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Risk Factors Associated With Acute Myocardial Infarction: A Review

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Abstract

Over the last four decades, our understanding of the pathogenesis of myocardial infarction has evolved and allowed new treatment strategies that have greatly improved survival. Acute myocardial infarction remains a leading cause of morbidity and mortality worldwide. It occurs when myocardial ischemia, a diminished blood supply to the heart, exceeds a critical threshold and overwhelms myocardial cellular repair mechanisms designed to maintain normal operating function and homeostasis. There has been a radical shift in therapy from passive healing of the myocardial infarction through weeks of bed rest to early discharge usually within 2 to 3 days as a result of immediate reperfusion strategies and other guideline-directed medical therapies. According to the INTERHEART study report, nine factors are responsible for 90% of myocardial infarctions. Modifiable risk factors include Diabetes mellitus, smoking, hypertension, hyperlipidemia, sedentary life style, obesity, stress and depression. The combination of several risk factors further enhances the risk. This review highlights risk factors that are responsible for the onset of infraction. Therefore management of these risk factors plays a vital role in order to prevent the development of acute myocardial infarction. Patients who develop cardiogenic shock still face a high 30-day mortality of at least 40%. Perhaps even more important is how do we identify and prevent patients from developing myocardial infarction in the first place. It is utmost important to understand and consider the risk factors during the management and treatment of acute myocardial infarction.

Keywords: Acute myocardial infarction, risk factors, Management of AMI

Introduction

Our understanding of the causes, diagnosis, and treatment of acute myocardial infarction (AMI) has evolved significantly over the decades. In the early 20th century, AMI was generally considered a fatal event diagnosed only at autopsy. Until the 1970s, with appropriate understanding of its usual clinical Presentation, risk factors and diagnosis, it was conservatively managed with prolonged bed rest and afterwards with a sedentary lifestyle. Since then, there has been an sudden increase of information which

has changed our understanding of its pathogenesis and markedly altered our treatment options, leading to vastly improved outcomes.

Acute myocardial infarction is one of the most common diseases among the developing countries [1]. These diseases have caused mortality in developed countries more than other diseases and impose numerous social and economic costs. This heart disease has emerged as a major health problem in developing countries including India. Precious life is snatched away when person is in most productive stage of life, when the social and family responsibilities are the greatest. These diseases are now seen in countries with low or average income which also have the majority of population. These diseases will probably turn into the most common cause of death in world till 2020 [2]. AMI is defined by the ischeamia and succeeding necrosis of the heart muscles that follows from a dramatic reduction of the blood flow in the heart. This blood flow reduction is caused by a thrombosis formation that can be initiated from erosion or from a disruption of an atherosclerotic plaque in the coronary artery [3]. Atherosclerosis relates to the accumulation of lipids and lipoproteins in the endothelium and is characterized by a chronic inflammation [4] that is also involved in the plague rupture and thrombosis [5].

It is commonly known as a heart attack, which occurs when there is a sudden block in blood flow in one or more of the coronary arteries and this cut off blood supply to a part of the heart muscle, causing necrosis (massive cell death, a permanent damage). If the block is severe, the heart can stop beating (cardiac arrest). This is most commonly due to occlusion or blockage of a coronary artery following the rupture of a vulnerable atherosclerotic plaque which is an unstable collection of lipids (cholesterol and fatty acids) and white blood cells (especially macrophages) in the wall of an artery. Myocardial infarction usually begins in the endocardium and spread towards the epicardium [6-8]. There are many symptoms of acute myocardial infarction but the most common is chest pain, which may travel into the shoulder, arm, back, neck or jaw. This type of pain always starts from the center or left side of the chest and remains for few minutes. The onset of symptoms in acute myocardial infarction is usually gradual, over several minutes and rarely instantaneous [9]. It seems that the mortality of these diseases will increase in developing countries due to lack of familiarity with the risk factors associated and failure to comply preventive principles. Identification of risk factors is an essential prerequisite to contain this menacing problem. Present study was conceived which is an attempt to study the risk factors associated with the development of acute myocardial infarction (A.M.I.).

