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Review Article

Pathophysiological Approach of Myocardial Infarction: A Review

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Abstract: Coronary artery disease (CAD) is the primary cause of myocardial infarction (MI), which is defined by a decreased or stopped blood supply to a portion of the heart myocardium, resulting in cardiac cell death. Because CAD robs the heart of oxygen supply, it frequently presents as silently or with symptoms like radiating chest pain to the shoulder, arm, neck, or jaw. Elevated cardiac troponins and ECG alterations are involved in the diagnosis. MI is caused by prolonged blockage of a major coronary artery; collateral circulation, hemodynamics, and residual blood flow all affect the size of the infarct. Atherosclerosis, thrombosis, and plaque erosion are the primary causes of ST-elevation MI (STEMI) and non-ST-elevation MI (NSTEMI). Male pattern baldness, advancing age, a family history of the condition, high cholesterol, smoking, hypertension, diabetes, obesity, and stress are risk factors for MI. Aortic dissection, drug misuse, and anomalies in the heart are further causes. Anaerobic respiration and lactate buildup are the results of tissue oxygen deprivation, which starts both reversible and irreversible phases of Myocardial infarction. Oxidative stress and endothelial dysfunction, in conjunction with inflammation and susceptible plaques, worsen the development of MI. Significant MI risks are highlighted by epidemiological research on smoking, obesity, diabetes, hypertension, and socioeconomic variables. Other factors include family history and genetic predisposition. Given that MI continues to be a major cause of morbidity and death worldwide, an understanding of its pathophysiology and risk factors is essential for both prevention and intervention. To lessen the effects of this terrible illness, effective strategies require prompt intervention and thorough risk factor control.
Keywords: Myocardial infarction, Oxidative stress, Diabetes mellitus, Hypertension, Blood lipid.

INTRODUCTION

The term "heart attack" refers to Myocardial infarction (MI), which is the result of reduced or stopped blood supply to a section of the heart myocardium. Myocardial infarction can occur "silently" and go unnoticed, or it can be a devastating incident that resulting in hemodynamic decline and abrupt death (Thygesen *et al.*, 2007). The primary cause of death in the US, coronary artery disease, is the root cause of the majority of myocardial infarctions. Oxygen is not available to the myocardium when coronary artery blockage occurs. Long-term oxygen supply deprivation of the heart can result in necrosis and death of cardiac cells (Reimer *et al.*, 1983). Individuals may exhibit pain or pressure in the chest, which can spread to the jaw, arm, neck, or shoulder. Myocardial ischemia may be linked to altered ECG patterns and increased biochemical markers such cardiac troponins in addition to the history and physical examination (Apple *et al.*, 2016; Goodman *et al.*, 2006).

Development and Determinants of Myocardial Infarction:

Myocardial infarction occurs when a large epicardial coronary artery is acutely blocked for longer than 20 to 40 minutes. The forming necrosis moves in a wave front toward less ischemic parts in the sub epicardium from the centre to the edge of the occluded vascular territory and from the endocardial layers that are most severely ischemia. The size and location of the perfusion territory distal to the coronary occlusion, temperature, hemodynamic state during ischemia, residual blood flow through collaterals (degree of ischemia), and the ischemia-related residual blood flow are the main factors that determine the final infarct size, with heart rate having a significant influence (Fig 1). In small rats with a fast heart rate and low collateral blood flow, the final infarct size is reached in 30 minutes;

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however, the species-specific time course of infarct development varies. In substantially larger species like humans the infarct size grows much more slowly over hours. Therefore, if there is sufficient collateral blood flow, reperfusion can preserve the ischemic myocardium even hours after the coronary blockage begins (Skyschally *et al.*, 2008).

