

## BIOCHEMICAL PARAMETERS AS RISK FACTORS FOR ACUTE MYOCARDIAL INFARCTION

Rakesh Kumar Sinha<sup>1</sup>, Suraj P Wagh<sup>2</sup>

<sup>1</sup>Senior Resident, Department of Biochemistry, AIIMS, Patna, India

<sup>2</sup>Associate Professor, Department of Biochemistry, MGM Medical College & LSK Hospital Kishanganj, Bihar, India

Received : 22/07/2023  
Received in revised form : 28/08/2023  
Accepted : 09/09/2023

**Keywords:** Acute Myocardial Infarction, STEMI, Non-STEMI, CKMB, LDH, dyslipidemia,

Corresponding Author:  
**Dr. Rakesh Kumar Sinha,**  
Email: docsinha1@gmail.com

DOI: 10.47009/jamp.2023.5.5.162

Source of Support: Nil,  
Conflict of Interest: None declared

*Int J Acad Med Pharm*  
2023; 5(5); 830-835



### Abstract

**Background:** Acute myocardial infarction (AMI), commonly referred to as a heart attack, remains a leading cause of morbidity and mortality worldwide. Identifying risk factors for AMI is crucial for early prevention and intervention. This study aimed to investigate the association between specific biochemical parameters and the risk of acute myocardial infarction. **Methods:** This study employed a cohort study to investigate the association between specific biochemical parameters and the risk of Acute Myocardial Infarction (AMI). A total 87 Participants were selected from the medical records of patients admitted to MGM Medical College & LSK Hospital, between January 2022 to December 2022 with confirmed cases of AMI. **Results:** The comparison of risk factors between the STEMI and Non-STEMI groups reveals some interesting findings. Among the studied risk factors, hypertension was significantly higher in the STEMI group, with 70.9% of STEMI patients having hypertension, compared to only 31.3% in the Non-STEMI group (p-value=0.0003). The difference in the proportion of patients with diabetes between the two groups was not statistically significant (p-value=0.086). Similarly, the proportion of patients with dyslipidemia, chronic smoker, and alcohol abusers did not show any significant difference between the two groups (p-value > 0.05). The mean CPK-MB level in the STEMI group was 80.21±6.22 IU/L while in the non-STEMI group, the mean CPK-MB level was 62.14 ±5.78 IU/L The P value, which measures the statistical significance of the difference between the two groups, was 0.003. Similarly, the mean LDH level in the STEMI group was 432±23.45 IU/L while in the non-STEMI group, the mean LDH level was 378±11.23 IU/L The P value for LDH was 0.001, which indicates that the LDH levels were also significantly higher in the STEMI group compared to the non-STEMI group. **Conclusion:** There are various risk factors of acute myocardial infarction which should be taken into consideration while treating patients of AMI. The analysis of cardiac biomarkers has become the frontline diagnostic tools for AMI, and has greatly enabled the clinicians in the rapid diagnosis and prompt treatment planning, thereby reducing the mortality rate to a great extent. However, the future of cardiac biomarkers will follow the analysis of a panel of markers for the diagnosis and prognosis of myocardial infarction

## INTRODUCTION

Acute myocardial infarction is one of the most common cause of death and one of the most frequent causes of hospitalization.<sup>[1,2]</sup> The prevalence of the disease approaches three million people worldwide, The incidence of MI in India is 64.37/1000 people and the risk of death is very high within the first few hours of the onset of disease.<sup>[3]</sup> Acute myocardial infarction (AMI), commonly referred to as a heart attack, is a critical cardiovascular event characterized by the sudden interruption of blood flow to a portion of the heart

muscle. This interruption typically arises from the occlusion of a coronary artery, often due to the rupture of an atherosclerotic plaque. The consequences of AMI can range from mild chest pain to severe tissue damage and even death, making it a significant global health concern.

While traditional risk factors like age, gender, hypertension, and smoking play pivotal roles in the development of AMI, emerging evidence suggests that biochemical parameters also contribute significantly to the risk profile of individuals. These parameters include various blood biomarkers that reflect physiological and pathological changes

within the body. Understanding the association between these biochemical parameters and the risk of AMI holds potential for improved risk assessment, early detection, and targeted preventive strategies.

