Journal of Community Health Provision
Vol. 6, Issue 2, 2026
Page 385-396
DOI: https://doi.org/10.55885/jchp.v6i2.978

Pathophysiological Mechanisms Linking Myocardial Infarction to Cardiac Arrest: A Literature Review

Andi Suleha[1], Akina Maulidhany Tahir[2], Fadillah Maricar[2], Priscilla Sasmita Rido[2], Nurhikmawati[2]

[1]Department of Medical Profession, Faculty of Medicine, Universitas Muslim Indonesia, Indonesia
[2]Departement of Cardiovascular, Faculty of Medicine, Universitas Muslim Indonesia, Indonesia

Email: andieha21@gmail.com

Abstract. *Myocardial infarction (MI) is a leading cause of cardiac arrest and sudden cardiac death worldwide. The pathophysiological link involves complex interactions between cellular, electrophysiological, hemodynamic, and neurohormonal mechanisms. The objective of the research to comprehensively review the pathophysiological mechanisms linking myocardial infarction to cardiac arrest. The methods of the research was a descriptive-analytic literature review was conducted using PubMed, ScienceDirect, SpringerLink, and Google Scholar databases (2021–2025). Study selection followed PRISMA guidelines. The results of the showed that eight studies were included. Key mechanisms include myocardial ischemia leading to ATP depletion and acidosis, ionic imbalance, arrhythmogenic substrate formation, sympathetic overactivation, inflammatory response and oxidative stress, and hemodynamic collapse. Cardiac arrest following MI is primarily driven by fatal ventricular arrhythmias and pump failure. Understanding these mechanisms is essential for improving prevention and management strategies.*

Keywords: *Myocardial Infarction, Cardiac Arrest, Pathophysiology*

Received: April 24, 2026 | Received in Revised: May 22, 2026 | Accepted: June 8, 2026

INTRODUCTION

Cardiovascular disease remains the leading cause of death globally, with myocardial infarction (MI) contributing significantly to the incidence of sudden cardiac arrest. Globally, 32.4 million MI events are recorded per year, with an annual mortality risk of 5%. In the United States, sudden cardiac arrest affects 180,000 to 450,000 people per year, with 90% of these resulting in sudden cardiac death. According to the 2018 Basic Health Research (Riskesdas), coronary heart disease is most common in the 65–74 age group (3.6%), followed by those aged ≥75 years (3.2%). Myocardial infarction is one condition that can trigger cardiac arrest through the mechanism of coronary artery thrombosis. Myocardial infarction occurs due to acute occlusion of the coronary arteries, causing prolonged myocardial ischemia and leading to myocardial cell necrosis. This structural and functional damage not only reduces the heart's pumping ability but also causes electrical instability, increasing the risk of fatal arrhythmias and heart attacks as early manifestations of the disease (Zheng et al., 2025; Yu, 2025; Indonesia, 2020; Husna & Fauzan, 2025; Sirait et al., 2025).

In out-of-hospital cardiac arrest (OHCA) patients with VF/pulseless VT rhythms exhibiting ST-elevation (STEMI) after return of spontaneous circulation (ROSC), the prevalence of significant coronary artery disease (CAD) reaches 70–95%, with acute lesions found in 70–80% of cases (Lott et al., 2021). A prospective study (2020–2023) of OHCA patients admitted to the intensive care unit (ICCU) found that the most common cause was acute coronary syndrome (ACS) in 59% of patients, of which 85% were STEMI and 15% were NSTEMI (Tabi et al., 2024). In 2023, STEMI as the leading cause of death was recorded in 18,132 deaths in the US, with an age-adjusted mortality rate of 4.3 per 100,000. The survival rate to hospital discharge in adult OHCA is 10.5% (American Heart Association, 2026).

Acute myocardial ischemia causes significant electrophysiological changes in myocardial cells, including disruption of ionic gradients, shortening of action potential duration, and heterogeneity of repolarization between myocardial regions (Song et al., 2024; Martinez-Navarro et al., 2025; Lei et al., 2024). This condition creates an arrhythmyogenic substrate that triggers malignant ventricular arrhythmias such as ventricular fibrillation and pulseless ventricular tachycardia, which are the most common causes of cardiac arrest in the early phase of myocardial infarction. This electrical instability often appears before medical intervention can be administered, contributing to high prehospital mortality rates (Zheng et al., 2025; Corrall et al., 2023; Alzahrani et al., 2025; Gamberini et al., 2025).

In addition to electrophysiological mechanisms, excessive sympathetic nervous system activation after myocardial infarction plays a crucial role in increasing the risk of cardiac arrest. Post-infarction sympathetic nervous system remodeling increases catecholamine release, which further increases myocardial oxygen demand and worsens ischemia. Increased sympathetic activity also increases myocardial electrical irritability, increasing the likelihood of ventricular arrhythmias leading to cardiac arrest (Yu, 2025; Moras et al., 2024; Menon et al., 2024).

The systemic inflammatory response and oxidative stress that occur during the acute phase of myocardial infarction further exacerbate the condition of the injured myocardium. Inflammatory mediators and immune cell activation can disrupt myocardial cell ionic homeostasis and prolong the arrhythmyogenic state. The complex relationship between inflammation and cardiac arrhythmias is a key factor in increasing the incidence of ventricular arrhythmias and cardiac arrest after myocardial infarction (Taghdiri, 2025; Buja, 2023).

