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ORIGINAL RESEARCH

# Importance of H-FABP in Early Diagnosis of Acute Myocardial Infarction

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**Background:** Over the years, troponins have aced the para-clinical tests for confirming the diagnosis of acute myocardial infarction. However, the rise in their levels is entirely time-dependent, which can cause a delay in the initiation of treatment protocols. Heart fatty acid binding protein (H-FABP) can serve comparatively as a better biological marker for overcoming this flaw of troponins, as it is quickly released into the bloodstream once the myocardial injury occurs due to decreased blood supply. This study aimed to evaluate the usefulness of this marker as well as establish the specificity and sensitivity of testing the H-FABP, if it adds to early diagnosis and can be relied upon in the future.

**Material and Methods:** We evaluated 83 patients and their H-FABP levels, along with the standard cardiac markers like hsTni and CK-MB, in patients presenting with symptoms indicating an ongoing coronary event, who had presented to our hospital between August 2020 and June 2021. The patients were divided into two groups: group 1 comprised patients who had first medical contact within 4 hours of the onset of chest pain, and group 2 patients who had first medical contact after 4 hours of the appearance of symptoms. Statistical analysis was performed using MedCalc v20.023, considering statistical significance values of $p < 0.05$. Results for targeted variables are presented using descriptive statistics (mean, standard deviation, range, median, and associated interquartile range) for continuous data, and counts with associated percentages for categorical data.

**Results:** H-FABP was found to have better sensitivity and specificity of 89.67 and 95.65 in group 1 patients and 86.73 and 49.84, respectively, in group 2 patients. The other two cardiac biomarkers evaluated had lower values in response to H-FABP in the first 4 hours of presentation. Results for group 2 showed that specificitivity for hsTni is higher than that of H-FABP, that is, 69.98.

**Conclusion:** Heart fatty acid binding protein (H-FABP) should be included in the protocol for biochemical evaluation of all patients presenting to the emergency services with a suspicion of possible myocardial infarction. Early detection of this protein can help in effective and timely management of myocardial infarction, thus further decreasing mortality rates and the financial burden on healthcare systems worldwide.

**Keywords:** heart fatty acid binding protein, H-FABP, cardiac biomarker, troponin, myocardial infarction, acute coronary syndrome

## Introduction

Acute myocardial infarction (AMI) is a critical cardiovascular event that demands immediate medical attention and medical intervention.[1] Recognizing the symptoms, seeking prompt medical attention, running confirmatory tests and treating accurately all play a crucial role when the clock is ticking at a faster rate in this emergency situation. Despite the latest advancements, mortality rates still remain high in AMI patients.[2] Early diagnosis strategies can help rule out non-cardiac causes of chest pain and also help in cases presenting with non-ST elevation myocardial infarction (NSTEMI), whereby physicians have to keep a continuous watch on the troponin levels of the patients.[3] Lifestyle changes and

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adherence to post-AMI treatment can all lead to better outcomes and improve quality of life (QOL) in patients suffering from coronary artery disease (CAD); however, early diagnosis remains crucial for treating this devastating disease.[4]

The diagnosis of AMI is currently based on clinical and paraclinical findings. Ischemic changes lead to symptoms of typical chest pain, for which patients usually approaches the emergency services. Electrocardiogram (ECG) results show ST–elevation or depression and/or pathological Q-waves or they can be normal. A typical increase in cardiac troponin T or I levels and a rapid rise in creatine kinase isoenzyme myocardial band (CK-MB) levels usually confirms diagnosis. Coronary angiography can be used as a confirmatory test and as a therapeutic procedure but, in many instances, healthcare facilities lack these units or specialized teams for interventional cardiology, which further increases the difficulty of treating this life-threatening condition.[5,6]

Troponin is a widely used cardiac biomarker for the diagnosis and risk stratification of CAD, a condition characterized by narrowing or blockage of coronary arteries, thus compromising blood supply to the myocardium. Troponins are a group of proteins found in cardiac myocytes, which play a crucial role in regulating muscle contraction. During myocardial injury due to decreased perfusion of the muscle wall, such as in the case of myocardial infarction (heart attack) or unstable angina, troponins are released into the bloodstream, leading to elevated levels of this biomarker.[7]

