The Review Of
DIABETIC
STUDIES

OPEN ACCESS

Clinical Study Of Conduction Block In Acute St Elevation Myocardial Infarction

Dr Parminder Singh Manghera*

*Assistant professor Cardiology, Government Medical college, Amritsar Punjab Parminder.manghera@gmail.com

Abstract

Background: There is clinical importance in identifying abnormalities as a complication of acute ST-elevation myocardial infarction (STEMI), but current-day statistics of conducting abnormalities during the post-reperfusion period are scarce, particularly in developing countries.

Objective: The purpose of this research was to examine the occurrence, trend, and prognostic significance of conduction abnormalities in STEMI and their relationship with the site of infarction and in-hospital outcomes.

Methods: In this cross-sectional study, 100 patients with acute STEMI were assessed by using a clinical approach, serial electrocardiography, and other pertinent biochemical examinations. To establish associations, chi-square and logistic regression tests were used to conduct a statistical analysis.

Results: Abnormalities in conduction were detected in 29% of patients, most often atrioventricular (AV) nodal blocks (74%), the first-degree AV block (27.6) and complete heart block (20.7) were the most common ones. AV nodal blocks (p < 0.05) were significantly associated with inferior-wall infarctions, with anterior-wall infarctions more frequently being complicated by intraventricular conduction delays. The majority of the conduction blocks (93.1) were present on admission and temporary. Permanent pacing was necessary in 5 % of cases, and mortality (4%) was much greater in patients with conduction disturbances (p = 0.031).

Conclusion: Conduction abnormalities are also still prevalent in STEMI, particularly in inferior-wall infarctions. Undertaking early detection, rhythm monitoring, and timely reperfusion are all important in minimising the morbidity and mortality of acute coronary care.

Keywords: Acute Myocardial Infarction, Atrioventricular Block, Conduction Disturbances, Electrocardiography, Reperfusion Therapy.

Introduction

Acute myocardial infarction (AMI) is among the leading causes of mortality and morbidity on the planet, regardless of significant achievements in cardiovascular care [1]. CVDs are the leading cause of death in the world, with ischemic heart disease causing a portion of mortality of about 16.7% of the total mortality [2]. This burden is ever on the increase because of the increasing prevalence of hypertension, diabetes mellitus, smoking, and dyslipidemia, in addition to inequality in access to cardiac care. ST-elevation myocardial infarction (STEMI) is the most severe manifestation of AMI, which is caused by the total occlusion of the coronary artery and the consequent extensive myocardial infarction [3].

STEMI triggers a series of ischemia, cell death, and electrophysiological impairment [4]. Conduction block is one of its most common and clinically important complications that can happen at any point along the cardiac conduction system, including the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His, and intraventricular routes [5]. These shocks take the form of AV blocks, bundle branch blocks or fascicular blocks, and these represent various locations of myocardial injury. They not only occur as diagnostic but also prognostic, meaning how much ischemic damage has taken place and how it is likely to lead to adverse outcomes [6].

Geographical differences in the involvement of coronary arteries determine the pattern of conduction abnormalities. Poor quality infarctions of the wall, often caused by right coronary artery occlusion, are

common causes of SA or AV nodal block, which is frequently temporary in nature [7]. Conversely, infarction of the anterior wall of the left anterior descending artery is more commonly linked to intraventricular conduction delays, including right bundle branch block (RBBB) or left anterior hemiblock (LAHB), which are also evidence of extensive myocardial infarction and worse prognosis [8]. Therefore, the nature of the conduction block offers a good understanding of the localisation and severity of the infarct, and can be helpful in the early recognition and management of risk [2].

Although there are developments concerning reperfusion therapy, especially through percutaneous coronary intervention (PCI) and thrombolytic therapy, conduction blocks are still a significant contributor to morbidity and mortality [9]. Although some abnormalities disappear with reperfusion, their existence normally indicates a large ischemic region and increased chances of complications like cardiogenic shock, arrhythmia and heart failure [10]. New-onset AV or bundle branch block in STEMI usually indicates critical myocardial infarction and is linked to augmented in-hospital fatality [11]. Therefore, in patients with Type 1 diabetes, continuous electrocardiographic monitoring is necessary for the early detection and treatment of such patients [12].