In addition to arrhythmias, extensive myocardial necrosis can also lead to acute cardiac pump failure, which progresses to cardiogenic shock. A drastic decrease in ventricular contractility results in decreased cardiac output and systemic hypoperfusion, which in later stages can lead to pulseless electrical activity, or asystole. This hemodynamic mechanism is associated with a poor prognosis and a very high mortality rate in patients with myocardial infarction complicated by heart attack (Zheng et al., 2025; Buja, 2023)

Cardiac arrest is defined as the sudden cessation of cardiac mechanical activity, characterized by the loss of effective blood circulation, resulting in cessation of perfusion to vital organs, particularly the brain and heart (Greif et al., 2025). The American Heart Association states that cardiac arrest is a life-threatening emergency resulting from primary cardiac electrical disturbances or mechanical failure. This can occur suddenly and is often associated with structural heart disease, particularly acute myocardial infarction (Panchal et al., 2025).

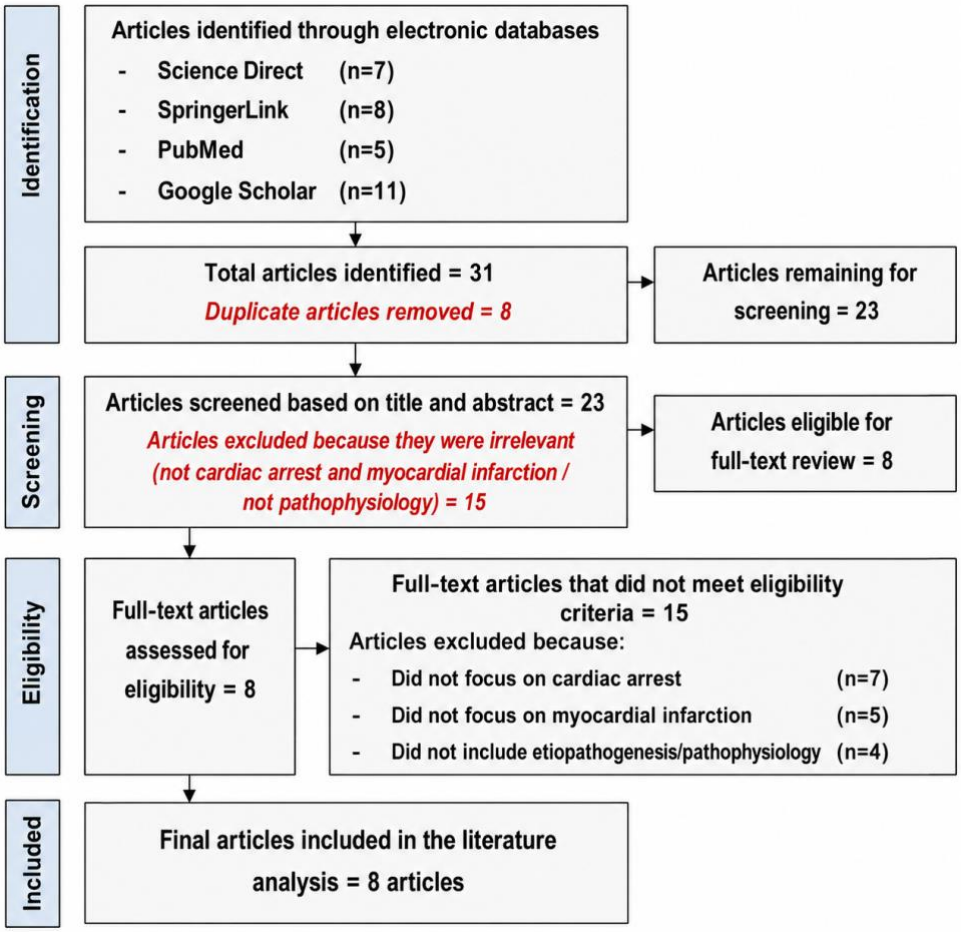
Although various mechanisms have been identified, the pathophysiological relationship between myocardial infarction and cardiac arrest still requires a more comprehensive understanding, particularly regarding the interactions between these simultaneous mechanisms. Therefore, this study aims to examine in depth the pathophysiological mechanisms linking myocardial infarction and cardiac arrest as a basis for developing more effective prevention and management strategies.

METHODS

This research is a literature review using a descriptive analytical approach that aims to examine the pathophysiological mechanisms linking myocardial infarction and cardiac arrest. Data sources were obtained through a systematic search of several electronic databases, namely PubMed, ScienceDirect, SpringerLink, and Google Scholar. The search was conducted using a combination of the keywords "myocardial infarction," "pathophysiology," and "cardiac arrest" in both English and Indonesian. Articles considered were published within the last five years (2021–2025) to ensure relevance to current scientific developments.

Inclusion criteria for this study included articles written in English or Indonesian, available in full-text format, and specifically discussing the pathophysiology of myocardial infarction, cardiac arrest, and the relationship between the two, from cellular, molecular, electrophysiological, and hemodynamic perspectives. Articles in the form of editorials, opinion pieces, single case reports without a discussion of pathophysiology, and articles irrelevant to the research topic were excluded from the analysis.

The literature selection process was conducted based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, which include identification, screening, eligibility assessment, and inclusion. Of the 31 articles found, 8 met the inclusion criteria and were further analyzed. The data obtained were then analyzed using narrative synthesis and thematic analysis methods by grouping findings based on main themes, namely the pathophysiological mechanisms of myocardial infarction, the process of cardiac arrest, aggravating factors, and related clinical implications.