The use of troponin as a diagnostic marker for CAD has revolutionized the field of cardiovascular medicine. Traditionally, the diagnosis of CAD relied on clinical symptoms, electrocardiogram findings, and imaging techniques, which were limited in their sensitivity and specificity. Lately, troponins, particularly cardiac troponin I (cTnI) and cardiac troponin T (cTnT), have emerged as highly sensitive and specific biomarkers for detecting myocardial injury associated with CAD. Troponin levels are measured upon admission to the hospital and at subsequent time points to assess the extent of myocardial injury and guide clinical decision-making.[8]

The development of high-sensitivity troponin assays (Hs-T) has further improved the detection and risk stratification of CAD. These high-sensitivity assays can measure troponin levels with greater precision at lower concentrations, enabling the detection of even minor myocardial damage. Hence, it can be said that the high-sensitivity troponin assays have improved the early diagnosis of CAD, allowing prompt initiation of appropriate treatment strategies and thus achieving better patient outcomes. However, a means of diagnosing myocardial infarction as quickly as possible, given the fact that the time elapsed from the occurrence of the infarction to the opening of the coronary artery is directly proportional to the amount of necrotic myocardium, remains elusive. Unfortunately, the detection of cardiac troponins is done a few hours after the occurrence of myocardial necrosis.[9,10] In addition to these markers, the neutrophil to albumin ratio can be considered as a predictor of severity of CAD in NSTEMI patients.[11] Global longitudinal strain (GLS) value is now used for prediction of mortality rates in STEMI patients.[12]

Hence, it can be said that troponin, specifically cTnI and cTnT, is a valuable biomarker for the diagnosis and risk stratification of CAD. Its high sensitivity and specificity, along with the advent of high-sensitivity troponin assays, have transformed the approach to CAD diagnosis, enabling earlier detection of myocardial injury and improved risk assessment.[13]

The timeline for troponin may vary from individual to individual and is based on the severity of the myocardial infarction. A general timeline of troponin levels in myocardial infarction calculated from the appearance of symptoms is illustrated in Table 1. (Table 1)

Another cardiac marker routinely used is CK-MB, an enzyme found predominantly in the heart muscle. It is a subtype of the creatine kinase isoenzyme, which is responsible for catalyzing the conversion of creatine to phosphocreatine, playing a role in energy metabolism within cells. CK-MB is commonly used as a diagnostic marker for myocardial infarction. It is released in the bloodstream, indicating damage to the myocytes. Therefore, elevated levels of CK-MB in the blood can indicate acute myocardial infarction (AMI) or other heart-related conditions.[14,15]

Traditionally, CK-MB was measured through laboratory blood tests. However, in recent years, the use of other cardiac biomarkers, such as troponin, has become more prevalent due to their higher specificity and sensitivity in diagnosing myocardial injury.[15]

Lactate dehydrogenase (LDH) is an enzyme found in various tissues of the body, including the heart. LDH exists in different forms, or isoenzymes, which are designated LDH-1 to LDH-5. LDH-1 is mainly found in the heart, and LDH-2 is found in lesser amounts in the heart as well as in other tissues.

**Table 1** Timelines Concerning Troponin Levels in Myocardial Infarction from the Appearance of Symptoms

| Timeline | Troponin Levels |
|---|---|
| Time of Onset | Troponin levels may initially be normal or slightly elevated. |
| 3–6 Hours | Troponin levels start to rise, indicating cardiac muscle damage. |
| 2–24 Hours | Troponin levels continue to increase, reaching their peak. The magnitude of the rise depends on the extent of the myocardial injury. |
| 48–72 Hours | Troponin levels start to decrease as the damaged cardiac muscle begins to heal. However, they may still remain elevated above the baseline |
| 3–5 Days | Troponin levels continue to decline further, gradually returning to normal. |

Karakayalı et al demonstrated that the white blood cell count to mean platelet volume ratio is associated with early risk stratification in patients with ST-segment elevation myocardial infarction.[16] Similarly, the authors also stated that the prognostic value of the hemoglobin, albumin, lymphocyte, and platelet (HALP) score, which indicates the nutritional and inflammatory status of the body, can be helpful in predicting in-hospital mortality in patients with ST-elevation myocardial infarction undergoing coronary intervention.[17]