Conduction block in STEMI is caused by pathophysiological ischemic damage to specialised conduction tissue, augmented by autonomic imbalance, electrolyte imbalances and metabolic imbalance [13]. Hypoxia induced by ischemia affects impulse formation and conductance, whereas changes in autonomic tone affect conduction velocity and refractoriness [14]. Conduction abnormalities are usually self-limiting or may develop into irreversible injury, which in some cases leads to temporary pacing or permanent pacing [15]. These processes support the fact that structural myocardial injury is closely interconnected with electrical instability in STEMI.

In previous studies, it has been proven that certain conduction defects are associated with certain infarct territories and prognostic outcomes [16,17]. As an example, the presence of RBBB and LAHB is frequently related to the proximal left anterior descending artery occlusion, but left bundle block (LBBB) is often linked to multivessel disease [10]. This is because such electrical disturbances are surrogate indicators of coronary pathology that offer useful information about anatomical as well as functional myocardial compromise [18]. In the Indian scenario, the outcomes of patients affected by conduction disturbances are more significant through the influence of various factors, such as late presentations, inadequate access to more complex interventions, and the presence of a high proportion of comorbidities [19].

Conduction block as a prognostic factor in STEMI has been under-evaluated in developing regions. Information on tertiary care hospitals in North India is still lacking and therefore requires dedicated clinical investigations. In this research, the gap is filled by examining the incidence, distribution, and prognostic implications of conduction blocks among patients with STEMI hospitalised in one tertiary care centre in Amritsar. The study will be able to produce regionally applicable evidence to support clinical decisions by comparing the patterns of conduction with the site of infarction, demographics, and hospital outcomes. The characterisation of the nature and prevalence of conduction disturbances can enhance the early triage, risk stratification, and management protocols, especially in resource-constrained environments.

Finally, the conduction block in STEMI is not just an electrical complication per se; it is an electrophysiological manifestation of the underlying ischemic burden. Early identification of such abnormalities provides prognostic value and treatment advice. This study will add to the understanding of the dynamics of myocardial conduction in acute ischemia and will support the clinical importance of continuous cardiac monitoring in better survival and recovery of patients with STEMI.

Objectives of the Study

The main aim of the study is to establish the occurrence and localisation of conduction blocks in patients with acute ST-elevation myocardial infarction. The secondary goals are to match the type of conduction block with the location of infarct, demographic and clinical factors, and assess the implications of such factors in prognosis in terms of mortality and in-hospital outcomes.

Methodology

Study Design and Participants

One hundred sequential adult patients, both male and female, aged 30 years and above, who presented with acute ST-elevation myocardial infarction (STEMI) were recruited. The criteria used to establish the diagnosis were AHA/ESC, which needed characteristic ischemic pains of the chest with new ST-segment elevation at the J-point in at least two consecutive leads (≥0.1 mV in all but V2, V3, and 0.2 mV in V2, 0.25 mV in V3 and V new in V2 0.15 mV in V4).

Patients who had a known diagnosis of acute STEMI during the previous 24 hours were included. Exclusion: A prior conduction abnormalities or pre-existing cardiomyopathy, congenital or rheumatic heart disease, conduction delay caused by drugs (clonidine, methyldopa, verapamil or digoxin), and known electrolyte imbalance before admission. These exclusions were necessary to make sure that the conduct defects that were observed could be attributed to the acute ischemic event as opposed to underlying pathology.

Data Collection and Clinical Evaluation

A thorough clinical evaluation of all patients with a detailed history, risk factor profile, and systematic inspection was performed. The first 24 hours of electrocardiographic (ECG) monitoring were followed by continuous analysis with the additional 12-lead ECG recording every 12 hours and other tracings on the appearance of new symptoms or changes in rhythm.