RESULTS AND DISCUSSION

Several previous studies have been conducted to identify the understanding of the mechanism by which myocardial infarction can cause cardiac arrest.

ISSN 2776-1150 (Print), ISSN 2776-1169 (online)

Table 1. Literature Search Results

No	Researcher (Year)	Research Title	Research Design	Factors Studied	Key Results
1	Buono, M et al. (2022)	*Ischemic Cardiomyopathy and Heart Failure After Acute Myocardial Infarction* (Current Cardiology Reports)	Article review	The main pathophysiological mechanisms and pathways of heart failure in ischemic cardiomyopathy, therapeutic opportunities and knowledge gaps in this field	Pathophysiological mechanisms, ranging from the extent of post-infarction scarring to the phenomenon of hibernating myocardium and the role of systemic inflammation in damaging cardiac structures.
2	Hørsdal, OK et al. (2022)	*The Immediate Cardiovascular and Mitochondrial Response in Ischemic Cardiogenic Shock* (Journal of Cardiovascular Translational Research)	Experiment	Pathophysiological dynamics of ischemic cardiogenic shock in the hyperacute phase in a large animal model	Systolic and diastolic dysfunction and increased cardiac workload occur almost immediately, followed by decreased mitochondrial metabolic efficiency. identifying mitochondrial function as a novel therapeutic target for early intervention.
3	Laksono,S. Harsas, N. (2022)	*Arrhythmia In Acute Coronary Syndrome* (Al-Iqra Medical Journal)	Mini review	The relationship between acute coronary syndrome and the emergence of various types of arrhythmias, a comprehensive guide to diagnosing and managing cardiac electrical complications in coronary heart disease patients.	gElectrophysiological disturbances and metabolic changes due to ischemia can trigger dangerous conditions such as ventricular tachycardia and atrial fibrillation. Rapid treatment through pharmacological interventions such as amiodarone or beta-blockers, as well as non-pharmacological measures such as defibrillation and pacemaker implantation, is crucial.
4	Tsalamandris, S et al. (2025)	*Endothelial Function and Pro-Inflammatory Cytokines as Prognostic Markers in Acute Coronary Syndromes* (Article)	Studi observasional	Endothelial dysfunction and inflammatory cytokine levels in patients with ACS	Significant endothelial dysfunction and elevated levels of inflammatory cytokines, particularly IL-6 and TNF-α, are closely associated with the development of coronary heart disease. The combination of blood flow restriction and systemic inflammation is

					key to understanding plaque vulnerability and the risk of cardiac death.
5	Frampton, J et al. (2023)	*Arrhythmias After Acute Myocardial Infarction* (Yale Journal of Biology and Medicine)	Review	Post-myocardial infarction arrhythmias (ventricular arrhythmias, supraventricular arrhythmias)	PGuidelines on pathophysiology, risk identification, and appropriate clinical management strategies, ranging from the use of beta-blockers to consideration of pacemaker or defibrillator implantation. Improving the stability of the infarcted myocardium with reperfusion has been effective in reducing atrial fibrillation.
6	Heusch, Gerd (2024)	*Myocardial ischemia/reperfusion: Translational pathophysiology of ischemic heart disease* (Med Review)	Review	Myocardial ischemia/reperfusion in the coronary vascular and myocardial aspects	The mechanisms of myocardial ischemia and reperfusion, from vessel occlusion to cardiac tissue damage that occurs when blood flow is restored. Regulated modes of cardiomyocyte cell death and the crucial role of coronary microvascular dysfunction.
7	Wallentin, L et al. (2021)	*Plasma proteins associated with cardiovascular death in patients with chronic coronary heart disease: A retrospective study* (PLoS Medicine)	Study Retrospektif	Circulation biomarkers, Cardiovascular Death	1Eight biomarkers independently associated with cardiac death, with NTproBNP and cTnT-hs emerging as the most dominant predictors compared to other clinical characteristics. These findings confirm that biological processes such as myocardial dysfunction, kidney damage, inflammation, and hemodynamic stress are key mechanisms determining patient prognosis.
8	Plott, C et. Al (2024)	*Neurocardiac Axis Physiology and Clinical Applicatioans* (IJC	Article Review	Neurocardiac Axis, cardiovascular disease, innovative	Chronic stress and excessive activation of the sympathetic nervous system can accelerate the

		Heart and Vasculature)		medical interventions	formation of cholesterol plaques and lead to heart failure and arrhythmias.

A review of eight articles that met the inclusion criteria indicates that the relationship between myocardial infarction and cardiac arrest involves complex and multifactorial pathophysiological mechanisms. Generally, this process begins with myocardial ischemia due to coronary artery occlusion, which disrupts cellular metabolism, characterized by decreased adenosine triphosphate (ATP) production and increased anaerobic metabolism. This condition triggers intracellular acidosis and dysfunction of energy-dependent ion pumps, resulting in ionic imbalance, particularly increased intracellular sodium and calcium concentrations.

This ionic imbalance contributes to changes in myocardial electrophysiological properties, including shortened action potential duration and increased repolarization heterogeneity. These changes create an arrhythmyogenic substrate that allows for reentry mechanisms and other abnormal electrical activity, which can ultimately trigger fatal ventricular arrhythmias such as ventricular tachycardia and ventricular fibrillation. Furthermore, post-infarction activation of the sympathetic nervous system has been found to play a key role in increasing cardiac electrical irritability through increased catecholamine release, which exacerbates the electrophysiological imbalance and increases myocardial oxygen demand.

