and CK isoforms are rapidly released, and blood levels rise quickly above the cut-off limit, they get cleared from the blood very early, hence not much of importance. [33]

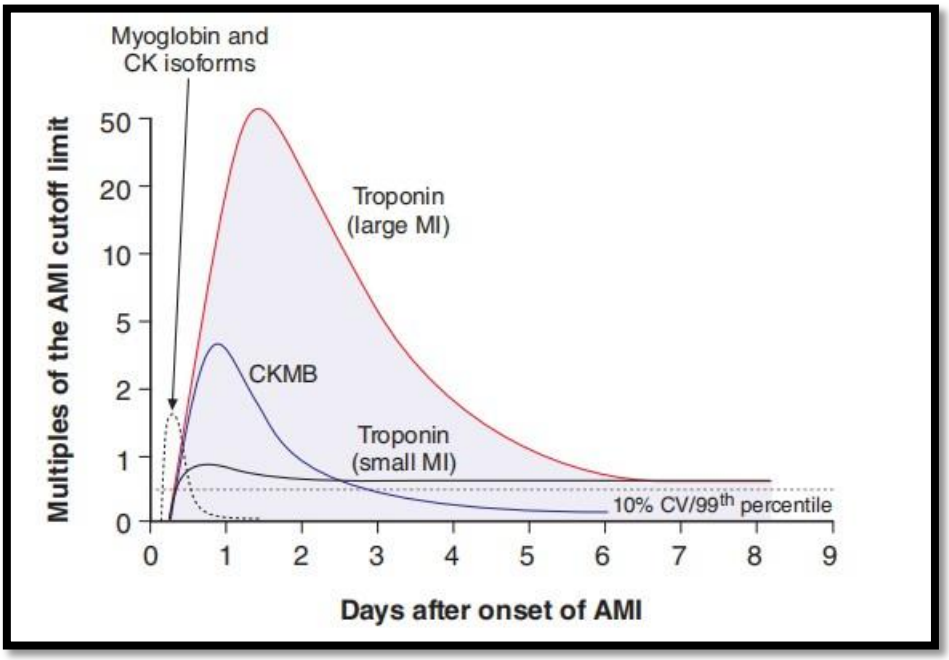


Figure 10: Biomarkers in Acute Myocardial Infarction. [33]

While it has long been recognized that the total quantity of protein released correlates with the size of the infarct, the peak protein concentration correlates only weakly with infarct size. Recanalization of a coronary artery occlusion in the early hours of STEMI causes earlier peaking of biomarker measurements because of a rapid washout from the interstitium of the infarct zone, quickly overwhelming lymphatic clearance of the proteins.

The nonspecific reaction to myocardial injury is associated with polymorphonuclear leukocytosis, which appears within a few hours after the onset of pain and persists for 3–7 days; the white blood cell count often reaches levels of 12,000–15,000/μL.

The erythrocyte sedimentation rate rises more slowly than the white blood cell count, peaking during the first week and sometimes remaining elevated for 1 or 2 weeks.

Various studies on Inflammatory markers, Acute phase reactants such as C-reactive protein (CRP), Fibrinogen, Cytokines, and Lactate dehydrogenase (LDH) levels correlate with the extent of myocardial injury and, in addition, have shown to be associated with worse clinical outcome in ACS patients.

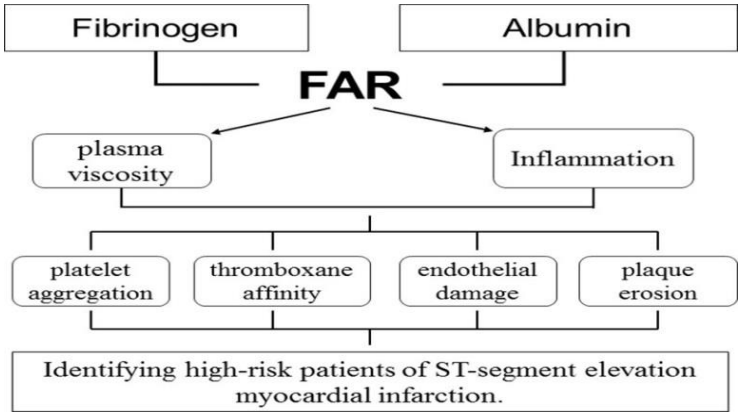
Fibrinogen is a protein in the blood that functions as an acute-phase reactant in addition to blood clotting. Furthermore, fibrin degradation products bind to platelets and increase platelet aggregation. Fibrinogen is a widely used marker and an important factor in response to systemic inflammatory and hemorheological alterations.

Fibrinogen, produced by the liver, serves as an indicator of the procoagulant state and plays a role in inflammatory responses at various levels. Fibrinogen increases thrombosis risk, increases expression of pro-inflammatory cytokines and is an independent predictor of CAD and MI. Recent clinical studies demonstrated that fibrinogen was associated with the presence and extent of atherosclerosis in patients with coronary artery disease in addition to its role in acute thrombus formation. [(1)]

Albumin is a major protein in human serum. It has many functions that can affect the cardiovascular system besides the regulation of osmotic pressure in extracellular fluid. In addition to inhibiting platelet aggregation by increasing the production of prostaglandin D2. It has been proposed that the serum albumin concentration is related to inflammatory and hemostatic processes. It was shown that decreased levels of albumin lead to an increase in blood viscosity and impaired endothelial dysfunction because of increased concentrations of free Lysophosphatidylcholine.[(2)]

Albumin has antioxidant properties and an inverse relationship with inflammation, hence a negative acute phase reactant. It was also demonstrated in some studies that low Albumin levels were associated with cardiovascular disease and increased incidence of morbidity and mortality in them. [4]

Although both high fibrinogen and low albumin levels have poor prognostic effects in cardiovascular diseases independently. The fibrinogen-albumin Ratio (FAR) may provide better prognostic information. It may form a significant biochemical parameter in determining the severity of CAD and in predicting outcomes of CAD.



CARDIAC IMAGING:

Echocardiography:

Echocardiography in the acute phase of STEMI is valuable in clarifying the diagnosis in patients presenting with non-diagnostic ECG changes. Regional wall motion abnormalities (RWMA) appear early after coronary artery occlusion.

Abnormalities of wall motion on two-dimensional echocardiography are almost universally present. Although acute STEMI cannot be distinguished from an old myocardial scar or from acute severe ischemia by echocardiography, the ease and safety of the procedure make its use appealing as a screening tool in the emergency department setting.

When the ECG is not diagnostic of STEMI, early detection of the presence or absence of wall motion abnormalities by echocardiography can aid in management decisions, such as whether the patient should receive reperfusion therapy (e.g., fibrinolysis or a percutaneous coronary intervention (PCI).

Echocardiographic estimation of left ventricular (LV) function is useful prognostically; detection of reduced function serves as an indication for therapy with an inhibitor of the RAAS. Echocardiography may also identify the presence of right ventricular (RV) infarction, ventricular aneurysm, pericardial effusion, and LV thrombus.

Coronary Angiography:

Coronary angiography comprises of visualization of the coronary anatomy under fluoroscopy, facilitated by direct injection of contrast media into the epicardial coronary arteries through a catheter advanced from a peripheral artery to the aortic root and finally into the coronary ostia.

The history of coronary angiography started in the 19th century with the discovery of X-rays by Roentgen in 1895 AD. One month later, Haschek and Lindenthal injected a mixture of calcium carbonate in the blood vessels of an amputated hand and were able to visualize the vascular bed using a roentgenogram. Selective coronary angiography was first attempted in 1958 by Mason Sones, who cannulated a right coronary artery with a catheter inserted through a brachial artery.[34] In the 1960s, angiographic studies for the determination of CAD were performed in extremely ill patients in the few tertiary care centres in the United States with the necessary resources. Coronary angiography had remained a purely diagnostic technique until 1977AD when Andreas Gruentzig performed the first percutaneous transcatheter coronary angioplasty.

Coronary Angiography Technique:

Patient Preparation:

Patients should receive a comprehensive explanation of the diagnostic angiographic procedure and of the coronary intervention potentially required. The risks of angiography should be discussed in-depth and weighed against both the clinical benefit and the risks related to refusal of the procedure. Patients are required to provide written informed consent before coronary angiography. Women of childbearing age should be questioned on their pregnancy status and advised on the additional risks of radiation exposure for pregnant women. In the event of an emergency procedure, as with a STEMI presentation, a brief evaluation of the patient history with particular attention to known chronic kidney disease (CKD) and known allergies to contrast media should be obtained.

Access Sites:

Possible access sites for coronary angiography are the femoral artery and the radial artery. Although the radial access approach is associated with fewer vascular and bleeding complications, femoral access remains the most common as it allows for larger-diameter equipment that could be necessary in case of PCI. In addition, accessing the femoral artery usually grants a more straightforward advancement of the catheter to the aortic root due to the lack of tortuosity in the descending aorta. Radial access should always be considered first, before resorting to the femoral approach, especially for diagnostic coronary angiography. [35]

Basic Technique:

Coronary angiography is an invasive procedure based on the intravascular advancement of angiographic guidewires and catheters from percutaneous access using the Seldinger technique. After a valved sheath is inserted into the access site artery, a

flexible metallic J-tipped guidewire is inserted through the sheath and advanced slowly under fluoroscopic imaging through the arterial axis until the aortic root is reached. A fluid-filled catheter is then advanced over the angiographic guidewire, while the wire itself is maintained in place.