The interpretation of ECGs was done by two senior cardiologically-trained physicians independently, and any disparity was sorted out by agreement with a senior cardiologist to guarantee accuracy in diagnosis. Standardised institutional tests and calibrated devices were used to conduct cardiac biomarkers (CK-MB, Troponin I/T), serum electrolytes, and usual biochemical studies. Patient care, such as reperfusion therapy (thrombolysis or percutaneous coronary intervention), pharmacologic therapy and pacing when necessary, was based on standard clinical practice and was not dependent on study enrollment.

Ethical considerations

It is a cross-sectional hospital-based study that was carried out at the Department of Medicine, Guru Nanak Dev Hospital, Government Medical College, Amritsar, between January 2021 and December 2022. The Institutional Ethics Committee gave ethical approval, and informed consent was signed by all participants. The research did not violate the standards of the Declaration of Helsinki.

Conduction Block in Acute STEMI

Definition: Conduction Block Conduction Block A conduction block is defined by a specific vibration and can be classified as an abrupt block, a gradual block, or a progressive block.

Conduction abnormalities were categorised using traditional electrophysiological definitions. These were sinoatrial block, first-, second, and third-degree atrioventricular (AV) block, and bundle branch or fascicular block. All the conduction disturbances were recorded with reference to the infarction site (anterior or inferior wall) and its progression throughout the hospitalisation.

The first and most important was the incidence and trend of conduction block in acute STEMI. The secondary outcomes were the correlation of the type of conduction block and infarct location, demographic and clinical factors and in-hospital outcomes (mortality, arrhythmias, and heart failure).

Sample Size Determination

Sample size was estimated using the standard formula for prevalence studies:

$$n = \frac{(Z_{1-\alpha/2})^2 pq}{d^2}$$

Where,

Z = 1.96 for 95% confidence

p = 34% (based on prior regional data)

q = 66%

d = 0.05

The calculated sample size was 87, and to accommodate potential dropouts, a total of 100 patients were included to maintain statistical power and representation.

Statistical Analysis

The data were inserted into a master database and analysed with SPSS software (Version 22, IBM Corp., Armonk, NY, USA). Continuous variables were given in terms of mean and standard deviation (SD), whereas categorical data were represented as frequency and percentage. Comparisons of conduction block patterns and clinical or demographic variables were considered with the Chi-square test or Student t-test, respectively. The p-value less than 0.05 was taken to be statistically significant. Binary logistic regression analysis was conducted as appropriate to provide independent predictors of conduction block by adjusting for the effects of possible confounders like age, sex, hypertension, diabetes, and smoking status.

Results

Demographic and Clinical Characteristics

The study included 100 patients who had acute ST-elevation myocardial infarction (STEMI). The cohort mean age was 52.04 (SD 8.66) age group with a range of 30-70 years, with 35 % in the 51-60-year age bracket. The number of males was higher, with a male-to-female ratio being about 4:1. The most common cardiovascular risk factors included hypertension (60 %) and diabetes mellitus (50 %), smoking (37 %), and dyslipidemia (22 %). Hypertension and diabetes were both found to have statistically significant associations with STEMI occurrence (p = 0.046 and 0.029, respectively). These results emphasise the association of metabolic and vascular risk factors in patients with acute myocardial infarction. Figure 1 presents the age distribution of the patients, with the highest number of patients being in the 51-60 years age bracket.

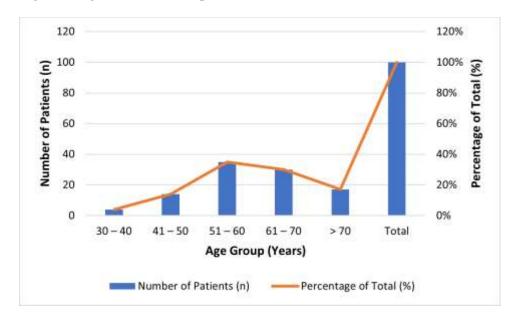


Figure 1. Age distribution of patients with acute STEMI

Distribution of Myocardial Infarction by Site

In terms of localisation of infarct, inferior-wall myocardial infarction (IWMI) was most common with a 54 ts or 34 ts rate, respectively, with the other types being anterolateral MI 6 ts, anteroseptal MI 3 ts and lateral-wall MI 2 ts. A single patient had been showing a combined superior and posterior wall infarction (1%). A worse-wall injury was more common in hypertensive and diabetic patients, and priorwall injury was most common in young men (Figure 2).

