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Assessment of factors responsible and clinical outcomes in patients diagnosed with acute myocardial infarction from Bihar region

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Abstract
Myocardial Infarction (MI) also known as heart attack is one of the most common non-communicable diseases/ chronic diseases, which cannot be transmitted from person to person. It gradually progresses and affects the blood supply to heart. A myocardial infarction is a serious medical emergency that occurs due to the blockage of one of the arteries which is supplying blood to the heart. Due to which, lack of oxygen to heart causes characteristic chest pain and death of myocardial tissue. Hence based on above findings the present study was planned for Assessment of Factors Responsible and Clinical Outcomes in Patients Diagnosed with Acute Myocardial Infarction from Bihar Region.
Total 50 cases of the patients diagnosed with the Myocardial Infarction from the both the sexes were included in the present study. The present study was planned in Department of Pharmacology, Anugrah Narayan Magadh Medical College, Gaya, Bihar, India. The study was planned from duration of October 2018 to November 2019.
The data generated from the present study concludes that Better management of co-morbid condition like diabetes melitus, hypertension, obesity etc. and better access to treatment facilities for diagnosis and intervention thereof are need of the hour. A number of elderly patients reported 12hrs after experiencing chest pain, compared to younger ones who appeared much earlier, a typical symptoms like breathlessness nausea/vomiting were more common in elderly.

Keywords: acute myocardial infarction, thrombolytics, fibrinolytic, acute coronary syndrome, etc

Introduction
Myocardial infarction (MI) (ie, heart attack) is the irreversible death (necrosis) of heart muscle secondary to prolonged lack of oxygen supply (ischemia). Initial stabilization of patients with suspected MI and ongoing acute chest pain should include administration of sublingual nitro glycerin 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.
Coronary care units have reduced early mortality rates from acute MI by approximately 50% by providing immediate defibrillation and by facilitating the implementation of beneficial interventions. These interventions include the administration of intravenous (IV) medications and therapy.
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. 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 ongoing 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].

1. Detection of an increase or decrease in cardiac biomarker values (preferably using cardiac troponin [cTn]) with at least one value above the 99th percentile of the upper reference limit (URL).
2. Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic changes or injury or new BBB on ECG, but death occurred before cardiac biomarker levles were obtained, or before cardiac biomarker values would be increased.

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 remodelling [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 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. 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. 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. Myocardial Infarction (MI) also known as heart attack is one of the most common non-communicable diseases/ chronic diseases, which cannot be transmitted from person to person. It gradually progresses and affects the blood supply to heart. A myocardial infarction is a serious medical emergency that occurs due to the blockage of one of the arteries which is supplying blood to the heart. Due to which, lack of oxygen to heart causes characteristic chest pain and death of myocardial tissue. Hence based on above findings the present study was planned for Assessment of Factors Responsible and Clinical Outcomes in Patients Diagnosed with Acute Myocardial Infarction from Bihar Region.

Methodology

Total 50 cases of the patients diagnosed with the Myocardial Infarction from the both the sexes were included in the present study. The present study was planned in Department of Pharmacology, Anugrah Narayan Magadh Medical College, Gaya, Bihar, India. The study was planned from duration of October 2018 to November 2019. 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. Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria

Patients diagnosed with the Myocardial Infarction from the both the sexes.

Exclusion Criteria

Patients with rein farction, history of renal disorders, congenital cardiac defects, cardiomegaly, and peripheral vascular diseases.

Results and Discussion

Acute myocardial infarction (AMI) is one of the most common diagnoses in hospitalized patients in industrialized countries. Patients with ischemic heart disease are divided into two large groups: patients with chronic coronary artery disease (CAD), presenting commonly with stable angina and patients with acute coronary syndromes (ACSs). The latter group is composed of patients with acute myocardial infarction (MI) with ST-segment elevation on their presenting electrocardiogram (ECG) (STEMI), those with unstable angina (UA) and non-ST-segment elevation MI (NSTEMI). MI is a clinical diagnosis based on the presence of myocardial injury or necrosis as indicated by a rise and fall of serum cardiac biomarkers. In unstable angina, myocardial injury is absent and cardiac biomarkers are normal. In myocardial infarction (MI) [both NSTEMI and STEMI] cardiac biomarkers are raised. In the recent decade, several observations led to the confirmation of the benefits of evaluation and monitoring of ST resolution indices after occurrence of STEMI. For the first time, Schroder *et al* found that ST resolution predicts the risk of mortality and congestive heart failure in patients treated by fibrinolytic therapy, effectively [18-19]. Another studies confirmed the relationship between the degree of ST resolution and mortality. In another study by Ito *et al*., it was determined that epicardial normal blood store is not sufficient for evaluation and providing adequate myocardial reperfusion. [20-21] In fact, new reperfusion regimens were developed to improve the limitations of two fibrinolytic and anticoagulant therapies [22-23]. and these therapies may be particularly useful in providing coronary micro-circulation [24]. ST resolution is currently evaluated in many clinical trials and managing patients. Primary studies on ST resolution showed this fact that patients with sharper ST resolution had a lower level of infarction compared to the patients with stable ST elevation [25].