During an AMI, due to the adverse effect of reduced perfusion on the myocardial cells, LDH-1 levels in the bloodstream can increase (Table 2). However, LDH is not considered a specific marker for myocardial infarction, as LDH is also present in other tissues, such as the liver, kidneys, and red blood cells. Therefore, LDH alone is not commonly used as a definitive diagnostic tool for MI. Heart fatty acid binding protein (H-FABP) is a cardiac biomarker that has gained attention over recent years for its potential role in the diagnosis and management of myocardial infarction, commonly known as a heart attack. H-FABP is a small protein primarily found in cardiac muscle cells and is involved in the transport of fatty acids within the heart. During a myocardial infarction, damaged heart cells release H-FABP into the bloodstream, leading to elevated levels of this biomarker.[18–20] Measurement of this biomarker is accurate, non-invasive, easy to perform, reveals infarct-related artery patency, and can be used to check if thrombolysis is adequate or not.[21] The urine and plasma levels of H-FABP can also be loyal indicators of myocardial perfusion injury in patients undergoing cardiac surgery, according to a study conducted on ten patients by Hayashida et al[22]

The use of H-FABP as a cardiac marker has been investigated recently to determine its diagnostic value and potential clinical applications. Early detection of myocardial infarction is crucial for prompt intervention and optimal patient outcomes. Traditional cardiac biomarkers, such as troponin, have been widely used for the diagnosis of myocardial infarction, but they may take several hours to become elevated after the onset of symptoms. It can also be used a prognostic tool for determining a 30-day risk evaluation for patients suffering from acute coronary syndrome.[23] In a study conducted by O'Donoghue et al involving 2287 patients suffering from ACS, the clinical importance of prognostic evaluation with H-FABP was proven.[24] The study demonstrated how H-FABP can be considered superior to other cardiac biomarkers in terms of mortality risk prediction.

The unique characteristic of H-FABP is its rapid release into the bloodstream following cardiac injury, making it a promising candidate for early detection of myocardial infarction. Studies have shown that H-FABP can be detected in the blood as early as 1–3 hours after the onset of symptoms, providing a potential advantage over other cardiac biomarkers.[25,26]

However, the clinical utility of H-FABP as a stand-alone marker for myocardial infarction diagnosis is still under investigation. Its specificity to myocardial infarction has been questioned, as elevated H-FABP levels can also occur in

**Table 2** Timeline Comparison of Different Cardiac Markers in Relation to Acute Myocardial Infarction

| Cardiac Marker | Detectable | Peak Levels are Achieved | Returns to Normal |
|---|---|---|---|
| Troponin- I | 4 to 6 hours | 12 hours | 3 to 10 days |
| Troponin-T | 4 to 8 hours | 12 to 48 hours | 7 to 10 days |
| CK-MB | 4 to 8 hours | 12 to 24 hours | 72 to 96 hours |
| LDH | 2 to 5 days | | 10 days |

**Abbreviations**: CK-MB, creatine kinase isoenzyme myocardial band; LDH, lactate dehydrogenase.

other conditions that cause damage to the heart muscle, such as myocarditis or congestive heart failure. Therefore, H-FABP is often used in combination with other markers, clinical symptoms, and diagnostic tools to improve the accuracy of myocardial infarction diagnosis.

The study aims to explore iwhether H-FABP can be beneficial in the context of early diagnosis of acute myocardial infarction compared to other standard cardiac markers, to evaluate its diagnostic value, potential use in risk stratification, and limitations.

## Materials and Methods

We evaluated 83 consecutive COVID-negative myocardial infarction patients admitted between August 2020 to June 2021 in the intensive coronary care unit of the County Hospital, Baia Mare, Romania, out of the total of 485 coronary patients addressed in that period. The patients were tested for H-FABP levels along with standard cardiac markers at the time of presentation to the hospital with symptoms indicating an ongoing coronary event. The patients were divided into two groups. Patients presenting to the hospital between 0 to 4 hours of the onset of chest pain were labelled group 1 and those presenting later than 4 hours were termed group 2. Patients were also divided according to having STEMI or not having STEMI. Statistical analysis of the results was performed to identify the specificity and sensitivity of testing the H-FABP and whether it contributes to early diagnosis and can be relied upon.

Before initiating the study, approval from the relevant ethical committees was obtained, according to the tenets of the Declaration of Helsinki. The study was approved by the Comisia de Etică a Cercetării Stiintifice (Ethics Committee for Scientific Research) of the Dr. Constantin Opriș County Emergency Baia Mare Hospital, Baia Mare (24406, 27.08.2021) and by the ethics committee of the Victor Babes University of Medicine and Pharmacy, Timisoara (approval reg. no. 74/2020, 22/2020 rev. 01.04.2024).