Once the catheter is in the aortic root, the wire is fully extracted from the sheath, and the catheter is flushed and connected to the contrast media injection apparatus. Under fluoroscopic imaging, and with the help of small injections of contrast, the coronary ostium is engaged with the tip of the catheter. At this point, the x-ray tube is positioned appropriately, and angiographic images are obtained while injecting contrast directly into the cannulated coronary artery.

Selective coronary angiography is almost always performed during cardiac catheterization and is used to define the coronary anatomy and determine the extent of epicardial coronary artery and coronary artery bypass graft disease. Specially shaped coronary catheters are used to engage the left and right coronary ostia. Hand injection of radiopaque contrast agents creates a coronary "luminogram" that is recorded as radiographic images (cine angiography). Because the coronary arteries are three-dimensional objects that are in motion with the cardiac cycle, angiograms of the vessels using several different orthogonal projections are taken to best visualize the vessels without overlap or foreshortening.

The normal coronary anatomy is highly variable between individuals, but, in general, there are two coronary ostia and three major coronary vessels—the left anterior descending, the left circumflex, and the right coronary arteries with the left anterior descending and left circumflex arteries arising from the left main coronary artery When the right coronary artery is the origin of the atrioventricular nodal branch, the posterior descending artery (PDA), and the posterior lateral vessels, the circulation is defined as

right dominant; this is found in ~85% of individuals. When these branches arise from the left circumflex artery, as occurs in ~5% of individuals, the circulation is defined as left dominant. The remaining ~10% of patients have a codominant circulation with the posterior descending vessel arising from both the right coronary and the posterior lateral vessels from left coronary circulation. In some patients, a ramus intermedius branch arises directly from the left main coronary artery that trifurcates into the left anterior descending, ramus, and circumflex arteries; this finding is a normal variant.

Coronary artery anomalies occur in 1–2% of patients, with separate ostia for the left anterior descending and left circumflex arteries being the most common (0.41%).

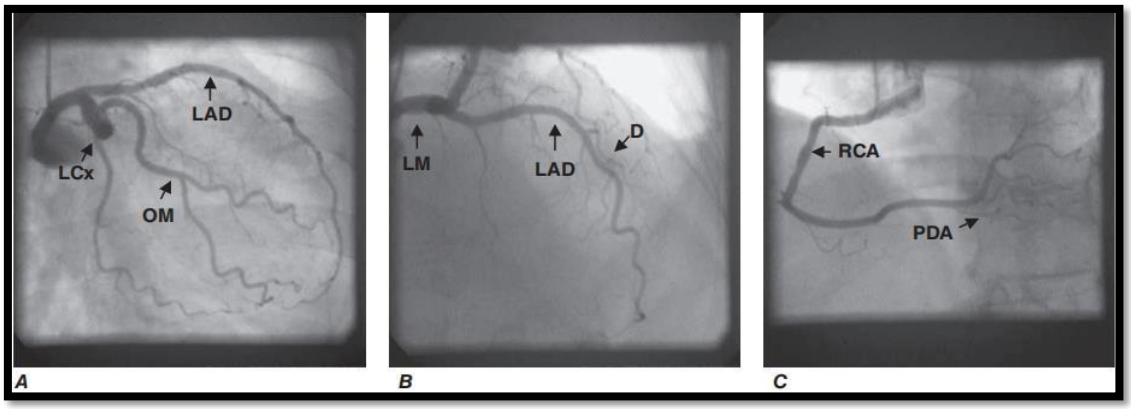


Figure 11: **Normal coronary artery anatomy**.

a) Coronary angiogram showing the left circumflex (LCX) artery and its obtuse marginal (OM) branches. The left anterior descending (LAD) artery is also seen but may be foreshortened in this view.
b) The LAD and its diagonal branches are best seen in cranial views. In this angiogram, the left main coronary artery is also seen.
c) The right coronary artery (RCA) gives off the posterior descending artery (PDA), so this is a right dominant circulation.

Angiogram Evaluation:

When reading coronary angiograms, the entire extension of every coronary artery and its branches should be carefully evaluated in all the acquired views.

First, the coronary dominance can be assessed. Next, the presence of abnormalities within the coronary arteries should be investigated.

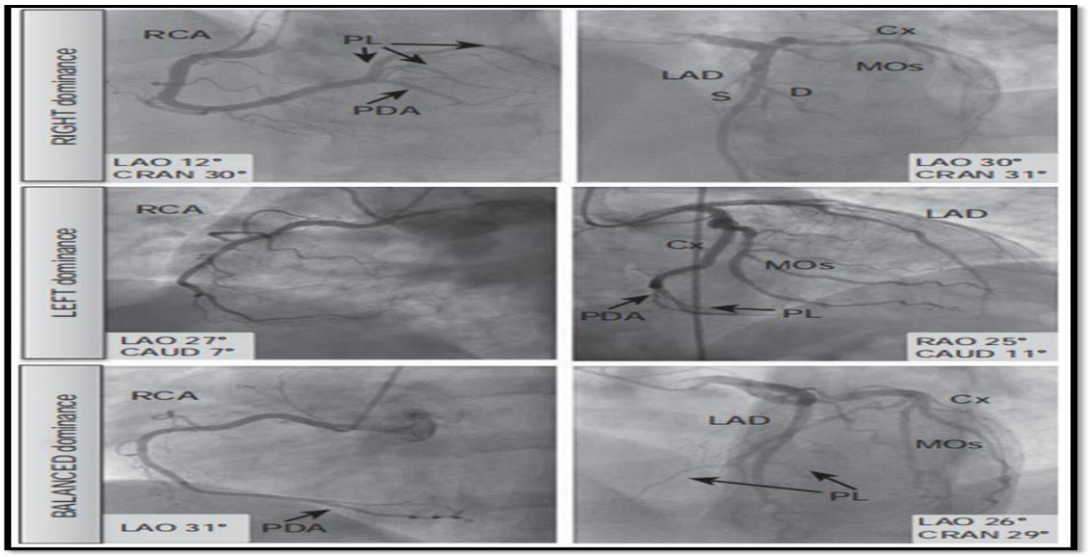


Figure 12: Coronary artery dominance. Upper panels- Example of right coronary dominance. **Middle panels**- Left coronary dominance. **Bottom panels**- Balanced dominance. LAD-Left anterior descending artery; Cx-circumflex artery; RCA-right coronary artery; D- diagonal branches; S-septal branches; OM, obtuse marginal branches; PDA, posterior descending artery, PL-posterolateral branches.[36]

The following elements should be part of the evaluation of diseased coronary vessels:

1. extension and localization of the lesion,
2. severity of the stenosis,
3. morphologic characteristics of the lesion,
4. evaluation of the downstream flow,
5. presence of collateral and changes compared to previous angiograms, if available.

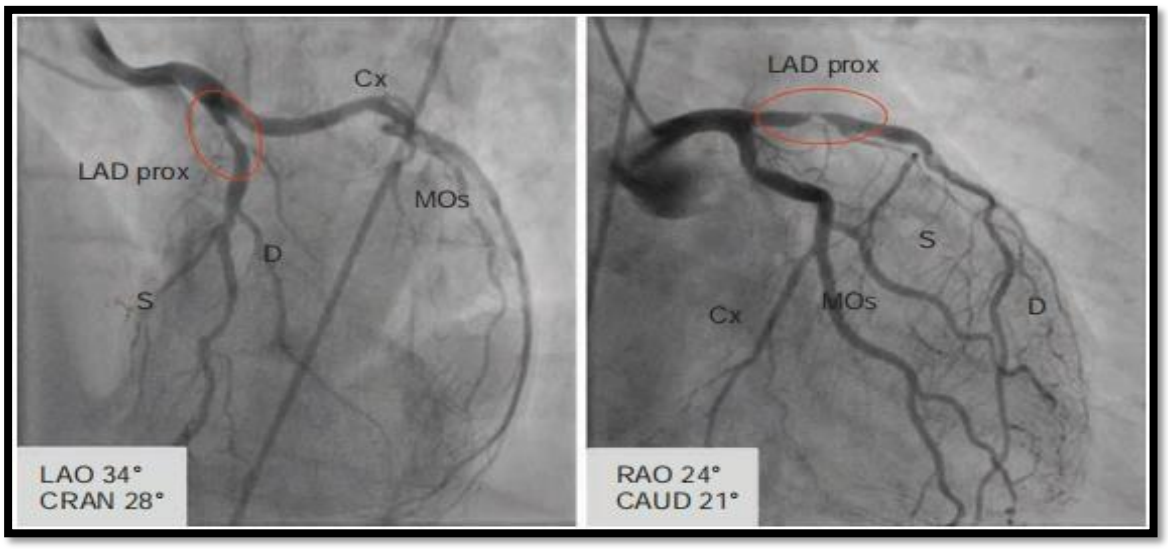


Figure 13: Example of eccentric coronary lesion in the proximal LAD, uncovered with the use of different angiographic projections. LAD prox-proximal segment of a left anterior descending artery; Cx-circumflex artery; D-diagonal branch(es); S-septal branch(es); MO- obtuse marginal branches. (LAO- Left Anterior Oblique, RAO-Right Anterior Oblique, CRAN- Cranial view, CAUD-Caudal view) [36]

Quantification of the Stenosis: A coronary stenosis is a reduction of the calibre of the vessel that is not caused by the progressive thinning of the vessel along its course but rather by pathologic local conditions. The degree of stenosis can be evaluated by comparing the minimum diameter of the vessel at the level of the lesion to the diameter of the adjacent segment upstream of the stenosis.

