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A Study on Evaluation of Coronary Artery Disease and its Risk Factors

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Abstract: Coronary artery disease (CAD) remains a leading global health burden, driven by a combination of modifiable and non-modifiable risk factors. This study evaluates key risk factors, including dyslipidemia, hypertension, diabetes mellitus, smoking, obesity, physical inactivity, and emerging contributors such as chronic inflammation and elevated lipoprotein(a). Non-modifiable factors like age, gender, and genetic predisposition are also discussed. Preventive strategies involving lifestyle changes, pharmacological interventions, and advancements in precision medicine are highlighted. Effective CAD prevention and treatment requires a multi-pronged strategy that targets these risk factors; doing so has the ability to lower death and morbidity rates.

Keywords: Coronary artery disease, risk factors, dyslipidemia, hypertension, diabetes mellitus, smoking, obesity, inflammation, prevention, precision medicine

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INTRODUCTION

From an anatomical perspective, the heart is a muscle that pumps blood. Its job is to periodically check the blood vessels to make sure that different areas of the body are getting the oxygen and substrates needed for metabolic processes. This is the primary role it plays. It maintains a steady blood pressure and a steady flow of blood, both of which are critical for the elimination of metabolic waste from tissues. Typically, a person's heart should continue to beat nonstop throughout their whole lives. A key component in defining the regularity of the heart's pumping activity is the coronary circulation system's ability to remove metabolic by-products and ensure that the myocardium obtains an appropriate supply of substrates. Because of this, the ability of the cardiovascular system to perform its activities properly is significantly jeopardized when coronary blood flow is reduced or stops entirely.

As we enter a new century, coronary artery disease (CAD), a kind of heart disease, is a major public health concern. This illness has the potential to progress to cancer1, is more common, more severe, and often detected at a younger age among Indians. Despite their scary appearance from a distance, these local concerns that continue to threaten our healthcare system are considerably less serious than the impending worldwide catastrophe of cardiovascular health. Cardiovascular disease is now experiencing a real epidemic. Worldwide, cardiovascular disease has become the biggest cause of mortality, disability, and healthcare spending in the last decade. According to the World Health Organization (WHO), 14.7 million people died in 1990 from cardiovascular disease (CVD), and 17 million people died in 1999 from the same reason. Cardiovascular disease kills 30% of the world's population every year.

This issue has lately grown in severity, posing a challenge to many research institutes worldwide. This is because research comparing local people in different areas of the globe with Indian immigrants has shown that the former are less prone to have cardiovascular disease.

In addition to leading a sedentary lifestyle and eating more calories, saturated fat, salt, and smokes, those who have become wealthy quickly tend to be overweight. All of these conditions—diabetes, obesity, dyslipidemia, hypertension, and hyperuricemia—are more likely to manifest when these variables are present. Living in this country has made Indians very suspicious about coronary artery disease. This is particularly true for Asian Indians, thus it's critical that we take a stronger stance in the battle against and treatment of current and new risk factors. Coronary artery disease (CAD) may be predicted, prevented, and treated using the current knowledge if risk factor screening begins promptly. The good news is that current knowledge allows for the prediction, prevention, and treatment of the disease.

Coronary artery disease is characterized by atherosclerosis in the coronary arteries and the lack of symptoms. Coronary heart disease (CHD) and ischaemic heart disease (IHD) are broad terms that include a lot of different medical issues (Ralapanawa U and Sivakanesan R 2021). Some examples of these disorders include coronary artery disease (CAD)–related CHD mortality, silent myocardial ischemia, stable angina, and acute coronary syndrome (ACS). Acute coronary syndrome (ACS) is characterized by myocardial infarction and unstable angina, and symptoms are present in every case. ACS is a potentially deadly disease. When plaque builds up within the arteries, a condition known as atherosclerosis, the most prevalent cause of coronary artery disease (CAD) occurs. The development of atherosclerosis is caused by a multitude of pathogenic processes. Endothelial dysfunction, lipid accumulation in the tunica intima, heightened innate and adaptive immune responses, vascular smooth muscle cell proliferation, and extracellular matrix remodeling are all involved in these processes. There are two main schools of thinking when it comes to the causes of atherosclerosis: The lipogenic hypothesis states that an imbalance between the systems that accumulate and eliminate lipids is the likely cause of lipid deposition within arterial walls (Lop L. 2021). One view is that thrombogenicity is due to fibroblast thickening and subsequent lipid enrichment in the intima layer of arteries brought about by fibrin organization. The heart-related condition known as cardiovascular disease (CVD) has been associated with oxidative stress in many investigations.