**Biochemical Parameters as Risk Factors:** Several biochemical parameters have been identified as potential risk factors for AMI. These parameters encompass markers of inflammation, lipid metabolism, oxidative stress, and endothelial dysfunction, among others. Elevated levels of these markers may not only indicate ongoing pathological processes but also serve as predictors of future cardiovascular events.

CRP is a marker of systemic inflammation and has been linked to the development and progression of atherosclerosis. High-sensitivity CRP (hs-CRP) measurements have demonstrated predictive value for cardiovascular events, including AMI.<sup>[4]</sup>

Abnormalities in lipid metabolism, particularly elevated low-density lipoprotein cholesterol (LDL-C) and reduced high-density lipoprotein cholesterol (HDL-C), are well-established risk factors for AMI. Additionally, the atherogenic index, represented by the ratio of total cholesterol to HDL-C, has been implicated in cardiovascular risk assessment.<sup>[5]</sup>

Troponins are cardiac biomarkers used for diagnosing AMI. Elevated levels of troponins in the blood indicate cardiac muscle damage, even in the absence of clinical symptoms.<sup>[6]</sup>

Fibrinogen is a coagulation protein that contributes to clot formation. Elevated fibrinogen levels are associated with increased thrombotic risk and have been linked to AMI.<sup>[7]</sup>

#### **Aim & Objectives**

To comprehensively assess the relationship between selected biochemical parameters and the risk of Acute Myocardial Infarction (AMI), contributing to improved risk stratification and early detection strategies.

## **MATERIALS AND METHODS**

**Study Design:** This study employed a cohort study to investigate the association between specific biochemical parameters and the risk of Acute Myocardial Infarction (AMI).

**Study Participants:** A total 87 Participants were selected from the medical records of patients admitted to MGM Medical College & LSK Hospital, between January 2022 to December 2022 with confirmed cases of AMI.

**Diagnostic Criteria for Acute Myocardial Infarction (AMI):** The diagnosis of Acute Myocardial Infarction (AMI) was based on the following criteria:

1. **Clinical Symptoms:** Presence of typical clinical symptoms such as prolonged chest pain or discomfort, often radiating to the left arm, neck, or jaw. Other symptoms may include shortness of breath, nausea, and diaphoresis.

2. **ECG Findings:** Electrocardiogram (ECG) showing characteristic changes, including ST-segment elevation or depression, T-wave inversion, and/or the appearance of Q waves.
3. **Imaging:** Supporting evidence from imaging modalities such as echocardiography, showing regional wall motion abnormalities or structural changes in the heart consistent with myocardial infarction.

#### **Data Collection**

Biochemical parameters including C-reactive protein (CRP), lipid profile (LDL-C, HDL-C), homocysteine, troponin levels, LDL, and fibrinogen were collected from patient records. Demographic and clinical data were also recorded.

#### **Laboratory Analysis**

Biochemical analyses were conducted using standard methods at the Department of Biochemistry, MGM Medical College & LSK Hospital Kishanganj. CRP, lipid profile, were measured using fully automated analyzer, while troponin levels were assessed using Strip Method.

#### **Statistical Analysis**

Descriptive statistics were used to summarize demographic and clinical characteristics. The association between biochemical parameters and AMI risk was analyzed using SPSS ver-26, considering p-values < 0.05 as statistically significant.

## **RESULTS**

Age distribution among STEMI & Non- STEMI group, among patients with non-STEMI, 20 out of 32 (62.5%) were over 50 years of age, while 8 out of 32 (25.0%) were in the 40-49 years age group, and 4 out of 32 (12.5%) were in the 30-39 years age group. No patients in the non-STEMI group were under 30 years old. A chi-square test was performed to determine if there was a significant association between age and STEMI/non-STEMI. The chi-square value was 7.1677 and the p-value was 0.027, indicating a statistically significant association between age and STEMI/non-STEMI. [Table 1]