Stenoses are defined as minimal if the narrowing is less than 50%, moderate between 50% and 70%, and severe or significant for a diameter reduction of 70% or more.54 Evaluation of stenosis severity can be estimated visually by the interventional cardiologist reading the angiogram, or it can be measured with quantitative coronary angiography (QCA) methodologies based on the selection of the area of interest and vessel diameter measurements, which can be automatic, semiautomatic, or manual.

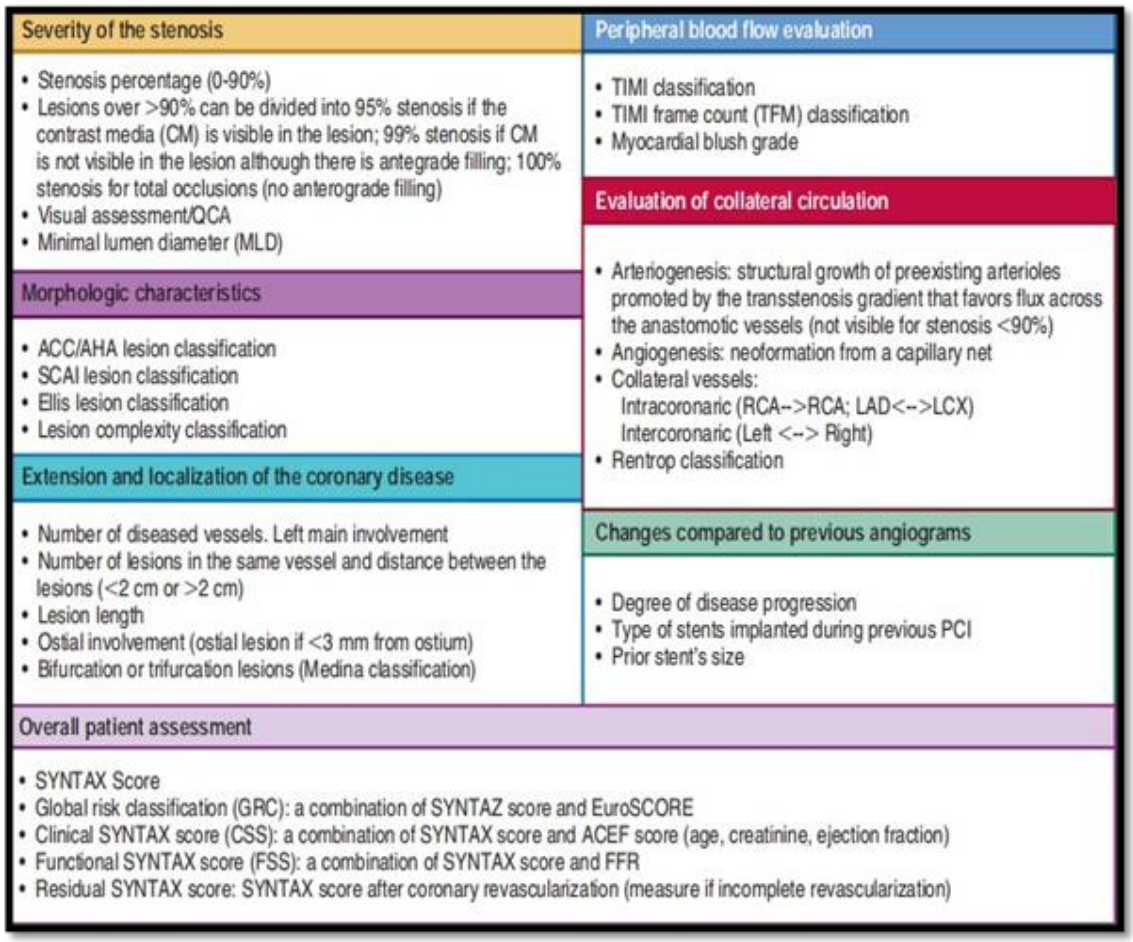


Figure 14: Evaluation of coronary stenosis. [36]

Myocardial Bridge:

Myocardial bridging is not a coronary lesion per se, the presence of a myocardial bridge, which most commonly involves the LAD, may be mistaken for a significant stenosis; this occurs when a portion of the vessel dips below the epicardial surface into the myocardium and is subject to compressive forces during ventricular systole. The key to differentiating a myocardial bridge from a fixed stenosis is that the "stenosed" part of the vessel returns to normal during diastole. Although bridging is not thought to be of any hemodynamic significance in most cases, myocardial bridging has been associated with angina, arrhythmia, depressed LV function, myocardial stunning, early death after cardiac transplantation, and sudden cardiac death (SCD).[37] Coronary calcification is also seen during angiography prior to the injection of contrast agents.

Collateral blood vessels may be seen traversing from one vessel to the distal vasculature of a severely stenosed or totally occluded vessel.

Gensini scoring system:

The GS system is a technique developed by Gensini et al. [38] for the assessment of the severity of CAD. This scoring system is based on the artery morphology, coronary anatomy, and severity of stenosis in lesions. [39] A strong association of GS was observed with long and short- term cardiovascular risk and is a simple, feasible scoring system for assessing the severity of CAD. [40]

The advantages of this scoring method are as follows:[40]

1. it provides an accurate stratification of patients according to the functional significance of their disease.
2. it lends itself to computer elaboration, storage, retrieval, and analysis.
3. it provides an opportunity to match patients with similar degrees of coronary artery disease who are receiving different forms of treatment.
4. it allows for continuous, microprocessor-assisted studies of inter-observer and intra-observer variability.

Treatment:

1. Early triage and pre-hospital management of STEMI.
2. Pre-determined hospital destination protocols.
3. Timely establishment of reperfusion.

Early triage and pre-hospital management of STEMI:

Efficient protocols of early triage of patients with STEMI should primarily aim to reduce time delays in patient care since these are associated with adverse outcomes. Morbidity and mortality due to acute STEMI can be considerably reduced if symptoms

are recognized early, allowing timely access to emergency medical services (EMS) and the institution of appropriate therapeutic measures. Since approximately one-third of patients with STEMI have symptoms other than chest pain, healthcare providers need to educate the public that the presence of any of the following symptoms (especially in a patient with a history of cardiac disease) should raise suspicion, prompting a call for medical help.

Immediate adjunctive treatment:

Nitrates	Decrease anginal symptoms by inducing coronary vasodilation and improving myocardial O2 supply and by decreasing myocardial O2 demand by decreasing preload through vasodilatation.
Beta-blockers	Decrease myocardial O2 demand by decreasing heart rate and contractility; in addition, it also contributes to electrical stability.
Calcium channel blockers	Decrease myocardial O2 demand by decreasing heart rate and contractility, decreasing wall stress via decreased blood pressure, and decreasing preload via vasodilatation.
Morphine	Reduces myocardial oxygen demands by decreasing chest pain and anxiety.
Oxygen	Improves oxygen supply in patients with hypoxemia.

TABLE 1: Drugs used as an immediate adjunctive treatment in acute MI.

Antiplatelet therapy:

Aspirin	Prevents further thrombus formation by inhibiting platelet synthesis of thromboxane A2, an important mediator of platelet activation
ADP receptor blockers	Inhibit ADP-mediated activation of platelets, thereby preventing expansion of the existing thrombus; have superior outcomes when used in combination with Aspirin
GP IIb/IIIa inhibitors	Potent antiplatelet agents that block the final common pathway of platelet aggregation; are often used in patients undergoing PCI as they are very effective in reducing cardiac events in these patients

TABLE 2: Antiplatelet drugs used in AMI.

Anticoagulant therapy:

Heparin (UFH/LMWH)	preferentially bind to antithrombin III and factor Xa, respectively, slow thrombin formation and impede clot development

TABLE 3: Anticoagulant drugs used in AMI.

Pre-determined hospital destination protocols:

A set of pre-defined instructions should be made available for the EMS personnel that guide them to appropriate hospitals designated for STEMI care. Pre-decided destination protocols are important to avoid the predicament of EMS personnel being forced to bear the responsibility of deciding where to take a patient for care. Destination protocols need to be formulated with the close involvement of emergency physicians and cardiologists. Local physicians should have a list and contact details of nearby PCI-capable centres/non-PCI hospitals so that fast-track transfers can be organized and further plans of action can be discussed with patients and cardiologists.

Timely establishment of reperfusion:

There are currently three reperfusion strategies recommended worldwide.

- Thrombolysis
- Primary PCI
- Pharmaco-invasive (PI) strategy

Thrombolysis/Fibrinolysis:

Recombinant tissue-type plasminogen activators (tPA, rPA and TNK-tPA)	Transform the inactive precursor plasminogen into the active protease plasmin, which lyses fibrin clots, thereby accelerating lysis of the occlusive intracoronary thrombus and restoring blood flow.

TABLE 4: Thrombolytic agents used in STEMI.

Primary percutaneous coronary intervention (PCI):

Plain old balloon angioplasty (POBA)	Inflation of a balloon within a stenosed coronary artery mechanically dilates the affected vessel to restore blood flow, both by compressing the atherosclerotic plaque and stretching the underlying media.
Bare metal stents	Mechanically maintain the patency of coronary arteries occluded by atherosclerotic plaques
Drug-eluting stents (DES)	In addition to maintaining patency, these stents release antiproliferative agents such as sirolimus or paclitaxel, which prevent neointimal proliferation, thereby decreasing the rate of in-stent restenosis.

