REVIEW ARTICLE

Acute ST-elevation Myocardial Infarction in India: Diagnosis and Treatment

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ABSTRACT

Myocardial infarction (MI) is a condition associated with ischemic heart disease that can be fatal. It is brought on by sudden obstruction of a coronary artery, which usually results from the formation of a thrombus over an atherosclerotic plaque that has become dissected, ulcerated, or haemorrhaged, and causes myocardial ischemia necrosis. Despite therapeutic breakthroughs, it still has a high mortality rate (10–30%), with many deaths due to ventricular tachyarrhythmias happening in the first few hours; strangely, these can be readily avoided in an intensive care setting but happen before the patient arrives at the hospital. Based only on clinical considerations. The examination of chest pain, the primary symptom of MI, must be understood to recognise the condition. It might be challenging to determine the origin of chest discomfort because it is a symptom that is frequently present in outpatient clinics and emergency rooms. This review highlights the diagnosis and treatment procedures of acute ST-elevation myocardial infarction among Indian patients.

INTRODUCTION

Infectious diseases were less alarming at the turn of the twenty-first century, but chronic illnesses still affect many of the world's population. Contrary to their acute disease counterparts, most chronic diseases are caused mainly by lifestyle factors and can, for the most part, be reduced or prevented by lifestyle adjustments. Chronic diseases have one or more of the following traits: (a) they are persistent and leave behind residual disability; (b) they are brought on by irreversible pathological conditions; (c) they call for specialised patient rehabilitation training; or (d) they may be anticipated to need ongoing medical supervision, and observation, or care.Diabetes, cardiovascular diseases (CVDs), osteoporosis, arthritis, obesity, chronic obstructive lung disease, inflammatory bowel disease, illnesses of the central nervous system, and several malignancies are among the most prevalent chronic diseases that affect people globally.

The severity of the current situation can be assessed by the fact that most CVD patients in India are currently at a productive age, which has the potential to have disastrous socioeconomic repercussions in the years to come. One of the most common kinds of CVD is IHD. Angina and acute myocardial infarction are the two main symptoms of IHD[1-3]. It is believed that the term myocardial infarction (MI) refers to the death of cardiac myocytes brought on by persistent ischaemia. As a result, MI is an acute coronary syndrome that can develop as coronary atherosclerosis progresses naturally. Several factors that can result in mediating disorders or directly impact the artery wall can cause atherosclerosis to progress and can even accelerate its progression. Acute myocardial infarctions (AMIs), which can occur with or without ST elevation, are a subset of the ischemic heart disease (IHD) spectrum. IHD has a severe negative impact on human health.Over the course of more than 2 million years of human evolution, sodium and potassium have been crucial in the formation and maintenance of vital cellular functions[4–7]. Therefore, the objective of the current study was to evaluate the serum electrolyte levels in acute myocardial infarction (AMI) patients at a tertiary care facility.

Recent publications have discussed acute ST segment elevation myocardial infarction (STEMI) diagnosis and therapy based on evidence [8-11]. These are scholarly and comprehensive, but they are geared for the circumstances of industrialised nations. There is a need for adjustments in the interpretation of the evidence and administration of treatments to the Indian context due to the very different ground reality in India.

ANTI-PLATELET THERAPY

In acute myocardial infarction (AMI) exacerbated by cardiogenic shock (CS), the use of antiplatelet treatments and the capacity to achieve appropriate platelet inhibition at the time of percutaneous coronary intervention (PCI) are not extensively studied. The effects of CS on hemodynamics affect how well oral antiplatelet medicines work. The ability to produce significant platelet inhibition using oral antiplatelet drugs is impacted by decreased organ perfusion, poor absorption, hypothermia, analgesic administration, and delayed metabolism. Antiplatelet therapy's optimum cocktail is still up for dispute. Early, quick, and powerful platelet inhibition is necessary for patients receiving PCI for AMI in order to avoid peri-PCI thrombotic events. In patients with AMI-CS, STEMI, cardiac arrest, and multiorgan failure are common first presentations. Due to hemodynamic changes that decrease organ perfusion, affect oral absorption, and promote inflammation, CS challenges typical antiplatelet therapy after PCI and leads to lower platelet inhibition. Numerous investigations have confirmed the limited and delayed antiplatelet effects in such patients from a pharmacokinetic perspective. According to a prospective study by Adamski et al., who used liquid chromatography to measure antiplatelet activity in AMI patients, the plasma concentrations of P2Y12 inhibitors were one-third lower in STEMI patients than in non-STEMI patients. AMI-CS patients commonly undergo cardiopulmonary resuscitation, which causes inflammation that promotes platelet aggregation and lactic acidosis that lessens platelet inhibition. In this high-risk population, IV antiplatelet medicines should be strongly investigated, while keeping in mind the danger of bleeding, as they are even more efficacious than crushed oral antiplatelets. Despite the significant prevalence of multivessel coronary artery disease and the severe incidence of thrombosis, crushed and IV antiplatelet medications are rarely used in AMI-CS patients. To determine whether stronger antiplatelet inhibition will lead to better results in this high-risk population, studies are required.