The cholesterol hypothesis states that atherosclerosis develops because of the presence of cholesterol in arterial plaques, which was found in the early 1900s. Numerous population studies have shown a correlation between elevated levels of bad cholesterol (LDL) and the main structural protein of LDL, apolipoprotein B (apo B) 100. Research has linked this association to ASCVEs, or atherosclerotic cardiovascular events. An important first step in the inflammatory cascade that causes atherosclerosis is the infiltration and retention of lipoproteins carrying ApoB in the artery wall. Atherosclerosis develops in part because of this. An artery wall lesion triggers endothelial dysfunction, which prompts apoB-containing lipoprotein modification and monocyte circulation into the subendothelial zone. The consumption of apoB-containing lipoproteins by macrophages triggers the development of foam cells, a hallmark of atherosclerosis. Macrophage cytokine and chemokine release is associated with LDL oxidation, endothelial cell activation, monocyte recruitment, and foam cell formation.

Evidence suggests that endogenous apoE, HDL, and apoA I all work together to boost cholesterol export,

which lowers inflammation, antioxidant stress, and oxidative stress (Yanez & Leiva 2022). Macrophages facilitate smooth muscle cell invasion and proliferation via their production of inflammatory chemoattractants. According to the smooth muscle cells' extracellular matrix, platelets and plaque prothrombotic components are distinct. Prolonged inflammation causes these vulnerable plaques to exhibit a number of undesirable characteristics, including an upregulation of necrotic cell death, a downregulation of extracellular matrix formation, and an inhibition of collagen breakdown by macrophage proteases. All of these things work together to cause these plaques to form. Thrombus formation due to a burst fibrous cap is more common in patients with ischemic acute coronary venous embolism. As it turns out, macrophages in vitro don't eat conventional LDL, so we need to change its makeup to encourage foam cell production. The inflammatory reactions that happen all across the body are caused by the conversion of LDL into atherogenic particles by oxidative mechanisms. In a perfect world, macrophage uptake and subsequent development of oxidatively damaged LDL (ox LDL) would be the cause of atherosclerotic plaque formation. According to Stone and Grundy (2019), lowering LDL cholesterol with statins is associated with a reduced risk of cardiovascular events. The cholesterol concept has been around for a while, and this lends credence to it.

CONVENTIONAL RISK FACTORS

High blood pressure, diabetes, excessive cholesterol, smoking, tobacco use, excess body fat, and insufficient physical activity are the conventional risk factors for coronary artery disease (CAD). Being overweight and not exercising enough are other contributors. Coronary artery disease (CAD) is more common in men, however there is a specific group of women who are more vulnerable and need extra precautions. Members of this subset are women who have experienced menopause. Compared to coronary artery disease (CAD), a form of heart disease, hypertension is linked to a higher risk of stroke. Recent research has shown that even at high normal levels, the risk of coronary artery disease (CAD) is twice as great. Diastolic blood pressure readings are in the 85–89 mm Hg range, while systolic readings are in the 130–139 mm Hg range.