TABLE 5: Various modes of angioplasty with stents.

Pharmaco-invasive (PI) strategy

The PI strategy consists of early thrombolysis followed by either rescue PCI for patients with failed thrombolysis, or non-urgent coronary angiography to determine the need for additional revascularization within 3–24 hr. [41]

Initial timely thrombolysis to open the infarct-related artery and early PCI, if required, to improve the patency rates, is an attractive option for reperfusion in STEMI and has gained momentum recently. It differs from a 'facilitated' approach, which consists an immediate PCI following fibrinolysis and has shown adverse outcomes. [42]

PCI performed 3 h after thrombolysis precludes the early pro-thrombotic phase and reduces the chances of re-occlusion. Furthermore, this delay may also be the reason for the decrease in bleeding complications that were seen with the facilitated approach. Multiple studies have subsequently shown that this strategy reduces the rate of re-infarction and is superior to the widely prevalent approach of thrombolysis followed by catheterization only for demonstrable ischemia. Most PI trials were performed using Tenecteplase.

The recent STREAM data and the Indian data from the STEPP AMI study showed that the PI strategy compared well with primary PCI in reducing overall morbidity and mortality. [43,44] Hence, the PI strategy is appropriate for patients with STEMI who are eligible for treatment with thrombolytic drugs and in whom First Medical Contact (FMC) to balloon time is ≥120 min.

Current STEMI guidelines from the American College of Cardiology/American Heart Association (ACC/AHA) and the European Society of Cardiology (ESC) also recommended that patients going to a non-PCI-capable hospital should receive fibrinolysis immediately if the expected FMC to device time is more than 120 min, and then be transferred to a PCI- capable hospital within 24 h for coronary angiogram and if needed PCI.

All patients with STEMI should be admitted to intensive coronary care units (ICCU). Though the initial emphasis is on reperfusion therapy, adjunct pharmacotherapy is no less important. Also, the routine investigations and discharge medications need to be optimised.

METHODOLOGY

Source of Data.

Cases were selected from patients of the General Medicine and Cardiology department in BLDE (Deemed to be University) Shri B M Patil Medical College Hospital and Research Centre Vijayapura, Karnataka. who satisfied the inclusion criteria.

The duration of the study was one and a half years, with a minimum of 70 patients from May 2023 to December 2024.

Method of Collection of Data

Study design: Cross-sectional observational study.

Place of study: BLDE (Deemed to be University) Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka.

Inclusion criteria

The study included,

1. Patients admitted to the ICCU, emergency wards with STEMI
2. Written informed consent.

Exclusion criteria:

The following patients were excluded from the study

1. Patients with severe liver disease and end-stage renal disease.
2. Patients with autoimmune diseases.
3. Patients with cancer and chronic inflammatory diseases.
4. Patients with severe valvular heart disease.
5. Patients with hemorheological disorders and bleeding diathesis.

Methodology:

1. After approval from the institutional ethical committee and written informed consent, the patients were recruited for this study. All the patients with ST-segment elevation myocardial infarction after fulfilling the inclusion criteria were enrolled for the study.
2. All the patients from the Department of General Medicine and the Department of Cardiology were included.
3. The socio-demographic data (age and gender) clinical presentation and past history including hypertension, diabetes mellitus, thyroid disorders, treatment and drug history, personal history including smoking and alcohol consumption and thorough physical examination with height, weight, BMI, pulse rate, blood pressure, jugular venous pressure and systemic examination were done.
4. A baseline 12 lead ECG was done to look for ST-T changes and interpreted. An initial diagnosis of STEMI is made as per criteria of "Fourth Universal Definition of Myocardial Infarction" 2018 ESC/ACC/AHA/WHF. which includes the following.
    a. A new ST elevation at the J point in 2 contiguous leads with the cut-off point >=1mm in all leads other than V2 and V3.
    b. For leads V2, V3 the cut-off points apply: >=2mm in men >=40years; >=2.5mm in men <40years, or >=1.5mm in women regardless of age.
5. Complete blood count, random blood glucose, Creatinine, serum albumin, and serum fibrinogen were analysed.

6. 2-Dimensional Echocardiography was performed to look for RWMA and left ventricular ejection fraction (LVEF).
7. PCI procedure was performed for the patients without contra-indications, which is the diagnostic as well as therapeutic procedure for acute ST-segment elevation myocardial infarction.
8. GS was calculated based on the coronary angiographic findings, coronary lesions leading to diameter stenosis in vessels were scored separately and added together to provide cumulative GS as interpreted.

Outcomes of the study.

1. Using the above assessment tools data was interpreted and fibrinogen-albumin ratio (FAR) was calculated as: fibrinogen (g/L)/albumin (g/L).
2. The primary end point was to measure the severity of coronary artery disease using GS system and correlate with FAR.

SAMPLE SIZE ESTIMATION

Sample size:

- With the Anticipated correlation between FAR and GENSINI score based on coronary angiography -0.38 from the study by Lior Charach et al. Using these values at a 95% confidence level and 90% power in the study, the sample size was 70.
- The formula used is

  $N=[(Z_{\alpha}+Z_{\beta})/C]^2+3$
- The standard normal deviate for $\alpha = Z_{\alpha} = 1.9600$
- The standard normal deviate for $\beta = Z_{\beta} = 1.0364$
- $C=0.5*\ln[(1+r)/(1-r)] =0.4001$
- N=70

Statistical methods:

- The data obtained was entered into a Microsoft Excel sheet, and statistical analysis will be performed using Pro 16 and the statistical package for the social sciences (Version 20).
- Association between Categorical variables will be computed using the Chi-square test.
- The correlation coefficient was used to find the correlation between quantitative variables. $p<0.05$ was considered statistically significant. All statistical tests will be performed in two-tailed.

RESULTS

Age	Frequency	Percent
<40yrs	2	2.86
40-60yrs	28	40
>60yrs	40	57.14
Total	70	100

Table 6: - Distribution of subjects according to age group.

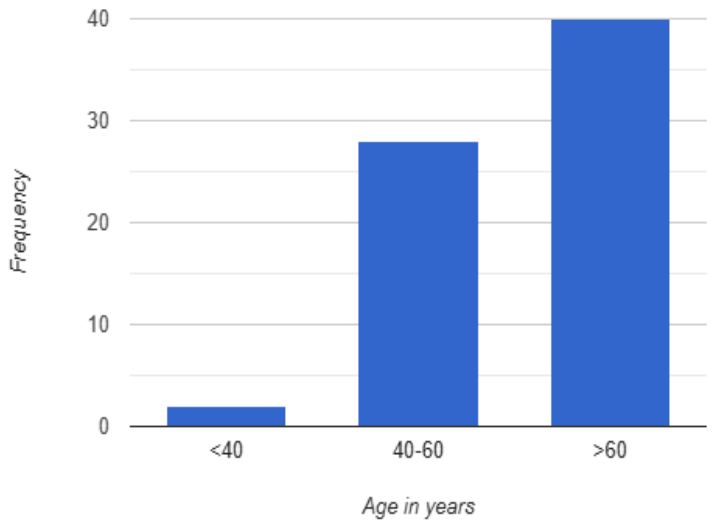


Figure 15: - Graph showing subjects according to age group.

Sex	Frequency	Percent
Male	47	67.1
Female	23	32.9
Total	70	100.0

Table 7: - Distribution of subjects according to sex.

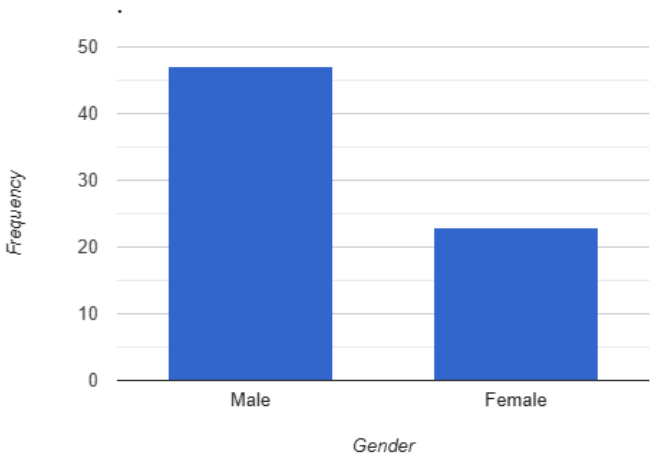


Figure 16: - Graph showing subjects according to gender.

BMI	Frequency	Percent
Underweight (<18.5)	3	4.28
Normal (18.5-22.9)	25	35.70
Overweight (23.0-24.9)	13	18.60
Obese 1 (25.0-29.9)	22	31.42
Obese 2 (≥30.0)	7	10
Total	70	100

Table 8: - Distribution of subjects according to BMI.

