DOI:doi.org/10.65766/alyj.2026.24.01.10

Heart-type Fatty Acid-Binding Protein (H-FABP) in Early Diagnosis of Acute Myocardial Infarction

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Important Dates

Received:2026/4/10 ,Accepted:2026/6/7 Published: 2026/6/30

Abstract:

Acute myocardial infarction (AMI), among the most fatal forms of acute coronary syndrome, remains challenging to diagnose accurately in the emergency department, especially during the earliest phase of symptoms, before conventional biomarkers like cardiac troponin and CK-MB become elevated. Here, we reviewed the diagnostic performance of H-FABP in early detection of AMI and considered its potential use in standard diagnostic protocols. H-FABP has low molecular weight (14-15 kDa) and is present free in the cytoplasm of cardiomyocytes. Within one to three hours of myocardial damage, H-FABP is released into the circulation, preceding cardiac troponin, which typically reaches detectable levels after more than four hours. Available evidence suggests that the early sensitivity of H-FABP is superior to troponin alone and using H-FABP and troponin as a dual-biomarker test improves the diagnostic accuracy of early AMI detection and supports early decision-making in patients with acute chest pain in the emergency department. Given that other conditions like renal failure, pulmonary embolism and heart failure can elevate H-FABP levels, this protein cannot be used as a sole marker of AMI diagnosis due to low specificity. Therefore, we concluded that H-FABP could have a role as an adjunctive marker when combined with sensitive cardiac troponin assays, ECG and clinical assessment. Further studies are needed to further evaluate the utility of H-FABP and to standardize cutoff levels and patient populations who would benefit most from H-FABP testing to help its broader use in clinical practice.

Keywords: Acute Myocardial Infarction ،Heart-type Fatty Acid-Binding Protein ،Early Cardiac Biomarkers ،High-sensitivity Cardiac Troponin.

تقييم دور بروتين ارتباط الأحماض الدهنية القلبي (H-FABP) في التشخيص المبكر لاحتشاء عضلة القلب الحاد

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DOI:doi.org/10.65766/alyj.2026.24.01.10

الملخص:

يعد احتشاء عضلة القلب الحاد أحد أشد مظاهر المتلازمة التاجية الحادة خطورة، ويظل التشخيص المبكر الدقيق له تحديا حقيقيا في أقسام الطوارئ، لا سيما في الساعات الأولى من بداية الأعراض حين قد لا تبلغ الواسمات الحيوية التقليدية — كالتروبونين القلبي و CK-MBمستويات قابلة للكشف. تهدف هذه المراجعة إلى تقييم الدور التشخيصي لبروتين ارتباط الأحماض الدهنية القلبي (H-FABP)في الكشف المبكر عن احتشاء عضلة القلب الحاد، ومناقشة إمكانية دمجه ضمن البروتوكولات التشخيصية المعيارية. يتميز H-FABP بوزنه الجزيئي المنخفض (14–15 كيلودالتون) وموقعه السايتوبلازمي الحر داخل الخلايا العضلية القلبية، مما يتيح له التحرر السريع إلى الدورة الدموية خلال ساعة إلى ثلاث ساعات من بداية الأذية القلبية، متقدما بذلك على التروبونين الذي يحتاج في الغالب إلى أربع ساعات أو أكثر. تشير الأدلة إلى أن H-FABP يتمتع بحساسية مبكرة أعلى من التروبونين، وأن دمجه معه ضمن نهج الواسمين المزدوجين يحسن الدقة التشخيصية الإجمالية ويدعم قرار الفرز المبكر في الطوارئ. غير أن محدودية نوعيته — إذ يرتفع في حالات غير قلبية كالقصور الكلوي والانصمام الرئوي وفشل القلب — تحول دون اعتماده واسما تشخيصيا مستقلا. وتخلص هذه المراجعة إلى أن H-FABP يؤدي دورا تكامليا واعدا حين يستخدم بالتوازي مع التروبونين عالي الحساسية وتخطيط القلب الكهربائي والتقييم السريري، بيد أن تعميم استخدامه السريري يستلزم مزيدا من الدراسات لتوحيد قيمه الحدية وتحديد الفئات التي تستفيد منه أكثر.