There has been a roughly 24% rise in the risk of stroke and a 12% increase in the risk of coronary artery disease (CAD) since these alterations occurred. On the other hand, among those with a history of cardiovascular disease or an acute myocardial infarction, there is a negative correlation between overall obesity (as measured by BMI) and death rates. Worldwide, the risk of cardiovascular disease is elevated in those with abdominal obesity. Obesity in the central region of the body is an independent risk factor for coronary artery disease (CAD) since the risk of CAD increases with even a little increase in the quantity of fat therein. When asked about their cholesterol levels, Indian patients with coronary artery disease are more likely to reveal their triglyceride levels. The association between atherosclerotic cardiovascular disease and increased triglyceride (Tg) levels is a contentious topic.

EMERGING RISK FACTORS

Although there is a lengthy list of these variables, several of the conventional and traditional risk factors for coronary artery disease (CAD) remain unknown. Because of this, researchers have been looking for other risk factors that might exacerbate coronary artery disease. These risk factors may contribute, at least in part, to the greater incidence of coronary artery disease (CAD) among Indians, according to studies

conducted on populations of Indian migrants. Coronary artery disease (CAD) diagnosis requires careful assessment of many risk variables, such as cholesterol, homocysteine, plasma fibrinogen, C-reactive protein, and other developing risk factors.

It has been extensively shown in the last several decades that oxidative stress is a major factor in the development and consequences of coronary atherosclerosis. Recent years have seen a plethora of research linking common risk factors for atherosclerosis—such as diabetes, hypertension, cigarette smoking, and dyslipidemia—to elevated levels of oxidative stress. As a result, it seems that oxidative stress contributes to vascular disease and coronary artery disease (CAD). Phenotypes A, B, and C distinguish among the genetically determined forms of LDL cholesterol.

The risk of coronary artery disease (CAD) is significantly increased in individuals with LDL phenotype-B because their LDL particles are tiny and dense. Phenotype-B is much more common among Asian Indians (75% vs. 25% in the white population). In comparison to native, unmodified LDL, LDL that has undergone oxidative change is a more efficient proatherosclerotic mediator, and several investigations have shown that lipid oxidation is caused by reactive oxygen species (ROS). The levels of oxidized-LDL (ox-LDL) in the plasma of atherosclerosis patients are higher than those of healthy individuals. Additionally, the idea was based on the fact that most of these individuals also had antibodies against ox-LDL in their plasma. Multiple studies have shown the harmful effects of ox-LDL on different parts of the artery wall, providing strong evidence that ox-LDL plays a role in atherosclerosis.

The endothelium layer present on the artery wall is now being damaged by free radicals that are being created in an uncontrolled manner. Diffuse lesions may spread to other arteries, including the coronary arteries, as a systemic disease progresses. Smoking, hypertension, diabetes, and dyslipidemia are risk factors for atherosclerosis, which might worsen the severity of these lesions. An acute coronary event (ACE) may occur when inflammation is accelerated by a systemic infection, which in turn speeds up the process of plaque production and thrombosis. The reversal of this process is possible after atherosclerotic plaques have formed in the endothelium of the coronary arteries due to traditional risk factors. More research is needed to establish the likelihood of either fuel being added to the fire or fire being added to the fuel.

Coronary artery disease (CAD) and other cardiovascular diseases remain the top causes of mortality and disability worldwide, regardless of country of origin. It has already risen from fourteenth to fourth place on the 'Charts' in India, behind only tuberculosis, infectious diseases, and starvation. In 30–40% of people with coronary artery disease (CAD), lipids and lipoproteins are not the illness itself, but they are major risk factors for the condition. In Indians, additional risk factors including smoking, high blood pressure, diabetes, and similar illnesses do not reliably predict the likelihood of developing coronary artery disease in the future. Coronary artery disease (CAD) is unfortunately rather common in India, and this isn't only due to the usual suspects—traditional risk factors and emerging ones.

NON-MODIFIABLE RISK FACTORS

Age, gender, and hereditary susceptibility are some of the immutable risk factors for coronary artery disease (CAD). An individual's risk of coronary artery disease (CAD) increases dramatically beyond the

ages of 45 for men and 55 for women, highlighting the importance of age as a predictor (Go et al., 2014). Similarly, CAD is more common in males, but declining estrogen levels put postmenopausal women at risk as well (Mehta et al., 2016). Studies show that those with a first-degree relative afflicted by CAD have a twofold higher risk, making family history of early CAD a key non-modifiable risk factor (Kullo et al., 2005).

