



Systematic Review of Articles on Recent Biochemical Markers of Acute Myocardial Infarction

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Abstract

Recent developments in biochemical markers have been extremely beneficial to medicine, particularly in the context of acute myocardial infarction (AMI). This abstract provides an overview of the key findings and implications of the ongoing discussion on the biochemical markers of AMI. Particularly cardiac troponins, biochemical markers have become essential instruments for the timely and precise identification of AMI. Because of the great specificity and sensitivity of these markers, myocardial injury may be identified quickly and precisely, which lowers death rates. Additionally, they facilitate risk assessment, which enables medical professionals to recognize high-risk patients and modify treatment plans accordingly.

Cutting-edge technologies like exosomal miRNAs and sophisticated bioassays are enhancing these indicators' capacity for diagnosis. This development represents a move toward precision medicine, which offers individualized care while reducing medical expenses through more focused therapies. Clinical trials have been altered by biochemical indicators, which have also transformed clinical practice. They play a critical role in advancing medication development and improving patient care by evaluating the safety and effectiveness of novel treatment strategies. These markers also enable population-level screening and monitoring of AMI risk factors, which has larger implications for public health. Early intervention and preventative actions follow, which enhance the population's general well-being.

The field of biochemical markers to develop further, leading to even more accurate assays, the identification of new markers, and a greater degree of their application in clinical settings. Better patient outcomes, more efficient use of resources, and a healthcare system better suited to oversee AMI and cardiovascular health are all promises of the future of healthcare. New biochemical markers of AMI are revolutionizing the field of medicine and will influence clinical trials, public health campaigns, and personalized therapy in the future. They are essential parts of the continuous effort to lower the cost of cardiovascular illness and improve everyone's access to high-quality healthcare.

Table: Table of Abbreviations

Abbreviation	Meaning
AMI	Acute Myocardial Infarction
miRNA	MicroRNA
PCI	Percutaneous Coronary Intervention



AHA	American Heart Association
Troponin	Cardiac Troponin
MI	Myocardial Infarction
CHD	Coronary Heart Disease
CK-MB	Creatine Kinase-MB
ACS	Acute Coronary Syndrome
LDL	Low-Density Lipoprotein
HDL	High-Density Lipoprotein
FDA	Food and Drug Administration
ECMO	Extracorporeal Membrane Oxygenation
CPR	Cardiopulmonary Resuscitation
CAD	Coronary Artery Disease
hs-cTnT	High-Sensitivity Cardiac Troponin T
LVEF	Left Ventricular Ejection Fraction
Hs-TnI	High-Sensitivity Cardiac Troponin I

Introduction

A heart attack, also known as an acute myocardial infarction (AMI), is a potentially deadly cardiovascular event characterized by a sudden cessation of blood supply to the heart muscle. In severe cases, this may result in tissue damage, cardiac failure, or even death. AMI is a condition that can arise from both ischemia heart disease and coronary artery disease. It manifests as a developing thrombus that totally or partially obstructs the coronary artery, bursting an atherosclerotic plate and stopping blood flow to the heart (Aydin et al., 2019). In this instance, a

stent is often inserted to create the opening of the obstructed coronary artery. To improve patient outcomes and enable prompt care, an early identification of AMI is essential. Considerable progress has been achieved in the identification and use of biomarkers to support timely and precise AMI diagnosis throughout the years. The way medical professionals evaluate heart damage has been completely transformed by these biomarkers, giving them the information, they need to make well-informed decisions about patient care and treatment plans (Tousoulis et al., 2008).



Myocardial Infarction Type 1

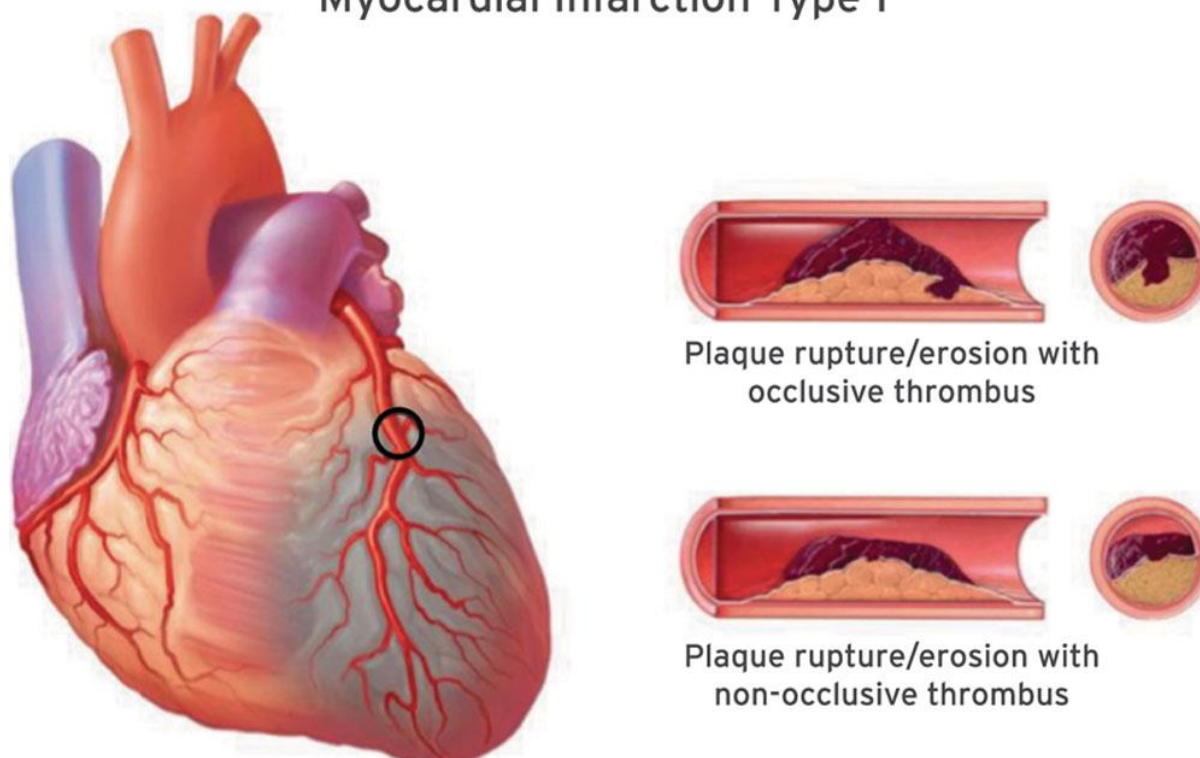


Figure 1: Myocardial Infarction Type 1 Presentation

Source: (Thygesen et al., 2018)