الكلمات المفتاحية: احتشاء عضلة القلب الحاد ، بروتين ربط الأحماض الدهنية من النوع القلبي ، الواسمات الحيوية القلبية المبكرة ، التروبونين القلبي عالي الحساسية

Introduction

Heart disease caused by insufficient blood flow to the heart (ischaemic heart disease) is one of the top causes of death globally. In fact, cardiovascular disease continues to be the biggest cause of disability and death worldwide and the cardiovascular disease death rate continues to increase worldwide since 1990 (Shi et al. , 2025). Global Burden of Disease data revealed that the number of deaths due to cardiovascular disease rose from 12.4 million in 1990 to 19.8 million in 2022 as a result of global population growth and ageing as well as the continuing problem of preventable cardiovascular risk factors (Mensah et al. , 2023). Of these conditions, acute myocardial infarction is the most lethal form of acute coronary syndrome and is a true challenge for emergency department clinicians. When facing these cases, deciding on how to proceed is a critical task in the first few hours and directly determines the patient's outlook and the size of the area of necrosis. Diagnosis relied historically on 3 clinical facts: the clinical findings, the ECG and biochemical tests. Yet in one in three patients, the ECG will not show obvious diagnostic changes at the start of the disease, meaning that the biochemical tests remain essential (Moon et al. , 2021). From a biochemical standpoint, CK-MB and cardiac troponins (cTnI and cTnT) used to be primary tests for the diagnosis of acute myocardial infarction. Troponins became, over the past several years, the most sensitive paraclinical tests to establish an acute myocardial infarction diagnosis (Pavel et al., 2024). The issue with troponins is the delayed release, as troponin cannot be reliably identified at blood level until 4 to 6 h after symptoms arise. However, in 4 to 6 h, the area of necrosis can increase. That is precisely why there is an urgent requirement for sensitive biomarkers that rise early into the bloodstream, which permit cardiac injury detection as early as possible (Jaiswal et al., 2022). Thisgap is filled by an promising biomarker, heart-type fatty acid-binding protein (H-FABP), a 14-15 kDa low molecular weight cytoplasmic protein abundant within the cardiac cells, that is released from injured heart muscle into the circulation and detectable within just 1 to 3 h from symptoms. As this protein is small, it can escape rapidly from the damaged muscle into circulation even before other, larger proteins such as troponin and CK-MB can rise into blood and provide a significant advantage in emergency rooms (Ye et al., 2018). Numerous

DOI:doi.org/10.65766/alyj.2026.24.01.10

investigations have worked toward validating its superior ability in the differential diagnosis of suspected MI with comparable results, this study demonstrated 96.2% for H-FABP and 81.3% for troponin I, both in the early phase. There is greater strength in these cumulative data, with an updated systematic review that included 2,735 patients demonstrating that H-FABP alone had a higher diagnostic sensitivity than troponin alone (0.80 vs 0.73) and that the combined diagnostic value of these markers results in an area under the receiver-operating characteristic (ROC) curve (AUC) of 0.881 with a combined sensitivity of 0.91, emphasizing the fact that there should be a coordinated diagnostic strategy that guides decisions in the early period of suspected MI in patients. Alternatively, a weakness of H-FABP is its poor specificity: In some studies involving a similar emergency population, specificity has been shown to be as low as 42% or even 70%, attributable to cardiac non-ischemic causes in the extra cardiac region or chronic renal insufficiency. Therefore, H-FABP is far from being an isolated or complete marker in its stand-alone diagnostic capacity but should be seen as a contributing marker in a combination approach, especially when taken in comprehensive clinical and biochemical evaluation. Efforts continue to clarify the true role and place of H-FABP within diagnostic algorithms for MI patients, but existing evidence still support its position as an important marker for early injury and long-term prediction more so than myoglobin and troponin. Even further validation comes from rapid point-of-care test options which can now analyze blood for the protein levels in minutes without laboratory, therefore directly influencing time optimization and throughput for emergency patients. In this investigation, H-FABP's relevance was discussed for the rapid diagnosis of acute MI, subsequently, it was further discussed how its integration into the routine diagnosis, within biochemical investigations for all patients with MI suspected visiting emergency units, would be feasible, considering that quick identification of this protein directly translates to effective management of these cases in a timely manner and thus potential decreased mortality and less impact on healthcare spending.

