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Assessment of different risk factors for patients suffered from acute myocardial infarction from Bihar State

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Abstract
Traditional/existing risk factors have been the major cause of all major cardiovascular events. Despite adjustment of these risk factors, cardiovascular related death and disability is still progressing in the developed and developing nations, indicating absence of reliable and effective risk predicting biomarkers. Hence based on above findings the present study was planned for Assessment of Different Risk factors for Patients Suffered from Acute Myocardial Infarction from Bihar State.
The present study was planned in Department of General Medicine, Indira Gandhi Institute of Medical Sciences, Patna, Bihar. The 50 cases of the acute myocardial infraction were enrolled and evaluated. Detailed history was taken in each patient and information of every patient was recorded in a separate proforma. Informed consent was obtained from all patients. The serum cardiac enzyme level was measured and ECG was done at the time of admission and repeated as necessary.
The data generated from the present study concludes that smoking, sedentary lifestyle and low HDL levels were the most common conventional risk factors found in the study population especially in younger age group. Stress did not contribute significantly. There was no statistically significant difference in risk factors between rural and urban population. Large case-control studies with multivariate logistic regression are needed to stratify, which conventional risk factor independently contributes the most in occurrence of coronary artery disease.

Keywords: risk factors, Acute myocardial infarction, Bihar, etc

Introduction
Myocardial infarction (MI) (i.e., heart attack) is the irreversible death (necrosis) of heart muscle secondary to prolonged lack of oxygen supply (ischemia). Cardiac biomarkers/enzymes: The American College of Cardiology/American Heart Association (ACC/AHA) and the European Society of Cardiology (ESC) guidelines recommend that cardiac biomarkers should be measured at presentation in patients with suspected MI, and that the only biomarker that is recommended to be used for the diagnosis of acute MI at this time is cardiac troponin due to its superior sensitivity and accuracy [1, 2, 3, 4].
Initial stabilization of patients with suspected MI and on-going acute chest pain should include administration of sublingual nitro-glycerine if patients have no contraindications to it. The American Heart Association (AHA) recommends the initiation of beta blockers to all patients with STEMI (unless beta blockers are contraindicated) [1, 2]. If STEMI is present and the patient is within 90 minutes of a PCI-capable facility, the patient should undergo emergent coronary angiography and primary PCI. If the patient is longer than 120 minutes from a PCI-capable facility, fibrinolysis should be considered [2].
Although patients presenting without ST-segment elevation (non-STE-ACS) are not candidates for immediate administration of thrombolytic agents, they should receive anti-ischemic therapy and may be candidates for PCI urgently or during admission.
Myocardial infarction (MI) usually results from an imbalance in oxygen supply and demand, which is most often caused by plaque rupture with thrombus formation in an epicardial coronary artery, resulting in an acute reduction of blood supply to a portion of the myocardium. Although the clinical presentation of a patient is a key component in the overall evaluation of the patient with MI, many events are either "silent" or are not clinically recognized by patients, families, and health care providers. (See Presentation.) The appearance of cardiac biomarkers in the circulation generally indicates myocardial necrosis and is a useful adjunct to diagnosis.
MI is considered part of a spectrum referred to as acute coronary syndrome (ACS). The ACS continuum representing on going myocardial ischemia or injury consists of unstable angina, non–ST-segment elevation MI (NSTEMI)—collectively referred to as non–ST-segment acute coronary syndrome (NSTE ACS)—and ST-segment elevation MI (STEMI). Patients with ischemic discomfort may or may not have ST-segment or T-wave changes denoted on the electrocardiogram (ECG). ST elevations seen on the ECG reflect active and ongoing transmural myocardial injury. Without immediate reperfusion therapy, most patients with STEMI develop Q waves, reflecting a dead zone of myocardium that has undergone irreversible damage and death.
Those without ST elevations are diagnosed either with unstable angina or NSTEMI—differentiated by the presence of cardiac enzymes. Both these conditions may or may not have changes on the surface ECG, including ST-segment depressions or T-wave morphological changes. MI may lead to impairment of systolic or diastolic function and to increased predisposition to arrhythmias and other long-term complications. Coronary thrombolysis and mechanical

revascularization have revolutionized the primary treatment of acute MI, largely because they allow salvage of the myocardium when implemented early after the onset of ischemia.