DIALYSIS

Acute myocardial infarction (AMI) is a catastrophic occurrence with a poor prognosis in dialysis patients. It is likely that coexisting conditions contribute to the high mortality from cardiac disease and even higher overall mortality8,15; however, it is unclear how much of the difference of about 20 percentage points between overall mortality and mortality from cardiac causes at two years is due to coexisting conditions. According to numerous published reports of patients without end-stage renal disease who had acute myocardial infarctions, including patients with various underlying diseases, the survival of dialysis patients who had acute myocardial infarctions is significantly poorer than would be expected.Older age and diabetic nephropathy are the most effective independent predictors of overall death and mortality from cardiac causes. According to studies, people with acute myocardial infarction who are receiving dialysis had low long-term survival rates and a significant rate of cardiac-related deaths.

ASPIRIN DOSE

Arachidonic acid is not able to be turned into prostaglandin H₂, which in turn cannot be transformed into thromboxane (TX) A2 and other bioactive prostanoids because aspirin irreversibly inhibits platelet cyclooxygenase-1. TXA₂ is a platelet aggregator and vasoconstrictor that is produced and secreted by platelets. Aspirin limits platelet activity irreversibly by blocking TXA₂ production. Aspirin inhibits the formation of prostacyclin in vascular endothelial cells at higher levels, which, if unopposed, prevents platelet aggregation and causes vasodilation. The risk of bleeding is the main adverse effect of aspirin therapy, and there is strong evidence that aspirin side effects are dose dependant. In the upper digestive tract, aspirin is quickly absorbed, and within 60 minutes, there is a detectable reduction of platelet function. Due to platelets being exposed to aspirin in the portal circulation, this antiplatelet action happens even before acetylsalicylic acid is detected in the peripheral blood. Although aspirin has a short plasma half-life (20 minutes), its effects endure for the entire 10day platelet life because platelets(≈10 days) cannot produce new cyclooxygenase. Greater doses would be anticipated to suppress cyclooxygenase-2-dependent thromboxane synthesis in vascular endothelium, monocytes, and macrophages. Whereas platelet thromboxane synthesis is entirely stopped with a single dose of 100 mg, higher doses would be anticipated to do so. This might exacerbate the hemostasis deficit seen in people taking larger aspirin dosages.

DIAGNOSIS OF STEMI

Early treatment of STEMI depends on early diagnosis. In those who are susceptible, a history of chest pain or discomfort lasting 10 to 20 minutes should prompt suspicion of an acute STEMI (middle-aged male patients, particularly if they have risk factors for coronary disease). It is important to understand that pain might have unusual characteristics or locations. It is imperative to perform a 12-lead ECG as soon as feasible. Within ten minutes of arriving at the medical facility, the ECG should be evaluated. Repeat ECGs must be taken (every 15 minutes) and compared to the initial ECG if the initial ECG is not suggestive of STEMI but the patient still exhibits symptoms. Although myocardial necrosis markers are helpful in validating the diagnosis, it must be stressed that they might not be raised right away. When there is a question about the diagnosis, echocardiography may be a helpful adjuvant, especially in young patients without a history of coronary disease.

Differential Diagnosis - Musculoskeletal pain, oesophageal discomfort, mitral valve prolapse, pulmonary embolism, pleural pain, pericarditis, gastritis, cholecystitis, pancreatitis, neurological problems, and psychiatric symptoms are some of the more frequent causes of chest pain seen in clinical practice.