The inclusion criteria in the study were represented by the presence of the diagnosis of MI established in the following cumulative conditions: patients being diagnosed based on the European Society of Cardiology guidelines[27] for acute myocardial infarction andpresenting at the hospital up to a maximum of 24 hours after the onset of pain. Those with thrombotic occlusion or stenotic lesion in the left coronary artery, circumflex coronary artery, and/or right coronary artery were included.

The exclusion criteria were as follows: a diagnosis of neoplasm, degenerative disease, atrial fibrillation, or COVID-19 infection; presenting at the hospital more than 24 hours after the onset of pain; participation in any clinical study in the previous month; or having hematological, hepatic, or renal pathologies.

The patients' written informed consent was obtained before enrollment in our study.

There were no exclusion criteria based on sex, age, political or religious affiliation, socio-economic status, place of origin (city or village), or means of transport to hospital.

After expressing voluntary agreement to participate in the study based on the inclusion and exclusion criteria, 83 patients were selected.

To establish the diagnosis of acute myocardial infarction, at the time of presentation to the emergency department of the hospital the patients biological samples were sent for the determination of enzymes of myocardial necrosis (high-sensitivity troponin I, total CK, CK-MB), blood count, serum creatinine, serum urea, serum glucose, ASAT, ALAT, and coagulation levels. For H-FABP evaluation, blood collection was also carried out, but its determination required a special regime.

The plasma, obtained by blood centrifugation, was stored at −40°C until the number of patients required to open the H-FABP determination kit was reached. For the determination of H-FABP, the Human FABP3 (cardiac) ELISA Kit was used, which had a maximum possible number of 96 samples.

In the shortest possible time from the first medical contact, the patients were transferred to the cardiac catheterization laboratory for emergency coronary angiography to detect the culprit lesion, as well as for interventional de-blockage through the percutaneous intravascular placement of pharmacologically active stents in accordance with the current recommendations of the practice guidelines of the European Society of Cardiology.[27] The drug treatment instituted after the coronary revascularization procedures was according to the recommendations of good practice guidelines.[27] The data were centralized using Microsoft Excel and their statistical processing was carried out using MedCalc v20.023,

considering statistical significance values of p <0.05. We use descriptive statistics, figures, and tables to summarize our findings. Results for targeted variables are presented using descriptive statistics (mean, standard deviation, range, median, and associated interquartile range) for continuous data, and counts with associated percentages for categorical data.

## Results

We analyzed 83 patients diagnosed with MI, who underwent angio coronagraphy and were divided according to the time elapsed from the onset of pain to the first medical contact into two groups. Group 1 patients had their first medical contact at 0–4 hours after the onset of pain (36 patients) and the second group at 4–24 hours after (47 patients). Further, they were divided according to the number of arteries causing the vascular disease, monovascular, bivascular or trivascular, and the type of myocardial infarction, that is, whether STEMI or non-STEMI (Figure 1). The basal demographic data are shown in Table 3. (Table 3)

Among these patients, three in group 1 were suffering from multi-vessel disease and 12 from group 2. There were 33 patients with monovascular disease in group 1 and 35 in group 2. Regardless of when the patient presented, the presence of non-STEMI myocardial infarction was statistically significant (chi-square test, *p*-value = 0.0029). It can also be observed that the probability of presenting with a STEMI increases significantly if the duration from the onset of pain to the first medical contact is over 4 hours (two-sample *z*-test for proportions, *p*-value = <0.0001). Even in this situation, the number of non-STEMI coronary events is significantly higher during the same time interval (two-sample *z*-test for proportions, *p*-value = <0.0001). It is also observed that the incidence of non-STEMI type events predominates regardless of time interval until the moment of first medical contact.

To analyze the usefulness of H-FABP compared to already used enzyme markers (CK-MB respectively hsTnI) in the diagnosis of patients with acute coronary syndromes to confirm the diagnosis of myocardial infarction, an ROC curve was used. An ROC curve (receiver operating characteristic curve) is a graph showing the performance of a classification model at all classification thresholds. The statistical test used was a comparison of ROC curves and areas under the curve (AUC) for several classification variables of MI.