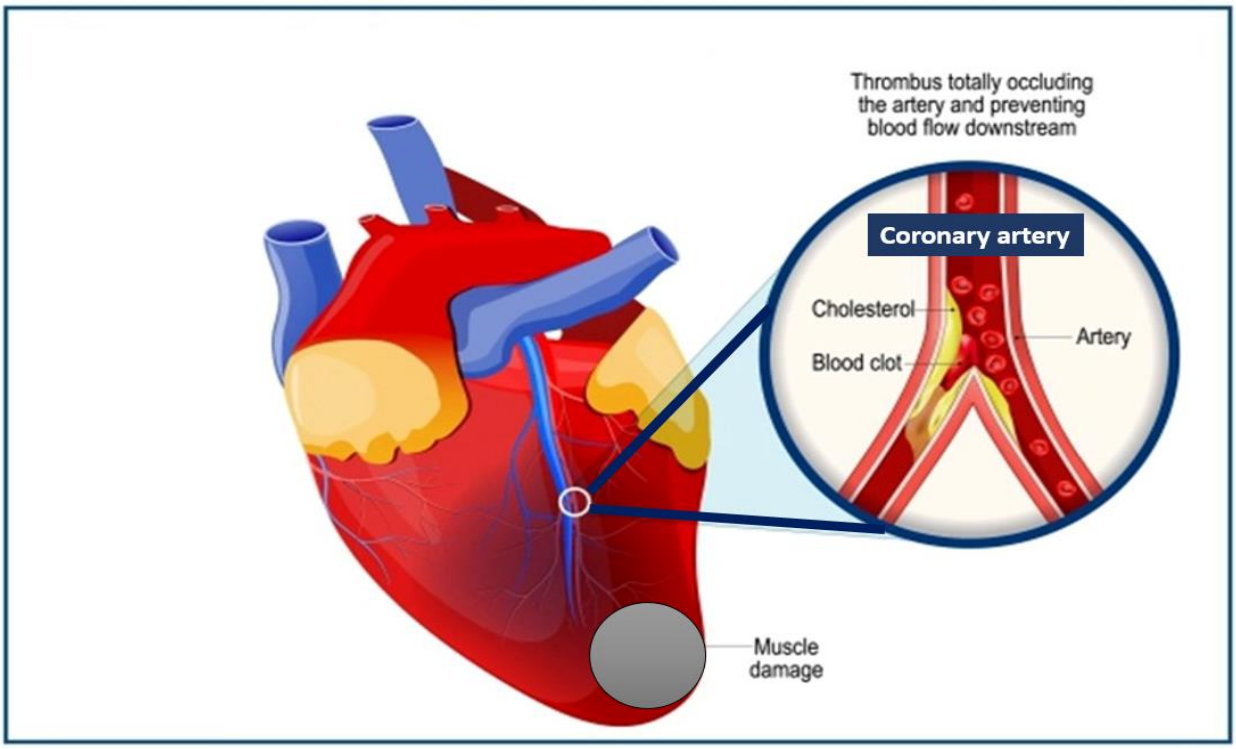

Figure 1: The image shows that a blocked artery due to thrombus reduces blood flow and damages the heart muscle

Types of Myocardial Infarctions

The differentiate between STEMI (ST-elevation myocardial infarctions) and NSTEMI (non-ST-elevation myocardial infarction), the two forms of MI that serve as the basis for clinical decision-making. Patients with STEMI are referred to immediate reperfusion treatment techniques, as opposed to those with NSTEMI. Additionally, there are five different types of MI based on its pathophysiology, clinics, and prognostics (Thygesen *et al*., 2012).

The fact that the culprit artery is typically prone with a non-occlusive thrombus in NSTEMI, whereas it is typically occluded by a thrombus in STEMI, is most likely the reason why intravenous thrombolytic therapy fails to improve clinical outcomes in the absence of AMI with ST-segment elevation. It doesn't seem likely that STEMI develops before NSTEMI. Research has shown that the majority of patients who experience recurrent MI episodes will either experience repeated STEMI or NSTEMI episodes, but not both. This suggests that some individuals are more likely to experience repeated occlusive thrombi episodes, while others may experience repeated non-occlusive thrombi episodes (Rott D *et al*., 2006). Therefore, the boundary between STEMI and NSTEMI is not as sharp as it once was. As of right now, it has been agreed that the initial course of care for a patient who comes with or without ST-elevation differs, but ST-elevation is not always permanent, which makes the case a "non- STEMI" for the sake of reperfusion decision-making. It is also true in the other way. The idea that early reperfusion of NSTE-ACS (non-ST elevation-acute coronary syndrome) is a better technique than a delayed approach is finally being investigated by ongoing randomized research; if these studies are successful, it will bring the two entities closer than they are now (Rott D *et al*., 2007).

Type I: Spontaneous MI:

Disturbance of the atherosclerotic plaque leading to the production of thrombi, reduced cardiac blood flow, or distant platelet emboli with subsequent myocyte necrosis.

Type II: MI secondary to an ischemic imbalance:

Imbalance in the supply and/or demand of oxygen in the heart caused by non-CAD conditions, such as coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, heart failure, anemia, respiratory failure, hypotension, hypertension, renal failure.

Type III: When biomarker values are not available, myocardial infarction that results in death.

Type IVa; Cardiomyopathy associated with percutaneous coronary intervention (PCI) is referred to as type IVa.

Type IVb: Stent thrombosis-related myocardial infarction

Type V: Coronary artery bypass grafting (CABG)-related myocardial infarction (Fig 2) (Thygesen *et al*., 2012)