Furthermore, the inflammatory response and oxidative stress that occur in the acute phase of myocardial infarction exacerbate tissue damage and expand the area of injury. The production of reactive oxygen species (ROS) and inflammatory mediators not only damages cell structure but also modifies ion channel function and the cardiac conduction system, thereby increasing the risk of arrhythmias. From a hemodynamic perspective, a large myocardial infarction can cause a significant decrease in ventricular contractility, thereby reducing cardiac output and triggering cardiogenic shock. This condition can progress to pulseless electrical activity or asystole, which is the terminal form of cardiac arrest.

The study results indicate that cardiac arrest in myocardial infarction occurs through two main pathways: the electrophysiological pathway, which involves fatal ventricular arrhythmias, and the hemodynamic pathway, which involves heart pump failure. These two mechanisms often interact and are exacerbated by other factors such as the size of the infarct, the location of the lesion, delayed reperfusion, and the presence of comorbidities in the patient

The mechanism is preceded by systemic endothelial dysfunction that decreases the production of nitric oxide (NO), which disrupts vascular homeostasis. Low-grade systemic inflammation, characterized by increased cytokines such as IL-6 and TNF-α, plays a key role in making plaques vulnerable and triggering acute events. Several plasma proteins have been identified as strong indicators of cardiovascular mortality risk in patients with chronic coronary heart disease, including NTproBNP, cTnT-hs, GDF-15, and IL-6. Specifically in ACS patients, TNF-α levels > 5.19 pg/mL are associated with a 2.5-fold higher risk of major cardiovascular events (MACE). Immediately after coronary occlusion, mitochondrial respiration ceases and ATP reserves decline drastically. This leads to mitochondrial damage, decreased oxidative capacity, especially in Complex I, and increased proton leakage that worsens energy failure. Ionic disorders, failure of the Na+/K+-ATPase pump, lead to the accumulation of extracellular potassium and excess intracellular calcium through reverse Na+/Ca2+ exchange. In addition to necrosis, myocyte death also occurs through regulated mechanisms such as apoptosis, pyroptosis (inflammasome response), and ferroptosis (Heusch, 2024; Wallentin et al., 2021; Tsalamandris et al., 2025).

Ischemia and reperfusion create electrical instability that triggers fatal arrhythmias (cardiac arrest). Ventricular arrhythmias are a re-entry mechanism, and abnormal automaticity triggers ventricular tachycardia (VT) and ventricular fibrillation (VF). Furthermore, conduction disturbances and reperfusion arrhythmias occur; reestablishing blood flow can trigger accelerated idioventricular rhythm (AIVR) and a surge of free radicals (ROS) that damage cell

membranes. After a myocardial infarction, the heart undergoes a healing process involving debris removal by pro-inflammatory macrophages, followed by a reparative phase by anti-inflammatory macrophages and fibroblasts. However, the extensive infarct burden and uncontrolled inflammation trigger detrimental left ventricular remodeling in the form of dilation and fibrosis, ultimately leading to post-infarction heart failure. This condition can be exacerbated by hyperactivity of the sympathetic nervous system and activation of the renin-angiotensin-aldosterone system (RAAS) (Heusch, 2024; Laksono & Harsas, 2022; Frampton et al., 2023).

The mechanism of myocardial infarction (MI) generally begins with an imbalance between oxygen supply and demand in the heart muscle (myocardium), leading to cell death (Heusch, 2024). In Type 1 MI, atherosclerotic plaque ruptures or erodes in the coronary arteries. When the plaque's fibrous cap ruptures, the thrombogenic lipid core is exposed to the bloodstream, triggering platelet aggregation and thrombus formation. This thrombus can completely or partially occlude the vessel, resulting in a sudden cessation of oxygen-rich blood flow. In Type 2 MI, infarction can also occur without plaque rupture, but rather due to an imbalance in supply such as coronary vasospasm, spontaneous coronary artery dissection (SCAD), severe anemia, or arrhythmias that worsen blood flow (Acharya et al., 2017).

Immediately after blood flow is interrupted (occlusion), cardiomyocytes undergo rapid chemical changes. Oxygen deprivation causes mitochondrial respiration to cease within seconds, resulting in a drastic decrease in energy (ATP) production. Cells switch to anaerobic metabolism, which produces little ATP and accumulates lactate and hydrogen ions, leading to intracellular acidosis. Due to energy deprivation, the heart muscle loses its ability to contract within approximately 60 seconds of occlusion (Buja, 2023; Heusch, 2024; Del Buono et al., 2022; Hørsdal et al., 2025).

The decrease in ATP causes the ion pumps in the cell membrane to fail. Potassium accumulates outside the cell and sodium accumulates inside, drawing water in and causing the cell to swell (oncosis). Then, a surge in intracellular calcium activates destructive enzymes (proteases and phospholipases) that damage the cell's skeleton and membrane. This severe damage triggers the opening of the Mitochondrial Permeability Transition Pore (MPTP), leading to permanent loss of mitochondrial membrane potential and complete cell death (Buja, 2023; Hørsdal et al., 2025).

Cell damage is reversible if blood flow is restored within 15–20 minutes. However, if ischemia persists for more than 20 to 40 minutes, cells begin to die permanently through necrosis. Necrosis begins in the most vulnerable inner layer of the heart (subendocardium), then spreads toward the outer layer (subepicardium) if the blockage is not immediately resolved. This cardiomyocyte cell death then triggers the release of proteins into the blood, such as troponin, which is a key marker in the clinical diagnosis of myocardial infarction. If the infarction is extensive, it can lead to severe complications such as acute heart failure or cardiogenic shock ((Buja, 2023; Heusch, 2024). The process of arrhythmia and cardiac arrest in patients with myocardial infarction (MI) results from metabolic disturbances, electrolyte imbalances, and nervous system activity that create electrical instability in the heart (Heusch, 2024; Frampton et al., 2023; Hørsdal et al., 2025).