The modest prognostic benefit of an opened infarct-related artery may be realized even when recanalization is induced only 6 hours or more after the onset of symptoms; that is, when the salvage of substantial amounts of jeopardized ischemic myocardium is no longer likely. The opening of an infarct-related artery may improve ventricular function and collateral blood flow; prevent ventricular remodeling, as well as decrease infarct expansion, ventricular aneurysm formation, and left ventricular dilatation; and reduce late arrhythmia associated with ventricular aneurysms, and mortality [5, 6, 7].

Evidence suggests a benefit from the use of beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, and statins. The American College of Cardiology (ACC)/American Heart Association (AHA)/European Society of Cardiology/World Heart Federation released the Observations From the TRITON-TIMI 38 Trial (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition With Prasugrel–Thrombolysis in Myocardial Infarction 38), which better outlines a universal definition of MI, along with a classification system and risk factors for cardiovascular death [8].

Myocardial infarction (MI), commonly known as a heart attack, is defined pathologically as the irreversible death of myocardial cells caused by ischemia. Clinically, MI is a syndrome that can be recognized by a set of symptoms, chest pain being the hallmark of these symptoms in most cases, supported by biochemical laboratory changes, electrocardiographic (ECG) changes, or findings on imaging modalities able to detect myocardial injury and necrosis. According to the third universal definition of MI, implemented by a joint task force from the European Society of Cardiology (ESC), American College of Cardiology (ACC) Foundation, American Heart Association (AHA), and the World Heart Federation (WHF), MI is diagnosed when either of the following two criteria are met [9].

For the normal heart to continue to function and to steadily pump blood efficiently to meet the demands of the body, it needs to have a constant supply of oxygen and nutrients provided mainly by the coronary circulation. A condition called myocardial ischemia happens if blood supply to the myocardium does not meet the demand. If this imbalance persists, it triggers a cascade of cellular, inflammatory and biochemical events, leading eventually to the irreversible death of heart muscle cells, resulting in MI. The spectrum of myocardial injury depends not only on the intensity of impaired myocardial perfusion but also on the duration and the level of metabolic demand at the time of the event. Severe loss of the ability of the heart muscle cell to contract can be observed as early as within 60 seconds. Persistence of oxygen deprivation to the myocardium through the cessation of blood supply will lead to irreversible myocardial injury within 20 to 40 minutes and up to several hours, depending on several factors including the existing metabolic state of the body and presence of coronary collateral blood flow [10].

Typical MI initially manifests as coagulation necrosis that is ultimately followed by a healing process characterized by formation of myocardial scarring, known as myocardial fibrosis. This mechanism allows significant architectural changes to the composition, shape and contractile function of the myocardium, especially in the left ventricle, which is the major contributor to the contractile function of the heart. Eventually the left ventricle dilates and changes to a more spherical shape, in a process known as ventricular remodeling. Despite being an irreversible process, ventricular remodeling is a regulated process, therefore, specific treatment strategies and agents should be used in acute MI management in order to reduce the occurrence and severity of ventricular remodeling [11].

In some occasions, restoration of blood flow to the damaged myocardium triggers further ischemic cellular damage, this paradoxical effect is known as reperfusion injury. This process involves a complex interaction between oxygen free radicals and intracellular calcium, leading to acceleration of myocardial damage and death, microvascular dysfunction and fatal arrhythmias. The role of nitric oxide (an endothelium-derived relaxing factor) as a cardioprotective agent against reperfusion injury, has been demonstrated, as nitric oxide works to inactivate oxygen free radicals, therefore, ameliorating the process of reperfusion injury [12]. Despite the improved understanding of the process of reperfusion injury, there are no specific therapies to prevent it.

Stunned myocardium is a condition of transient left ventricular dysfunction following an ischemic event to the myocardium. It occurs if coronary blood flow was impaired for a brief period of time (5 to 15 minutes). Usually, stunned myocardium persists for hours or days following the re-establishment of coronary blood flow. However, prolonged exposure of the myocardium to an ischemic state, results in an impairment of its contractile function, which can be partial or complete, this is known as myocardial hibernation, and is reversible with revascularization. Both myocardial stunning and hibernation occur because of loss of essential metabolites required for normal myocardial contractility, such as adenosine, which is needed for adenosine triphosphate (ATP)-dependent contraction [13].

The atheromatous plaque responsible for acute MI develops in a dynamic process in multiple stages. Starting with arterial intimal thickening, which consists of vascular smooth muscles with very minimal or no inflammatory cells, this process can be observed soon after birth. Subsequently, the formation of fibrous cap atheroma occurs, which has a lipid-rich necrotic core that is surrounded by fibrous tissue. Eventually, a thin-cap fibroatheroma develops, this is also known as a vulnerable plaque which is composed mainly of a large necrotic core separated from the vascular lumen by a thin fibrous cap that is infiltrated by inflammatory cells and is deficient of smooth muscle cells, making it vulnerable to rupture [14, 15].