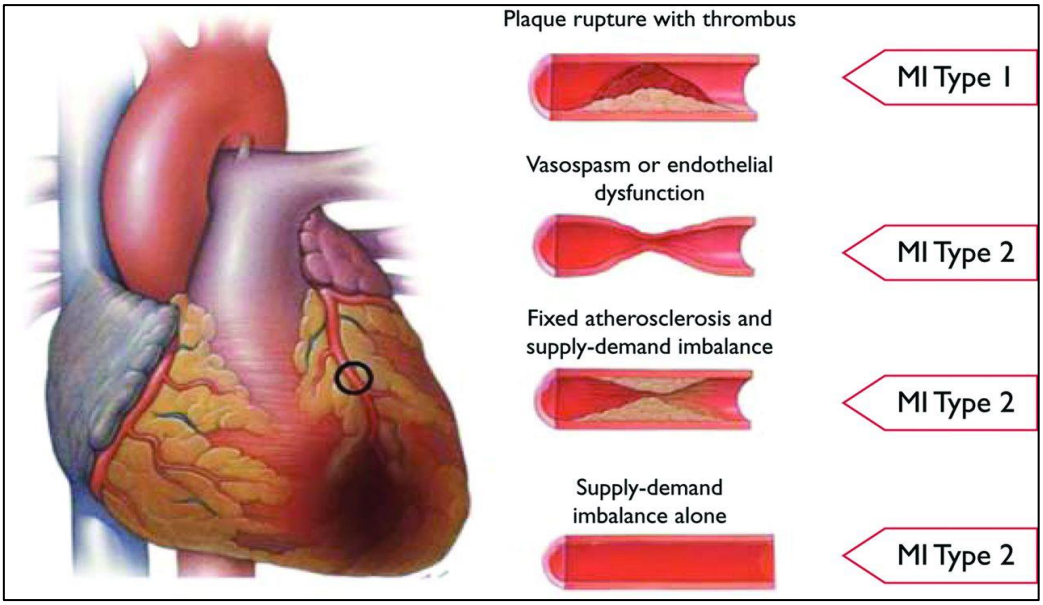

Figure 2: Types of Myocardial Infarction (MI)

Etiology

The majority of occurrences of acute coronary syndrome (ACS) are caused by atherosclerosis. Ninety percent of myocardial infarctions (MIs) are caused by an acute thrombus that obstructs a coronary artery with atherosclerosis. It is thought that erosion and plaque rupture are the main causes of coronary thrombosis. Plaque erosion or rupture is followed by endothelial vasoconstriction, platelet activation and aggregation, and activation of the coagulation cascade, which resulting in coronary thrombosis and occlusion. Flow dynamics and endothelial shear stress within the coronary vasculature are implicated in the etiology of susceptible plaque development (Chatzizisis *et al*., 2007). A substantial amount of data suggests that the responsible lesions are often proximally positioned within the coronary tree and have stenoses of less than 70% in many cases (Wang *et al*., 2003; Falk *et al*., 1995). Coronary atherosclerosis is more noticeable in the vicinity of vascular branching points (Daniel *et al*., 2008). Atheroma's with a big lipid-rich core encircled by a thinning fibrous crown are considered culprit lesions that are especially prone to rupture. These lesions also include an abundance of macrophages.

MI can also occur for causes other than atherosclerosis. Nonatherosclerotic causes of MI include the following:

Coronary artery emboli caused by sepsis products, air, cholesterol, or ventricle hypertrophy are some of the conditions that might cause coronary occlusion secondary to vasculitis and ventricle hypertrophy. Aortic dissection, with retrograde involvement of the coronary arteries; Coronary trauma; Primary coronary vasospasm (variant angina); Drug use (e.g., cocaine, amphetamines, ephedrine) Arteritis, Coronary anomalies, including aneurysms of coronary arteries; Factors that increase oxygen requirement, such as heavy exertion, fever, or hyperthyroidism; Factors that decrease oxygen delivery, like hypoxemia or severe anaemia (Macintyre *et al*., 2013; Kwong *et al*., 2018).

Risk factors

Certain risk factors have a substantial correlation with the onset of acute myocardial infarction (AMI) (RFs). These risk factors (RFs) are characterized as clinical or laboratory factors linked to the probability of illness start and progression over a given duration. Approaches to preventing the majority of premature MI depend on the identification of RFs (Yusuf *et al*., 2004). The risk factors include central obesity, hypertension, diabetes with a blood glucose level of 126 mg/dL or more, smoking five or more cigarettes a day, and low-density lipoprotein cholesterol between 100 and 120 mg/dL (Piegas *et al*., 2003). Other variables, referred to be non-modifiable RFs, include African American ancestry, age above 45 for men and above 55 for women, and a prior family history of early chronic artery disease (CAD) (Harrington *et al*., 2019).

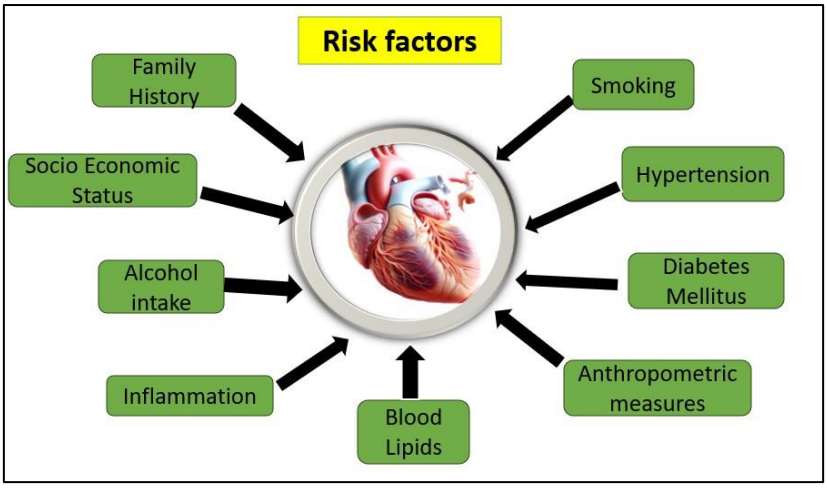

Figure 3: Risk Factors of Myocardial Infarction

Smoking

The primary independent risk factor for the morbidity and death of AMI is smoking. The duration and quantity of tobacco use are directly correlated with the development of CAD (Prescott *et al*., 1998). Approximately 10% of fatalities are caused by smoking, and by 2030, that percentage is predicted to rise to roughly 17% (Piegas *et al*., 2003).