Immediately after blood flow stops, mitochondrial respiration stops and energy reserves (ATP) decrease drastically within seconds, causing the Na+/K+-ATPase pump to fail, resulting in an accumulation of extracellular potassium that depolarizes surrounding cells. Pump failure also causes an accumulation of intracellular calcium through a reversed Na+/Ca2+ exchanger. This increase in cytosolic calcium triggers abnormal depolarization that manifests as ectopic beats. Metabolism shifts to anaerobic glycolysis, producing lactate and lowering cellular pH, further impairing cardiac function (Heusch, 2024; Frampton et al., 2023; Hørsdal et al., 2025).

These chemical changes create conditions conducive to arrhythmias, which trigger spontaneous electrical activity, particularly in subendocardial Purkinje fibers or infarcted areas that are still excitable. Conduction blockages occur due to damage to gap junctions. The difference

in electrical velocity between healthy and ischemic/scarred tissue creates a closed electrical circuit (re-entry), which is the primary cause of ventricular tachycardia (VT) and ventricular fibrillation (VF). Activation of certain potassium currents shortens the duration of the action potential, increasing susceptibility to arrhythmias (Heusch, 2024; Frampton et al., 2023; Acharya et al., 2017). While restoring blood flow (reperfusion) is essential for saving the heart muscle, this process can also trigger reperfusion arrhythmias, leading to the formation of large amounts of reactive oxygen species (ROS), which damage cell membranes and contractile proteins. Uneven ion washout creates an electrical gradient that favors the re-entry circuit (Heusch, 2024; Del Buono, 2022).

Central nervous system activity plays a major role in exacerbating this condition through the neurocardiac axis. Ischemia triggers a massive release of norepinephrine from cardiac nerve terminals that cannot be reabsorbed, triggering action potentials in remaining active cells. Physical and emotional stress increase amygdala activity, which sends strong sympathetic signals to the heart, increasing heart rate and lowering the threshold for fatal arrhythmias (Heusch, 2024; Plott et al., 2024). Unstable VT and VF cause the heart to lose its ability to pump blood to the brain and other vital organs. Ischemia affecting the conduction system (such as occlusion of the right coronary artery supplying the AV node) can cause high-degree atrioventricular block (AVB) or asystole. Extensive myocardial damage can lead to severe cardiogenic shock, leading to cardiac arrest (Frampton et al., 2023; Del Buono et al., 2022; Merchant et al., 2024).

Factors that exacerbate cardiac arrest due to myocardial infarction (MI) involve a combination of the characteristics of the heart damage, patient comorbidities, and technical challenges during resuscitation. Infarct Size. The extent of tissue loss is a major determinant of prognosis; loss of 40% or more of left ventricular mass due to a single or multiple infarcts leads to a lethal syndrome of cardiogenic shock and uncontrolled ventricular arrhythmias. Infarcts in the anterior wall of the heart are associated with a higher risk of pump failure and pathological remodeling than other locations. Patients with a left ventricular ejection fraction (LVEF) ≤ 40% have a significantly higher risk of death from arrhythmias. Events such as ventricular septal rupture, myocardial free wall rupture, or acute mitral valve regurgitation can cause sudden, fatal cardiovascular collapse (Buja, 2023; Frampton et al., 2023; Acharya et al., 2027; Del Buono et al., 2022).

Electrolyte Imbalance. Extreme potassium levels, either severe hypokalemia or hyperkalemia, and hypomagnesemia directly trigger cardiac electrical instability, leading to malignant arrhythmias (VT/VF). 3,6,17 Cellular energy (ATP) deficiency and intracellular acidosis due to anaerobic glycolysis impair myocyte contractility and electrical stability. 17,23 Even when blood flow is restored, a surge of free radicals (ROS) and excess calcium can lead to stunning (temporary cardiac muscle paralysis) or permanent microvascular damage (no-reflow) (Buja, 2023; Heusch, 2024; Del Buono et al., 2022). Diabetes Mellitus, Chronic Kidney Disease (CKD), and anemia significantly increase the risk of recurrent ischemic events and poor neurological outcomes after cardiac arrest. Elderly patients tend to have multiple comorbidities and low physiologic reserve, which are independent predictors of post-interventional mortality. Obesity presents unique technical challenges to effective CPR, ranging from difficult airway management and vascular access to decreased efficacy of vasoactive drugs and defibrillation (Acharya et al., 2017).

Reperfusion Delay. The time between symptom onset and opening of the blood vessel (door-to-balloon) is crucial. Every minute of delay increases the risk of irreversible cell death. Out-of-Hospital Cardiac Arrest (OHCA): Patients experiencing cardiac arrest outside of a medical setting often experience delays in access to defibrillation and advanced life support, which decreases the chance of survival (Lazzarin et al., 2022). Cardiogenic Shock: A state of low cardiac output characterized by hypotension (systolic blood pressure <90 mmHg), cool extremities, and oliguria is the primary cause of death in 6-10% of MI cases. Malignant Arrhythmia: The presence of Ventricular Fibrillation (VF) within the first 48 hours is an indicator of a significantly increased risk of short-term mortality (Frampton et al., 2023).