The process of acute coronary thrombosis leading to ACS involves the pathogenic mechanism of plaque rupture, and less frequently plaque erosion. The Brasilia Heart Study Group indicates that changes in high-density lipoprotein (HDL) during an MI may alter the antiatherogenic function of HDL to transport lipids from arterial walls [16]. The investigators noted a simultaneous decrease in lipid transfer to HDL and in the capacity of HDL to efflux cholesterol from cells occurs in the acute period after an MI. In a nested case-control study that evaluated the associations of plasma metabolic markers with the risks of incident MI, ischemic

stroke, and intracerebral hemorrhage, investigators found positive associations of lipoproteins and lipids with MI and ischemic stroke but not with intracerebral hemorrohage, as well as positive associations between triglyceride concentrations and MI [17]. Except for small HDL, there was also an inverse association of HDL particles with MI, and an inverse association of cholesterol in large HDL with MI and ischemic stroke. The study cohort included 912 patients with MI, 1146 with ischemic stroke, 1138 with intracerebral hemorrhage, and 1466 control subjects [17].

Atherosclerosis is the disease primarily responsible for most acute coronary syndrome (ACS) cases. Approximately 90% of myocardial infarctions (MIs) result from an acute thrombus that obstructs an atherosclerotic coronary artery. Plaque rupture and erosion are considered to be the major triggers for coronary thrombosis. Following plaque erosion or rupture, platelet activation and aggregation, coagulation pathway activation, and endothelial vasoconstriction occur, leading to coronary thrombosis and occlusion.

Within the coronary vasculature, flow dynamics and endothelial shear stress are implicated in the pathogenesis of vulnerable plaque formation [18]. A large body of evidence indicates that in numerous cases, culprit lesions are stenoses of less than 70% and are located proximally within the coronary tree [19, 20]. Coronary atherosclerosis is especially prominent near branching points of vessels. Culprit lesions that are particularly prone to rupture are atheromas containing abundant macrophages, a large lipid-rich core surrounded by a thinned fibrous cap.

Traditional/existing risk factors have been the major cause of all major cardiovascular events. Despite adjustment of these risk factors, cardiovascular related death and disability is still progressing in the developed and developing nations, indicating absence of reliable and effective risk predicting biomarkers. Hence based on above findings the present study was planned for Assessment of Different Risk factors for Patients Suffered from Acute Myocardial Infarction from Bihar State.

Methodology

The present study was planned in Department of General Medicine, Indira Gandhi Institute of Medical Sciences, Patna, Bihar. The 50 cases of the acute myocardial infraction were enrolled and evaluated. Detailed history was taken in each patient and information of every patient was recorded in a separate proforma. Informed consent was obtained from all patients. The serum cardiac enzyme level was measured and ECG was done at the time of admission and repeated as necessary.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

All patients diagnosed as Acute Coronary Syndrome according to current guidelines, were included in the study. All patients with stable angina, cerebrovascular disease or stroke, acute or chronic liver disease, renal impairment or age less than 18 or above 40 years were excluded from the study. Patients having prior cardiac conditions that could affect outcome like valvular heart disease, cardiomyopathy.

Results & Discussion

Myocardial infarction (MI) is a pathological process due to a compromise in the blood supply of such severity to an area of myocardium that adequate oxygen cannot be obtained even after prolonged rest [21]. Acute coronary syndrome has two subtypes which refer to acute myocardial infarction, namely non-ST-elevated myocardial infarction and ST levated myocardial infarction, which are not always but most frequently a manifestation of coronary artery disease (CAD) [22]. Coronary artery disease is a chronic disease with stable and unstable periods. During unstable periods patients may develop MI with the involvement of activated inflammation in vascular wall. A myocardial infarction may be the first manifestation of CAD and in patients with established disease, it may occur repeatedly [23]. MI means, a part of heart muscle loses its blood supply due to sudden formation of blood clot in coronary artery, leading to death of the affected part if no prompt treatment is devised. It may sometimes call as coronary thrombosis or more commonly heart attack [24, 25].