Hypertension

One significant risk factor for both CAD and AMI is hypertension. High blood pressure levels are directly linked to the morbidity and death from CAD. Moreover, appropriate hypertension control may decrease the morbidity and death burden associated with AMI (Kaplan *et al*., 1999). It has been discovered that elevated blood pressure increases the risk of AMI more than twice (Piegas *et al*., 2003).

Diabetes mellitus

Diabetes mellitus (DM) is an established risk factor for AMI and CAD. Individuals without diabetes are less likely to experience a myocardial infarction (MI) than those with the disease. Moreover, diabetes and hyperglycemia are significant risk factors for death in MI cases (Stevens *et al*., 2004). Long-term insulin resistance linked to hyperglycaemia is the pathophysiology of diabetes mellitus (DM), which results in compensatory hyperinsulinemia.

Anthropometric measures

Body weight does not distinguish between muscle mass and body fat, despite the fact that it seems to be a predictor of obesity. Furthermore, peripheral obesity has less of an impact on the development of atherosclerosis than central fat (Siavash *et al*., 2008). Consequently, it's proposed that other anthropometric measurements such as circumference (WC), waist/hip ratio (WHR), and waist/thigh ratio (WTR) are better representative of the risk of CAD (Zhang *et al*., 2004).

Blood lipids

Low-density lipoprotein (LDL) cholesterol is linked to the development of arterial plaques, endothelial dysfunction, and atherosclerosis, which may be the cause of coronary artery disease (CAD) and stroke (Linton *et al*., 2019). The activation of the lectin-like oxLDL receptor-1 (LOX-1) is thought to have a role in the pathophysiology of atherosclerosis and endothelial damage and dysfunction caused by oxidized LDL (oxLDL) (Szmitko *et al*., 2003). Such LOX-1 is the primary oxLDL receptor on endothelial cells. It might be expressed by the macrophages and smooth muscle cells (SMCs) that are encroaching (Fig 4) (Yoshida *et al*., 1998; Aoyama *et al*., 2000).

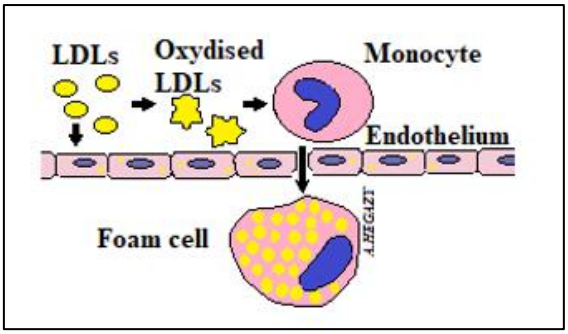

Figure 4: Differentiation of Monocytes to Phagocytes and Foam Cells

Inflammation

There is mounting evidence that inflammation is a significant risk factor for the development of atherosclerosis, possibly even more so than LDL (Libby *et al*., 2011; Smit *et al*., 2020). Regardless of LDL levels, elevated levels of highly sensitive C-reactive protein (CRP) may be a marker of CHD risk in otherwise healthy individuals. Furthermore, it is thought that the statins' ability to lower inflammation is what protects against CAD. This is demonstrated by a drop in inflammatory marker levels, such as CRP (Diamantis *et al*., 2017).

Alcohol intake

When compared to abstaining from alcohol, binge drinking doubles the chance of developing AMI. On the other hand, according to some researchers, minimal consumption can be advantageous in the case of myocardial infarction (Ilic *et al*., 2018). Increasing HDL cholesterol is anticipated to provide this advantage as opposed to affecting platelets and fibrinolysis (Suh *et al*., 2001; Piegas *et al*., 2003). Although there may be benefits to reducing alcohol use, it is not advised because excessive alcohol consumption has been shown to have detrimental effects (Suh *et al*., 2001).

Socioeconomic status

The risks of AMI and CAD are correlated with socioeconomic level. AMI and education level are inversely correlated; people with only an elementary education are most impacted, irrespective of social status (González-Zobl *et al*., 2010). Furthermore, those who are materially deprived and have low income are more likely to experience excessive psychological stress, which is bad for their general health, especially their cardiovascular system. Adopting harmful habits like smoking can also lead to this, as they increase the chances (Piegas *et al*., 2003).