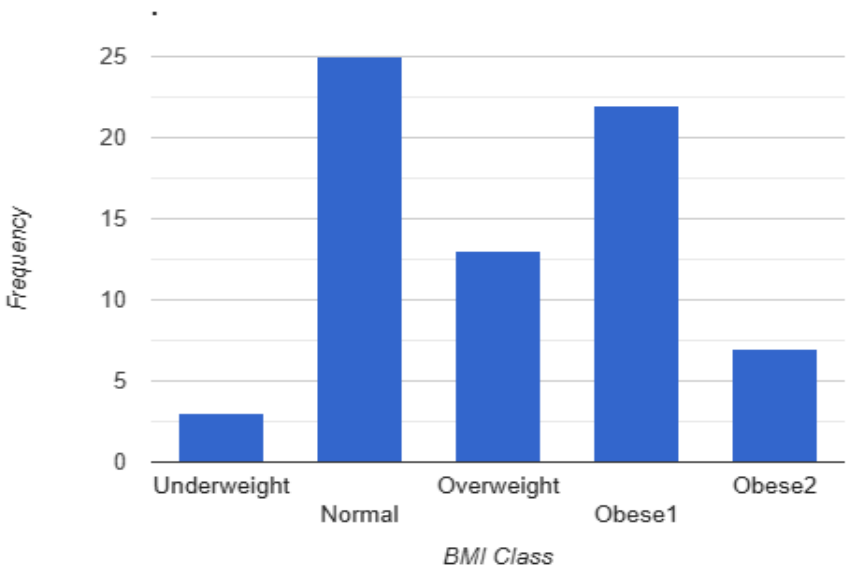


Figure 17 : - Graph showing subjects according to BMI.

Risk factors	Frequency	Percent
Hypertension	23	32.86
Diabetes	21	30
Diabetes and Hypertension	09	12.9
Smoking	17	24.28
Tobacco	17	24.28
Alcohol	07	10
Overweight	13	18.57
Obese	29	41.43

Table 9: - Frequency distribution of co-morbidities and risk factors.

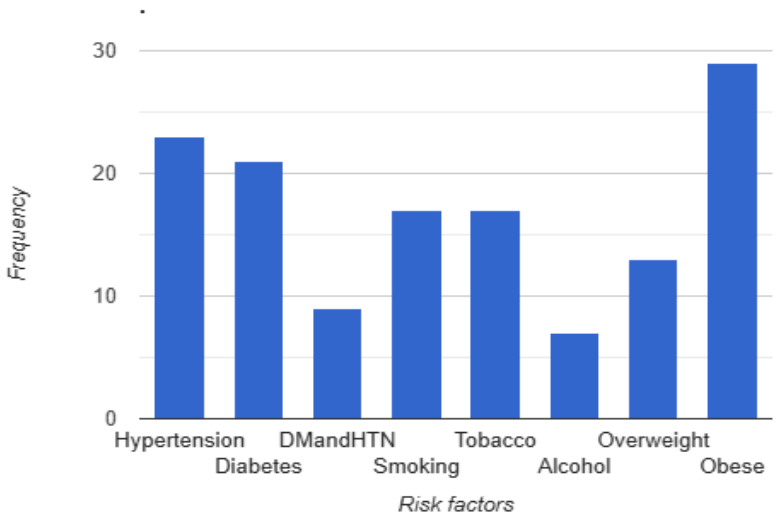


Figure 18: - Graph showing the distribution of risk factors.

Myocardial infarction	Frequency	Percent
Anterior wall	38	54.28
Inferior wall	26	37.14
Lateral wall	1	1.44
Posterior wall	5	7.14
Total	70	100.0

Table 10: - Distribution of subjects according to location of Myocardial infarction.

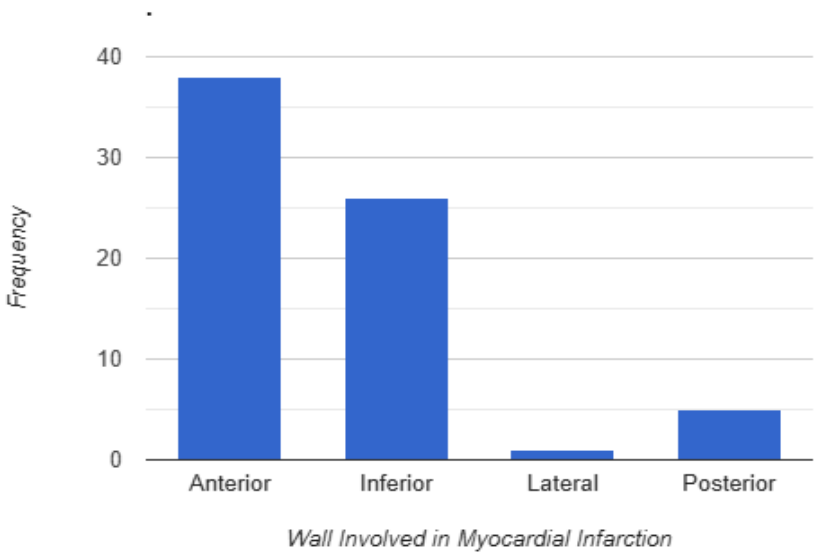


Figure 19: - Graph showing subjects according to location of Myocardial infarction.

CAD- Different vessels involved	Frequency	Percent
SVD	24	34.3
DVD	24	34.3
TVD	20	28.6
RECANALIZED	2	2.8
Total	70	100.0

Table 11: - Distribution of subjects according to CAD.

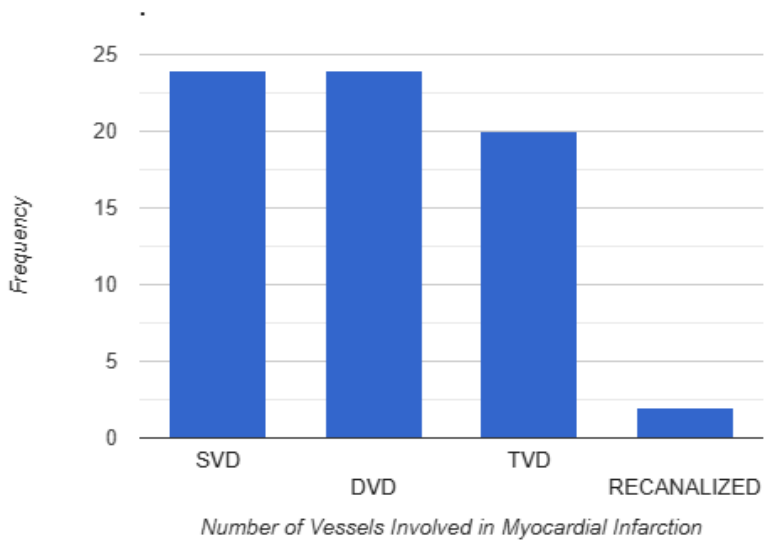


Figure 20: - Graph showing subjects according to CAD.

FAR	FREQUENCY	PERCENT
<0.05	4	5.72
0.05-0.1	29	41.42
>0.1	37	52.86
TOTAL	70	100

Table 12: Distribution of subjects according to FAR

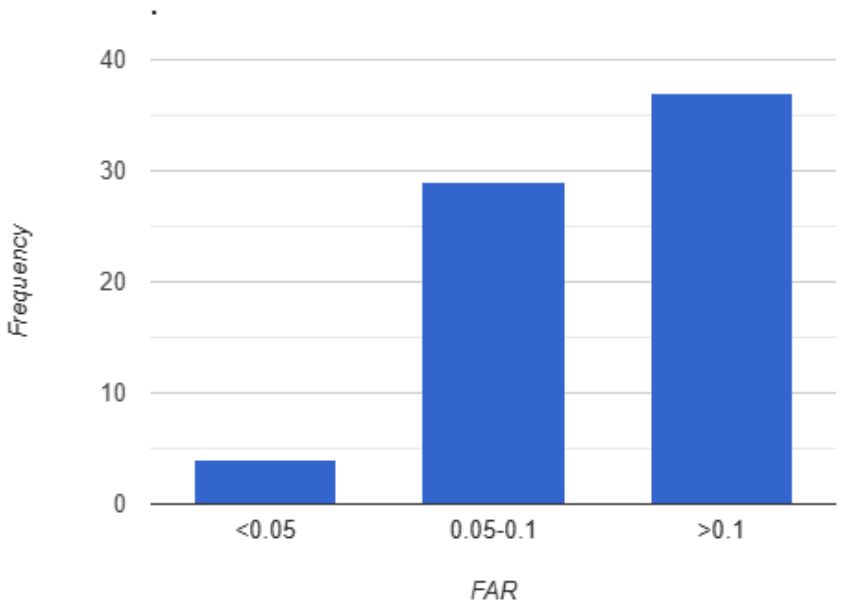


Figure 21: Graph showing subjects according to FAR

		Frequency	Percent
ADVERSE OUTCOME	HFrEF (<40)	41	58.6
	HFmrEF (41-50)	21	30
	HFpEF (>50)	8	11.4
	Total	70	100.0

Table 13: Distribution of subjects according to Heart failure.