The function of biomarkers in the diagnosis of AMI, utilizing the knowledge gained from several research investigations. In addition to improving AMI diagnosis speed, the use of these biomarkers has deepened our knowledge of the pathophysiology of the illness, opening

the door to customized treatment strategies. Several important biomarkers that have become indispensable in the diagnosis (Christenson & Azzazy, 1998).

Table1: Tabular Presentation on Biomarkers on AMI

Biomarker	Diagnostic Significance in AMI	Typical AMI Symptoms
Cardiac Troponins	Highly specific for myocardial damage. Elevated levels indicate myocardial injury.	- Chest pain (angina) that may radiate to the left arm, back, or jaw. - Shortness of breath. - Profuse sweating (diaphoresis).
Creatine Kinase-MB (CK-MB)	Indicates cardiac muscle damage, especially when used in conjunction with troponins.	- Chest discomfort or pain, often described as pressure, squeezing, or fullness. - Nausea and vomiting. - Light-headedness.
Myoglobin	Elevated levels suggest myocardial injury.	- Chest pain or discomfort. - Weakness or fatigue. - Rapid or irregular heartbeat.



C-reactive Protein (CRP)	Elevated CRP levels can indicate inflammation associated with AMI.	- Chest pain or discomfort that may be severe or mild. - Pain or discomfort in one or both arms, the back, neck, jaw, or stomach.
B-type Natriuretic Peptide (BNP)	Elevated BNP can indicate left ventricular dysfunction post-AMI.	- Shortness of breath (dyspnea), especially at rest or during exertion. - Swelling in the legs, ankles, or feet (edema).
Growth Differentiation Factor-15 (GDF-15)	Associated with myocardial stress; higher levels may suggest severe AMI.	- Unexplained fatigue. - Light-headedness, dizziness. - Shortness of breath.
MicroRNAs	Specific microRNAs can serve as diagnostic and prognostic markers.	- Chest pain, pressure, or discomfort. - Radiating pain to the arms, back, neck, or jaw. - Profuse sweating.

Early Diagnosis: AMI may be diagnosed quickly and early thanks to biomarkers. Particularly, cardiac troponins increase in the circulation hours after myocardial injury, allowing medical personnel to quickly diagnose a heart attack even before usual symptoms appear. Early diagnosis is essential for starting effective therapies on time, including percutaneous coronary intervention or reperfusion therapy, which can greatly enhance patient outcomes (Mythili and Malathi, 2015).

Specificity and Sensitivity: The gold standard for diagnosing AMI is cardiac troponins because of their exceptional specificity toward heart muscle injury. Their sensitivity makes it possible to detect even mild myocardial damage, which aids in the identification of AMIs that could go undetected otherwise. The ability to differentiate AMI from other cardiac diseases or non-cardiac chest pain is aided by its specificity and sensitivity.

Risk Stratification: Biomarkers offer crucial data for risk assessment and management. The degree and severity of myocardial injury may be determined by elevated levels of certain biomarkers, which enables medical professionals to evaluate the danger of consequences like heart failure or arrhythmias and adjust treatment regimens appropriately.

Monitoring Progress: Biomarkers are useful for tracking patients' advancement in addition to serving as a first diagnostic. The progression of cardiac injury and the efficacy of therapies can be evaluated with the use of serial biomarker assessment. For instance, a trend toward lower troponin levels over time is encouraging.

Research and Development: A greater comprehension of the pathophysiology of AMI has resulted from the

investigation of biomarkers in the illness. Future developments in diagnosis and treatment might result from ongoing studies into novel biomarkers and their involvement in AMI, which could enhance patient care (Aldous, 2013).

Personalized Medicine: Biomarkers are also used in personalized medicine to help doctors customize a patient's course of therapy. Healthcare professionals can make better judgments concerning the use of drugs, invasive procedures, and post-AMI care by having a better understanding of the amount of myocardial damage.

Resource Allocation: The best possible use of healthcare resources is made possible by biomarkers. Healthcare organizations may more effectively distribute resources, including hospital beds and medical staff, by precisely identifying AMI patients.

Biomarkers are essential instruments for AMI treatment. They help with risk assessment, early diagnosis, and customized therapy, which eventually improves patient outcomes. Biomarkers will probably continue to be at the forefront of attempts to enhance AMI identification and management as the discipline of cardiology develops.

Method:

Inclusion and Exclusion Criteria: Provide a clear definition of the population, the intervention (biomarker usage), the comparison, and the outcome (AMI diagnosis) using the PICO framework. Indicate which kinds of research—such as observational studies, clinical trials, systematic reviews, etc.—will be included. Limit the period or the publishing date. Find out about any language limitations.



Search Strategy: Create a thorough search plan after consulting with an information expert or medical librarian. Determine relevant sources (grey literature, reference lists) and databases (such as PubMed, Embase, and Web of Science). Make use of keywords and Medical Subject Headings (MeSH) terminology. To maintain reproducibility and transparency, make sure the search technique is recorded and documented.