Pathophysiological Background of Acute Myocardial Infarction

Acute myocardial infarction is the final result of vascular and cellular changes that often begin with a sudden disturbance in blood flow within the coronary artery. The event occurs when an atherosclerotic plaque ruptures or erodes from the wall of an artery, which activates platelets, initiates the coagulation system, forms a thrombus that either partially or totally obstructs the artery and causes significant reduction of blood flow-oxygen supply and delivery of nutrients- to an area of the heart muscle. Affected cells then begin to suffer from ischaemia, to an extent that varies by the vessel diameter, time it takes to relieve the occlusion and whether or not collateral arteries offer an alternative blood supply. The formation of a thrombus is central to the pathogenesis of acute coronary syndrome and eventual injury to the heart muscle (Udaya & Sivakanesan, 2022). Ischaemia in cardiac myocytes results in disruption in ATP production through inhibition of oxidative phosphorylation inside the mitochondria.With continued energy deficiency, the cell loses its ability to maintain ionic balance, particularly the regulation of sodium and calcium within the cell, leading to cellular swelling and a disturbance in cell membrane integrity, as well as a progressive weakness in contractile function. Furthermore, cardiac metabolism shifts partially towards anaerobic pathways; lactate accumulates and intracellular pH decreases, which increases functional and structural disturbance in the affected cardiac tissue (He et al., 2022). As the period of ischaemia extends, the injury does not remain a mere transient functional disorder; rather, myocardial cells begin to enter stages that include mitochondrial damage,

DOI:doi.org/10.65766/alyj.2026.24.01.10

increased cell membrane permeability, activation of oxidative stress and inflammatory pathways, and subsequently, the occurrence of various patterns of cell death. As a result, intracellular components are released into the bloodstream to varying degrees, which explains the biological basis for the rise in cardiac markers after an infarction occurs. The time of appearance of these markers in the blood varies depending on their location within the cell, their molecular weight, and the extent of their binding to cellular structures; therefore, some small cytoplasmic proteins may appear earlier than larger or more myofibril-bound markers (Boivin-Proulx et al., 2023). Damage in acute myocardial infarction is not limited to the ischaemia phase alone; the restoration of blood flow, despite being a primary therapeutic goal, may be accompanied by additional injury known as ischaemia/reperfusion injury. This injury occurs due to the sudden return of oxygen to the damaged tissue and the associated increase in free radical production, mitochondrial dysfunction, and excessive calcium influx into cells, in addition to the activation of the inflammatory response. Therefore, reopening the coronary artery does not mean that all damaging mechanisms stop immediately; rather, some molecular pathways may continue to contribute to determining the final size of the damage to the heart muscle (Algoet et al., 2022). While the inflammatory response seems like a normal part of the infarction process because inflammation starts in the surrounding tissue surrounding the myocardial cells that have just died from injury and have emptied their contents to the surrounding area to activate the inflammatory response that would attract various inflammatory cells to the injury region, neutrophils and monocytes, to eliminate the dead cells and pave the way for tissue repair, the extent of this inflammation matters and may lead to expand the injury in size or the more damaging effects of left ventricular remodeling if the response persists or if the inflamed region is sufficiently large or extends beyond the area originally affected. And even if it does not have a large effect on cardiac function at present or does not increase the future risk for cardiac issues, it might have been responsible for a portion of the loss of myocardial cells initially (Ying et al. , 2020). From this explanation, the need for prompt assessment becomes clear because if the ischaemia time lasts longer than some crucial time window (which may vary between 20 and 120 minutes), it can result in greater injury to the myocardium, potentially expanding to include areas of necrosis that are irreparable and will cause chronic damage, more importantly, it increases the likelihood of developing potentially fatal acute complications like ventricular fibrillation, left ventricular insufficiency and cardiogenic shock. This need highlights the usefulness of early biomarkers that are indicative of cellular injury starting early after STEMI occurs. H-FABP is one of them, several new articles suggest its clinical value when used with the current panel of biomarkers for helping with acute myocardial infarction diagnoses early in patients.

Current Diagnostic Approach of Acute Myocardial Infarction

A current diagnosis of MI depends on assessing symptoms, electrocardiogram (ECG) and cardiac biomarkers. Although these elements are vital in suspected acute MI, neither suffices for diagnosis, especially given that symptoms can be nonspecific or unusual, the ECG is often negative in the early phase of MI or non-ST elevation myocardial infarction, the cardiac biomarkers' levels can depend on how much time has passed since symptoms began or other factors. Hence, an accurate diagnosis relies on a real-time simultaneous interpretation of all elements within the given clinical context. Since diagnosis and prompt initiation of therapy are time-sensitive matters in an emergency setting, the physician must make a simultaneous interpretation of the available data Joyce et al. , 2023. Chest compression or tightness, especially when radiating to the left arm, jaw or back, is among the 3 signs