Family history

Family history is thought to represent a separate risk factor for AMI, particularly for first-degree relatives. However family history is not a single element; rather, it is the product of several interactions between variables including genetic predisposition and environmental influences (Ranthe *et al*., 2015). According to certain claims, between 40% and 60% of cases of coronary heart disease are inherited (Roberts *et al*., 2012). More than 40 common genetic variations have been linked in previous publications to an increased risk of coronary artery disease (CAD) (Roberts *et al*., 2012; Deloukas *et al*., 2013). For this reason, gathering a thorough family history may be essential, particularly for patients between the ages of 35 and 55, in order to appropriately assess their risk for AMI (Ranthe *et al*., 2015).

Epidemiology

Ischemic heart disease, including MI, is a leading cause of death worldwide, accounting for almost 9 million deaths annually. Studies have revealed that 63% of patients in the UK experienced heart failure within six years. Cardiovascular diseases, especially coronary heart disease (CHD), are rather prevalent in India. The Registrar General of India reports that between 2001 and 2003, CHD was responsible for 17% of all deaths and 26% of adult deaths; between 2010 and 2013, this number increased to 23% of all deaths and 32% of adult deaths.

The World Health Organization (WHO) and the Global Burden of Disease Study have also highlighted growing patterns in the years of life lost (YLLs) and disability-adjusted life years (DALYs) attributable to CHD in India (Virani, *et al.*, 2021).

Pathophysiology of MI:

Reversible stage

Shortly after the ischemia episode began, low tissue oxygen levels prompted anaerobic respiration, which lowered ATP synthesis and raised pH due to lactate buildup (Solaro *et al*., 1988). Furthermore, early phases of the electron transport chain generate reactive oxygen species (ROS), which have the potential to damage contractile proteins (Steenbergen *et al*., 1977). As glycogen was increasingly utilized to compensate for decreasing ATP production, a common ultrastructural alteration in cardiomyocytes at this stage was the depletion of cytoplasmic glycogen granules (Jennings *et al*., 1985; RB, J. 1960). Additional alterations documented included the transverse tubular system's deformation and mitochondrial swelling brought on by intracellular Ca+2 excess (Coraboeuf, *et al*., 1980). Complete recovery is still achievable up to this point, but it is only feasible to restore the oxygen balance. Myocardial function can fully recover in 15-20 minutes if oxygen is quickly reintroduced (Frangogiannis 2015).

Vulnerable patient" instead of "vulnerable plaque

A severe and protracted imbalance between the myocardium's oxygen supply and demand leads to myocardial infarction (MI). This is typically caused by luminal thrombus on top of occlusive coronary atherosclerosis (Christia *et al*., 2013). Atherosclerosis is a dynamic, forward-moving condition. It is believed that endothelial dysfunction and inflammation work together to cause its consequences (Szmitko *et al*., 2003). About 10% of the time, coronary artery blockage goes undetected and results in myocardial infarction. These instances have a different prognosis and course of treatment than coronary artery blockage (Niccoli *et al*., 2020). In non-obstructive situations, the precise mechanism is still unclear. Nonetheless, a few possible reasons have been proposed, including microvascular dysfunction, spontaneous coronary artery dissection, coronary artery spasm, plaque disruption, and in-situ thrombosis (Tamis-Holland *et al*., 2019) (Hegazy *et al*., 2022). In these situations, it's critical to rule out additional causes of high troponin, such as pulmonary embolism, Takotsubo cardiomyopathy, and myocarditis

(Li *et al*., 2020). The most common underlying cause of MI is the rupture or erosion of a coronary atherosclerotic plaque. There has been intense discussion over the past 20 years on the structural traits of a susceptible plaque (a plaque that is more likely to rupture) but no clear consensus has been reached. Furthermore, there an ongoing debate on the presence of a susceptible plaque in animal models and human atherogenesis. More recently, it was proposed that a better way to determine the risk of MI would be to identify "vulnerable patients," taking into account many factors (such as the features of the plaque, circulating biomarkers, and the injured myocardium's reaction). Actually, it is probably unreasonable to try to pinpoint specific morphological traits that are predictive of plaque rupture, erosion, and clinical events in the future because they are subclinical processes with a dynamic, non-linear, and unpredictable trajectory. Instead, the prevailing pathophysiological paradigm views MI as the consequence of a "perfect storm" situation wherein a pro-thrombotic milieu at the site of plaque erosion or rupture overlaps a coronary artery stimulus for clinically significant thrombosis (Arbab-Zadeh *et al*., 2015).

Failing endothelium

The endothelium, which is the innermost layer of the blood vessel wall, is the target of numerous neurotransmitters, hormones, and physiological cues and is crucial in maintaining vascular homeostasis. Endothelium-derived autacoids, including prostacyclin, nitric oxide (NO), and endothelium-derived hyperpolarizing factor, regulate vascular tone. Reduced NO bioavailability is a significant factor in endothelial dysfunction (Cai H *et al*., 2000). NO is one of the most significant vasodilators secreted by the endothelium. It possesses anti-aggregant properties on platelets, works as a vasodilator, and suppresses growth and inflammation. It has frequently been observed that less NO is present when endothelial function is compromised. It could be brought on by decreased NO bioavailability as well as decreased activity of endothelial NO synthase (eNOS; due to endogenous or exogenous inhibitors or lower availability of its substrate, L-arginine). ROS are known to produce peroxynitrite, which quenches NO (Koppenol *et al*., 1992). Shear stress is the primary physiological trigger for the expression of the gene encoding endothelial NO synthase (eNOS) and the production of NO (Harrison *et al*., 2006). which is a cytotoxic oxidant and affects protein activity, which in turn affects endothelial function, by nitrating proteins. An essential mediator of LDL oxidation, peroxynitrite highlights the proatherogenic nature of LDL (Griendling *et al*., 2003). Endothelial dysfunction has a complicated pathophysiology that encompasses several pathways. Nonetheless, nitric oxide, asymmetric dimethylarginine, oxidative excess, Ang II, hyperhomocysteinemia, and diabetes appear to be linked to the majority of illnesses.