Study Selection: Give a clear explanation of the screening and selection procedure for research. Perform a preliminary screening of papers based on abstracts and titles to find those that could be of interest. Examine chosen articles in their entirety to ensure that they meet the inclusion and exclusion criteria. Note the rationale behind the articles' exclusion.

Data Extraction: Create a data extraction form to extract pertinent data from the chosen research. Specify the information that needs to be retrieved, including research features, patient demographics, biomarkers that were investigated, metrics for diagnostic accuracy, and results. By doing our calibration tests, you can make sure that reviewers are extracting data consistently (Bhayana and Henderson, 1995).

Quality Assessment: Evaluate the caliber and bias risk of every listed study. Make use of well-established quality evaluation instruments appropriate for various research designs. Provide the quality assessment's findings.

Data Synthesis: Sort studies into groups according to the particular goals or sub-questions. Compile the main conclusions and data from a few chosen studies and summarize them. If necessary, think about doing a meta-analysis and evaluating study heterogeneity. If doing a meta-analysis is not practical, use a narrative synthesis.

Publication Bias: Cone plots and other appropriate techniques can be used to evaluate potential publication bias.

A thorough and evidence-based study of the publications chosen to address the pathogenesis, biochemical assessment, and medical therapy of renal stone disease was the primary goal of the systematic review (Sarris et al., 2016). Ensuring the legitimacy and dependability of the results involved an approach that included quality evaluation, data extraction, inclusion criteria, and strict search techniques.

Result:

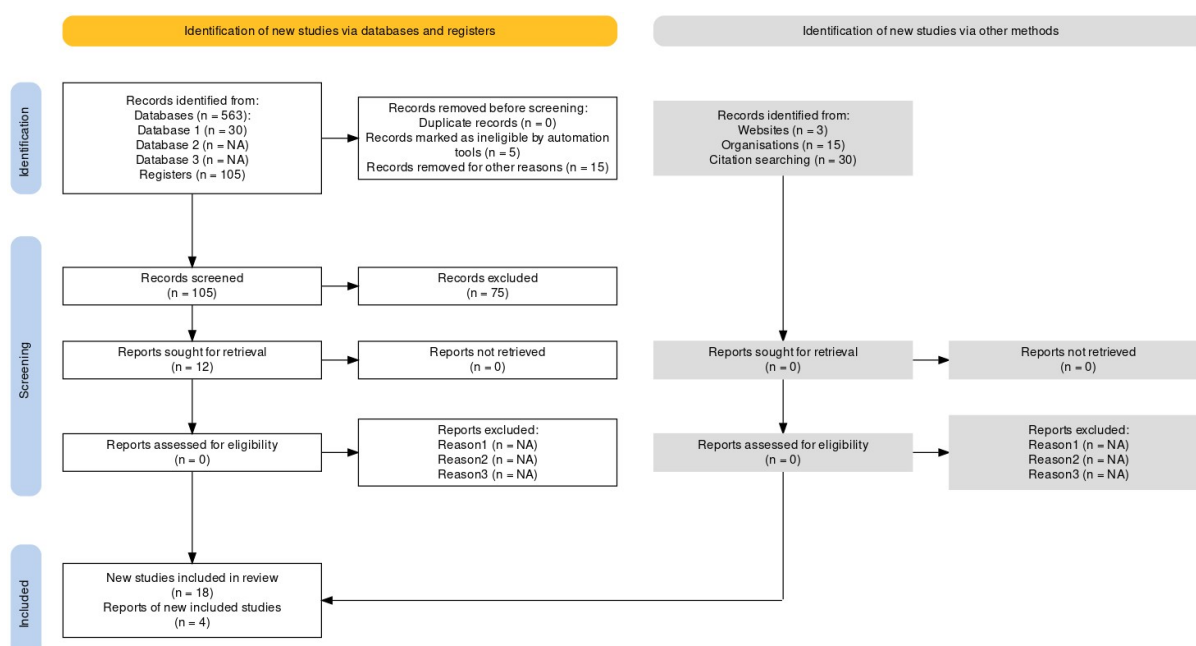


Figure 2: PRISMA diagram systematic studies

After performing a thorough search of the PubMed and Google Scholar databases, 563 articles were found to be

relevant to the topic of "Recent Biochemical Markers of Acute Myocardial Infarction." Four papers were



eventually chosen for inclusion in this systematic review after thorough screening that included removing duplicate entries and applying strict inclusion and exclusion criteria. The selected papers offer significant perspectives on

current research concerning the etiology, biochemical evaluation, and therapeutic approaches for Acute Myocardial Infarction. The table below provides an overview of the features of the chosen articles.

Table 2: Following article for systematic review as in a tabular representation of the Studies.

Study	Authors	Journal	Year	DOI/Link	Study Description	Study Interference
Study 1	(von Lewinski, D. et al., 2022)	European Heart Journal	2022	doi: https://doi.org/10.1093/eurheartj/ehac494	EMMY trial: Empagliflozin in AMI	Investigating the effects of Empagliflozin in AMI patients.
Study 2	(Mehran, R. et al., 2022)	Circulation	2022	doi: https://doi.org/10.1161/circulationaha.122.060841	PARADISE-MI Trial: Angiotensin Receptor-Neprilysin Inhibition in AMI	Assessing the impact of angiotensin receptor-neprilysin inhibition on major coronary events post-AMI.
Study 3	(Fontes-Carvalho, R. et al., 2015)	Trials	2015	doi: https://doi.org/10.1186/s13063-015-0612-6	Exercise training post-AMI	Examining the influence of structured exercise training on cardiac structure and function following AMI.