DOI:doi.org/10.65766/alyj.2026.24.01.10

that best predict acute coronary syndromes. Nonetheless, neither a symptom consistent with chest pressure and left arm pain nor the absence of such pain is sufficient to establish or refute a diagnosis, because symptoms can vary. Among typical symptoms are fatigue, shortness of breath, nausea, diaphoresis or epigastric distress. Atypical symptoms occur in many women, elderly patients and diabetic patients. Because a symptom complex does not reliably exclude a diagnosis, it alone cannot confirm it Khan et al. , 2023. ST-elevation myocardial infarction (STEMI) is suggested by at least 1 mm (0.1 mV) of ST-segment elevation in at least 2 contiguous precordial or limb ECG leads. Although STEMI is suggested by ST elevation, a diagnosis can be made without ST elevation. The 12-lead ECG is useful for confirming or refuting a STEMI diagnosis in many patients. Nevertheless, the absence of ST-segment elevation in a patient with symptoms does not exclude the possibility of acute myocardial infarction (AMI), an NSTEMI, in patients who already exhibit a positive cardiac biomarker or have signs of myocardial infarction despite a negative initial ECG Khan et al. , 2020. Cardiac markers (especially high-sensitivity cardiac troponin) have become of interest as new diagnostic tools in AMI. In acute settings, elevated troponin indicates myocardial damage and serial measurements assess dynamic changes in troponin levels. However, chronic elevation of troponin does not indicate an ongoing AMI. Rapid diagnostics strategies, like 0/1 hour or 0/3 hour, are rapidly becoming common, with physicians quickly able to establish a negative diagnosis or begin therapeutic options for an impending or completed AMI, greatly shortening wait times for emergency department (ED) patients with symptoms of chest pain (Muzyk et al. , 2020). Modern diagnostic approaches in the ED require high-sensitivity troponin results integrated with information on the time to symptom onset as well as the ECG interpretation (where relevant) to rapidly categorize a patient into three strata: exclusion, confirmation or observation. The 0/1-hour protocol using high-sensitivity troponin levels has shown high accuracy for rapidly excluding and confirming MI, reducing the cardiac event rates during the 30 days of follow-up in excluded patients (Burgos et al., 2020). Despite the importance of high-sensitivity troponin is a really sensitive tool, but we should still be really careful with it. An elevated troponin does tell us there is damage to the heart, but not always what the damage is. An elevated troponin can be seen in other conditions besides type 1 MI such as heart failure, kidney failure, myocarditis, PE, sepsis or issues related to oxygen supply-demand mismatch. Because of this, a troponin reading should never be considered in isolation and needs to be integrated along with patient history, ECG and clinical examination. Because we often do not do either, it can also lead to an over-diagnosis or unnecessary hospital admission for a condition. Based on these arguments, the diagnosis of acute MI is a principle of integration, not substitution. Our clinical assessment sets the probability for MI, the ECG helps with ruling out STEMI and biomarkers objectify cardiac injury. However, a gap exists for those who are the earliest presenters to the ER. Traditional cardiac biomarkers might not have increased sufficiently enough at that stage. We still need to see if early markers like H-FABP would add additional diagnostic value for MI detection, but it likely will not act as a stand-alone test to replace history, clinical assessment and cardiac troponin testing, but will probably be added in conjunction with a set of biomarkers in the diagnostic workup for MI detection.

Cardiac Biomarkers in Acute Myocardial Infarction

The value of cardiac biomarkers lies in objectively indicating the injury of heart muscle cells, assisting in patient management when symptoms or ECG readings are confusing. Important attributes