The clinical implications of cardiac arrest due to myocardial infarction (MI) are broad, encompassing the risk of immediate death, systemic organ damage, and long-term decline in cardiac function. Sudden cardiac arrest is the sudden cessation of cardiac activity, resulting in circulatory failure, loss of consciousness, and sudden cessation of breathing. Without immediate cardiopulmonary resuscitation (CPR) and defibrillation, irreversible brain damage or death can occur within minutes. Therefore, the availability of a defibrillator and minimizing delays in treatment are crucial in the early phase of an infarction (Lazzarin et al., 2022). In the clinical context of Acute Coronary Syndrome (ACS), patients experiencing cardiac arrest or life-threatening arrhythmias are classified into a very high-risk group. The implication of this classification is that patients should undergo immediate invasive strategies (coronary angiography) without waiting for cardiac biomarker results to identify and treat blood vessel obstruction (Abbas et al., 2023; Katsioupa et al., 2023; Maurovich-Horvat et al., 2022).

The presence of malignant arrhythmias such as ventricular tachycardia (VT) or ventricular fibrillation (VF) in the acute phase of myocardial infarction has serious prognostic implications. Patients with early VF/VT (<48 hours) have a 30-day mortality risk of 22%, significantly higher than patients without arrhythmias (5%). 3 Arrhythmias that appear more than 48 hours post-infarction are associated with a significantly worse risk of death and often indicate extensive myocardial damage or recurrent ischemia. 62 Infarct Size: Loss of 40% or more of left ventricular mass due to infarction often leads to cardiogenic shock and uncontrolled ventricular arrhythmias, which have a very high fatality rate. (Leancă et al., 2022; Chen et al., 2025; Thomas et al., 2025).

For patients who survive cardiac arrest but remain comatose, the clinical focus shifts to protecting brain function. Preventing fever (>37.7°C) is recommended to improve neurologic outcome. Evaluation of neurological recovery is usually performed at least 72 hours after hospital admission (Nikolovski et al., 2024; American Heart Association, 2020). Cardiac arrest due to MI often leaves significant structural damage. The process of changes in heart shape and size (dilation) due to myocyte death increases the risk of chronic heart failure and future recurrent cardiac arrest. Patients with a left ventricular ejection fraction (LVEF) ≤ 40% after an acute event have a higher 1-year mortality risk and may require assistive devices such as an Implantable Cardioverter Defibrillator (ICD) for secondary prevention (Mugnai et al., 2025; Del Buono et al., 2020). Thus, cardiac arrest in MI is the result of a complex interaction between electrophysiological and hemodynamic disturbances exacerbated by metabolic, inflammatory, and neurohormonal factors. A comprehensive and timely therapeutic approach is essential to reduce mortality in this condition.

CONCLUSION

Myocardial infarction is a condition that can lead to cardiac arrest through complex and multifactorial pathophysiological mechanisms. This process begins with myocardial ischemia, which disrupts cellular metabolism and causes ionic imbalance, which then triggers cardiac electrophysiological instability. These changes create a substrate and trigger for fatal ventricular arrhythmias, such as ventricular tachycardia and ventricular fibrillation, which are the primary causes of cardiac arrest in the acute phase of myocardial infarction. Furthermore, activation of the sympathetic nervous system, inflammatory responses, and oxidative stress contribute to worsening myocardial damage and increasing susceptibility to arrhythmias. From a hemodynamic perspective, decreased cardiac contractility can lead to pump failure and the development of cardiogenic shock, leading to pulseless electrical activity or asystole. Thus, cardiac arrest in myocardial infarction results from the interaction of electrophysiological and hemodynamic disturbances exacerbated by metabolic, inflammatory, and neurohormonal factors. A comprehensive understanding of these mechanisms is crucial for prevention, early detection, and optimal management to reduce mortality in myocardial infarction patients.

THANK-YOU NOTE

The author would like to express his gratitude to all parties who have provided support in the preparation of this article, both directly and indirectly. He also expresses his gratitude to the

educational institutions and supervisors who provided guidance, input, and academic support throughout the writing process. He also acknowledges the contributions of previous researchers and authors whose work served as the basis for this literature review.

REFERENCES

Abbas, A., Raza, A., Ullah, M., Hendi, A. A., Akbar, F., Khan, S. U., ... & Rizg, W. Y. (2023). A comprehensive review: epidemiological strategies, catheterization and biomarkers used as a bioweapon in diagnosis and management of cardio vascular diseases. *Current Problems in Cardiology*, *48*(7), 101661. https://doi.org/10.1016/j.cpcardiol.2023.101661

Acharya, U. R., Fujita, H., Oh, S. L., Hagiwara, Y., Tan, J. H., & Adam, M. (2017). Application of deep convolutional neural network for automated detection of myocardial infarction using ECG signals. *Information Sciences, 415*, 190–198. https://doi.org/10.1016/j.ins.2017.06.027

Alzahrani, R. S. M., Almotairi, R. A. G., Alessa, A. M. A., Alsahafi, W. A. H., Alabdali, N. M., Almotairi, M. A., ... & Algmani, F. M. S. (2025). Prehospital Cardiac Arrest Management: A Narrative Review. *The Review of Diabetic Studies*, 10-29. https://doi.org/10.1016/j.jen.2025.12.001

American Heart Association. (2020). *Highlights of the 2020 American Heart Association guidelines for CPR and ECC*. American Heart Association.