The incidence of myocardial infarction in the world varies greatly. As per Global Burden of Diseases (GBD) study reported the estimated mortality from CHD in India at 1.6 million in the year 2000. It has been predicted that by 2020 there would be a 111 percent increase in cardiovascular deaths in India. This increase is much more than 77 percent for China, 106 percent for other Asian countries and 15 percent for economically developed countries [10].

In the United States and United Kingdom, nearly 650.000 and 180.000 patients get an acute myocardial infarction every year, respectively[11]. Worldwide, more than 3 million people have STEMIs and 4 million have NSTEMIs [12]. Indians are four time more prone to AMI as compared to the people of other countries due to a combination of the genetic and lifestyle factors that promote metabolic dysfunction [13]. The incidence of myocardial infarction in India is 0.064%[14]. The

mortality rate of myocar-dial infarction is approximately 30% and for every 1 in 25 patients who survive the initial hospitalization, dies in the first year after AMI. In India, 31.7% of deaths occur due to myocardial infarction. Incidence of cardiovascular diseases was about 7% in 1970 and increased upto 32% in 2011 in India [15].

Some disease factors contribute to the risk of myocardial infarction and they include diabetes mellitus (type 1 or 2), high blood pressure, dyslipidemia/hypercholesterolemia and particularly high amount of low-density lipoprotein, low amount of high density lipoprotein, high triglycerides, and obesity [16, 17]. Atherosclerosis is by far the most common cause of myocardial infarction. According to the INTERHEART study report, nine factors are responsible for 90% of myocardial infarctions. Modifiable risk factors include Diabetes mellitus, smoking, hypertension, hyperlipidemia, sedentary life style, obesity, stress and depression. The combination of several risk factors further enhances the risk. Treatment of hypertension and dyslipidemia causes reduction in risk of myocardial infarction. As there is variability in the prevalence of risk factors in different populations, a highly potent factor in one country could be less important in another one [18]. According to the study from west Sweden, acute myocardial infarction (AMI) among women is commonly affected by multiple risk factors. Hence, women more commonly have diabetes and arterial hypertension, while cigarette smoking is the only factor of lower frequency. On the other side, men have 3-6 times higher risk of developing disease compared to women, since women develop disease approximately ten years later. The risk of developing diseases is increased with aging [19]. Diabetes increases the risk of cardiovascular diseases (CVD) and mortality by about four times in women and about two folds in men, 30% of patients with an episode of myocardial infarction had type II Diabetes mellitus.

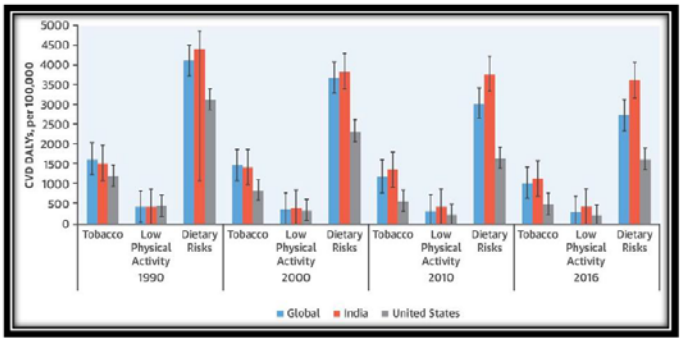

Figure 1: Cardiovascular Disease –Attributable to risk factors (Both Genders, Age Standardized, DALYS per 100,000) [20]

Risk Factors Associated with Acute Myocardial Infarction (AMI)

There are various risk factors of AMI. Among them, some are modifiable and others are non-modifiable. The major risk factors associated with AMI are explained as follows hereunder.