Atherosclerosis

Often referred to as Arteriosclerotic Vascular Disease, or ASVD, is a disorder where a buildup of fatty materials, such as cholesterol, causes an artery's wall to thicken. A syndrome known as "hardening or furring of the arteries" affects arterial blood vessels. It is characterized by a chronic inflammatory response in the artery walls, which is mostly caused by the accumulation of macrophage white blood cells and is encouraged by low density (especially small particle) lipoproteins (plasma proteins that carry cholesterol and triglycerides) without sufficient removal of fats and cholesterol from the macrophages by functional high-density lipoproteins (HDL). It results from many plaques building up inside the arteries (Fig 5) (Maton *et al*., 1993).

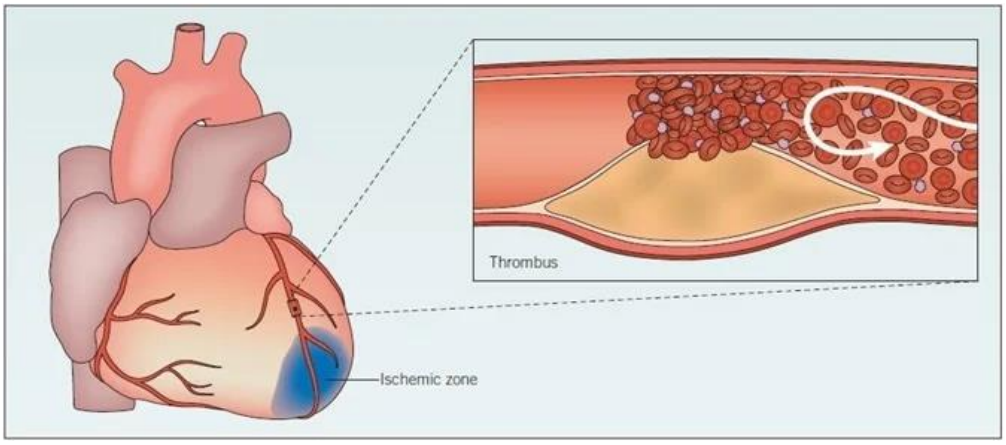

Figure 5: Image shows Coronary artery blocked by clot (thrombus), reducing blood flow and causing heart muscle damage

The progression of atherosclerosis

The intima thickens in areas with low shear stress, such as vascular curvatures and the outer walls of vessel branch points, most likely as a result of physiological adaptation. Beginning to form are the deeper musculoelastic layer containing smooth muscle cells (SMC) and elastic fibres, as well as the subendothelial proteoglycan-rich layer. The so-called intimal thickenings are areas that are prone to atherosclerosis (Bentzon *et al*., 2011).

Three separate components make up the atheromatous plaque:

1. The atheroma, or "lump of wax" (Greek: Athera, wax), is a nodular buildup of a soft, flaky, yellowish substance in the middle of massive plaques made up of macrophages closest to the artery's lumen.
2. Areas of cholesterol crystals beneath
3. Calculation in the periphery of more mature or advanced lesions.

Causes include:

- Elevated and modified low-density lipoprotein (LDL)
- Free radicals from smoking cigarettes
- Elevated plasma homocysteine
- Hypertension
- Diabetes mellitus
- Genetic changes
- Herpes virus infections chlamydia A case of pneumonia (Kakadiya 2009)

Coronary artery spasm

A abrupt, severe vasoconstriction of an epicardial coronary artery that results in vascular occlusion or near occlusion is referred to as a coronary artery spasm (CAS). While CAS may be implicated in other coronary syndromes, it is mostly responsible for variable angina (Prinzmetal *et al*., 1959)

Two factors interact to cause CAS:

1. A coronary artery anomaly that is often localized but can occasionally be diffuse and causes the artery to become hyperreactive to vasoconstrictor stimuli; and
2. A vasoconstrictor stimulus that can cause a spasm at the level of the hyperreactive coronary segment.

Three mechanisms have been proposed as the substrate for CAS susceptibility: (1) endothelial dysfunction; (2) vascular smooth muscle cells (VSMCs) primary hyperreactivity; and (3) other variables (Lanza *et al*., 2011).