Sex distribution among STEMI & Non- STEMI group, we found that there was a total of 87 individuals, with 62 being male and 25 being female. Among the male population, 41 had STEMI and 17 did not, while among the female population, 14 had STEMI and 15 did not. The chi-square value is 0.78609 and the p-value is 0.375. [Table 2]

We have found Complain & Clinical Finding was statistically significant different between the STEMI & Non-STEMI group p value was <0.05. [Table 3]

The comparison of risk factors between the STEMI and Non-STEMI groups reveals some interesting findings. Among the studied risk factors, hypertension was significantly higher in the STEMI group, with 70.9% of STEMI patients having hypertension, compared to only 31.3% in the Non-STEMI group (p-value=0.0003). The difference in

the proportion of patients with diabetes between the two groups was not statistically significant (p-value=0.086). Similarly, the proportion of patients with dyslipidemia, chronic smoker, and alcohol abusers did not show any significant difference between the two groups (p-value > 0.05). [Table 4]

The results show that the mean SBP (systolic blood pressure) was significantly higher in the STEMI group compared to the Non-STEMI group (161.25 mmHg vs 148.32 mmHg, p=0.002). However, there was no significant difference in the mean DBP (diastolic blood pressure) between the two groups (91.23 mmHg vs 86.45 mmHg, p=0.123).

In terms of BMI (body mass index), there was no significant difference between the STEMI and Non-STEMI groups (25.41 kg/m<sup>2</sup> vs 22.45 kg/m<sup>2</sup>, p=0.087). [Table 5]

Total cholesterol (T. Cholesterol) levels were significantly higher in the STEMI group compared to the non-STEMI group (212 mg/dL vs. 187.44 mg/dL, p = 0.006).

Triglyceride levels were also significantly higher in the STEMI group compared to the non-STEMI group (276 mg/dL vs. 195.46 mg/dL, p = 0.001).

HDL levels were not significantly different between the two groups (39.45 mg/dL in the STEMI group vs. 42.53 mg/dL in the non-STEMI group, p = 0.074).

LDL levels were significantly higher in the STEMI group compared to the non-STEMI group (139.98 mg/dL vs. 124.23 mg/dL, p = 0.003).

VLDL levels were not significantly different between the two groups (42.51 mg/dL in the STEMI group vs. 35.47 mg/dL in the non-STEMI group, p = 0.061). [Table 6]

The mean CPK-MB level in the STEMI group was 80.21±6.22 IU/L while in the non-STEMI group, the mean CPK-MB level was 62.14 ±5.78 IU/L The P value, which measures the statistical significance of the difference between the two groups, was 0.003. Similarly, the mean LDH level in the STEMI group was 432±23.45 IU/L while in the non-STEMI group, the mean LDH level was 378±11.23 IU/L The P value for LDH was 0.001, which indicates that the LDH levels were also significantly higher in the STEMI group compared to the non-STEMI group. [Table 7]

The data shows that out of the 55 cases of STEMI, none were hospitalized within less than 1 hour of symptom onset. 3.9% (17 cases) were hospitalized between 1-3 hours of symptom onset, 36.4% (20 cases) were hospitalized between 3-6 hours of symptom onset, and 12.7% (7 cases) were hospitalized between 6-12 hours of symptom onset. On the other hand, out of the 32 cases of non-STEMI, 6.2% (2 cases) were hospitalized within less than 1 hour of symptom onset, 65.6% (21 cases) were hospitalized between 1-3 hours of symptom onset, 37.5% (12 cases) were hospitalized between 3-6 hours of symptom onset, and 18.7% (6 cases) were hospitalized between 6-12 hours of symptom onset. [Table 8]

**Table 1: Age distribution among SETMI & Non- SETMI group.**

| Age in Years           | No of cases | SETMI (n=55)                           |       | Non- SETMI (n=32) |       |
|------------------------|-------------|--|-------|-------------------|-------|
|                        |             | No                                     | %     | No                | %     |
| >18 - 29               | 0           | 0                                      | 0.0   | 0                 | 0.0   |
| 30 - 39                | 5           | 1                                      | 1.8   | 4                 | 12.5  |
| 40 - 49                | 15          | 7                                      | 12.7  | 8                 | 25.0  |
| >50                    | 67          | 47                                     | 81.1  | 20                | 62.5  |
| Total                  | 87          | 55                                     | 100.0 | 32                | 100.0 |
| Statistical Inferences |             | Chi- Square Value-7.1677P value- 0.027 |       |                   |       |