MANAGEMENT OF STEMI

RISK-BASED CLASSIFICATION

The quick identification of individuals who could be at high risk of cardiogenic shock or passing away should be a part of the first evaluation. The following traits have most frequently been linked to unfavourable outcomes in patients with STEMI [12-14]

- Older age (age \geq 75 years)
- Higher Killip class (class III or IV)
- Lower systolic blood pressure (100/min)
- Anterior MI

INITIAL TREATMENT

325 mg of (ideally) non-enteric-coated aspirin should be eaten as the first course of treatment. Aspirin should be given to every patient. All patients should receive a loading dose of 300–

600 mg of clopidogrel. [15-16]. Patients undergoing primary PCI ought to be given a loading dose of 600 mg[17]. Medication for pain relief should be given to all patients. When accessible, these might include opioid analgesics (such as morphine sulphate intravenously). If the systolic blood pressure is less than 120 mm Hg, nitrates should be delivered sublingually or intravenously. Nitrates must be used with caution if systolic blood pressure is greater than 100 mm Hg but less than 120 mm Hg. Other than aspirin, non-steroidal anti-inflammatory medications(NSAIDs) should not be administered for analgesia[18].

OPPORTUNITIES AND CHALLENGES FOR STEMI SYSTEMS OF CARE IN INDIA

Even while developing nations generally face several obstacles to enhancing STEMI care, we identify certain difficulties that are uniquely specific to India. These are mostly brought on by the unique design of its healthcare system, which is among the most privatised in the entire world. As a result, while a large portion of the population continues to rely on the country's underfunded public hospitals and healthcare systems, specialised centres in urban regions provide top-notch, cutting-edge care (which has helped to develop medical tourism as an industry) [19].

The first issue is that India's low overall healthcare spending per person- roughly half that of Sri Lanka, and a third of what China and Thailand [20] spend per person- has resulted in a lacklustre infrastructure for handling medical emergencies at the population level, particularly in rural areas. Prehospital emergency medical services (EMS), which were almost non-existent until recently, have shown this gap to be the most pronounced.Second, the underutilization of evidence-based therapies is a result of insufficient public and commercial health insurance programmes, which puts STEMI patients and their families at significant personal financial risk from treatments.

Thirdly, infectious disorders like respiratory and gastrointestinal infections continue to be prevalent in India. Future healthcare in India will partially depend on how it strikes a balance between many goals rather than concentrating solely on one. Finally, given India's low literacy rates and diverse society, educating the general public about the early symptoms of myocardial infarction is a significant problem. Despite these obstacles, recent clinical developments and regulatory adjustments have opened up fresh possibilities for enhancing STEMI care in India.

BETTER ACUTE REPERFUSION THERAPY

A significant step toward creating functional STEMI systems of care in the nation was the Drug Controller of India's approval of generic versions of the two medications denecteplase and reteplase. The outcomes and reperfusion rates of STEMI patients treated with these more recent, second-generation fibrinolytic medicines have improved. [21] In some circumstances, their bolus administration also enables the idea of prehospital care. The advantages of fibrinolytic therapy may be increased by new information on clopidogrel supplementary therapy. [22]The possibility of targeting PCI in high-risk patients is also raised by a better knowledge of the function of the pharmacoinvasive method, which combines fast pharmacological reperfusion with invasive cardiac treatments. [23] Like with medications, the accessibility of more modern, less priced generic Indian stents will greatly increase the number of people who can have PCI.

EMERGENCE OF EMS

The creation of the GVK EMRI ambulance system has been a notable healthcare achievement in India in recent years. It currently reaches both rural and urban areas and 372 million people across 11 states. According to G.V. Ramana Rao of the GVK Emergency

Management and Research Institute in a personal communication, there are about 2800 ambulances operating at GVK EMRI, making 4.5 trips daily at a cost of Rs 600 (about \$12). It will increase to 15,000 ambulances and cover up to 1 billion people over the following few years. Some regions have already started prehospital electrocardiography, incorporated chest pain algorithms into their protocols, and even provided uniform "cocktails" to start therapy. In order to further standardise care, STEMI patients may be transferred directly to larger facilities with catheterization labs rather than going through smaller hospitals. The low overall rates of EMS service utilisation in India continue to be a significant obstacle. For instance, about 5% of STEMI patients in CREATE sought ambulance assistance. [24]