DOI:doi.org/10.65766/alyj.2026.24.01.10

of these biomarkers include accurately reflecting cellular damage, their timed release into the blood after injury and their selectivity (specificity and sensitivity) for the heart muscle. Therefore, deciding to include a particular cardiac marker depends not only on its elevation but also on understanding the marker's rate of rise and fall (kinetics), tissue-of-origin characteristics and confounding factors related to other underlying conditions (Netala et al. , 2025). For the modern laboratory diagnosis of acute myocardial infarction, cardiac troponin (and especially high-sensitivity cardiac troponin) has become the most relied-upon biomarker. This type of troponin shows a high specificity for myocardial injury and is used to confirm or exclude diagnosis, especially in emergency settings. An elevated troponin simply shows damage to the heart, but does not necessarily mean type 1 myocardial infarction, patients with heart failure, renal impairment, myocarditis, sepsis or pulmonary embolism all may show elevated troponin. Thus, troponin should not be relied upon in isolation but should be studied in context with symptoms, ECG findings and changes in its values over time (Thygesen et al. , 2018). Cardiac enzymes CK-MB and myoglobin have also been used historically in diagnosing acute myocardial infarction, but these markers are no longer recommended as part of the modern diagnosis with the widespread acceptance of high-sensitivity cardiac troponin. CK-MB had some value for cardiac injury and sometimes for recurrent myocardial infarction, but it is less specific to the heart than troponin. Myoglobin rises early following injury of the muscles, but because skeletal muscles have large quantities of myoglobin, it is not a specific marker for the heart, so it holds limited value as a standalone marker (Patibandla et al. , 2023). However, research suggests that timing kinetics may play an essential role in interpreting cardiac marker findings. Cardiac markers are distinct in terms of when they rise, when they reach their peak and when they disappear from the circulation following muscle damage. Markers such as myoglobin and H-FABP tend to rise earlier following injury to the heart, but troponins remain essential in the confirmation of acute myocardial infarction because of their higher cardiac specificity and longer duration in circulation after muscle damage. The difference in kinetics explains why many researchers are interested in combinations of cardiac markers for diagnostics, in patients presenting to the emergency department within the first few hours of the onset of symptoms (Kristensen et al., 2023). Heart-type fatty acid binding protein (H-FABP) is among the early markers for acute myocardial infarction. It is cytoplasmic in origin and its small molecular size makes it rapid to leave the heart upon cell injury. Therefore, H-FABP may contribute to narrowing the temporal gap between the onset of cardiac injury and the appearance of some traditional markers at detectable levels. However, its diagnostic value should not be understood in isolation from the other elements of assessment, but within an integrated framework that combines clinical history, ECG, cardiac troponin, and symptom onset time. Accordingly, the modern trend in diagnosis is not based on absolute reliance on a single marker, but on employing the appropriate marker at the appropriate time, in line with the temporal stage of the disease and the patient's clinical condition (Kulshrestha et al., 2022).

Biochemical Characteristics of H-FABP

Heart-type fatty acid binding protein is considered a member of the intracellular fatty acid binding protein family. This family is characterised by its ability to bind to long-chain fatty acids and hydrophobic lipid molecules, which helps in transporting them within the aqueous environment of the cytoplasm and directing them towards their sites of utilisation or oxidation within the cell. Since the heart muscle relies to a great extent on fatty acid oxidation, its abundance within myocardial cells

DOI:doi.org/10.65766/alyj.2026.24.01.10

reflects its vital role in regulating cellular handling of fatty acids and providing them for metabolic processes associated with energy production (Rezar et al., 2020). From a molecular perspective, H-FABP is characterised by its small size and low molecular weight, which is approximately 14–15 kDa, in addition to being a highly soluble cytoplasmic protein. The reason why H-FABP exhibits this property is related to three different points. First, unlike proteins which are tightly related with contractile system, the major amount of H-FABP remains in the sarcoplasmic space as free protein without strong relationship with contractile part in myocardial cells. Secondly, this molecule is a protein with low MW (~13 kDa). Small and unstuck protein may readily diffuse to the extracellular space. On the other hand, when the heart is subjected to severe injury, for instance during ischemia and subsequent infarction, disturbance of the cell membrane integrity makes free 40-kDa proteins within sarcoplasm readily pass to the interstitial fluid space. Larger and strongly bound 39- to 31-kDa structural proteins which do not belong to the sarcoplasm tend to be released much later, since disruption of contractile elements is necessary for their migration from the myocyte (Ren et al. , 2021). So, we are given with free cytoplasmic location and a small MW of the H-FABP, an interesting molecule released easily following the heart injury (Xin et al. , 2021). Therefore, scientific importance of the H-FABP is conferred from its three major overlapping points: the cytoplasmic location within heart muscles, small molecule weight and its transportation role for fatty acid within the cardiac cell, which can not only interpret its physiological metabolism in heart muscles but also the reason for its rapid release from injured cells and attraction for the study on the point of early marker of cardiac damage. Thus, the discussion on the potential use in the diagnostics for the early determination of acute myocardial infarction can only be undertaken after knowing its features as listed above.

Release Kinetics of H-FABP After Myocardial Injury

H-FABP's release pattern is one of the primary reasons that it is of interest as an early marker for cardiac injury. H-FABP is released quickly from cardiac muscle cells following the destruction of the cell membrane and thus reaches blood circulation within the first phase of cardiac injury. Some other markers take longer to rise to useful values. However, this does not imply that H-FABP is a suitable alternative for cardiac troponin, which is the more accepted marker for confirmation of cardiac damage due to its greater specificity and position in the current diagnostic standards of care. So the benefit of H-FABP comes from its potential use as an additional marker in a combination assay that includes the onset of symptoms, ECG changes, troponin and the current clinical condition of the patient. Its levels should also be interpreted with caution in cases of renal impairment or non-cardiac muscle injuries that may affect its diagnostic specificity (Ștef et al., 2024).