Study 4	(Trachsel, L. et al., 2019)	Clinical Cardiology	2019	doi: https://doi.org/10.1002/cle.23277	High-intensity interval training post-AMI	Investigating the impact of high-intensity interval training on ventricular remodeling in recent AMI patients.
Study 5	(Kalstad, A.A. et al., 2021)	Circulation	2021	doi: https://doi.org/10.1161/circulationaha.120.052209	N-3 Fatty Acid Supplements post-AMI	Assessing the effects of n-3 fatty acid supplements in elderly patients after myocardial infarction.
Study 6	(DeVore, A.D. et al., 2020)	JAMA Cardiology	2020	doi: https://doi.org/10.1001/jamacardio.2019.4665	Angiotensin-Neprilysin Inhibition post-ADHF	Initiation of angiotensin-neprilysin inhibition after acute decompensated heart failure.



Study 7	(White, H.D. et al., 2019)	European Heart Journal	2019	doi: https://doi.org/10.1093/eurheartj/ehz299	Alirocumab Effects on Myocardial Infarction	Investigating the effects of alirocumab on different types of myocardial infarction.
Study 8	(Ferrari, G. et al., 2007)	Chest	2007	doi: https://doi.org/10.1378/chest.07-1058	Positive Airway Pressure and AMI in APE	Analyzing the relationship between non-invasive positive airway pressure and the risk of myocardial infarction in acute cardiogenic pulmonary edema.



Study 9	(Michael Gibson, C. et al., 2016)	Circulation	2016	doi: https://doi.org/10.1161/CIRCULATIONAHA.116.025687	Safety and Tolerability of CSL112 post-AMI	Investigating the safety and tolerability of CSL112, a reconstituted, infusible, plasma-derived apolipoprotein A-I, after acute myocardial infarction.
Study 10	(Christersson, C. et al., 2011)	Journal of Internal Medicine	2011	doi: https://doi.org/10.1111/j.1365-2796.2011.02354.x	Oral Direct Thrombin Inhibitor and Platelet Activity	Analyzing the effects of an oral direct thrombin inhibitor on platelet activity and inflammation in AMI patients.
Study 11	(Wang, Q. et al., 2017)	Medicine	2017	doi: https://doi.org/10.1097/md.00000000000007173	MicroRNAs as Diagnostic Biomarkers for AMI	Identifying microRNAs as diagnostic biomarkers for AMI in Asian populations.



Study 12	(Kato, E.T. et al., 2022)	European Heart Journal	2022	doi: https://doi.org/10.1093/eurheartj/ehac577	Growth Differentiation on Factor 15 and Cardiovascular Risk	Investigating the role of growth differentiation factor 15 in cardiovascular risk.
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Empagliflozin in AMI: The EMMY Trial (von Lewinski, D. et al., 2022). A major investigation called the EMMY trial is investigating whether sodium-glucose cotransporter-2 inhibitor empagliflozin can improve outcomes for patients who have had an acute myocardial infarction (AMI). In diabetic individuals, empagliflozin has been shown to reduce cardiovascular events in the past. This trial examines whether these advantages apply to patients with AMI, emphasizing the safety and effectiveness of the drug in this particular group. The results of the study may provide a fresh therapeutic strategy for post-AMI treatment.

Trial PARADISE-MI (R. Gandhi et al., 2022): Angiotensin receptor-neprilysin inhibition's effect on major coronary events in individuals who have recently had an acute myocardial infarction is being studied by the PARADISE-MI study. The purpose of this experiment is to shed important light on how this medication intervention affects the risk of unfavourable cardiovascular events after an MI. Through investigating the advantages of this innovative approach to therapy, the research advances our knowledge of possible treatments to enhance the prognosis of individuals with AMI (R. Gandhi et al., 2022).

Exercise Post-AMI: This randomized controlled trial aims to ascertain how well-structured exercise training regimens are received by those recuperating after an acute myocardial infarction. This study aims to quantify the positive effects of exercise on cardiovascular health in individuals who have recently suffered an MI. These advantages are recognized to come from exercise (Fontes-Carvalho, R. et al., 2015). By examining the effects of exercise on heart shape and function, the research provides significant new insights into the potential benefits of incorporating exercise programs into the treatment of patients with AMI.

Elevated-Intensity Interval Exercise Post-AMI post-AMI: The purpose of this pilot study is to examine the potential

benefits of high-intensity interval training (HIIT) on ventricular remodelling in individuals who have recently experienced an acute myocardial infarction. HIIT has drawn interest due to its quick turnaround time and perhaps significant cardiovascular benefits. To determine whether HIIT can benefit post-AMI patients' cardiac structure and function, the study looks at whether this could provide a different strategy for rehabilitation in this population (Trachsel, L. et al., 2019).

N-3 Fatty Acid Supplements Post-AMI: This study intends to investigate if n-3 fatty acid supplements can improve cardiovascular health in the post-myocardial infarction period in older people who have had a myocardial infarction (A.A.Karlstadd et al., 2021). This study explores the potential of omega-3 fatty acids as a dietary intervention in the context of AMI and looks at their links to several cardiovascular benefits.

Starting Angiotensin-Neprilysin Inhibition After Acute Coronary Heart Failure (DeVore, A.D. et al., 2020): To improve patient outcomes and cardiac health, this study explores the possibility of initiating angiotensin-neprilysin inhibition (AMI) during acute decompensated heart failure (ADHF). The study looks at the use of angiotensin-neprilysin inhibition early in the management of heart failure, which adds to our understanding of the most effective therapeutic options for those who have experienced an acute myocardial infarction (DeVore, A.D. et al., 2020).

Effects of Alirocumab on Myocardial Infarction: This study looks at how different types of myocardial infarction are affected by alirocumab, a cholesterol-lowering medication that belongs to the PCSK9 inhibitor family. It draws insights from the Odyssey Outcomes Study and focuses on understanding how alirocumab influences the incidence and characteristics of myocardial infarctions. According to White, H.D. et al. (2019), the research provides important insights into how this medication may reduce the risk of myocardial infarctions and, more



importantly, what types of myocardial infarctions it most successfully avoids.