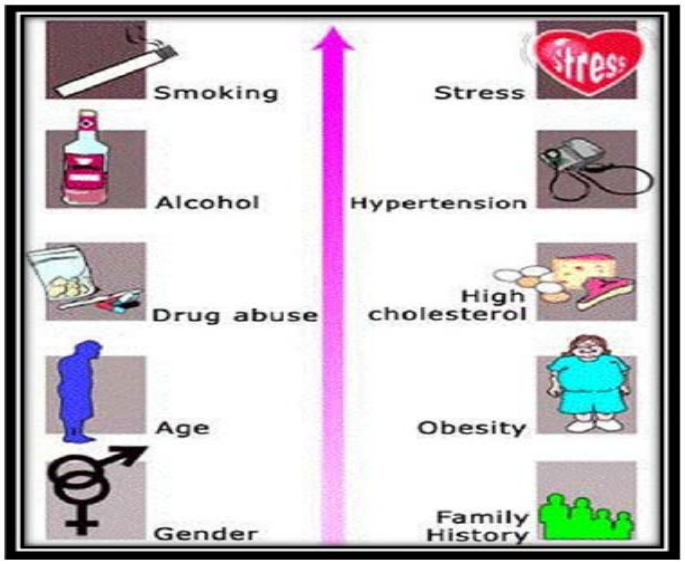

Figure 2: Showing the List of Risk Factors Associated With Acute Myocardial Infraction

Physical Activity

Inactive people with multiple cardiac risk factors are more likely to develop AMI [21]. Physical activity may

contribute up to 30%-35% reduced risk of coronary heart disease [22]. However, studies have shown that different types of physical activities 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 [23] whereas others, such as intensive domestic physical activity, may not offer protection against CVD. There are also interactive effects between lack of exercise and sitting at work and between demanding household work and sitting at work on the association with increased risk of acute myocardial infarction (AMI) [24]. Researchers examined the relationship between physical activity and acute myocardial infarction (AMI) and confirm that low physical activity is an indicator of subsequent risk of AMI [25]. Similarly, Gong et al., suggested that a light indoor activity pattern is associated with reduced AMI risk [26].

Smoking

Smoking is considered to be strong risk factor of myocardial infarction, premature atherosclerosis and sudden cardiac death. Smoking results in early STEMI especially in otherwise healthier patients [27]. Cigarette smoking increases the risk for AMI by multiple and complex mechanisms [28]. With respect to atherogenesis, smoking increases serum LDL-cholesterol and triglyceride concentrations and reduces serum HDL-cholesterol. Smoking appears to contribute vascular inflammation characteristic of atherosclerosis, as reflected by higher serum C-reactive protein levels in smokers than in nonsmokers [29]. Smoking, mainly through its nicotine content, activates the sympathetic nervous system (SNS), increasing both heart rate and systolic blood pressure. This increase in the rate-pressure product results in increased myocardial oxygen demands. Increased in activity of SNS also leads to coronary arterial vasoconstriction, decreasing myocardial blood flow at a time when oxygen demand is increasing. In addition to increasing myocardial oxygen demand and reducing coronary blood flow, cigarette smoking also causes increase in the levels of carboxyhemoglobin in the blood, with the potential to further reduce myocardial oxygen delivery from oxyhemoglobin.

Alcohol Consumption

Alcohol consumption is associated with an acutely higher risk of myocardial infarction in the subsequent hour among people who do not typically drink alcohol daily. There is consistent evidence that moderate habitual alcohol consumption is associated with a lower risk of cardiovascular events in subsequent months and years [30] and that heavy episodic (binge) drinking is associated with higher cardiovascular risk.

According to McKee and Britton [31] and research sources,[32] a number of possible mechanisms have been discussed explaining the acute effects of binge drinking on CHD events.

These include:

(i) Over drinking has been shown to increase low-density lipoproteins, which in turn are linked to cardiovascular morbidity and mortality. However, low to moderate steady drinking, heavy irregular drinking has not been associated with increased levels of high-density lipoproteins, which are linked to favorable cardiovascular outcomes.

(ii) Over drinking has been associated with an increased risk of thrombosis, occurring after cessation of a heavy drinking episode.

(iii) Over drinking appears to predispose drinkers to histological changes in the myocardium and conducting

system, as well as to a reduction in the threshold for ventricular fibrillation [33].