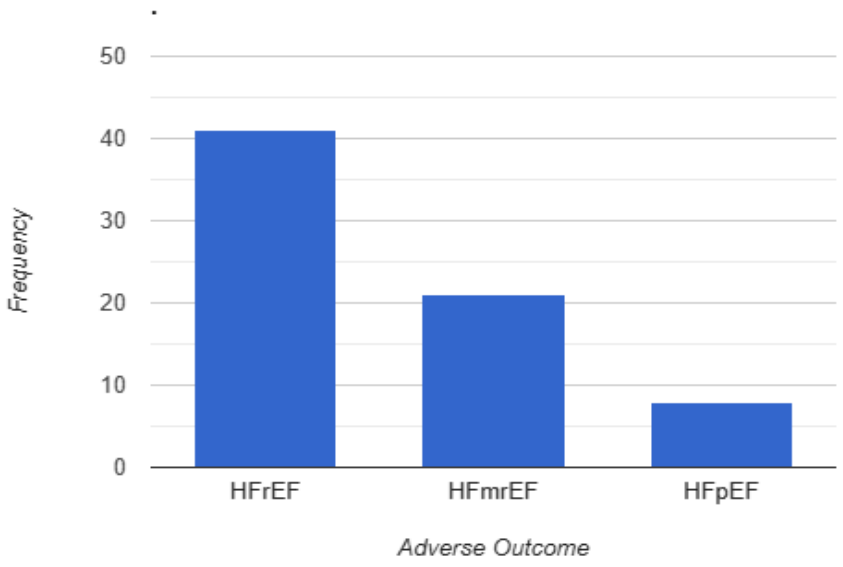


Figure 22: Graph showing subjects according to Heart failure.

		HFrEF	HFmrEF	HFpEF	TOTAL
GS	MILD (<30)	8	5	13	26
	MODERATE (30-60)	7	2	18	27
	SEVERE (>60)	6	1	10	17
TOTAL		21	8	41	70

Table 14: Correlation of Gensini score with Heart failure.

NO. OF VESSEL INVOLVED	GENSINI SCORE			TOTAL
	MILD	MODERATE	SEVERE	
SVD	15	6	3	24
DVD	8	13	3	24
TVD	1	8	11	20
RECANALISED	2	0	0	2
TOTAL	26	17	17	70

Table 15: Correlation of Gensini score with Number of Vessels involved.

		HEmrEF	HFpEF	HFrEF	TOTAL
FAR	<0.05	0	1	3	4
	>0.1	8	6	23	37
	0.05-0.1	13	1	15	29
Total		21	8	41	70

Table 16: Correlation of FAR with Heart failure.

		FIBRINOGEN	ALBUMIN	FAR
GS	R	-.032	-.164	-.002
	P	.790	.174	.989
	N	70	70	70

Table 17: Correlation of Fibrinogen, Albumin with Gensini score.

DISCUSSION

Coronary artery disease is a chronic inflammatory state; there is an ongoing inflammation throughout the pathological course of atherosclerosis. Various inflammatory markers are associated with the pathogenesis of the disease and play a significant role in predicting the outcome.

A particularly interesting molecule to assess the onset and progression of coronary atherosclerosis is plasma fibrinogen, an acute-phase reactant. Elevated fibrinogen levels were identified as a risk factor for coronary artery disease by the Atherosclerosis Risk in Communities (ARIC) research. However, compared to other well-established risk variables, their evaluation in healthy persons seems to add little to the ability to predict coronary events. [45]

Although serum albumin, a negative acute phase reactant, has been demonstrated to have anti-inflammatory properties, it is also implicated in acute inflammatory reactions like STEMI. Reperfusion damage and worsened ischaemia can be brought on by hypoalbuminemia. The two main components of whole-blood viscosity albumin and fibrinogen combine to provide a significant biomarker for cardiovascular disease. [46, 47]

Therefore, a high fibrinogen-to-albumin Ratio (FAR) results in an increased risk of ischemic events such as ST elevation or non-ST elevation myocardial infarction. [48]

ST-segment elevation myocardial infarction (STEMI) is one of the standard forms of acute myocardial infarction (AMI). Percutaneous coronary intervention (PCI) is performed as soon as possible to recanalize the infarct-related coronary artery, which has been recommended as the preferred method of reperfusion. [49]

The care of STEMI has advanced significantly in developed nations because of an increase in the percentage of patients receiving primary PCI within a window period, which lowers mortality and complications. However, it is still difficult to manage STEMI effectively in poorer nations because of a lack of resources and PCI-Centre availability. Therefore, a variety of biomarkers have been employed to diagnose coronary artery disease early and avoid the onset of STEMI. One such biomarker is the new, non-invasive test called FAR.

The mean age of the 70 patients who were enrolled in this study ranged from 33 to 78 years of age was 70 years. The majority of instances, 40 (57.14%), occurred in people over 60, followed by people between 40 and 60 years 28 (40%), and those under 40 were 2 (2.86%). There were 47 men and 23 women in this study, making up 67.1% and 32.9% of the total participants, respectively. As a result, there were more men than females.

A total of 78 STEMI patients with a mean age of 61.5 years were included in a study by **Oguz Karahan et al.** in 2016. Most of the cases in their study were males, about 73%, and 27% were females. Similar to our study, the majority of the cases were males. [50]

In our study, 34 (48.57%) patients out of 70 had a history of smoking and tobacco chewing, 23 (32.85%) of them had hypertension, and 21 (30%) of them had diabetes mellitus. Based on body mass index (BMI), patients were classified as obese1, obese2, overweight, normal weight and underweight. About 13 (18.6%) of them were overweight, and 29 (41.4%) were obese, which forms significant risk factors for developing coronary artery disease.

In a study by **F. Rasool et al.** To ascertain the prevalence of STEMI risk variables, 400 patients with STEMI were included in total in 2013. Similar to our study, it was observed that smoking was the most common risk factor (51.5%), followed by diabetes mellitus (28%) and hypertension (20.5%) [51].

An investigation by **Ajay Kumar Sharma et al.** To ascertain the ECG alterations and correlate the results of coronary angiography, 201 STEMI patients were enrolled in 2021. About 117 patients, or 58.2% of the total, experienced anterior wall myocardial infarction (AWMI). In contrast, inferior wall myocardial infarction (IWMI) occurred in 84 (41.8%) of the patients. According to coronary angiography, 34 patients (29.1%) had multivessel disease, which includes either double vessel disease (DVD) or triple vessel disease (TVD), while 83 patients (70.9%) had single vessel disease (SVD). [52]

In our study, 38 (54.3%) have AWMI, followed by IWMI 26(37.1%), 5 (7.1%) have PWMI, and 1 (1.4%) had LWMI. Coronary angiographic findings obtained after PCI showed that 24 (34.3%) patients had SVD, which is less commonly involved as compared with the above-mentioned study,44 (62.9) had multivessel disease in which 24 (34.3%) patients had DVD and 20 (28.6%) had TVD.

This study's small sample size, which is one of its limitations, and the fact that there aren't many studies of the Indian population could be the reason for the statistical insignificance of the lack of correlation between the FAR and the Gensini score with the Pearson correlation ($r = -0.002$, $P = 0.989$).

Strengths of the study:

- Fibrinogen-Albumin Ratio (FAR) is a non-invasive, convenient, less time-consuming laboratory test.
- It can be quickly done in patients who are denying coronary angiographic evaluation, which is an invasive procedure in determining coronary artery disease (CAD).
- As an add-on biomarker in patients with acute coronary syndrome for early detection of CAD.

Limitations

1. This study was conducted at a single centre.
2. Only baseline fibrinogen and albumin levels were measured at admission; changes observed by serial measurements could have given better results. However, this evaluation could not be completed in our study.
3. The Gensini scoring system used in the study for scoring the severity of CAD is a simple scoring system and is observer-dependent.
4. Other complex scoring systems like SYNTAX score and GRACE score could have been used, which have more functional parameters for scoring and assessing the severity of CAD.
5. FAR is a laboratory parameter, so there can be some possible sampling errors.

CONCLUSION

In our study, it was concluded that FAR, a serum biomarker index, has a Negative correlation with the severity of coronary artery disease, and there is a positive correlation between the Gensini score and the severity of coronary artery disease. FAR was statistically insignificant in predicting the outcome.

However, some research conducted in nations other than India has produced encouraging findings. This could be because of their high sample size, which turns into one of the study's significant flaws. Because of the expense, fewer patients chose percutaneous intervention, preferring intravenous thrombolysis instead. Therefore, once coronary artery disease (CAD) is suspected, it is critical to identify at-risk patients using a variety of non-invasive biomarkers or indices in order to prevent acute ischaemic events such as STEMI or NSTEMI and to begin treatment with anti-platelets, statins, and lifestyle changes.

Researchers have found that FAR may be utilised as a predictor of hemorheological anomalies after comparing it between patients with cerebrovascular accidents and healthy individuals in earlier investigations. [53] FAR has been linked to thrombotic events and has been successful in predicting outcomes in certain malignancies. [54] In addition, FAR's accessibility and affordability make it a promising serum biomarker that is crucial for assessing haemorrhage and inflammation.

In order to elucidate the significance of FAR during STEMI, future prospective studies with a sizable sample are necessary.

SUMMARY

Over the course of 18 months, a cross-sectional study was carried out in our hospital with 70 patients who received Percutaneous coronary intervention for ST-segment elevation myocardial infarction. The institutional ethics committee granted ethical approval, and the patient's informed consent was acquired before the study began.

The study was carried out with the following objectives in mind.

- To determine the Fibrinogen-Albumin Ratio in ST-segment elevation myocardial infarction.
- To correlate the Fibrinogen-Albumin Ratio with the Gensini Score based on coronary angiography.

Results showed that.