Acute coronary syndrome (ACS)

AMI with non-S (downward deflection immediately after ventricular contraction)-segment elevation, T (recovery of ventricles)-segment elevation, unstable angina pectoris, and AMI with ST-segment elevation and sudden death are among the clinical syndromes that make up ACS (Cervellin *et al*., 2016). AMI with a Q wave, or the downward deflection right before ventricular contraction, happens in most cases of patients with ST-segment elevation in the ECG and characteristic ischemic chest pain; AMI without a Q wave develops in a small percentage of cases. While some patients develop AMI with a Q wave, most cases without ST-segment elevation result in unstable angina pectoris or AMI without a Q wave. When an oxygen-free atmosphere lasts for an extended period of time, ST-segment elevation turns into ST-segment depression (Dizon *et al*., 2014). Electrocardiography's (ECG) sensitivity and specificity are low in ACS (Wang *et al*., 2018).

Oxidative stress

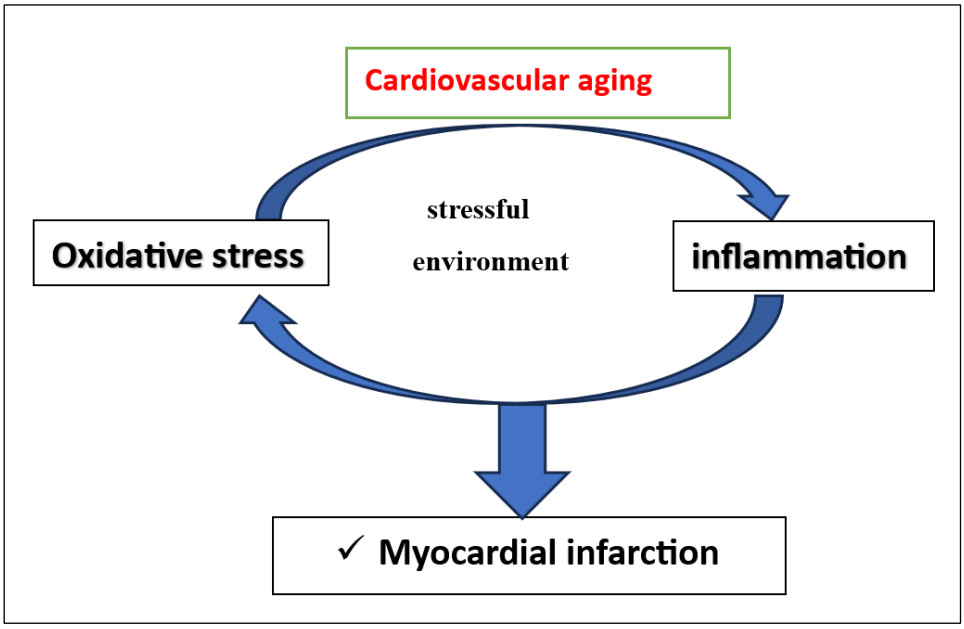

Figure 6: Ischemia, or low oxygen levels, produces a stressful environment that leads to oxidative stress, which harms cells. Stress and damaged cells cause inflammation, which weakens the heart muscle even more. A heart attack results from myocardial necrosis, the death of heart muscle cells, if blood flow is not rapidly restored

The major cause of death and disability worldwide is cardiovascular diseases (CVD), which are commonly defined as illnesses of the heart and blood vessels and include coronary heart disease, cerebrovascular disease, and other vascular problems. Fascinatingly, heart attacks and strokes account for four of every five deaths from CVD (Mozaffarian *et al*., 2015). Oxidative stress results from an unbalanced concentration of oxidants and antioxidants in the cardiomyocytes, which encourages the build-up of oxidants and causes cellular damage. Higher concentrations of reactive nitrogen species (RNS) or ROS in cellular and subcellular environments are more frequently linked to oxidative stress (Navarro-Yepes *et al*., 2014). On the other hand, at suboptimal levels, ROS and RNS can operate as signalling molecules to preserve cardiovascular function (Fig 6) (Penna *et al*., 2009). Superoxide anion (O2 ·−), hydroxyl radical (· OH), hydrogen peroxide (H2O2), singlet oxygen (O−), and hypochlorous acid (HClO) are examples of radicals that are included in the ROS. RNS consists of higher oxides of nitrogen, peroxynitrite (NO3 −), dinitrosyl iron complexes, nitroxyl anion (NO−), and nitrosonium cation (NO+) (Mozaffarian *et al*., 2015).