MODIFIABLE RISK FACTORS

1. Dyslipidemia: One known risk factor for coronary artery disease (CAD), which is characterized by abnormal lipid profiles such as low HDL-C levels, high LDL-C levels, and triglyceride levels (Nordestgaard et al., 2020). Low HDL-C levels hinder cholesterol removal from the circulation, which worsens plaque development, while high LDL-C levels promote atherosclerosis by accumulating cholesterol inside artery walls.

2. Hypertension: According to Williams et al. (2018), endothelial dysfunction and the acceleration of atherosclerosis are both caused by hypertension, which raises the mechanical stress on artery walls. In order to avoid coronary artery disease (CAD), it is essential to regulate blood pressure. Research has shown that a ten mmHg drop in systolic blood pressure lowers the risk of major cardiovascular events by around twenty percent (Ettehad et al., 2016).

3. Diabetes mellitus: coronary artery disease (CAD) Atherosclerosis is exacerbated by chronic hyperglycemia because it increases oxidative stress, inflammation, and endothelial dysfunction (Emerging Risk Factors Collaboration, 2010). Cardiovascular risks are enhanced by insulin resistance and dyslipidemia, which are both linked to diabetes.

4. Risk factor for coronary artery disease (CAD): Vascular damage and faster plaque development are consequences of smoking's oxidative stress, decreased nitric oxide bioavailability, and increased pro-thrombotic factors (Ambrose & Barua, 2004). A former smoker's risk of coronary artery disease (CAD) drops by as much as half after five years of giving up smoking (Rigotti, 2013).

5. Lack of Physical Activity and Obesity: Central obesity in particular promotes hypertension, insulin resistance, and dyslipidemia, all of which are risk factors for coronary artery disease (CAD) (Lavie et al., 2014). Sedentary lifestyles are associated with higher inflammation, worse lipid profiles, and worse cardiovascular fitness, which increases the risk of cardiovascular disease. Lipid profiles, blood pressure, and endothelial function may all be improved with regular exercise.

EMERGING RISK FACTORS

Chronic inflammation, increased lipoprotein(a) [Lp(a)], and psychosocial stress are rising to the forefront of CAD risk assessment alongside more conventional risk variables. Atherosclerotic plaque instability and rupture are significantly impacted by chronic inflammation, which is identified by high levels of C-reactive protein (CRP) (Ridker et al., 2002). Tsimikas (2017) found that having an elevated Lp(a) gene increases the risk of atherogenesis and thrombosis. Stress, sadness, and a lack of social support are psychosocial variables that increase the risk of coronary artery disease (CAD). This highlights the need of implementing comprehensive treatment techniques (Rosengren et al., 2004).

PREVENTIVE STRATEGIES

Preventing CAD involves a multifaceted approach targeting modifiable risk factors. Lifestyle interventions, including a heart-healthy diet, regular physical activity, and smoking cessation, form the cornerstone of prevention. Pharmacological therapies, such as statins, antihypertensive agents, and antiplatelet medications, are critical for high-risk individuals or those with established CAD (Arnett et al., 2019). Recent advancements in precision medicine, including genetic screening and biomarker identification, offer potential for personalized prevention strategies.

CONCLUSION

Effective prevention and therapy of coronary artery disease (CAD) relies on accurately assessing risk variables in order to identify at-risk patients. A more comprehensive approach is required to assess the risk of coronary artery disease (CAD), since new risk factors such as chronic inflammation and psychosocial stress have emerged alongside more established ones like smoking, high blood pressure, and dyslipidemia. Reducing the worldwide burden of coronary artery disease (CAD) may be possible with a mix of lifestyle changes, pharmaceutical therapies, and advances in personalized therapy.

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