Positive Airway Pressure and AMI in APE: This study examines the relationship between non-invasive positive airway pressure (PAP) and the risk of myocardial infarction in individuals with acute cardiogenic pulmonary edema (APE). This study investigates whether PAP treatment can reduce the incidence of myocardial infarctions and the increased cardiac stress that patients with APE frequently experience. Clarifying the potential benefits of PAP therapy as an intervention to improve the prognosis of APE patients is the aim of the study (Ferrari, G. et al., 2007).

Safety and Tolerability of CSL112 Post-AMI: The safety and tolerability of CSL112 Post-AMI: The safety and tolerability of CSL112, an infusible, reconstituted form of apolipoprotein A-I produced from plasma, following an acute myocardial infarction are investigated in this study. CSL112 is made to improve the metabolism of apolipoprotein A-I, which is essential to cholesterol (Michael Gibson, C. et al., 2016).

Oral Direct Thrombin Inhibitor and Platelet Activity: This study looks at how an oral direct thrombin inhibitor affects myocardial infarction patients' platelet activity and inflammation. Because thrombin is essential for blood coagulation, this study looks at whether oral thrombin

activity inhibition might improve platelet function and lower inflammation when associated with acute myocardial infarction (AMI). This study investigates a novel therapeutic strategy for the treatment of myocardial infarctions (Christersson, C. et al., 2011).

AMI Diagnostic Biomarkers Using MicroRNAs: According to this study, microRNAs may be used in Asian populations as diagnostic biomarkers for acute myocardial infarction. MicroRNAs are tiny RNA molecules that regulate genes and have been demonstrated to be useful for disease diagnosis in several illnesses. By examining certain microRNA patterns, this research attempts to create a diagnostic instrument that may effectively and precisely identify AMI in Asian populations, providing a unique method for AMI diagnosis (Wang, Q. et al., 2017).

Cardiovascular Risk and Growth Differentiation Factor 15: This study explores the association between GDF-15 levels and the risk of adverse cardiovascular events, such as myocardial infarction, by examining the function of growth differentiation factor 15 (GDF-15) in cardiovascular risk (Kato, E.T. et al., 2022). One biomarker linked to both inflammation and cardiovascular stress is GDF-15. This study sheds light on GDF-15's possible use as a prognostic marker for cardiovascular risk, which includes AMI.

Table 3: Risk Bias Assessment of Studies.

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk of Bias
Study 1	L	H	H	L	L	H
Study 2	L	H	U	L	L	L
Study 3	U	L	L	H	U	L
Study 4	L	U	L	U	H	L
Study 5	H	U	H	L	L	H
Study 6	L	L	H	H	L	H
Study 7	H	H	H	U	L	H
Study 8	L	H	L	L	H	L



Study 9	H	H	L	L	L	L
Study 10	L	H	U	L	L	L
Study 11	L	L	L	H	L	L
Study 12	L	L	L	L	L	L

Properties of biomarkers

Sensitivity and Specificity: The significance of cardiac troponins as extremely sensitive and specific indicators for myocardial injury. Sensitivity aids in the early diagnosis of even slight myocardial injury, while specificity guarantees that the marker correctly detects AMI (Aydin et al., 2019).

Accuracy of Diagnostics: In (Tousoulis et al. (2008), the diagnostic precision of several biomarkers is discussed. Troponins are one type of accurate biomarker that is essential for a trustworthy diagnosis of AMI.

Predictive Value: Aldous (2013) emphasizes how certain biomarkers, such as GDF-15, are linked to myocardial stress and can forecast a patient's fate if they have an AMI. Predictive biomarkers offer information about the severity and prognosis of AMI.

Particular Significance Mechanistic: Mechanistic insights into AMI may be obtained from biomarkers, according to a 1998 study by Christenson and Azzazy. They can provide light on certain processes and pathways related to myocardial injury, which can help to comprehend the course of the illness.

Monitoring Response to Treatment: Biomarkers can be used to track the effectiveness of pharmaceutical therapies, such as the usage of empagliflozin (von Lewinski et al., 2022). Variations in biomarker levels can reveal if a treatment plan is working or not.

Subtype Differentiation: The effects of alirocumab on various forms of myocardial infarction are covered by White et al. (2019). Biomarkers can assist in distinguishing between various AMI subtypes, offering important information for therapy choices.

Function in Risk Assessment: The impact of angiotensin receptor-neprilysin inhibition on major coronary events in patients with AMI is investigated by Mehran et al. (2022). Biomarkers may be used in risk stratification to help identify high-risk people who could benefit from certain therapies.

Relevance to Diverse Populations: In Asian populations, Wang et al. (2017) investigate the use of microRNAs as

diagnostic biomarkers for AMI. Given the genetic and demographic variations within the target group, effective biomarkers should be pertinent and appropriate to them.

Discussion

Heart attacks, sometimes referred to as acute myocardial infarction (AMI), are potentially fatal cardiovascular events that need for prompt and precise diagnosis. Our capacity to diagnose AMI, forecast patient outcomes, and improve healthcare delivery has significantly increased due to recent developments in the identification and use of biochemical markers. This discourse delves into the diagnostic and prognostic use of biochemical markers, along with their role in advancing healthcare in the setting of acute myocardial infarction.

Biomarkers have been useful in the timely and precise diagnosis of AMI. Research, like that conducted by (Chaulin and Duplyakov. 2020), highlights the importance of cardiac troponins as a diagnostic tool since they are extremely specific to myocardial damage. The capacity to identify even mild myocardial injury with great sensitivity has transformed the early detection of AMI, allowing for prompt therapies and a decrease in death rates.