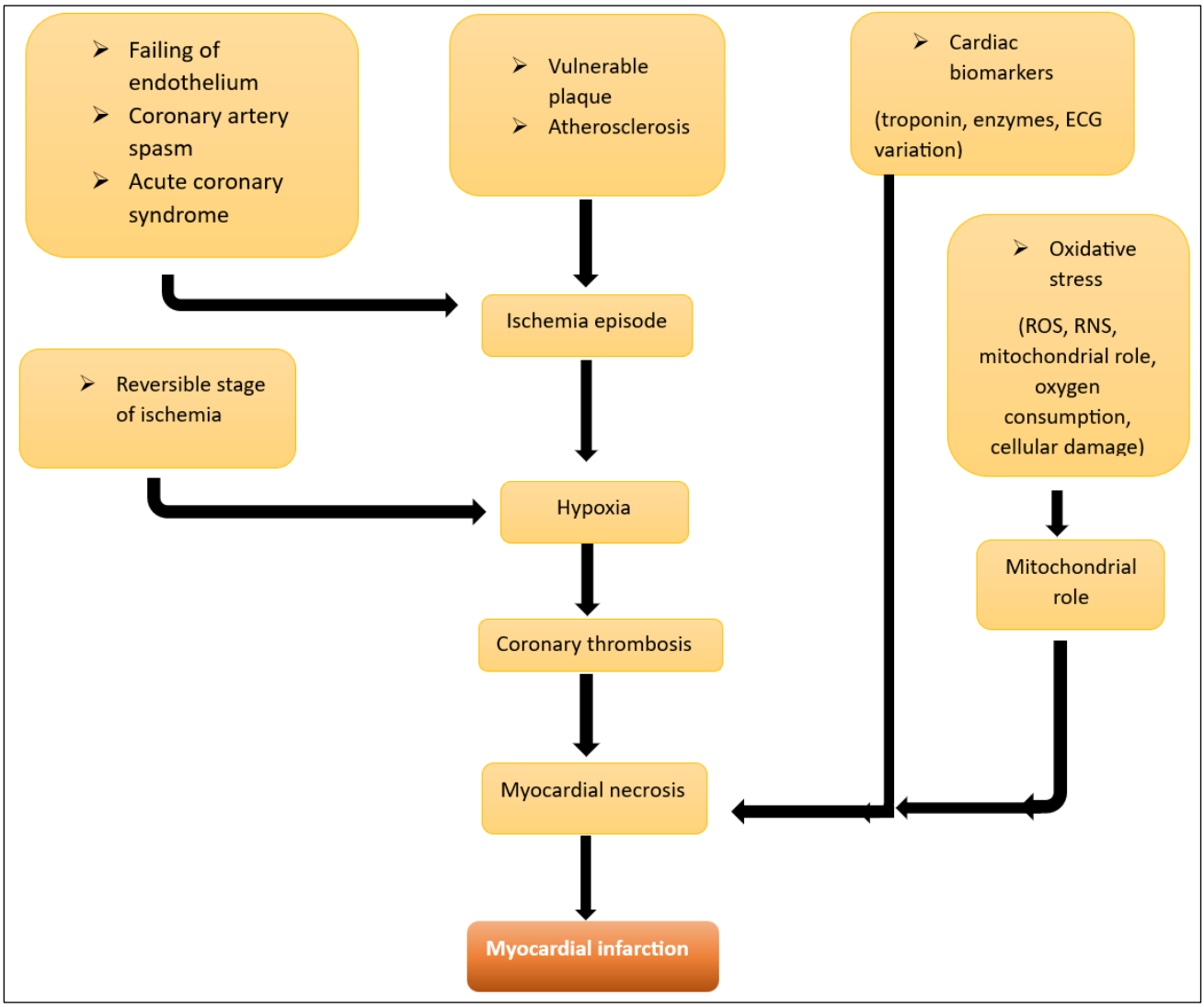

Figure 7: Image shows the steps leading to a heart attack (myocardial infarction) in an acute coronary syndrome (ACS) scenario. Damage to the artery lining, spasms, and plaque buildup reduce blood flow (ischemia) and damage heart muscle cells

The heart has a ratio of oxygen delivery to oxygen consumption that is 1.6–1.8 times higher than other tissues, which may indicate that the myocardium is consuming too much oxygen (C. B. Wolff 2008). By utilizing nitrone DMPO to trap these free radicals, it was confirmed that ROS formation and release during cardiac ischemia/reperfusion injury occurred (C. M. Arroyo *et al*., 1987). Because of their capacity to detect the amounts of cellular oxygen, mitochondria have become one of the most well-known cellular producers of reactive oxygen species (ROS). Actually, ROS is an inadvertent result of mitochondrial respiration in a healthy myocardium, where SOD strictly regulates its concentration to a low steady state level (F. J. Giordano 2005). demonstrated that the heart is made up of two spatially different types of mitochondria:

subsarcolemmal (SSM) and interfibrillar (IFM) mitochondria (J. W. Palmer *et al*., 1977). We have shown that the oxidative stress brought on by cardiac ischemia/reperfusion affects IFM and SSM populations differently (S. A. Banu *et al*., 2016).

CONCLUSION

A heart attack, sometimes referred to as a myocardial infarction (MI), is a condition in which the heart's blood supply is cut off, depriving the heart's cells of oxygen and causing them to die. Atherosclerosis, a disorder where fatty deposits accumulate in the arteries, is frequently the cause of this blockage. High cholesterol, diabetes, obesity, smoking, and hypertension are risk factors for both MI and atherosclerosis. Non-modifiable elements like sex, age, and family history also come into play. Comprehending the various forms, origins, and potential hazards of MI is essential for both mitigation and treatment. A complex web of interrelated variables, including oxidative stress, thrombosis, inflammation, and endothelial dysfunction, contribute to the pathogenesis of MI. Reducing the mortality rate and lessening the effects of MI need early identification using cardiac biomarkers and swift action.

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