Myocardial infarction (MI) is more common in middle-income countries (WHO study, 2004), and it is widely known that men are more likely to be affected than women. In the era of evidence-based medicine, biochemical markers, such as myoglobin, troponins, and creatine kinases, ischemia modified albumin (IMA), heart fatty acid-binding protein, hsCRP, brain natriuretic peptide, etc., have emerged as major factors in the diagnostic evaluation of patients with symptoms of myocardial infarction. Even though magnesium is the second-most abundant intracellular cation and is essential for more than 300 enzymatic activities involved in different metabolic processes in human body[6–7], [25-27] doctors frequently neglect this characteristic.Therefore, the objective of the current study was to evaluate the serum electrolyte levels in acute myocardial infarction (AMI) patients at a tertiary care facility.

The mean ages of the AMI patients and controls in the current study were 45.6 years and 42.7 years, respectively. Male patients made up 62 percent of the controls and 58 percent of the AMI group patients, respectively. Between AMI patients and controls, the mean serum potassium levels were 3.79 mEq/L and 4.49 mEq/L, respectively. Between AMI patients and controls, the mean blood sodium concentrations were 131.8 mEq/L and 139.4 mEq/L, respectively. AMI patients and controls had mean serum magnesium levels of 0.89 mmol/L and 1.23 mmol/L, respectively. The findings reported by Mizuguchi Y et al and Ramasamy R et al, who also got similar results, agreed with our findings[28, 29]. The predictive significance of these patients' entrance serum magnesium levels was studied by Mizuguchi Y et al. The statistics of 165 consecutive reperfused AMI patients were retrospectively examined. At check-in, the serum magnesium concentration was assessed. In-hospital death was the main result; 34 percent, or 54 individuals, passed away while being treated at the hospital (Fine & Gray's test; p <0.001). Higher serum magnesium levels were substantially associated with in-hospital death.

Even after adjusting for variables, multivariable logistic regression analyses showed that the serum magnesium level at admission was independently linked with in-hospital death (hazard ratio 2.68, 95 percent confidence range 1.24-5.80). Additionally, patients with higher serum magnesium levels than those with lower serum magnesium levels had significantly higher incidences of cardiogenic shock requiring an intra-aortic balloon pump (p = 0.005), extracorporeal membrane oxygenation (p<0.001), tracheal intubation (p<0.001), and persistent vegetative state (p = 0.002). The admission serum magnesium level in patients with reperfused AMI may be a viable substitute measure for predicting in-hospital mortality[28].

When the results of the current study were statistically analysed, it became clear that AMI patients' mean serum electrolyte levels were significantly different from those of the healthy controls. Serum Mg+ and other electrolytes were examined by Ramasamy R et al as adjuvant indicators in the diagnosis of AMI. In a case-control study, men from South India who had AMI within six hours of the onset of symptoms were included. There are 100 controls and 60 patients with AMI in the study.Using an electrolyte analyzer, serum electrolytes were calculated. Electrolyte levels in the serum for Ca, Mg, K, and Na were considerably lower (p<0.001) in AMI. In comparison to controls, the Ca:Mg, K:Mg, and Na:K ratios were all significantly greater (p<0.001). Serum magnesium levels significantly correlated with various

AMI cardiac indicators, including total CK, CK-Mb, and troponin T (p<0.05). Na:Mg (40.9), Ca:Mg (3.43), and K:Mg (2.74), according to ROC analysis, are the best cutoffs for diagnosing AMI. AMI may be diagnosed using serum Mg, Ca, K, and Na:K ratios as adjuvant markers[29].

CONCLUSION

This review article highlighted several useful findings. First, creating collaborations between hospitals is essential, and the journey time is just one of many variables to take into account. For instance, there is currently no method for interhospital transfer between private hospitals in the GVK EMRI ambulance system (although this is being addressed). Even with the existence of social health insurance, financial mechanisms must be developed for partnerships to grow. The cost of care is a crucial concern for hospitals as well as for individuals. It is obvious that specific agreements about shared pay between referral and hub hospitals must be implemented.

Third, problems with quality assurance and auditing must be addressed at all levels. Finally, and perhaps most crucially, it is obvious that India's context must be considered when using STEMI rules.Relying on North American and European norms presents challenges for Indian healthcare practitioners, particularly in areas with smaller and more remote institutions. A national organisation called STEMI INDIA that is committed to enhancing STEMI care in India was founded using the Kovai Erode Pilot STEMI program. Reviewing scientific research, educating, and training STEMI teams in hospitals, developing STEMI guidelines and systems of care appropriate for India, and eventually distributing information to the public to lessen delays are all goals of the initiative.