However, there is a controversy regarding consumption of alcohol and risk of AMI. Most researches showed that alcohol consumption can increase high density lipoprotein cholesterol (HDL-C), apolipoprotein-a1 and adiponectin and reduce fibrinogen level, in which the HDL-C was the main protective factor which could explain about 50% of the causal relationship [34]. But, some researchers believed that the value of HDL-C was low, which could only explain about 16% of the causal relationship [35]. Besides, some researchers found that alcohol consumption could increase the prostacyclin of blood vessel wall, improve functions of vascular endothelial cells, increase insulin sensitivity and resist thrombosis [36]. Moreover, long-term regular alcohol consumption could improve heart rate variability [37] and thus reduce MI onset risks. Most researches showed that alcohol consumption could increase LDH-C, triglyceride, heart rate, blood pressure [38] and thus increase the risks of atherosclerosis, atrial fibrillation and anoxia, resulting in damaging cardiac muscle cells and cardiovascular system and producing fibrinolytic enzyme inhibitor [39].

Dyslipidemia

Dyslipidemia, a major risk factor of cardiovascular disease, is generally defined as the total cholesterol, LDL, triglycerides, apo B or Lp (a) levels above the 90th percentile or HDL and apo A levels below the 10th percentile of the general population

[40]. Increased triglyceride levels and dense, small LDL particles act as predisposing risk factors for myocardial infarction. Non fasting triglyceride level appears to be a strong and independent predictor of future risk of AMI, particularly when the total cholesterol level is also increased. The reason behind it is that decreased HDL-C levels and increased triglyceride levels cause metabolic perturbations and thus causing adverse consequences [41]. A researcher by name Ali et al.,[42] concluded that there is a high frequency of dyslipidemia in young patients presenting with acute myocardial infarction, with descending order hypertriglyceridemia followed by hypercholesterolemia, raised LDL and low HDL. The prospective studies such as those of Framingham study established the relationship of dyslipidemia and coronary artery disease. High levels of total cholesterol, LDL and low level of HDL are major risk factors for coronary atherosclerosis. Correction of dyslipidemia can reduce the risk of myocardial infarction [43].

Diabetes Mellitus

Type 2 diabetes mellitus is on the verge of becoming a pandemic in India [44]. It is a chronic condition that occurs when the body can not produce enough or effectively use of insulin, and are induced by a genetic predisposition coupled with environmental factors [45]. Type 2 diabetes shares several risk factors in common with coronary artery disease (CAD), such as age, hypertension, dyslipidemia, obesity, physical inactivity and stress, an increase in the prevalence of diabetes indirectly implicates an escalating risk of CAD as well [46]. Diabetes mellitus is a well-established risk factor for cardiovascular disease (CVD). People with type 2 diabetes mellitus have a higher cardiovascular morbidity and mortality and are disproportionately affected by CVD compared with non-diabetic subjects. Diabetes increases risk of coronary heart disease (CHD) by two to four times. Patients with diabetes bear greater risk of atherosclerotic vascular disease in the heart as well as in other vascularized areas.

The life expectancy of people with diabetes is reduced by nearly eight years due to increased mortality [47].

Coronary artery disease accounts for more than 80% of all deaths and 75% of all hospitalizations in diabetic subjects [48]. It is also reported that plaques are more vulnerable to rupture among patients with diabetes. The protective female gender effect is lost in diabetic subjects, and indeed, women with diabetes are possibly more prone to develop CAD than men with diabetes.

Diabetes increases the risk of myocardial infarction because it increases the rate of atherosclerotic progression and adversely affects the lipid profile and facilitates formation of atherosclerotic plaque In a hospital based case control study, conducted in a rural population of India, cases of AMI were twice as likely to have history of diabetes compared to controls. According to Bibbins et al., that diabetes mellitus is the biggest individual risk factor of heart disease[49]. Diabetes is also a risk factor for myocardial infarction case fatality: that is, myocardial infarction is more often fatal in people with diabetes compared with myocardial infarction in those without diabetes [50].