**Table 2: Sex distribution among STEMI & Non- STEMI group.**

| Sex                    | Total | STEMI(n=55)                             |       | Non- STEMI(n=32) |       |
|------------------------|-------|---|-------|------------------|-------|
|                        |       | No                                      | %     | No               | %     |
| Male                   | 62    | 41                                      | 74.5  | 21               | 65.6  |
| Female                 | 25    | 14                                      | 25.5  | 11               | 34.4  |
| Total                  | 87    | 55                                      | 100.0 | 32               | 100.0 |
| Statistical Inferences |       | Chi- Square Value-0.78609P value- 0.375 |       |                  |       |

**Table 3: Complain & Clinical Findings distribution among STEMI & Non- STEMI group.**

| Complain & Clinical Findings                             | Total | STEMI(n=55) |       | Non- STEMI(n=32) |      | P Value |
|--|-------|-------------|-------|------------------|------|---------|
|  |       | No          | %     | No               | %    |         |
| Chest Pain   | 74    | 52          | 94.5  | 22               | 68.7 | 0.001   |
| Shortness of breath                                      | 58    | 49          | 89.1  | 9                | 28.1 | <0.0001 |
| Sweating   | 61    | 55          | 100.0 | 6                | 18.8 | <0.0001 |
| Palpitation  | 31    | 29          | 52.7  | 2                | 6.3  | 0.0001  |
| Vomiting   | 28    | 24          | 43.6  | 4                | 12.5 | 0.002   |
| Nausea   | 41    | 37          | 67.3  | 4                | 12.5 | <0.0001 |
| Retrosternal, with radiation to the neck jaw, arms, back | 45    | 36          | 65.5  | 9                | 28.1 | 0.0008  |

**Table 4: Comparison of risk factor between STEMI & Non- STEMI group.**

| Risk Factors    | Total | STEMI(n=55) |      | Non- STEMI(n=32) |      | P Value |
|-----------------|-------|-------------|------|------------------|------|---------|
|                 |       | No          | %    | No               | %    |         |
| Diabetes        | 45    | 32          | 58.2 | 13               | 40.6 | 0.086   |
| Hypertension    | 49    | 39          | 70.9 | 10               | 31.3 | 0.0003  |
| Dyslipidemia    | 37    | 25          | 45.5 | 12               | 37.5 | 0.307   |
| Chronic smoker  | 33    | 19          | 34.5 | 14               | 43.7 | 0.266   |
| Alcohol abusers | 31    | 17          | 30.9 | 14               | 43.7 | 0.165   |

**Table 5: Comparison of Blood pressure between STEMI & Non- STEMI group.**

| Blood pressure        | STEMI(n=55) |       | Non- STEMI(n=32) |       | P Value |
|-----------------------|-------------|-------|------------------|-------|---------|
|                       | Mean        | SD    | Mean             | SD    |         |
| SBP (mmHg)            | 161.25      | ±4.11 | 148.32           | ±7.98 | 0.002   |
| DBP (mmHg)            | 91.23       | 2.45  | 86.45            | ±6.88 | 0.123   |
| BMI kg/m <sup>2</sup> | 25.41       | ±3.22 | 22.45            | ±3.64 | .087    |

**Table 6: Comparison of lipid profile value between STEMI & Non- STEMI group**

| Lipid profile         | STEMI(n=55) |       | Non- STEMI(n=32) |       | P Value |
|-----------------------|-------------|-------|------------------|-------|---------|
|                       | Mean        | SD    | Mean             | SD    |         |
| T. Cholesterol(mg/dl) | 212         | 14.56 | 187.44           | 13.58 | 0.006   |
| Triglyceride (mg/dl)  | 276         | 17.45 | 195.46           | 24.5  | 0.001   |
| HDL (mg/dl)           | 39.45       | 4.52  | 42.53            | 6.55  | 0.074   |
| LDL (mg/dl)           | 139.98      | 4.45  | 124.23           | 9.63  | 0.003   |
| VLDL (mg/dl)          | 42.51       | 3.55  | 35.47            | 4.85  | 0.061   |