American Heart Association. (2026). *2026 heart disease and stroke statistics update fact sheet at a glance* [Preprint].

Buja, L. M. (2023). Pathobiology of myocardial ischemia and reperfusion injury: Models, modes, molecular mechanisms, modulation, and clinical applications. *Cardiology in Review, 31*(5), 252–264. https://doi.org/10.1097/crd.0000000000000440

Chen, L., Luo, S., Feng, M., Chandra, A., Baizabal-Carvallo, J. F., Mofatteh, M., ... & Liao, X. (2025). Decoding the link between ischemic stroke and myocardial infarction: a pathophysiological and clinical perspective. *European Journal of Medical Research*, *30*(1), 984. https://doi.org/10.1186/s40001-025-03221-y

Corrall, S., Laws, S., & Rice, A. (2023). Low-voltage electrical injuries and the electrocardiogram: is a 'normal'electrocardiogram sufficient for safe discharge from care? A systematic review. *British paramedic journal*, *8*(3), 27-36. https://doi.org/10.29045/14784726.2023.12.8.3.27

Del Buono, M. G., Moroni, F., Montone, R. A., Azzalini, L., Sanna, T., & Abbate, A. (2022). Ischemic cardiomyopathy and heart failure after acute myocardial infarction. *Current Cardiology Reports, 24*(10), 1505–1515. https://doi.org/10.1007/s11886-022-01766-6

Frampton, J., Ortengren, A. R., & Zeitler, E. P. (2023). Arrhythmias after acute myocardial infarction. *The Yale Journal of Biology and Medicine, 96*(1), 83–92. https://doi.org/10.59249/LSWK8578

Gamberini, L., Carinci, V., Pallavicini, P., Rovera, M., Tartaglione, M., Gioachin, R., ... & Sirugo, P. (2025). Prehospital management of supraventricular tachycardia: a multicentre study of current practices with a subgroup propensity score-based comparison of adenosine and electrical cardioversion in unstable patients. *Resuscitation*, *215*, 110707. https://doi.org/10.1016/j.resuscitation.2025.110707

Greif, R., Lauridsen, K. G., Djärv, T., Ek, J. E., Monnelly, V., Monsieurs, K. G., ... & Kiebooms, E. (2025). European Resuscitation Council guidelines 2025 executive summary. *Resuscitation, 215*, 110770. https://doi.org/10.1016/j.resuscitation.2025.110770

Heusch, G. (2024). Myocardial ischemia reperfusion: Translational pathophysiology of ischemic heart disease. *Med, 5*(1), 10–31. https://doi.org/10.1016/j.medj.2023.12.007

Hørsdal, O. K., Frederiksen, P. H., Helgestad, O. K. L., Ravn, H. B., Møller, J. E., Wiggers, H., ... & Berg Hansen, K. (2025). The immediate cardiovascular and mitochondrial response in ischemic cardiogenic shock. *Journal of Cardiovascular Translational Research*, 1–14. https://doi.org/10.1007/s12265-025-10647-6

Husna, A. F., & Fauzan, R. (2025). Diagnosis dan tata laksana cardiac arrest berdasarkan panduan AHA 2020: Tinjauan literatur terkini. *Inovasi Kesehatan Global, 2*(4), 185–198. https://doi.org/10.62383/ikg.v2i4.2373

Katsioupa, M., Kourampi, I., Oikonomou, E., Tsigkou, V., Theofilis, P., Charalambous, G., ... & Tousoulis, D. (2023). Novel biomarkers and their role in the diagnosis and prognosis of acute coronary syndrome. *Life*, *13*(10), 1992. https://doi.org/10.3390/life13101992

Laksono, S., & Harsas, N. A. (2022). Arrhythmia in acute coronary syndrome: Mini review. *Al Iqra Medical Journal, 5*(1), 41–47. https://doi.org/10.1016/j.hrtlng.2013.07.010

Lazzarin, T., Tonon, C. R., Martins, D., Fávero Jr, E. L., Baumgratz, T. D., Pereira, F. W. L., ... & Minicucci, M. F. (2022). Post cardiac arrest mechanisms management and future perspectives. *Journal of Clinical Medicine, 12*(1), 259. https://doi.org/10.3390/jcm12010259

Leancă, S. A., Crișu, D., Petriș, A. O., Afrăsânie, I., Genes, A., Costache, A. D., ... & Costache, I. I. (2022). Left ventricular remodeling after myocardial infarction: from physiopathology to treatment. *Life*, *12*(8), 1111. https://doi.org/10.3390/life12081111

Lei, M., Salvage, S. C., Jackson, A. P., & Huang, C. L. H. (2024). Cardiac arrhythmogenesis: roles of ion channels and their functional modification. *Frontiers in physiology*, *15*, 1342761. https://doi.org/10.3389/fphys.2024.1342761

Lott, C., Truhlář, A., Alfonzo, A., Barelli, A., González-Salvado, V., Hinkelbein, J., ... & Schmitz, J. (2021). European Resuscitation Council guidelines 2021: Cardiac arrest in special circumstances. *Resuscitation, 161*, 152–219. https://doi.org/10.1016/j.resuscitation.2021.02.011

Martinez-Navarro, H., Zhou, X., & Rodriguez, B. (2025). Mechanisms and implications of electrical heterogeneity in cardiac function in ischemic heart disease. *Annual Review of Physiology*, *87*(1), 25-51. https://doi.org/10.1146/annurev-physiol-042022-020541