Hypertension

The systolic and diastolic hypertension increase the risk of a myocardial infarction and the higher the pressure, the greater the risk [51]. It is major risk factor of causing atherosclerosis in coronary blood vessels, result in heart attack or myocardial infarction. Hypertension and myocardial infarction are closely linked. In old age, hypertension is even worse to heart and responsible for at least 70 percent of heart disease. Several mechanisms can account for the increased coronary risk in hypertensive patients. Hypertension accelerates the effects on atheroma, increases shear stress on plaques, exerts adverse functional effects on the coronary circulation, and impairs endothelial function and control of sympathetic tone. Ciruzzi et al.,[52] reported that in Argentine population, hypertension is a strong and independent risk factor for acute myocardial infarction. The control of hypertension with strict compliance of proper medication and adoption of lifestyle modifications reduce the risk of myocardial infarction significantly.

BMI/Obesity

Increased BMI is directly related to incidence of myocardial infarction. Infarction is greatly enhanced by extreme obesity because it is a recognized risk factor for myocardial infarction. Zhu et al.,[53] performed the meta-analysis of previous studies and suggested that overweight and obesity are associated with higher risk of AMI. Overweight and obesity may affect health, and it is necessary to control one's BMI to prevent AMI. Schargrodsky et al.,[54] demonstrated that overweight is an independent risk factor for MI. Yusuf et al.,[55] demonstrated that abdominal obesity increased the risk of AMI in both ages and sexes in all regions.

Stress

Chronic life stress, social isolation and anxiety increase the risk of heart attack and stroke. Acute psychological stress also is associated with increased risk for coronary heart disease, and it has been reported that intense grief in the days after death of a significant person may trigger the onset of myocardial infarction. The pathophysiological mechanism of acute emotional stress remains unclear, but it is assumed to be related to hemodynamic stress in the coronary arteries and rupture of an atherosclerotic plaque, with consequent thrombosis [56].

Gout

Patients with gout have an increased risk of myocardial infarction. In gout patients, the inflammatory response associated with gout plays a key role in the initiation and progression of atherosclerosis, and promotion of a pro-

thrombotic environment that leads to acute coronary events such as angina or myocardial infarction [57].

Periodontal Diseases

Periodontal diseases are a group of inflammatory diseases in which bacteria and their by-products are the principal aetiologic agents.[58] The first indication of association between dental disease and atherosclerosis was given in 1963. [59] Since then, there is growing evidence that poor dental health, especially the presence of periodontal disease, increases the risk of occurrence of CHD. The study done by Kaisare et al.,[60] also indicates an association between periodontal disease and acute myocardial infarction.

A Familial History

Family history of myocardial infarction is an independent risk factor for AMI. Several genetic variants are associated with increased risk of AMI and family history of AMI in a first-degree relative doubles AMI risk. A recent combined analysis of 12 cohort studies found a combined relative risk of 1.6 for future events in persons with a first-degree relative with cardiovascular disease, compared with persons without an affected first-degree relative [61]. Ciruzzi et al., reported that family history of acute myocardial infarction was a strong and independent risk factor in a study of 1.060 cases and 1.071 controls from Argentina. The antecedent of ≥1 first-degree relative with acute myocardial infarction increased the risk about twofold in men and nearly threefold in women, with a significant trend in risk in the number of relatives affected [62]. Friedlander et al.,[63] reported that family history of AMI is positively associated with the risk of early AMI in women. If a father develops heart attack before the age of 55 and mother before the age of 65 years, this positive family history becomes very significant for the next generation and mere presence of parental and maternal history for premature myocardial infarction may increase the risk to 7 folds in descendents [64].

Age

Advanced age is associated with an increased mortality in acute myocardial infarction [65]. The mechanism by which increasing age contributes so dramatically to mortality is unknown [66]. About 80% of heart disease deaths occur in people aged 65 or older.

Gender

Men tend to have heart attacks earlier in life than women. Women's rate of heart attack increases after menopause but does not equal men's rate. Even so, heart disease is the leading cause of death for both men and women [67].

Conclusion

There are various risk factors of acute myocardial infarction which should be taken into consideration while treating patients of AMI. Expression of coronary heart disease phenotype is feature of ageing and therefore increased life expectancy has resulted in the greater exposure to the risk factors of coronary heart disease. As a result of population growth and demographic trends, urban population of India is expected to double to 600 million by 2020, further expanding the coronary heart disease epidemic.13 In the present study except family history all other risk factors are modifiable, illustrating the enormous potential for prevention of acute myocardial infarction.

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