INFERIOR + POSTERIOR MI (IWMI + PWMI)

LATERAL-WALL MI (LWMI)

ANTEROSEPTAL MI (ASMI)

ANTEROLATERAL MI (ALMI)

ANTERIOR-WALL MI (AWMI)

INFERIOR-WALL MI (IWMI)

0% 20% 40% 60% 80% 100% 120%

Percentage of Total Cases (%)

Figure 2. Distribution of STEMI by infarct site

Incidence and Pattern of Conduction Abnormalities

The conduction abnormalities were noted in 29 % of patients with acute STEMI. First-degree AV block (27.6 %), complete heart block (20.7 %), second-degree AV block Mobitz I (10.4 %), Mobitz II (6.9 %) and 2:1 AV block (6.9 %) were the most common conduction abnormalities. Right bundle branch block (RBBB) and left anterior hemiblock (LAHB) were observed in 6.9 and 6.9, respectively, with another 6.9 being RBBB + LAHB. Left bundle of block (LBBB) and sinoatrial block were less common (3.4 and 3.4, respectively). In general, AV-nodal blocks were 74 % of the conduction disturbances, and intraventricular blocks were 26 %. Table 1 summarises the conduction disturbances and their distribution across the patients with acute STEMI.

Table 1. Distribution of conduction abnormalities in STEMI

Type of Conduction Block	No. of Cases	Percentage (%)
First-degree AV block	8	27.6
Complete heart block	6	20.7
Second-degree AV block (Mobitz I)	3	10.4
Second-degree AV block (Mobitz II)	2	6.9
2:1 AV block	2	6.9
RBBB	2	6.9
LBBB	1	3.4
LAHB	2	6.9
Combined RBBB + LAHB	2	6.9
SA block	1	3.4

Relationship Between Infarct Location and Conduction Block

The infarct site and the nature of conduction disturbance were significantly related (p < 0.05). Inferior-wall infarctions were mainly reported to be related to atrioventricular nodal blocks, such as first-degree, second-degree, and complete heart block, but anterior-wall infarction was more likely to have intraventricular conduction delays, such as RBBB, LBBB, and LAHB. This correlation indicates the variability of the anatomical and vascular platform of conduction defects among diverse infarction areas. Table 2 indicates that there was a strong relationship between the location of infarcts and the type of conduction block.

Table 2. Association Between Infarct Location and Conduction-Block Type

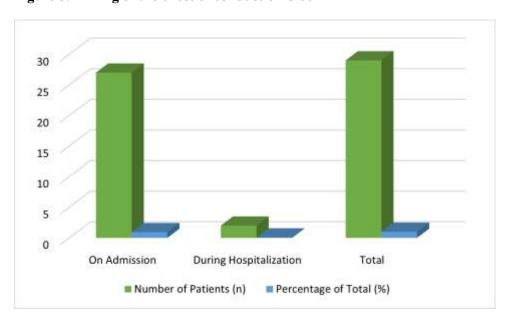
Type of Conduction Block	Inferior-Wall MI (IWMI)	Anterior-Wall MI (AWMI)	Total (n)
First-degree AV block	5	3	8
Second-degree AV block (Mobitz	4	1	5
I & II)			
Complete heart block	5	1	6
2:1 AV block	2	0	2
Right bundle branch block	0	2	2
(RBBB)			
Left bundle branch block (LBBB)	0	1	1
Left anterior hemiblock (LAHB)	0	2	2
Combined RBBB + LAHB	0	2	2
Sinoatrial (SA) block	1	0	1
Total (n)	17 (≈59%)	12 (≈41%)	29 (100%)

Note: χ^2 test = significant at p < 0.05

Temporal Evolution of Conduction Abnormalities

Out of the 29 patients who developed conduction disturbances, 27 (93.1) exhibited it at the time of hospitalisation, and 2 (6.9) developed it during hospitalisation. The majority of the conduction defects were experienced in the initial 24 hours after the onset of the symptoms, and the conduction defects were short-lived and relieved after reperfusion therapy or medical stabilisation. No patients showed progressive conduction loss. These observations indicate the ischemic but reversible nature of the majority of conduction blocks during acute STEMI. Figure 3 shows the time of conduction block onset in patients with STEMI.