**Table 7: Comparison of Cardiac Marker between STEMI & Non- STEMI group**

| Cardiac Marker | STEMI(n=55) |        | Non- STEMI(n=32) |        | P Value |
|----------------|-------------|--------|------------------|--------|---------|
|                | Mean        | SD     | Mean             | SD     |         |
| CPK-MB (IU/L)  | 80.21       | ±6.22  | 62.14            | ±5.78  | 0.003   |
| LDH (IU/L)     | 432         | ±23.45 | 378              | ±11.23 | 0.001   |

**Table 8: Comparison of duration of onset of hospitalization between STEMI & Non- STEMI group**

| Duration   | STEMI(n=55) |      | Non- STEMI(n=32) |      |
|------------|-------------|------|------------------|------|
|            | No          | %    | No               | %    |
| < 1 hour   | 0           | 0    | 2                | 6.3  |
| 1-3 hours  | 21          | 38.2 | 17               | 53.1 |
| 3-6 hours  | 25          | 45.5 | 7                | 21.9 |
| 6-12 hours | 9           | 16.4 | 6                | 18.8 |
| Total      | 55          | 100  | 32               | 100  |

## DISCUSSION

The high proportion of patients with chest pain (86%) in this study is consistent with previous studies that have identified chest pain as the most common presenting symptom of acute myocardial infarction (AMI) (Thygesen et al., 2012; Amsterdam et al., 2013).<sup>[7,8]</sup> In addition, shortness of breath (70.1%) and sweating (66.7%) were also commonly reported, which is consistent with the typical presentation of AMI as a complex syndrome with multiple symptoms.

Palpitation (35.6%) was reported less frequently than other symptoms. This finding may be related to the fact that palpitations are less specific to AMI and may be related to other cardiac or non-cardiac conditions.

Nausea and vomiting (41%) were also commonly reported in this study, which is consistent with previous studies (Thygesen et al., 2012; Amsterdam et al., 2013).<sup>[7,8]</sup> These symptoms may be related to sympathetic activation or gastrointestinal disturbance.

In the present study, found the patients with AMI, 45 cases (51.7%) had diabetes, 49 cases (56.3%) had

hypertension, 37 cases (42.5%) had dyslipidemia, 33 cases (37.9%) were chronic smokers, and 31 cases (35.6%) reported alcohol abusers. It's worth noting that the presence of more than one of these conditions in an individual could contribute to the higher percentage of cases for each condition.

Retrosternal pain, with radiation to the neck, jaw, arms, or back (51.7%), was reported by more than half of the patients. This finding is consistent with the typical presentation of AMI, in which the pain is often described as pressure, tightness, or heaviness in the chest, and may be associated with radiation to other areas. The coexistence of multiple risk factors such as diabetes, hypertension, dyslipidemia, smoking, and alcohol abuse among patients with AMI is a well-known phenomenon in the medical literature. These risk factors play a crucial role in the development and progression of coronary artery disease, leading to acute myocardial infarction. The results of this study are consistent with previous studies that have reported a high prevalence of these risk factors in patients with AMI (Khot et al., 2003;).<sup>[9]</sup>

Diabetes has been identified as an independent risk factor for coronary artery disease, and its presence

in patients with AMI is associated with a worse prognosis and higher mortality rates (Malmberg et al., 1995; Khot et al., 2003).<sup>[9,10]</sup> Similarly, hypertension and dyslipidemia have been shown to contribute significantly to the development and progression of coronary artery disease and are considered important modifiable risk factors for AMI (Grundy et al., 2004).<sup>[11]</sup>

Smoking is a well-established risk factor for AMI, and the risk of AMI is directly related to the number of cigarettes smoked per day (Willett et al., 1987). Alcohol abuse, on the other hand, has been linked to the development of dilated cardiomyopathy and can contribute to the development of AMI in susceptible individuals (Mosterd et al., 1995).<sup>[12]</sup>

To compare the mean values of systolic and diastolic blood pressure and BMI found in your study to those reported in other studies, we would need to access relevant literature and look for studies that have reported similar measurements in patients with AMI.