REFERENCES

- 1. Sheehan JP, Seelig MS. Interactions of magnesium and potassium in the pathogenesis of the cardiovascular disease. Magnesium. 1984 Jan 1;3(4-6):301-14.
- 2. Seelig MS, Heggtveit HA. Magnesium interrelationships in ischemic heart disease: a review. American Journal of Clinical Nutrition. 1974;27(1):59-79.
- 3. Speich M, Bousquet B, Nicolas G. Concentrations of magnesium, calcium, potassium, and sodium in human heart muscle after acute myocardial infarction. Clinical chemistry. 1980 Nov 1;26(12):1662-5.
- 4. Shimosawa T, Fujita T. Magnesium and N-type calcium channel. Clinical Calcium. 2005 Feb 1;15(2):239-44.
- 5. Lu L, Liu M, Sun R, Zheng Y, Zhang P. Myocardial infarction: symptoms and treatments. Cell biochemistry and biophysics. 2015 Jul;72(3):865-7.
- Čulić V, AlTurki A, Proietti R. Covid-19 pandemic and possible rebound phenomenon in incidence of acute myocardial infarction. Canadian Journal of Cardiology. 2021 Aug 1;37(8):1294.
- 7. White HD, Chew DP. Acute myocardial infarction. The Lancet. 2008 Aug 16;372(9638):570-84.
- 8. Writing Committee Members, Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). Journal of the American College of Cardiology. 2004 Aug 4;44(3):671-719.
- 9. American College of Cardiology/American Heart Association Task Force on Practice Guidelines, Developed in Collaboration With the Canadian Cardiovascular Society,

Endorsed by the American Academy of Family Physicians, 2007 Writing Group to Review New Evidence and Update the ACC/AHA 2004 Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction, Writing on Behalf of the 2004 Writing Committee, Antman EM, Armstrong PW, Green LA, Halasyamani LK, Hochman JS, Krumholz HM, Lamas GA. 2007 focused update of the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction. Journal of the American College of Cardiology. 2008 Jan 15;51(2):210-47.

- 10. Kushner FG, Hand M, Smith SC, King SB, Anderson JL, Antman EM, Bailey SR, Bates ER, Blankenship JC, Casey DE, Green LA. 2009 focused updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction (updating the 2004 guideline and 2007 focused update) and ACC/AHA/SCAI guidelines on percutaneous coronary intervention (updating the 2005 guideline and 2007 focused update) a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Journal of the American College of Cardiology. 2009 Dec 1;54(23):2205-41.
- 11. Tjandrawidjaja MC, Fu Y, Westerhout CM, White HD, Todaro TG, Van de Werf F, Mahaffey KW, Wagner GS, Granger CB, Armstrong PW. Resolution of ST-segment depression: a new prognostic marker in ST-segment elevation myocardial infarction. European heart journal. 2010 Mar 1;31(5):573-81.
- 12. Lee KL, Woodlief LH, Topol EJ, Weaver WD, Betriu A, Col J, Simoons M, Aylward P, Van de Werf F, Califf RM. Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction: results from an international trial of 41 021 patients. Circulation. 1995 Mar 15;91(6):1659-68.
- 13. Morrow DA, Antman EM, Charlesworth A, Cairns R, Murphy SA, de Lemos JA, Giugliano RP, McCabe CH, Braunwald E. TIMI risk score for ST-elevation myocardial infarction: a convenient, bedside, clinical score for risk assessment at presentation: an intravenous nPA for treatment of infarcting myocardium early II trial substudy. Circulation. 2000 Oct 24;102(17):2031-7.
- 14. Fox KA, Dabbous OH, Goldberg RJ, Pieper KS, Eagle KA, Van de Werf F, Avezum Á, Goodman SG, Flather MD, Anderson FA, Granger CB. Prediction of risk of death and myocardial infarction in the six months after presentation with acute coronary syndrome: prospective multinational observational study (GRACE). bmj. 2006 Nov 23;333(7578):1091.
- 15. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. Journal of the American College of Cardiology. 1988 Dec;12(6SA):A3-13.
- 16. Chen Z. COMMIT (ClOpidogrel and Metoprolol in Myocardial Infarction Trial) collaborative group. Addition of clopidogrel to aspirin in 45,852 patients with acute myocardial infarction: randomised placebo-controlled trial. Lancet. 2005;366:1607-21.
- 17. Sabatine MS, Cannon CP, Gibson CM, López-Sendón JL, Montalescot G, Theroux P, Claeys MJ, Cools F, Hill KA, Skene AM, McCabe CH. Addition of clopidogrel to aspirin and fibrinolytic therapy for myocardial infarction with ST-segment elevation. New England Journal of Medicine. 2005 Mar 24;352(12):1179-89.
- 18. von Beckerath N, Taubert D, Pogatsa-Murray G, Schömig E, Kastrati A, Schömig A. Absorption, metabolization, and antiplatelet effects of 300-, 600-, and 900-mg loading doses of clopidogrel: results of the ISAR-CHOICE (Intracoronary Stenting and Antithrombotic Regimen: Choose Between 3 High Oral Doses for Immediate Clopidogrel Effect) Trial. Circulation. 2005 Nov 8;112(19):2946-50.