Figure 3. Timing of the onset of conduction block



Treatment Interventions

Primary percutaneous coronary intervention (PCI) was carried out in 75% of the patients, thrombolysis and PCI in 13% and thrombolysis alone in 7%. Five % needed permanent pacemaker implants, primarily for the high-grade AV block and complete heart block. There was also a statistically significant difference between treatment modality and presence of conduction block (p = 0.001), which suggests

that patients whose conductors abnormalities were present tended to have more advanced management. Figure 4 illustrates the spread of treatment modalities and pacing demand among patients with STEMI.

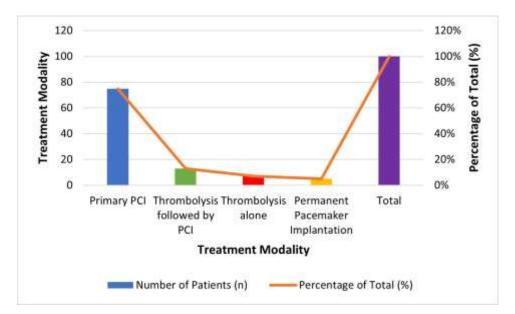


Figure 4. Treatment modalities and pacing requirements in STEMI

Clinical Outcomes

In total, 96 % of patients survived, and 4 % (n=4) died during hospitalisation. Three of those deaths were of patients with conduction abnormalities, and the conduction block and in-hospital mortality were statistically significantly related (p = 0.031). The majority of the patients (72 %) recovered successfully, although some developed transient arrhythmias or mild heart failure, which could be medically treated. There was no sudden cardiac death or ventricular asystole. Table 3 summarises clinical outcomes of patients with acute STEMI and their relationships to conduction block.

Table 3. In-hospital outcomes in patients with acute STEMI

Clinical Outcome	Number of	Percentage of	Remarks / Association
	Patients (n)	Total (%)	
Survived	96	96 %	The majority had a good
			recovery
Died	4	4 %	3 of 4 deaths had conduction
			block $(p = 0.031)$
Uncomplicated Recovery	72	72 %	Stable, no major complications
Transient Arrhythmias or Mild	24	24 %	Managed medically
Heart Failure			
Sudden Cardiac Death /	0	0 %	None reported
Ventricular Asystole			
Total	100	100 %	

Statistical Summary

Data were analysed through SPSS Version 22 (IBM Corp., USA), which showed significant correlations between the presence of conduction block, mortality, and treatment modality. There was no independent correlation to be found with age, sex or comorbidities after adjustment of logistic regression. These findings affirm conduction abnormalities as crucial prognostic variables in acute STEMI.

Discussion

This paper assessed the incidence, pattern and prognostic significance of the conduction abnormalities among patients who had acute ST-elevation myocardial infarction (STEMI) and were admitted to a

tertiary care hospital. It was confirmed that rhythm complications are still common and clinically important even in the reperfusion era, with conduction disturbances being identified in 29% of patients. The average age of the group was 52.04 with a standard deviation of 8.66 years, indicating that coronary artery disease in this group was relatively young [20]. The male dominance was evidenced in a unique manner, which is in line with gender patterns in ischemic heart disease [2]. The main risk factors were hypertension, diabetes, smoking, and dyslipidemia, making the central weight in promoting atherosclerosis and myocardial ischemia [21].