Table 1: Demographic Details

Age	No. of Cases
21 – 30 years	1
31 – 40 years	5
41 - 50 years	12
51 – 60 years	22
61 – 70 years	6
71 & above years	4
Total	50
Sex	
Males	22
Females	28
Total	50

Table 2: Comorbidities

Comorbidities	No. of Cases
Diabetes	16
Hypertension	18
COPD	2
Others	14
Total	50

Table 3: ECG changes

ECG changes	No. of Cases
T Wave inversion	3
Heart Block	2
ST segment depression	4
NSTE MI	3
STE MI	38
Total	50

Table 4: Associated symptoms

Symptoms	No. of Cases
Sweating	30
Breathlessness	26
Nausea/vomiting	28
Giddiness	12
Syncope	3
Palpitation	24
Altered sensorium	22
Pain in abdomen	8
Unconsciousness	1

Table 5: Clinical Outcomes

Clinical Outcomes	No. of Cases
Dead	2
Patients with Poor Prognosis	3
Thrombolysis, Stabilized & Discharged	45
Total	50

A study carried out by Aggarwal *et al*. (2010) also showed that, active family participation and support are one of the most important factors in following the regimen programs for cardiovascular patients after released from hospitals [26]. Therefore, involvement of families in patient education, especially chronic patients, such as cardiovascular patients who need comfort and appropriate psychological conditions can have a positive impact on improving patients' observance of regimen programs and the clinical outcomes accordingly.

Multivariate analysis by Lenzen MJ, Scholte op Reimer WJ, Boersma E, *et al*. showed that several of the predictors of death among patients with a preserved ejection fraction were related to those for patients with a decreased ejection fraction, as reported in previous studies [27]. But in another study by Yusuf S, Pfeffer MA, Swedberg K, *et al*. in the Candesartan in Heart Failure: Evaluation of Reduction in Mortality and Morbidity (CHARM) trials observed a variation in mortality between patients with preserved ejection fraction and those with decreased ejection fraction [28]. A substantial proportion of patients with coronary artery disease do not have traditional risk factors [29] of the disease. The common risk factors of atherosclerosis explain disease occurrence in only half of the diagnosed cases. In only 40% patients, risk factors modification inhibits the progression of atherosclerosis. This necessitates a context-specific and holistic model to explain the occurrence of AMI, including searching for new risk factors of atherosclerosis [30]. The present study identified 11 significant risk factors of AMI in the final model. These include conventional risk factors for coronary artery disease like obesity (estimated through waist-hip ratio and BMI), stress, hypertension, family history of CHD, tobacco smoking, raised total serum cholesterol, and past history of gingival sepsis. The INTERHEART study identified abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, consumption of fruits, vegetables, and alcohol, and regular physical activity account for most of the risk of myocardial infarction world-wide. The relationship between cholesterol and ischemic heart disease has been studied by the Prospective Studies Collaboration [31] wherein total cholesterol was positively associated with ischemic heart disease mortality in both middle and old age and at all blood pressure levels. The age-specific relevance of usual blood pressure to vascular mortality has been examined in a meta-analysis [32] wherein a meta-analysis of individual data for one million adults in 61 prospective studies was performed. The meta-analysis concluded that throughout middle and old age, usual blood pressure is strongly and directly related to vascular (and overall) mortality, without any evidence of a threshold down to at least 115/75 mmHg [32].

The pathophysiology of conventional risk factors of AMI is well understood. However, information on association of markers of infection and inflammation with AMI is sparse in this country. A wide variation in the prevalence of these microbial agents in different parts of the country; patient age and their level of immunity may modify this prevalence. Additionally, the extent of antibiotic usage in the treatment of other incidental infections may also alter this prevalence estimate. Consequently, we can expect a wide variation in the national prevalence of these infections among adults. If similar association between these agents and AMI is observed in other population groups in this country, this will have for reaching implications for the prevention and treatment of AMI [33].

Conclusion

The data generated from the present study concludes that Better management of co-morbid condition like diabetes melitus, hypertension, obesity etc. and better access to treatment facilities for diagnosis and intervention thereof are need of the hour. A number of elderly patients reported 12hrs after experiencing chest pain, compared to younger ones who appeared much earlier, a typical symptoms like breathlessness nausea/vomiting were more common in elderly.

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