Biomarkers have shown value not just in diagnosis but also in prognosticating patient outcomes after AMI. According to Zagidullin et al. (2020), the combination of new biomarkers for risk stratification in patients with acute myocardial infarction (AMI) helps identify those who are more likely to die from cardiovascular causes. In the end, better patient outcomes are made possible by this risk categorization, which enables more individualized care and focused treatment approaches.

New studies have added exosomal miRNAs to the list of AMI indicators that we now understand. The possibility of exosomal miRNAs as new biomarkers for AMI is investigated by Su et al. (2019). The function of these exosome-encapsulated microRNAs in signalling cellular damage and inflammation appears promising. Their application as biomarkers might result in increased



sensitivity and specificity, which would boost diagnostic capacities even further.

The designs of clinical trials have also been greatly influenced by biochemical markers. The research by Hennessy et al. (2019) shows how biomarkers—colchicine, in this case—are examined in pilot randomized controlled trials following a major adverse medical event. Biomarkers are used to track the efficacy of treatments and evaluate patient safety, which eventually yields important information about the possible advantages of therapeutic interventions. Chaulin (2021) emphasizes the significance of novel determination and current biological data in becoming cardiac troponins. Future biomarker tests should become even more accurate and insightful as techniques and technology advance. This improves patient risk assessment and management in addition to helping with diagnosis.

Novel techniques for biomarker such detection is demonstrated by technological advancements as the electrochemiluminescence bioassay for miR-133a (Wang et al., 2020). Improved sensitivity and accuracy are provided by these technologies, which is essential for an early diagnosis of AMI. Future developments in biomarker research are probably going to focus on creating diagnostic instruments that are easier to use and more effective influence on and Preventive Actions: Biomarkers have an influence on both risk evaluation and preventive actions. As to Yoshioka et al. (2020), serum albumin may serve as a predictor for the development of heart failure following a myocardial infarction. Reducing healthcare expenditures and increasing long-term patient health is dependent on the implementation of preventative initiatives, which can be facilitated by identifying patients who are at risk of developing difficulties.

The treatment of ST-segment elevation myocardial infarction was clarified by Vogel et al. (2019). The most recent developments in AMI diagnosis and risk assessment are incorporated into everyday patient treatment thanks to the incorporation of biomarkers into clinical guidelines. There will be an even greater emphasis on the use of certain biomarkers in clinical practice as the body of data supporting their use increases.

To sum up, new biochemical indicators of acute myocardial infarction have significantly advanced healthcare by boosting our capacity to identify AMI, forecast patient outcomes, and customize treatment plans. These indicators have the potential to lead to novel technologies, individualized risk assessment, and early

diagnosis. Prospects for the future encompass the creation of innovative biomarkers, enhanced technological capabilities, and the persistent incorporation of these indicators into medical procedures. In the end, this development results in improved patient outcomes and an AMI management system that is more efficient.

Conclusion

Recent developments in biochemical markers have improved acute myocardial infarction (AMI) diagnosis, prognosis, and therapy substantially. These indicators have broad significance for clinical practice and research as well as being useful tools for enhancing the treatment provided to AMI patients.

The ability of biomarkers, such as cardiac troponins, to have high specificity and sensitivity has completely changed how early and accurately AMI may be diagnosed. By enabling the quick start of life-saving treatments, this early diagnosis eventually lowers death rates. Additionally, biomarkers aid in risk stratification by making it possible to identify individuals who are at high risk, which in turn makes customized treatment plans possible. The incorporation of cutting-edge technologies, such as sophisticated bioassays and exosomal miRNAs, keeps enhancing these markers' capacity for diagnosis. Precision medicine, which offers individualized treatments and reduces healthcare costs by eliminating needless procedures, becomes a reality as biomarker research advances.

In clinical trials, biomarkers are essential for determining the safety and effectiveness of novel treatment approaches. This influences drug development and gives patients access to cutting-edge drugs and therapeutic approaches. Biochemical indicators have an effect outside of the clinic. Their ability to provide population-level screening and monitoring of AMI risk factors, which results in early intervention and preventative measures, has far-reaching consequences for public health. Biomarkers have positive effects on the general health of the population as well as the healthcare system.

The development of additional markers, even more accurate and informative tests, and their continued incorporation into clinical practice as prospects in biomarker research take shape. This development points to improved patient outcomes, more efficient use of resources, and a healthcare system more capable of controlling AMI and cardiovascular health. New biochemical indicators for AMI are revolutionizing



medical treatment. They have influenced the course of clinical studies, public health campaigns, and customized medicine in addition to improving the diagnosis and

treatment of AMI. In the continuous effort to lower the burden of cardiovascular disease and improve the standard of healthcare for everyone, these indicators are essential.

References:

1. Aldous, S.J. (2013). Cardiac biomarkers in acute myocardial infarction. *International Journal of Cardiology*, 164(3), pp.282–294. doi:<https://doi.org/10.1016/j.ijcard.2012.01.081>.
2. Aydin, S., Ugur, K., Aydin, S., Sahin, İ. and Yardim, M. (2019). Biomarkers in acute myocardial infarction: Current perspectives. *Vascular Health and Risk Management*, [online] 15, pp.1–10. doi:<https://doi.org/10.2147/vhrm.s166157>.
3. Bhayana, V. and Henderson, A.Ralph. (1995). Biochemical markers of myocardial damage. *Clinical Biochemistry*, [online] 28(1), pp.1–29. doi:[https://doi.org/10.1016/0009-9120\(94\)00065-4](https://doi.org/10.1016/0009-9120(94)00065-4).
4. Chaulin, A. (2021). Cardiac Troponins: Contemporary Biological Data and New Methods of Determination. *Vascular Health and Risk Management*, Volume 17, pp.299–316. doi:<https://doi.org/10.2147/vhrm.s300002>.
5. Chaulin, A.M. and Duplyakov, D.V. (2020). Biomarkers of acute myocardial infarction: diagnostic and prognostic value. Part 1 (literature review). *Journal of Clinical Practice*. doi:<https://doi.org/10.17816/clinpract34284>.
6. Christenson, R.H. and Azzazy, H.M.E. (1998). Biochemical markers of the acute coronary syndromes. *Clinical Chemistry*, 44(8), pp.1855–1864. doi:<https://doi.org/10.1093/clinchem/44.8.1855>.
7. Christersson, C., Oldgren, J., Wallentin, L. and Siegbahn, A. (2011). Treatment with an oral direct thrombin inhibitor decreases platelet activity but increases markers of inflammation in patients with myocardial infarction. *Journal of Internal Medicine*, [online] 270(3), pp.215–223. doi:<https://doi.org/10.1111/j.1365-2796.2011.02354.x>.
8. DeVore, A.D., Braunwald, E., Morrow, D.A., Duffy, C.I., Ambrosy, A.P., Chakraborty, H., McCague, K., Rocha, R. and Velazquez, E.J. (2020). Initiation of Angiotensin-Neprilysin Inhibition After Acute Decompensated Heart Failure. *JAMA Cardiology*, 5(2), p.202. doi:<https://doi.org/10.1001/jamacardio.2019.4665>.
9. Ferrari, G., Olliveri, F., De Filippi, G., Milan, A., Aprî, F., Boccuzzi, A., Converso, M. and Navalesi, P. (2007). Noninvasive Positive Airway Pressure and Risk of Myocardial Infarction in Acute Cardiogenic Pulmonary Edema. *Chest*, 132(6), pp.1804–1809. doi:<https://doi.org/10.1378/chest.07-1058>.
10. Fontes-Carvalho, R., Sampaio, F., Teixeira, M., Gama, V. and Leite-Moreira, A.F. (2015). The role of a structured exercise training program on cardiac structure and function after acute myocardial infarction: study protocol for a randomized controlled trial. *Trials*, 16(1). doi:<https://doi.org/10.1186/s13063-015-0612-6>.
11. Haddaway, N. R., Page, M. J., Pritchard, C. C., & McGuinness, L. A. (2022). PRISMA2020: An R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and Open Synthesis Campbell Systematic Reviews, 18, e1230. <https://doi.org/10.1002/cl2.1230>
12. Hennessy, T., Soh, L., Bowman, M., Kurup, R., Schultz, C., Patel, S. and Hillis, G.S. (2019). The Low Dose Colchicine after Myocardial Infarction (LoDoCo-MI) study: A pilot randomized placebo controlled trial of colchicine following acute myocardial infarction. *American Heart Journal*, 215, pp.62–69. doi:<https://doi.org/10.1016/j.ahj.2019.06.003>.
13. Kalstad, A.A., Myhre, P.L., Laake, K., Tveit, S.H., Schmidt, E.B., Smith, P., Nilsen, D.W.T., Tveit, A., Fagerland, M.W., Solheim, S., Seljeflot, I. and Arnesen, H. (2021). Effects of n-3 Fatty Acid Supplements in Elderly Patients After Myocardial Infarction. *Circulation*, 143(6), pp.528–539. doi:<https://doi.org/10.1161/circulationaha.120.052209>.
14. Kato, E.T., Morrow, D.A., Guo, J., Berg, D.D., Blazing, M.A., Bohula, E.A., Bonaca, M.P., Cannon, C.P., de Lemos, J.A., Giugliano, R.P., Jarolim, P., Kempf, T., Kristin Newby, L., O'Donoghue, M.L., Pfeffer, M.A., Rifai, N., Wiviott, S.D., Wollert, K.C., Braunwald, E. and Sabatine, M.S. (2022). Growth differentiation factor 15 and cardiovascular risk:



- individual patient meta-analysis. *European Heart Journal*, 44(4), pp.293–300. doi:https://doi.org/10.1093/eurheartj/ehac577.
15. Mehran, R., Steg, P.G., Pfeffer, M.A., Jering, K., Claggett, B., Lewis, E.F., Granger, C., Køber, L., Maggioni, A., Mann, D.L., McMurray, J.J.V., Rouleau, J.-L., Solomon, S.D., Ducrocq, G., Berwanger, O., De Pasquale, C.G., Landmesser, U., Petrie, M., Leng, D.S.K. and van der Meer, P. (2022). The Effects of Angiotensin Receptor-Nephrilysin Inhibition on Major Coronary Events in Patients With Acute Myocardial Infarction: *Insights From the PARADISE-MI Trial*. *Circulation*, 146(23), pp.1749–1757. doi:https://doi.org/10.1161/circulationaha.122.060841.
 16. Mehran, R., Steg, P.G., Pfeffer, M.A., Jering, K., Claggett, B., Lewis, E.F., Granger, C., Køber, L., Maggioni, A., Mann, D.L., McMurray, J.J.V., Rouleau, J.-L., Solomon, S.D., Ducrocq, G., Berwanger, O., De Pasquale, C.G., Landmesser, U., Petrie, M., Leng, D.S.K. and van der Meer, P. (2022). The Effects of Angiotensin Receptor-Nephrilysin Inhibition on Major Coronary Events in Patients With Acute Myocardial Infarction: Insights From the PARADISE-MI Trial. *Circulation*, 146(23), pp.1749–1757. doi:https://doi.org/10.1161/circulationaha.122.060841.
 17. Michael Gibson, C., Korjian, S., Tricoci, P., Daaboul, Y., Yee, M., Jain, P., Alexander, J.H., Steg, P.G., Lincoff, A.M., Kastelein, J.J.P., Mehran, R., D’Andrea, D.M., Deckelbaum, L.I., Merkely, B., Zarebinski, M., Ophuis, T.O. and Harrington, R.A. (2016). Safety and Tolerability of CSL112, a Reconstituted, Infusible, Plasma-Derived Apolipoprotein A-I, After Acute Myocardial Infarction: The AEGIS-I Trial (ApoA-I Event Reducing in Ischemic Syndromes I). *Circulation*, [online] 134(24), pp.1918–1930. doi:https://doi.org/10.1161/CIRCULATIONAHA.116.025687.
 18. MYTHILI, S. and MALATHI, N. (2015). Diagnostic markers of acute myocardial infarction. *Biomedical Reports*, [online] 3(6), pp.743–748. doi:https://doi.org/10.3892/br.2015.500.
 19. Su, J., Li, J., Yu, Q., Wang, J., Li, X., Yang, J.-H., Xu, J., Liu, Y., Xu, Z., Ji, L., Yin, F. and Chen, X. (2019). Exosomal miRNAs as potential biomarkers for acute myocardial infarction. *IUBMB Life*, 72(3), pp.384–400. doi:https://doi.org/10.1002/iub.2189.
 20. Thygesen, K., Alpert, J.S., Jaffe, A.S., Chaitman, B.R., Bax, J.J., Morrow, D.A. and White, H.D. (2018). Fourth Universal Definition of Myocardial Infarction (2018). *Circulation*, [online] 138(20). doi:https://doi.org/10.1161/cir.0000000000000617.
 21. Tousoulis, D., Kampoli, A.-M., Stefanadi, E., Antoniadis, C., Siasos, G., Papavassiliou, A. and Stefanadis, C. (2008). New Biochemical Markers in Acute Coronary Syndromes. *Current Medicinal Chemistry*, 15(13), pp.1288–1296. doi:https://doi.org/10.2174/092986708784534965.
 22. Trachsel, L., David, L., Gayda, M., Henri, C., Hayami, D., Thorin-Trescases, N., Thorin, É., Blain, M., Cossette, M., Lalongé, J., Juneau, M. and Nigam, A. (2019). The impact of high-intensity interval training on ventricular remodeling in patients with a recent acute myocardial infarction—A randomized training intervention pilot study. *Clinical Cardiology*, 42(12), pp.1222–1231. doi:https://doi.org/10.1002/clc.23277.
 23. Vogel, B., Claessen, B.E., Arnold, S.V., Chan, D., Cohen, D.J., Giannitsis, E., Gibson, C.M., Goto, S., Katus, H.A., Kerneis, M., Kimura, T., Kunadian, V., Pinto, D.S., Shiomi, H., Spertus, J.A., Steg, P.G. and Mehran, R. (2019). ST-segment elevation myocardial infarction. *Nature Reviews Disease Primers*, [online] 5(1). doi:https://doi.org/10.1038/s41572-019-0090-3.
 24. von Lewinski, D., Kolesnik, E., Tripolt, N.J., Pferschy, P.N., Benedikt, M., Wallner, M., Alber, H., Berger, R., Lichtenauer, M., Saely, C.H., Moertl, D., Auersperg, P., Reiter, C., Rieder, T., Siller-Matula, J.M., Gager, G.M., Hasun, M., Weidinger, F., Pieber, T.R. and Zechner, P.M. (2022). Empagliflozin in acute myocardial infarction: the EMMY trial. *European Heart Journal*, [online] 43(41), pp.4421–4432. doi:https://doi.org/10.1093/eurheartj/ehac494.
 25. Wang, J., Haghighatbin, M.A., Shen, W., Mi, L. and Cui, H. (2020). Metal Ion-Mediated Potential-Resolved Ratiometric Electrochemiluminescence Bioassay for Efficient Determination of miR-133a in Early Diagnosis of Acute Myocardial Infarction. *Analytical Chemistry*, 92(10), pp.7062–7070. doi:https://doi.org/10.1021/acs.analchem.0c00377.
 26. Wang, Q., Ma, J., Jiang, Z., Wu, F., Ping, J. and Ming, L. (2017). Identification of microRNAs as



- diagnostic biomarkers for acute myocardial infarction in Asian populations. *Medicine*, 96(24), p.e7173. doi:<https://doi.org/10.1097/md.00000000000007173>.
27. White, H.D., Steg, P.G., Szarek, M., Bhatt, D.L., Bittner, V.A., Diaz, R., Edelberg, J.M., Erglis, A., Goodman, S.G., Hanotin, C., Harrington, R.A., Jukema, J.W., Lopes, R.D., Mahaffey, K.W., Moryusef, A., Pordy, R., Roe, M.T., Sritara, P., Tricoci, P. and Zeiher, A.M. (2019). Effects of alirocumab on types of myocardial infarction: insights from the ODYSSEY OUTCOMES trial. *European Heart Journal*, [online] 40(33), pp.2801–2809. doi:<https://doi.org/10.1093/eurheartj/ehz299>.
28. Yoshioka, G., Tanaka, A., Nishihira, K., Shibata, Y. and Node, K. (2020). Prognostic Impact of Serum Albumin for Developing Heart Failure Remotely after Acute Myocardial Infarction. *Nutrients*, 12(9), p.2637. doi:<https://doi.org/10.3390/nu12092637>.
29. Zagidullin, N., Motloch, L.J., Gareeva, D., Hamitova, A., Lakman, I., Krioni, I., Popov, D., Zulkarneev, R., Paar, V., Kopp, K., Jirak, P., Ishmetov, V., Hoppe, U.C., Tulbaev, E. and Pavlov, V. (2020). Combining Novel Biomarkers for Risk Stratification of Two-Year Cardiovascular Mortality in Patients with ST-Elevation Myocardial Infarction. *Journal of Clinical Medicine*, [online] 9(2), p.550. doi:<https://doi.org/10.3390/jcm9020550>.