Poorest-wall myocardial infarction (IWMI) was the most characteristic and the most frequent, with more than half of them. This right-sidedness is indicative of right coronary artery involvement, and this is the reason why atrioventricular (AV) nodal blocks are so common in IWMI patients [22]. Transient first-, second-, or complete heart block may be caused by ischemia of the AV nodal artery, which is typically a branch of the right coronary artery [23]. Conversely, anterior-wall infarctions, which were mostly due to left anterior descending artery occlusion, tended to be linked with intraventricular conduction defects, including right bundle branch block (RBBB), left bundle branch block (LBBB), and left anterior hemiblock (LAHB) [6]. These observations are based on the fact that infarct territory and vascular anatomy affect the site of conduction disturbance [24].

Atrioventricular nodal blocks included about three-fourths of all conduction abnormalities, the most frequent of them being first-degree AV block, then complete heart block. The majority of conduction abnormalities (93.1%) were evident at presentation, suggesting that conduction tissue ischemic injury takes place early in infarction. Most of them were transient, which corrected the situation following reperfusion or stabilisation, and it is important to highlight that the dysfunction of ischemic conduction is usually reversible, provided that the coronary flow is restored in time [25].

75 % of patients underwent primary percutaneous intervention (PCI), which is in line with guideline-based therapy [26]. Permanent pacemaker implantation was necessary in 5% which occurred in most cases of advanced AV block or complete heart block. The high correlation of the conduction abnormalities and treatment modality shows that these patients are usually demanding more aggressive treatment. Death rates were 4, and three out of four deaths happened in patients with conduction blocks (p = 0.031), which indicates that rhythm disorders are indicative of the extent of ischemic damage and increased in-hospital mortality despite low overall mortality.

The correlation between conduction anomalies and poor outcomes is probably due to the twofold impact of ischemia on myocardial contractility and the electrical stability [9]. The failure to transmit impulses is caused by ischemic injury, whereas decreased ventricular functioning preconditions the arrhythmia and heart failure [11]. This study reported cases of 24 % transient arrhythmia or mild heart failure, which could be treated medically. The fact that there were no cases of sudden cardiac death or refractory asystole indicates that fatal electrical complications can be appropriately prevented with the help of early reperfusion and close rhythm control.

The 29% incidence rate in this case is a positive effect of early intervention and enhanced control of ischemia compared with a pre-reperfusion data reporting incidence of over 40% [13]. However, the fact that conduction abnormalities were still present in almost a third of cases highlights the fact that these complications are still of clinical importance, especially when delays in presentation restrict the utility of revascularisation. The logistic regression analysis did not find an independent correlation between conduction block and age, sex, and comorbidities, which implied that the conduction size and ischemic burden, instead of demographic variables, were the key determinants of conduction involvement [10]. These findings have far-reaching clinical implications. The use of continuous electrocardiographic monitoring must be maintained in the centre of the acute STEMI management to enable the detection of conduction abnormalities in the early stages. The fact of the existence of the conduction block should make one alert about close hemodynamic monitoring, early pacing preparedness, and increased attention to post-reperfusion. These results also emphasise the need to have timely reperfusion in peripheral centres, as delayed intervention can turn a non-permanent conduction impairment into a permanent one.

This study has its limitations despite its strengths. It was performed in one tertiary-care facility and has a rather limited sample that can limit the ability to generalise the results. The causal inference is restricted due to the observational design, and the outcomes of the long-term post-discharge were not determined. In addition, more sophisticated electrophysiologic testing and imaging co-location were

not conducted, which might have offered more information about the structural foundation of conduction abnormalities. Multicentric studies using larger cohorts and then longer follow-up with further incorporation of image modalities are required to confirm these views and improve prognostic models.

Conduction anomalies are still a common complication of acute STEMI, especially in inferior-wall infarctions. The majority of them are transient and reversible with early reperfusion, although their occurrence indicates a greater amount of myocardial damage and increased mortality in the short term. The initial awareness, constant rhythm monitoring, and pacing in time are critical towards better patient outcomes. The results provide evidence of the existing knowledge of the conduction abnormalities in myocardial infarction and the necessity to remain attentive to acute coronary care.