Maurovich-Horvat, P., Bosserdt, M., Kofoed, K. F., Rieckmann, N., Benedek, T., Donnelly, P., ... & Dewey, M. (2022). CT or invasive coronary angiography in stable chest pain. *New England Journal of Medicine*, *386*(17), 1591-1602. https://doi.org/10.1056/nejmoa2200963

Menon, T., Ogbu, I., & Kalra, D. K. (2024). Sleep-Disordered Breathing and Cardiac Arrhythmias. *Journal of Clinical Medicine*, *13*(22), 6635. https://doi.org/10.1056/nejmoa2200963

Merchant, R. M., Becker, L. B., Brooks, S. C., Chan, P. S., Del Rios, M., McBride, M. E., ... & American Heart Association. (2024). The American Heart Association emergency cardiovascular care 2030 impact goals and call to action to improve cardiac arrest outcomes: A scientific statement from the American Heart Association. *Circulation, 149*(8), e914–e933. https://doi.org/10.1161/cir.0000000000001196

Moras, E., Yakkali, S., Gandhi, K. D., Virk, H. U. H., Alam, M., Zaid, S., ... & Krittanawong, C. (2024). Complications in acute myocardial infarction: Navigating challenges in diagnosis and management. *Hearts*, *5*(1), 122-141. https://doi.org/10.3390/hearts5010009

Mugnai, G., Genovese, D., Tomasi, L., & Ribichini, F. (2025). The role of implantable cardioverter-defibrillators in the elderly. *Progress in Cardiovascular Diseases*. https://doi.org/10.1016/j.pcad.2025.12.002

Nikolovski, S. S., Lazic, A. D., Fiser, Z. Z., Obradovic, I. A., Tijanic, J. Z., Raffay, V., ... & Tijanic, J. (2024). Recovery and survival of patients after out-of-hospital cardiac arrest: A literature review showcasing the big picture of intensive care unit-related factors. *Cureus*, *16*(2). https://doi.org/10.7759/cureus.54827

Panchal, A. R., Bartos, J. A., Wyckoff, M. H., Drennan, I. R., Mahgoub, M., Schexnayder, S. M., ... & Del Rios, M. (2025). Part 2: Evidence evaluation and guidelines development. *Circulation, 152*(16 Suppl. 2), S313–S322. https://doi.org/10.1161/CIR.0000000000001373

Plott, C., Harb, T., Arvanitis, M., Gerstenblith, G., Blumenthal, R., & Leucker, T. (2024). Neurocardiac axis physiology and clinical applications. *IJC Heart & Vasculature, 54*, 101488. https://doi.org/10.1016/j.ijcha.2024.101488

Sirait, N. E., Larasati, T. A., Azka, L., & Saftarina, F. (2025). Systematic literature review: Validitas prognostik NIHSS sebagai prediktor luaran fungsional pada pasien stroke tinjauan periode 2020–2025. *Journal of Medical Practice and Research, 1*(2), 1–9. https://doi.org/10.65310/4rte7j40

Song, T., Hui, W., Huang, M., Guo, Y., Yu, M., Yang, X., ... & Chen, X. (2024). Dynamic changes in ion channels during myocardial infarction and therapeutic challenges. *International journal of molecular sciences*, *25*(12), 6467. https://doi.org/10.3390/ijms25126467

Tabi, M., Perel, N., Taha, L., Amsalem, I., Hitter, R., Maller, T., ... & Jerusalem Platelets Thrombosis and Intervention in Cardiology Study Group. (2024). Out of hospital cardiac arrest new insights and a call for a worldwide registry and guidelines. *BMC Emergency Medicine, 24*(1), 140. https://doi.org/10.1186/s12873-024-01060-4

Taghdiri, A. (2024). Inflammation and arrhythmogenesis: A narrative review of the complex relationship. *International Journal of Arrhythmia, 25*(1), 4. https://doi.org/10.1186/s42444-024-00110-z

Thomas, K. S., Puthooran, D. M., Edpuganti, S., Reddem, A. L., Jose, A., & Akula, S. S. M. (2025). Reperfusion injury in STEMI: a double-edged sword. *The Egyptian Heart Journal*, *77*(1), 83. https://doi.org/10.1186/s43044-025-00683-7

Tsalamandris, S., Koliastasis, L., Miliou, A., Oikonomou, E., Papageorgiou, N., Antonopoulos, A., ... & Tousoulis, D. (2025). Endothelial function and pro inflammatory cytokines as prognostic markers in acute coronary syndromes. *Diagnostics, 15*(8), 1033. https://doi.org/10.3390/diagnostics15081033

Wallentin, L., Eriksson, N., Olszowka, M., Grammer, T. B., Hagström, E., Held, C., ... & Siegbahn, A. (2021). Plasma proteins associated with cardiovascular death in patients with chronic coronary heart disease: A retrospective study. *PLoS Medicine, 18*(1), e1003513. https://doi.org/10.1371/journal.pmed.1003513

Yu, T. (2025). Neurocardiac crosstalk: Sympathetic remodeling and arrhythmogenesis after myocardial infarction. *Current Issues in Molecular Biology, 47*(12), 1037. https://doi.org/10.3390/cimb47121037

Zheng, M., Zhou, Z., Deng, K. Q., Zhang, H., Zeng, Z., Zhang, Y., ... & Lu, Z. (2025). Ventricular arrhythmias and myocardial infarction: Electrophysiological and neuroimmune mechanisms. *Biomedicines, 13*(6), 12 90. https://doi.org/10.3390/biomedicines13061290