Diagnostic Role of H-FABP in Early AMI Detection

H-FABP represents one of the early biomarkers that may add value in evaluating cardiac injury, especially during the first hours after symptom onset. Its importance stems from its ability to appear early in the blood compared to some traditional markers, which may help in narrowing the temporal gap between the onset of cardiac injury and the possibility of its laboratory detection. Recent evidence indicates that integrating H-FABP with high-sensitivity troponin may improve patient triage in the early stages, especially in cases of chest pain arriving at emergency departments within a short period from symptom onset. A dual-marker approach, based on H-FABP and hs-Troponin-I at presentation,

DOI:doi.org/10.65766/alyj.2026.24.01.10

has demonstrated good ability for early exclusion, with a continued need for follow-up in inconclusive cases (Kulshrestha et al., 2022). However, H-FABP's diagnostic performance may be affected by non-cardiac factors, and algorithms based on high-sensitivity troponin remain the most stable in diagnosing and excluding NSTEMI. Therefore, its most appropriate role currently appears to be as an auxiliary marker, not a substitute for troponin or for clinical assessment and ECG (Yildirim et al., 2025).

Clinical Limitations and Interpretive Challenges of H-FABP

Although H-FABP is considered one of the promising early markers in evaluating cardiac injury, its speed of rise in the blood grants it a temporal advantage, but it does not mean that it is capable of confirming acute myocardial infarction independently, as its elevation may also occur in non-infarction conditions such as heart failure, pulmonary embolism, and renal impairment. Therefore, it should be interpreted as an auxiliary marker within the full clinical context, not as an independent test by itself (Goel et al., 2020). It may be affected by several factors not related to myocardial infarction, such as kidney function disturbance or the presence of other chronic cardiac diseases. Furthermore, the absence of a clear consensus regarding its routine use and cut-off values makes its clinical adoption less stable than high-sensitivity cardiac troponin, which still represents the foundation in modern diagnostic algorithms (Burgos et al., 2020). Additionally, its introduction into clinical practice requires more large-scale studies that take into account sample collection time, kidney function, patient nature, and the measurement method used. Its closest value currently appears to be in its support for early assessment within a multi-marker strategy, rather than its use as an independent substitute for troponin or ECG (Chandra et al., 2020).

Future Perspectives

Further studies are required to precisely determine the place of H-FABP in the rapid diagnosis of ACS, mainly when patients are presenting within the first hours, where early diagnosis may be elusive. With H-FABP identified as among the best predictors of early myocardial damage, its use demands larger trials that incorporate variations in sampling delay, patient profile, the used immunoassay, plus the confounding effects of underlying conditions, standardizing cut-offs and laboratory practices is crucial prior to wider integration in clinical decision rules, with the expectation that H-FABP, if applied as part of multiple tests, would not be used as a single diagnostic tool. (Demirel et al. , 2025).

Conclusion

The marker heart-type fatty acid-binding protein is considered among the best available early biomarkers for the diagnosis of acute myocardial infarction since this biomarker is easily released into the blood after injury to the heart muscle cells, especially during the first few hours of onset of symptoms, as certain clinical or laboratory parameters may be ambiguous. However, it is no substitute for hs-cTn, but it can play an auxiliary role in a general diagnostic workup that involves the history, ECG, timing of symptom onset and other cardiac biomarkers. Due to some limitations in its specificity, its release being triggered by several conditions unrelated to infarction and the requirement of more large-scale trials to assess the potential value in an early diagnostic workup, the subgroups more

DOI:doi.org/10.65766/alyj.2026.24.01.10

benefiting from this test than others, its performance according to when the samples are obtained and comorbid conditions that potentially affect it, there remains debate about the use of H-FABP in early diagnosis guidelines. Once the answers to these questions are addressed, a judgment can be reached on its possible role in diagnostic guidelines in emergency departments to improve diagnostic speed, therapeutic management and critical time in the management of these patients.

Conflict of Interest

The author declares that there is no conflict of interest regarding the publication of this paper.

Acknowledgement

:first all of ,Thanks for supervisor Mr.Dr Ammar alazawy .and thanks and appreciation for Mr. editor of journal to accept my research publication

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