Conclusion

This research presents geographically localised, up-to-date evidence on the spectrum and prognostic worth of conduction abnormalities in acute ST-elevation myocardial infarction (STEMI) in a North Indian tertiary-care group setting poorly represented in post-reperfusion literature. In 29 % of patients, it was found that conduction was blocked, although this was most often in the case of inferior-wall infarction, and was most often associated with disturbances of the atrioventricular nodes. The transience and the early development of most blocks indicate ischemic and reversible nodal dysfunction in the case of timely reperfusion. Notably, the conduction abnormalities were closely linked to the necessity to use more complicated management, such as pacing, and an increase in in-hospital mortality, which confirms their role as prognosticators even in the era of primary percutaneous coronary intervention. This study provides useful information on the early risk stratification and management of STEMI patients by relating the pattern of conduction defects with the location of an infarct and clinical outcome. The outcomes of the research indicate the value of constant ECG monitoring, timely reperfusion, and pre-readiness to pacing to alleviate electrical instability and enhance survival rates in acute myocardial infarction.

References

- 1. Nikus K, Birnbaum Y, Fiol-Sala M, Rankinen J, de Luna AB: Conduction disorders in the setting of acute STEMI. Curr Cardiol Rev. 2021, 17(1):41-49. 10.2174/1573403X16666200702121937
- 2. Ram R, Devi KB, Chanu KJ, Devi TS, Naorem S, Chongtham DS: Study of conduction blocks in acute myocardial infarction. J Med Soc. 2016, 30(3):149-152. 10.4103/0972-4958.191179
- 3. Shinde S, Jadhav N: Study of conduction blocks in ST elevation myocardial infarction—a cross-sectional analysis. Eurasian J Med. 2024, 56(3):148. 10.5152/eurasianjmed.2024.20164
- 4. Lahti R, Rankinen J, Lyytikäinen LP, Eskola M, Nikus K, Hernesniemi J: High-risk ECG patterns in ST elevation myocardial infarction for mortality prediction. J Electrocardiol. 2022, 74:13-19. 10.1016/j.jelectrocard.2022.07.068
- 5. Harikrishnan P, Gupta T, Palaniswamy C, et al.: Complete heart block complicating ST-segment elevation myocardial infarction: temporal trends and association with in-hospital outcomes. JACC Clin Electrophysiol. 2015, 1(6):529-538. 10.1016/j.jacep.2015.08.007
- 6. Meyer MR, Radovanovic D, Pedrazzini G, et al.: Differences in presentation and clinical outcomes between left or right bundle branch block and ST segment elevation in patients with acute myocardial infarction. Eur Heart J Acute Cardiovasc Care. 2020, 9(8):848-856. 10.1177/2048872620905101
- 7. Pokorney SD, Radder C, Schulte PJ, et al.: High-degree atrioventricular block, asystole, and electromechanical dissociation complicating non-ST-segment elevation myocardial infarction. Am Heart J. 2016, 171(1):25-32. 10.1016/j.ahj.2015.09.004
- 8. Tsutsumi K, Tsukahara K: Is the diagnosis ST-segment elevation or non–non-ST-segment elevation myocardial infarction? Circulation. 2018, 138(23):2715-2717. 10.1161/CIRCULATIONAHA.118.037818
- 9. Frampton J, Ortengren AR, Zeitler EP: Arrhythmias after acute myocardial infarction. Yale J Biol Med. 2023, 96(1):83. 10.59249/LSWK8578