Based on the data analyzed in this study, it can be said that, despite intense efforts via social media to highlight the possibility of experiencing a myocardial infarction in the case of chest pain, the presentation of patients to the emergency room is still quite delayed. Further, a significant number of patients who could benefit from interventional myocardial reperfusion methods fail to present themselves during the optimal therapeutic window. In the situation of patients presented in the first 4 hours from the onset of pain, a comparison of ROC curves is presented in Table 4, the variables used being CK-MB, H-FABP, and hsTni for patients who presented with MI. (Table 4) As can be seen from Table 5 and Figure 2, (Table 5) (Figure 2) there are statistical differences between the AUC for the three elements under discussion, between CK-MB and hsTni, registering a *p*-value of 0.035, and for the comparison between H-FABP and hsTni,

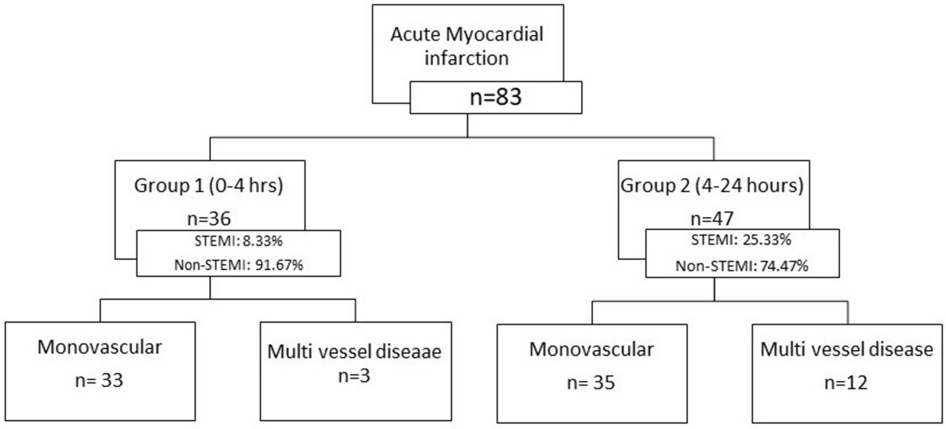


**Figure 1** Patient distribution according to the moment of first medical contact, type of myocardial infarction, and number of vessels involved.

**Table 3** Characteristics of the Study Population

| Parameter | Value (n=83) | Percentage Value (%) |
|---|---|---|
| Gender | | |
| Male | n=50 | 60.24 |
| Female | n=33 | 39.73 |
| Age above 60 years | n= 67 | 80.72 |
| Urban | n=66 | 79.51 |
| Rural | n=17 | 20.48 |
| Obesity | n=48 | 57.83 |
| Hypertension | n=55 | 66.26 |
| Known cardiac co-morbidities | n= 31 | 37.34 |
| Dyslipidemia | n=74 | 89.15 |
| Diabetes | n=49 | 59.03 |

**Table 4** Comparison of AUC for Patients Presenting in Less than 4 Hours from the Onset of Chest Pain

| Variable | AUC | SE[a] | 95% CI[b] |
|---|---|---|---|
| CK_MB | 0.884 | 0.0667 | 0.733 to 0.966 |
| H_FABP | 0.960 | 0.0346 | 0.835 to 0.997 |
| hsTni | 0.753 | 0.103 | 0.581 to 0.881 |

**Notes**: [a] DeLong et al (1988);[18] [b] Binominal exact.

**Table 5** Pairwise Comparison of ROC Curves for Patients Presenting in Less than 4 Hours from the Onset of Chest Pain

| CK_MB–H_FABP | |
|---|---|
| Difference between areas | 0.0758 |
| Standard error[a] | 0.0737 |
| 95% confidence interval | −0.0686 to 0.220 |
| z statistic | 1.028 |
| Significance level | *p*-value = 0.3038 |
| CK_MB ~ hsTni | |
| Difference between areas | 0.131 |
| Standard error[a] | 0.0621 |
| 95% confidence interval | 0.00957 to 0.253 |
| z statistic | 2.114 |
| Significance level | *p*-value = 0.0345 |
| H_FABP ~ hsTni | |
| Difference between areas | 0.207 |
| Standard error[a] | 0.100 |
| 95% confidence interval | 0.0103 to 0.404 |
| z statistic | 2.063 |
| Significance level | *p*-value = 0.0391 |