1. In our study males 47 (67.14) were predominantly affected than (23) females.
2. In our study, 24 (34.3%) had double vessel disease (DVD), and about 24 (34.3%) had single vessel disease (SVD), 20 (28.6%) had triple vessel disease (TVD), 2 (2.9%) were recanalised.
3. In our study, 34 (48.57%) patients out of 70 had a history of smoking and tobacco chewing, which was a major risk factor. Based on body mass index (BMI), About 13 (18.6%) of them were overweight, and 29 (41.4%) were obese, which forms significant risk factors for developing coronary artery disease.
4. In our study, there was a Negative correlation between the FAR and the Gensini score with a Pearson correlation ($r = -0.002$, $P = 0.989$), which was not statistically significant.
5. In our study, there was a positive correlation between the Gensini score and the severity of coronary artery disease.

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ANNEXURE -1

ETHICAL CLEARANCE CERTIFICATE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 929/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinized the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "CORRELATION OF SERUM FIBRINOGEN TO ALBUMIN RATIO WITH GENSINI SCORE BASED ON CORONARY ANGIOGRAPHY IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.ABHIJIT HANAMANTARAYA UTNAL

NAME OF THE GUIDE: DR.SIDDANANGOUDA M.BIRADAR, PROFESSOR, DEPT. OF GENERAL MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE –2

INFORMED CONSENT FORM

BLDE (DEEMED TO BE UNIVERSITY) SHRI B M PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA, KARNATAKA.

TITLE OF THE PROJECT - *"*CORRELATION OF SERUM FIBRINOGEN TO ALBUMIN RATIO WITH GENSINI SCORE BASED ON CORONARY ANGIOGRAPHY IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION*"*

PRINCIPAL INVESTIGATOR - Dr. ABHIJIT HANAMANTARAYA UTNAL

GUIDE NAME - Dr. S M BIRADAR
PROFESSOR OF GENERAL MEDICINE

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to routine care received, the investigator will ask me a series of questions. I have been asked to undergo the necessary investigations and treatment to help the investigator in this study.

3) RISK AND DISCOMFORTS:

I understand that I may experience pain and discomfort during the examination or treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate the feelings associated with the usual course of treatment.

4) BENEFITS:

I understand that my participation in this study will help the patient's survival and better outcomes.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality and privacy regulations of the hospital.

No name will be used if the data is used for publication in the medical literature or for teaching purposes.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study time.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or withdraw consent and discontinue participation in the study at any time.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided.

_____

Signature of patient/attender

Date:

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APPENDIX –3

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B M PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA, KARNATAKA.

SCHEME OF CASE TAKING

"CORRELATION OF FIBRINOGEN-ALBUMIN RATIO WITH GENSINI SCORE IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION"

Informant:

Name:	CASE NO:
Age:	IP NO:
Gender:	DOA:
DOE:	DOD:
Address:	
Occupation:	

Chief complaints:

History of present illness:

Past History:

Personal History:

Family History:

Menstrual History:

General Physical Examination

Height:

Weight:

Body Mass Index:

Vitals

PR:

BP:

RR:

Temp:

JVP:

Head-to-toe examination:

SYSTEMIC EXAMINATION - CARDIOVASCULAR SYSTEM

Inspection of Neck-

Inspection of chest-

Palpation-

Percussion-

Auscultation-

Mitral area

Tricuspid area

Pulmonary area

Aortic area

OTHER SYSTEMS

RESPIRATORY SYSTEM:

PER ABDOMEN:

CENTRAL NERVOUS SYSTEM:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS

1. Electrocardiography

2. Serum Fibrinogen

3. Serum Albumin

4. FAR

5. Coronary Angiography

6. GENSINI score-

Date-

Signature- **Signature of guide**

6. GENSINI Scoring System (Multiplication Factor Used):

Vessel	Involved	Stenosis (%)	Score
LEFT MAIN(x5)			
LAD	Proximal (x2.5)		
	Midpart (x1.5)		
	Distal (x1)		
D1 (x1)			
D2 (x2)			
LCX	Proximal (x2.5)		
	Midpart (x2)		
	Distal (x1)		
OM1 (x1)			
OM2 (x2)			
RCA	Proximal (x1)		
	Midpart (x1)		
	Distal (x1)		

PDA (x1)		
FINAL SCORE		

Degree of Stenosis **Severity Score**

- **1 - 25%** **1**
- **26 – 50%** **2**
- **51 – 75%** **4**
- **76 – 90%** **8**
- **91 – 99%** **16**
- **100%** **32**

MASTER CHART

- **KEY**
- **SL NO. – SERIAL NUMBER**
- **HTN – HYPERTENSION**
- **DM – DIABETES MELLITUS**
- **BMI – BODY MASS INDEX**
- **IP NO. – ADMISSION NUMBER**
- **CO-MORBID – CO MORBIDITIES**
- **WALL – WALL INVOLVED IN ECG**
- **IWMI – INFERIOR WALL MYOCARDIAL INFARCTION**
- **AWMI – ANTERIOR WALL MYOCARDIAL INFARCTION**
- **LWMI – LATERAL WALL MYOCARDIAL INFARCTION**
- **PWMI – POSTERIOR WALL MYOCARDIAL INFARCTION**
- **FAR – FIBRINOGEN-ALBUMIN RATIO**
- **SVD – SINGLE VESSEL DISEASE**
- **DVD – DOUBLE VESSEL DISEASE**
- **TVD – TRIPLE VESSEL DISEASE**

SL No.	NAME	AGE	GENDER	OCCUPATION	IP NO.	FIBRINOGEN	ALBUMIN	FAR	GS	CO MORBIDS	VESSEL	WALL	HABITS	BMI
1	SHANTABAI RATHOD	70yrs	FEMALE	HOUSEWIFE	213936	5.39g/L	34g/L	0.16	16	NIL	DVD	IWMI	NIL	35.55
2	BHIMSING T RATHOD	53yrs	MALE	TAILOR	218223	2.05g/L	40g/L	0.05	29	DM, HTN	TVD	AWMI	NIL	31.14
3	LAXMIBAI NIDONI	75YRS	FEMALE	HOUSEWIFE	224594	3.59g/L	36g/L	0.1	51	HTN	TVD	AWMI	NIL	21.06
4	SHANTAWWA DODAMANI	63YRS	FEMALE	LABOUR	256651	5.14g/L	28g/L	0.18	11	DM, HTN	SVD	IWMI	NIL	24.41
5	BALAPPA KAREKAL	70YRS	MALE	FARMER	K1928	6.0g/L	33g/L	0.18	58	NIL	SVD	AWMI	NIL	30.14
6	BASAVARAJ MALI	58YRS	MALE	TEACHER	256642	2.22g/L	28g/L	0.08	26	NIL	DVD	IWMI	NIL	25.6
7	BABU KATTAPPAPILLAI	51YRS	MALE	FARMER	258658	3.44g/L	39g/L	0.09	19	DM, HTN	DVD	AWMI	NIL	26.35
8	BASAVARAJ MOPAGAR	33YRS	MALE	BUSINESS	260612	5.32g/L	40g/L	0.13	16	NIL	DVD	AWMI	SMOKE,ALCOHOL	26.56
9	RACHAPPA HADAPAD	62YRS	MALE	EX ARMY	340574	4.25g/L	30g/L	0.14	45	COPD	TVD	AWMI	SMOKE	27.77
10	IRAPPA TELASANG	65YR	MALE	DAILY WAGE	347901	1.68g/L	26g/L	0.06	100	T2DM	TVD	IWMI	NIL	22.96
11	SOMANING BAJANTRI	49YRS	MALE	BUSINESS MAN	347338	1.78g/L	48g/L	0.04	13	NIL	SVD	IWMI	TOBACCO,ALCOHOL	20.31
12	BHIMASHYA	71YRS	MALE	FARMER	180596	6.2g/L	22g/L	0.28	16	NIL	SVD	AWMI	TOBACCO,ALCOHOL	29.85
13	KASHINATH ZIPARE	78YRS	MALE	FARMER	269289	2.42g/L	30g/L	0.08	76	NIL	TVD	AWMI	SMOKE	24
14	VITTAL HIREKURUBUR	62YRS	MALE	FARMER	269961	5.12g/L	28g/L	0.18	80	NIL	SVD	AWMI	NIL	23.44
15	HUNNU RATHOD	59YRS	MALE	FARMER	276778	6.2g/L	33g/L	0.24	88	HTN	DVD	AWMI	SMOKE,ALCOHOL	25
16	ARJUN SURYAVANSHI	45YRS	MALE	SHOP KEEPER	280662	5.1g/L	36g/L	0.14	36	NIL	DVD	AWMI	SMOKE,ALCOHOL	27.85
17	SHARANAGOUDA BIRADAR	52YRS	MALE	FARMER	283568	5.06g/L	23g/L	0.22	12	DM,HTN	SVD	AWMI	NIL	34.37
18	GURUBAI SUDAM	55YRS	FEMALE	HOUSE WIFE	283630	4.45g/L	37g/L	0.12	8	NIL	SVD	IWMI	NIL	35.93
19	GOPU CHAVAN	72YRS	MALE	FARMER	288016	3.56g/L	34g/L	0.1	53	DM	SVD	AWMI	TOBACCO,SMOKE,ALCOHOL	28.24
20	REVANASIDDAPPA	69YRS	MALE	FARMER	290730	2.43g/L	33g/L	0.07	56	NIL	DVD	IWMI	SMOKE	22.06
21	SANJEEV GUDDEVADI	53YRS	MALE	TEACHER	290487	3.79g/L	34g/L	0.11	96	NIL	TVD	IWMI	NIL	28.9
22	BANUMA MULLA	66YRS	FEMALE	HOUSEWIFE	SSH00037	5.32g/L	32g/L	0.17	62	HTN,DM	TVD	AWMI	TOBACCO	21.09
23	RAMANNA MADAR	71YRS	MALE	SHEPHARD	2514	3.45g/L	34g/L	0.1	8	NIL	SVD	IWMI	NIL	19.03
24	MUNEERA JATTA	50YRS	FEMALE	FRUIT VENDOR	SSH00020	4.56g/L	37g/L	0.12	16	NIL	SVD	IWMI	NIL	33.75
25	LAKKAPPA BIJJARAGI	70YRS	MALE	FARMER	SSH00038	5.27g/L	44g/L	0.12	32	DM	DVD	AWMI	NIL	20.76
26	UMAKANTH SONNAD	73YRS	MALE	FARMER	SSH00042	4.57g/L	35g/L	0.13	36	NIL	DVD	AWMI	NIL	28.12
27	SHANTABAI JADHAV	74YRS	FEMALE	HOUSE WIFE	SSH00055	5.7g/L	22g/L	0.26	23	DM	SVD	IWMI	NIL	22.22
28	BASAPPA NARALI	75YRS	MALE	SHEPHARD	2034	2.42g/L	40g/L	0.06	8	NIL	SVD	IWMI	TOBACCO,SMOKE	19.25
29	KASTURIBAI NAVI	55YRS	FEMALE	HOUSE WIFE	2857	5.57g/L	32g/L	0.17	88	IHD DM	TVD	AWMI	NIL	22.22
30	PREMANAND SHATAGAR	54YRS	MALE	ATTENDER	2816	6.0g/L	34g/L	0.17	51	NIL	TVD	IWMI	TOBACCO,SMOKE	20.76
31	SHARANAYYA HIREMATH	70YRS	MALE	SAINT	2866	4.03g/L	32g/L	0.12	39	NIL	TVD	AWMI	NIL	20.31
32	RENUKA KAMBLE	50YRS	FEMALE	HOUSEWIFE	2614	6g/L	30g/L	0.2	32	HTN	SVD	IWMI	TOBACCO	31.66
33	REVANSIDDA JOGUR	64YRS	MALE	BUSINESS MAN	3438	6g/L	34g/L	0.18	56	HTN CVA	DVD	AWMI	NIL	25.89
34	MALAMMA KUMBAR	52YRS	FEMALE	FARMER	3487	4.37g/L	44g/L	0.1	20	NIL	SVD	AWMI	NIL	23.91
35	LAXMIBAI GANGASETTY	51YRS	FEMALE	HOUSEWIFE	4068	6g/L	41g/L	0.15	50	DM	DVD	AWMI	NIL	27.34