Cardiovascular disease (CVD) is the leading cause of death and disability worldwide. It is expected that by 2020, CVD would prevail as the leading cause of death and disability over infectious diseases globally [26]. Cardiovascular disease encompass atherosclerotic vascular diseases like coronary heart disease (CHD), cerebrovascular disease (CBVD), and peripheral arterial diseases. In recent years, demographics and health surveys have reported increasing malaise of CVD among individuals of all socioeconomic strata. According to recent statistics, incidences of CVD-related death and disability in low-income countries have grown at an alarming pace.

In 2008, Gupta *et al*. reported that India alone is burdened with approximately 25% of cardiovascular-related deaths and would serve as a home to more than 50% of the patients with heart ailments worldwide within next 15 years [27]. The seriousness of current scenario could be gauged by the fact that most CVD sufferers in India happens to be in their productive age which may potentially impose huge socioeconomic burden and devastating consequences over the coming years. In 2005, Reddy *et al*. reported that India has incurred the highest loss in productive years of life worldwide [28]. Presently, the greatest public health challenge to developing countries is to control epidemics of chronic noncommunicable diseases, specifically CVD, CHD, diabetes and stroke which have caused almost doubled mortality rates than other communicable diseases in India [29].

Table 1: Demographic Details

Age	No. of Cases
21 – 30 years	1
31 – 40 years	7
41 – 50 years	10
51 – 60 years	20
61 – 70 years	8
71 & above years	4
Total	50
Sex	
Males	35
Females	15
Total	50

Table 2: Predisposing Factors

Factors	No. of Cases
Social class	
Lower	29
Middle	10
Upper	11
Residential Status	
Rural	41
Urban	9
Medical History	
No	23
Family CHD History	13
Hypertension	9
Diabetes mellitus	5
Consumption of Tobacco	
No	16
Chewing	10
Smoking	24
Alcohol consumption	
No h/o alcohol consumption	22
Less than once a week	13
1-2 times a week	12
3times/week to daily	3
Occupational physical activity	
Light (sedentary)	27
Moderate	21
Heavy	2
Body mass index	
18.5 - 24.99	29
25 - 29.99	17
30 - 39.99	4

Hypertension, smoking, and dyslipidemia were leading cause of deaths and other major hazardous outcome mainly attributed to smokers and dyslipdemia. There are numerous case-control studies exist in India. The largest of these case-control studies is the Interheart study [30]. This study was performed in 27 000 cases of acute myocardial infarction and controls in 52 countries of the world. This study reported that standard risk factors such as smoking, abnormal lipids, hypertension, diabetes, high waist-hip ratio, sedentary lifestyle, psychosocial stress, and a lack of consumption of fruit and vegetables explained more than 90% of acute CHD events in South Asians. Another study, the Interstroke study [32] reported 10 common risk factors explained more than 90% of incident hemorrhagic and thrombotic strokes, Similar conclusions were reached in smaller case-control studies [31].

Recently, a case- control study by Panwar R B *et al*. showed that the thrombotic (smoking, low fruit/vegetables intake, fibrinogen, homocysteine) as well as atherosclerotic (hypertension, high fat diet, dyslipidemia) risk factors were important in premature coronary heart disease [33]. Reviews of epidemiological studies suggest that all the major cardiovascular risk factors are increasing in India. In this study, most of young patients had risk factors like smoking, hypertension and dyslipidemia. Obesity noted to double the prevalence of cardiovascular disease in men and women under the age of 50 years, has been reported between 30% to 58% of younger patients [34, 36]. Interestingly, a much higher percentage of young patients (almost 20%) were unaware of their hypertension, dyslipidemic status before the index MI and, thus, were not able to benefit from prior therapeutic interventions. Younger patients were more likely to have an MI as their first event (70.5%), whereas heart failure was a more common first event in older patients (60.5%). Importantly, the relative proportion of sudden death events was similar across age groups.

However, careful scrutiny of available scientific evidence for modifiable CAD risk factors (elevated serum total and low-density lipoprotein cholesterol [LDL-C], low high-density lipoprotein cholesterol [HDL-C], smoking, diabetes, hypertension, low level of physical activity, and obesity) in this population may be helpful in formulating a more immediate CAD prevention strategy. A cost-effective preventive strategy is needed to focus on reducing risk factors both in the individual and in the population at large.

Conclusion

The data generated from the present study concludes that smoking, sedentary lifestyle and low HDL levels were the most common conventional risk factors found in the study population especially in younger age group. Stress did not contribute significantly. There was no statistically significant difference in risk factors between rural and urban population. Large case-control studies with multivariate logistic regression are needed to stratify, which conventional risk factor independently contributes the most in occurrence of coronary artery disease.

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