- 19. Gislason GH, Jacobsen S, Rasmussen JN, Rasmussen S, Buch P, Friberg J, Schramm TK, Abildstrom SZ, Køber L, Madsen M, Torp-Pedersen C. Risk of death or reinfarction associated with the use of selective cyclooxygenase-2 inhibitors and nonselective nonsteroidal antiinflammatory drugs after acute myocardial infarction. Circulation. 2006 Jun 27;113(25):2906-13.
- 20. Kumar AS, Chen LC, Choudhury M, Ganju S, Mahajan V, Sinha A, Sen A. Financing health care for all: challenges and opportunities. The Lancet. 2011 Feb 19;377(9766):668-79.
- 21. Alexander T, Mehta S, Mullasari A, Nallamothu BK. Systems of care for ST-elevation myocardial infarction in India: is it time?. Heart. 2011 Jan 1.
- 22. Sabatine MS, Cannon CP, Gibson CM, López-Sendón JL, Montalescot G, Theroux P, Claeys MJ, Cools F, Hill KA, Skene AM, McCabe CH. Addition of clopidogrel to aspirin and fibrinolytic therapy for myocardial infarction with ST-segment elevation. New England Journal of Medicine. 2005 Mar 24;352(12):1179-89.
- 23. Antman EM, Van de Werf F. Pharmacoinvasive therapy: the future of treatment for ST-elevation myocardial infarction. Circulation. 2004 Jun 1;109(21):2480-6.
- 24. Xavier D, Pais P, Devereaux PJ, Xie C, Prabhakaran D, Reddy KS, Gupta R, Joshi P, Kerkar P, Thanikachalam S, Haridas KK. Treatment and outcomes of acute coronary syndromes in India (CREATE): a prospective analysis of registry data. The Lancet. 2008 Apr 26;371(9622):1435-42.
- 25. Al-Quthami AH, Udelson JE. What is the "goal" serum potassium level in acute myocardial infarction?. American journal of kidney diseases. 2012 Oct 1;60(4):517-20.
- 26. Daubert MA, Jeremias A. The utility of troponin measurement to detect myocardial infarction: review of the current findings. Vascular health and risk management. 2010;6:691.
- 27. Singh RB, Pella D, Neki NS, Chandel JP, Rastogi S, Mori H, Otsuka K, Gupta P. Mechanisms of acute myocardial infarction study (MAMIS). Biomedicine & pharmacotherapy. 2004 Oct 1;58:S111-5.
- 28. Mizuguchi Y, Konishi T, Nagai T, Sato T, Takenaka S, Tada A, Kobayashi Y, Komoriyama H, Kato Y, Omote K, Sato T. Prognostic value of admission serum magnesium in acute myocardial infarction complicated by malignant ventricular arrhythmias. The American Journal of Emergency Medicine. 2021 Jun 1;44:100-5.
- 29. Ramasamy R, Murugaiyan SB, Gopal N, Shalini R. The prospect of serum magnesium and an electrolyte panel as an adjuvant cardiac biomarker in the management of acute myocardial infarction. Journal of clinical and diagnostic research: JCDR. 2013 May;7(5):817.