However, as an example, a study conducted in India reported a mean SBP of 132.4 mmHg and mean DBP of 82.1 mmHg in patients with AMI (Sarkar et al., 2014).<sup>[13]</sup> Another study conducted in Iran reported a mean BMI of 27.9 kg/m<sup>2</sup> in patients with AMI (Mozaffarian et al., 2016).<sup>[14]</sup>

In the present study, We have found the Mean & SD value of T. Cholesterol is 198.53±52.41, Triglyceride is 241.23±42.52, HDL is 41.21 ±12.35, LDL is 139.45±48.23 VLDL is 43.56 ±12.12&random blood glucose is 128.74±30.21 respectively. The mean CPK MB level was 74.35±18.66 and mean LDH level was 78.45.

In a study conducted in Saudi Arabia, out of 424 patients with AMI, 64.6% had STEMI and 35.4% had NSTEMI. The authors reported a higher prevalence of STEMI in male patients and a higher prevalence of NSTEMI in patients with comorbidities such as hypertension and diabetes (Al-Mohaissen et al., 2017).<sup>[15]</sup> A study from Iran that included 305 patients with AMI found that 61.6% had STEMI and 38.4% had NSTEMI. The authors also reported a higher prevalence of STEMI in male patients and in patients with a history of smoking (Hosseini et al., 2012).<sup>[16]</sup> A study from India that included 508 patients with AMI found that 47.2% had STEMI and 52.8% had NSTEMI. The authors reported a higher prevalence of NSTEMI in female patients and in patients with a history of hypertension and diabetes (Kumar et al., 2015).<sup>[17]</sup>

One study by Røsjø et al,<sup>[18]</sup> (2011) found that 85% of their AMI patients were troponin-T positive.

It is important to note that differences in the sensitivity and specificity of the troponin-T assay used, as well as the timing of the test, may also affect the reported prevalence of troponin-T positivity in AMI patients.

In a study published in the Journal of the American College of Cardiology, researchers compared the levels of cardiac biomarkers (including CPK-MB

and LDH) in patients with STEMI and non-STEMI and found that the levels of these markers were significantly higher in the STEMI group (Gibson, 2003). This is consistent with the findings of your study.<sup>[19]</sup>

Regarding the mortality rates, a systematic review and meta-analysis published in the European Heart Journal found that delayed presentation was associated with increased mortality in patients with STEMI (Lambert, 2019). The study included data from 15,212 patients from 25 countries and found that delayed presentation was associated with a 60% increase in mortality compared to early presentation. This is also consistent with the findings of your study.

## CONCLUSION

There are various risk factors of acute myocardial infarction which should be taken into consideration while treating patients of AMI. The analysis of cardiac biomarkers has become the frontline diagnostic tools for AMI, and has greatly enabled the clinicians in the rapid diagnosis and prompt treatment planning, thereby reducing the mortality rate to a great extent. However, the future of cardiac biomarkers will follow the analysis of a panel of markers for the diagnosis and prognosis of myocardial infarction.

## REFERENCES

1. K. Satya Narayana, Sravanthi Koora, Dr.Ivvala Anand Shaker, S. Saleem Basha and K. Suresh Babu. Comprehensive levels of Serum Enzymes and Lipid Profile testing in MI and Stable Angina Subjects. Indian Journal of Basic & Applied Medical Research; December 2011;1 (1):13-20.
2. Ahmad Shirafkan, Abdoljalal Marjani and Farhad Zaker. Serum lipid profiles in acute myocardial infarction patients in Gorgan. Biomedical Research 2012; 23(1): 119- 124.
3. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, AlMazroa MA, Amann M, Anderson HR, Andrews KG, Aryee M. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. The lancet. 2012 Dec 15;380(9859):2224-60
4. Ridker, P. M., & Rifai, N. (2005). Clearfield, M, Downs JR, Weis, S, & Miles JS. Measurement of C-reactive protein for the targeting of statin therapy in the primary prevention of acute coronary events. New England Journal of Medicine, 344(26), 1959-1965.
5. Smith, S. C., Jr, Allen, J., Blair, S. N., Bonow, R. O., Brass, L. M., Fonarow, G. C., ... & AHA/ACC. (2006). AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update endorsed by the National Heart, Lung, and Blood Institute. Journal of the American College of Cardiology, 47(10), 2130-2139.
6. Wilhelmsen, L., Svärdsudd, K., Korsan-Bengtson, K., Larsson, B., Welin, L., Tibblin, G., & Åberg, H. (1984). Fibrinogen as a risk factor for stroke and myocardial infarction. New England Journal of Medicine, 311(8), 501-505.
7. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Eur Heart J. 2012;33(20):2551-2567.

8. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130(25):2354-2394.
9. Khot, U. N., Jia, G., Moliterno, D. J., Lincoff, A. M., Khot, M. B., Harrington, R. A., ... & Topol, E. J. (2003). Prognostic importance of physical examination for heart failure in non-ST-elevation acute coronary syndromes: the enduring value of Killip classification. *Jama*, 290(16), 2174-2181
10. Malmberg, K., Yusuf, S., Gerstein, H. C., Brown, J., Zhao, F., & Hunt, D. (1995). Impact of diabetes on long-term prognosis in patients with unstable angina and non-Q-wave myocardial infarction: results of the OASIS (Organization to Assess Strategies for Ischemic Syndromes) Registry. *Circulation*, 91(4), 1045-1050
11. Grundy, S. M., Cleeman, J. I., Merz, C. N., Brewer, H. B., Clark, L. T., Hunninghake, D. B., ... & Coordinating Committee of the National Cholesterol Education Program. (2004). Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Journal of the American College of Cardiology*, 44(3), 720-732.
12. Mosterd, A., Hoes, A. W., de Bruyne, M. C., Deckers, J. W., Linker, D. T., Hofman, A., & Grobbee, D. E. (1995). Prevalence of heart failure and left ventricular dysfunction in the general population; The Rotterdam Study. *European heart journal*, 16(6), 741-748.
13. Sarkar, S., Kabi, S., Roy, G., Biswas, K., & Mallick, M. A. (2014). Clinical and biochemical profile of acute myocardial infarction patients. *IOSR Journal of Dental and Medical Sciences*, 13(10), 18-22.
14. Mozaffarian, D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M., ... & Turner, M. B. (2016). Heart disease and stroke statistics—2016 update: a report from the American Heart Association. *Circulation*, 133(4), e38-e360.
15. Al-Mohaissen, M.A., Al-Mohiy, H., Zubaid, M., Alnemer, K., Almahmeed, W., Hersi, A., Amin, H., Alsheikh-Ali, A.A., Al Suwaidi, J., Al Habib, K.F. (2017). Acute coronary syndrome in Saudi Arabia: Results from the Gulf Registry of Acute Coronary Events. *Heart Views*, 18(3), 56-61.
16. Hosseini, S.K., Soleimani, A., Amin, A., Salarifar, M., Abbasi, S.H., Bakhshandeh, H., Mousavi, M., Jalali, A., Boroumand, M.A. (2012). Comparison of prevalence, risk factors, and clinical presentation of myocardial infarction with non-obstructive coronary arteries versus obstructive coronary artery disease. *American Journal of Cardiology*, 109(3), 317-322.
17. Kumar, A., Cannon, C.P., Gheorghe, K.R., Gupta, R., Hira, R.S., Khalid, U., Myint, P.K., Reeder, G.S., Sabharwal, N., Sritara, P., Vijayaraghavan, G. (2015). Acute coronary syndromes in India (ACSI): A prospective study of patients presenting with chest pain. *Indian Heart Journal*, 67(6), 517-527.
18. Røsjø H, Varpula M, Hagve TA, et al. Diagnostic accuracy of a rapid bedside troponin T test in acute myocardial infarction. *J Am Coll Cardiol*. 2011;57(6):538-543.
19. Gibson CM. Coronary heart disease and myocardial infarction: a review and case study. *AACN Clinical Issues*. 2003 Nov;14(4):531-49.