36	GANGADHAR S MASHYAL	36YRS	MALE	FARMER	4742	4.16g/L	45g/L	0.09	24	HTN	SVD	AWMI	NIL	22.43
37	ALLAMMA KALADAGI	60YRS	FEMALE	HOUSE WIFE	4463	2.43g/L	40g/L	0.06	81	NIL	TVD	AWMI	TOBACCO	22.22
38	SIDDARAM WALI	56YRS	MALE	FARMER	4726	3.43g/L	44g/L	0.08	12	HTN	DVD	AWMI	NIL	24.22
39	BASAVARAJ PYATI	61YRS	MALE	BUSINESSMAN	6467	6.0g/L	38g/L	0.16	50	NIL	SVD	AWMI	NIL	25
40	NANDABASAPPA GANIGER	75YRS	MALE	FARMER	6556	3.63g/L	58g/L	0.06	36	DM HTN	DVD	IWMI	SMOKE	24.22
41	GANGABAI S BIRADAR	60YRS	FEMALE	FARMER	6606	5.3g/L	39g/L	0.13	73	DM HTN	TVD	AWMI	NIL	23.8
42	SOMANAGOUDA S PATIL	72YRS	MALE	BUSINESSMAN	6962	2.2g/L	37g/L	0.06	3	DM	RECAN L	IWMI	SMOKE	22.97
43	YALLAVVA ADIN	57YRS	FEMALE	FARMER	6986	2.2g/L	27g/L	0.08	46	NIL	DVD	IWMI	NIL	26.72
44	SHAKIL NADAF	48YRS	MALE	BROKER	6987	4g/L	48g/L	0.08	50	NIL	DVD	IWMI	TOBACCO	25.73
45	SAKARAM JADHAV	75YRS	MALE	FARMER	7339	4.7g/L	25g/L	0.18	38	NIL	DVD	IWMI	TOBACCO,SMOKE	17.78
46	SHANKARGOWDA S N	65YRS	MALE	SHOPKEEPER	7382	5.2g/L	35g/L	0.15	57	DM	TVD	IWMI	TOBACCO	23.3
47	SHEVU RATHOD	75YRS	MALE	FARMER	7895	2.55g/L	17g/L	0.15	89	DM	TVD	IWMI	TOBACCO,SMOKE	14.5
48	KAMRUNNISA GOLASANGI	65YRS	FEMALE	HOUSEWIFE	8405	1.70g/L	46g/L	0.04	15	DM	DVD	IWMI	NIL	27.34
49	BHIMSING RATHOD	55YRS	MALE	FARMER	8248	1.31g/L	30g/L	0.04	20	DM	SVD	AWMI	SMOKE	25.6
50	YANKAVVA METI	52YRS	FEMALE	HOUSEWIFE	8427	3.02g/L	31g/L	0.1	96	HTN	SVD	AWMI	NIL	17.5
51	HALIMA MULLA	64YRS	FEMALE	HOUSEWIFE	8470	2.91g/L	26g/L	0.11	42	HTN	SVD	AWMI	NIL	27.16
52	RAMU NAMANE	45YRS	MALE	LABOUR	8562	3.14g/L	16g/L	0.2	5	NIL	RECANA	AWMI	NIL	21.45
53	MALLAMMA GULED	49YRS	FEMALE	HOUSEWIFE	8764	2.32g/L	30g/L	0.08	38	NIL	SVD	AWMI	NIL	22.05
54	AMEERABI GOUNDI	74YRS	FEMALE	HOUSEWIFE	9197	1.22g/L	29g/L	0.04	39	HTN	DVD	IWMI	TOBACCO	21.39
55	SHANKREPPA KERUR	79YRS	MALE	FARMER	9992	6.0g/L	17g/L	0.35	43	DM	TVD	AWMI	NIL	21.27
56	RUKMAVVA WALIKAR	74YRS	FEMALE	HOUSEWIFE	10103	4.58g/L	35g/L	0.13	92	NIL	DVD	AWMI	NIL	26.97
57	GURUBASAPPA BILIJADAR	58YRS	MALE	FARMER	11199	4.11g/L	39g/L	0.1	58	NIL	TVD	AWMI	TOBACCO	22.83
58	MAHADEVI NAVADAGI	65YRS	FEMALE	HOUSEWIFE	11324	1.88g/L	30g/L	0.06	84	DM, HTN	TVD	IWMI	NIL	21.66
59	SANGAPPA BADRAGONDA	65YRS	MALE	FARMER	12816	2.66g/L	29g/L	0.09	16	NIL	SVD	IWMI	NIL	21.69
60	GIREPPA K K	70YRS	MALE	FARMER	13682	1.5g/L	30g/L	0.05	46	NIL	TVD	IWMI	NIL	20.61
61	SHIVANAGOUDA PATIL	65YRS	MALE	BUSINESSMAN	7671	1.5g/L	28g/L	0.05	5.5	DM, HTN	SVD	IWMI	SMOKE	27.62
62	VITTHAL WALIKAR	65YRS	MALE	FARMER	7832	6.0g/L	32g/L	0.18	15	HTN	DVD	IWMI	SMOKE,TOBACCO,ALCOHOL	18.75
63	JAINABEE TASHEEDAR	55YRS	FEMALE	FRUIT VENDOR	7874	6.0g/L	24g/L	0.25	86	HTN	SVD	AWMI	TOBACCO	25.6
64	SHEELABAI KALE	52YRS	FEMALE	HOUSE WIFE	8061	6.0g/L	39g/L	0.15	21	HTN	DVD	IWMI	NIL	23.44
65	MAHEBOOBSAB B	62YRS	MALE	FARMER	8833	2.1g/L	39g/L	0.05	47	HTN	DVD	AWMI	NIL	28.37
66	SOMANING PUJARI	77YRS	MALE	FARMER	9563	2.2g/L	39g/L	0.06	64	IHD	DVD	AWMI	TOBACCO	22.48
67	AYUBA KHAN NADAF	67YRS	MALE	BUSINESSMAN	9701	5.6g/L	24g/L	0.23	20	NIL	SVD	IWMI	SMOKE	24.42
68	SHEKHARAYYA HIREMATH	54YRS	MALE	FARMER	9783	3.7g/L	32g/L	0.11	43	NIL	DVD	AWMI	NIL	22.22
69	BASAGOND TORVI	65YRS	MALE	FARMER	9845	1.5g/L	30g/L	0.05	80	NIL	TVD	LWMI	NIL	23.44
70	MALLIKARJUN KALASGOND	64YRS	MALE	TEACHER	9869	2.4g/L	26g/L	0.09	119	HTN	TVD	IWMI	NIL	22.13