- 10. Alahmad Y, Al-Tamimi H, Khazaal F, et al.: Atrioventricular conduction disorders as a complication of inferior ST-elevation myocardial infarction in patients with COVID-19 infection. Case Rep Cardiol. 2022, 2022(1):3621799. 10.1155/2022/3621799
- 11. Uddin M, Mir T, Khalil A, et al.: ST-elevation myocardial infarction outcomes: a United States nationwide emergency departments cohort study. J Emerg Med. 2022, 62(3):306-315.10.1016/j.jemermed. 2021.10.028
- 12. Bruña V, Velásquez-Rodríguez J, Valero-Masa MJ, et al.: Prognostic of interatrial block after an acute ST-segment elevation myocardial infarction. Cardiology. 2019, 142(2):109-115. 10.1159/000499501
- 13. Okada JI, Fujiu K, Yoneda K, Iwamura T, Washio T, Komuro I, et al.: Ionic mechanisms of ST-segment elevation in electrocardiogram during acute myocardial infarction. J Physiol Sci. 2020, 70(1):36. 10.1186/s12576-020-00760-3
- 14. Misumida N, Ogunbayo GO, Kim SM, Abdel-Latif A, Ziada KM, Elayi CS: Frequency and significance of high-degree atrioventricular block and sinoatrial node dysfunction in patients with non-ST-elevation myocardial infarction. Am J Cardiol. 2018, 122(10):1598-1603. 10.1016/j.amjcard.2018.08.001
- 15. Auffret V, Loirat A, Leurent G, et al.: High-degree atrioventricular block complicating ST-segment elevation myocardial infarction in the contemporary era. Heart. 2016, 102(1):40-49. 10.1136/heartjnl-2015-308260
- 16. Çinier G, Tekkeşin Aİ, Genç D, et al.: Interatrial block as a predictor of atrial fibrillation in patients with ST-segment elevation myocardial infarction. Clin Cardiol. 2018, 41(9):1232-1237. 10.1002/clc.23029
- 17. de Bliek EC: ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation. Turk J Emerg Med. 2018, 18(1):1-10. 10.1016/j.tjem.2018.01.008
- 18. Ullah F, Din IU, Adil I, Ali U, Khan MA, Abbas F: High degree AV block in patients presenting with acute myocardial infarction. Pak Heart J. 2019, 52(3). 10.47144/phj.v52i3.1784
- 19. Vogel B, Claessen BE, Arnold SV, et al.: ST-segment elevation myocardial infarction. Nat Rev Dis Primers. 2019, 5(1):39. 10.1038/s41572-019-0090-3
- 20. Abu Fanne R, Kleiner Shochat M, Shotan A, et al.: Acute diagonal-induced ST-elevation myocardial infarction and electrocardiogram-guidance in the era of primary coronary intervention: new insights into an old tool. Eur Heart J Acute Cardiovasc Care. 2020, 9(8):827-835. 10.1177/2048872619828291
- 21. Bhasin D, Kumar R, Agarwal T, Gupta A, Bansal S: A case with inferior wall myocardial infarction and conduction abnormalities: addressing the diagnostic challenges. Cureus. 2022, 14(3).10.7759/cureus.23614
- 22. Herweg B, Marcus MB, Barold SS: Diagnosis of myocardial infarction and ischemia in the setting of bundle branch block and cardiac pacing. Herzschrittmacherther Elektrophysiol. 2016, 27(3):307-322. 10.1007/s00399-016-0439-1
- 23. Chera HH, Mitre CA, Nealis J, Mironov A, Budzikowski AS: Frequency of complete atrioventricular block complicating ST-elevation myocardial infarction in patients undergoing primary percutaneous coronary intervention. Cardiology. 2018, 140(3):146-151. 10.1159/000491076
- 24. Dodd KW, Elm KD, Smith SW: Comparison of the QRS complex, ST-segment, and T-wave among patients with left bundle branch block with and without acute myocardial infarction. J Emerg Med. 2016, 51(1):1-8. 10.1016/j.jemermed.2016.02.029
- 25. Mansoor A, Chang D, Mitra R: Rhythm, conduction, and ST elevation with COVID-19: myocarditis or myocardial infarction? HeartRhythm Case Rep. 2020, 6(10):671-675. 10.1016/j.hrcr.2020.08.001
- 26. Alam MK, Mahmood M, Adhikary DK, et al.: The pattern of cardiac arrhythmias in acute ST-elevated myocardial infarction and their in-hospital outcome. Univ Heart J. 2020, 16(1):16-21. 10.3329/uhj.v16i1.44817