**Notes**: [a]DeLong et al (1988).[18]

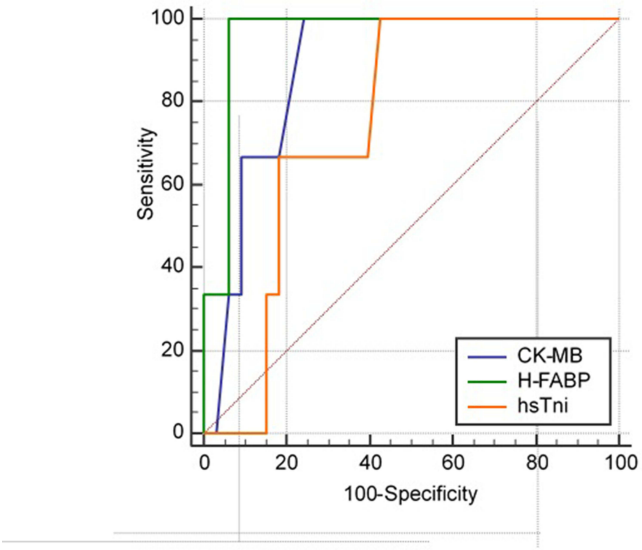


**Figure 2** Pairwise comparison of ROC curves for patients included in group 1.

registering a *p*-value of 0.039. In other words, hsTni is a weaker discriminating element than H-FABP and CK-MB in the case of patients presenting at the hospital up to a maximum of 4 hours after the onset of chest pain.

Regarding group 2, the analyzed parameters were the same (CK-MB, hsTni, H-FABP). The comparison of AOC is presented in Tables 6 and 7 and Figure 3 shows the pairwise comparison of ROC curves. (Tables 6 and 7)

As results from the data show, there are statistical differences between the AUC of H-FABP and hsTni (*p*-value = 0.035) and, in this situation, H-FABP proves to be a significantly better discriminator in detecting acute myocardial infarction at an early stage (Figure 3).

The sensitivity and specificity of all three biomarkers studied, in both groups based on time of presentation, are shown in Table 8. (Table 8)

**Table 6** Comparison of AUC for Patients Presenting After 4 Hours from the Onset of Chest Pain

| Variable | AUC | SE[a] | 95% CI[b] |
|---|---|---|---|
| CK_MB | 0.581 | 0.103 | 0.428 to 0.723 |
| H_FABP | 0.876 | 0.0889 | 0.747 to 0.954 |
| hsTni | 0.565 | 0.101 | 0.413 to 0.709 |

**Notes**: [a]DeLong et al (1988;[18] [b]Binominal exact.

**Table 7** Pairwise Comparison of ROC Curves for Patients Presenting After 4 Hours from the Onset of Chest Pain

| CK_MB–H_FABP | |
|---|---|
| Difference between areas | 0.295 |
| Standard error[a] | 0.163 |
| 95% confidence interval | −0.0243 to 0.615 |
| z statistic | 1.811 |
| Significance level | *p*-value = 0.0702 |

(*Continued*)

**Table 7** (Continued).

| | |
|---|---|
| CK_MB ~ hsTni | |
| Difference between areas | 0.0155 |
| Standard error[a] | 0.124 |
| 95% Confidence Interval | −0.228 to 0.259 |
| z statistic | 0.125 |
| Significance level | *p*-value = 0.9008 |
| H_FABP ~ hsTni | |
| Difference between areas | 0.311 |
| Standard Error[a] | 0.148 |
| 95% confidence interval | 0.0213 to 0.600 |
| z statistic | 2.104 |
| Significance level | *p*-value = 0.0354 |

**Notes**: [a]DeLong et al (1998).[18]

## Discussion

The data presented in our research clearly demonstrates that H-FABP can be a useful tool for diagnosing acute myocardial infarction at an early stage, especially when clinical and paraclinical evaluations are not clear or require time. A simple cardiac biomarker like H-FABP can help clinicians save time and reach accurate diagnoses both in STEMI and non-STEMI cases.

Serial troponin levels are required to rule out the chance of having a myocardial injury if their level at the time of the first determination does not fall within the diagnostic limits for acute myocardial infarction.

During COVID-19, many doctors struggled to differentiate between the most common symptom, myalgia, and chest pain due to a cardiac cause or entirely due to COVID-19. Indeed, some researchers described myocardial issues post-COVID-19 infectionas increasing the complexity involved in diagnosing and managing already complex entities.[28,29] Various studies reported the presence of chest pain mimicking myocardial infarction and a rise in new-onset rhythm disturbances, in previously healthy individuals,[30] especially when anxiety levels were high due to the devastating nature

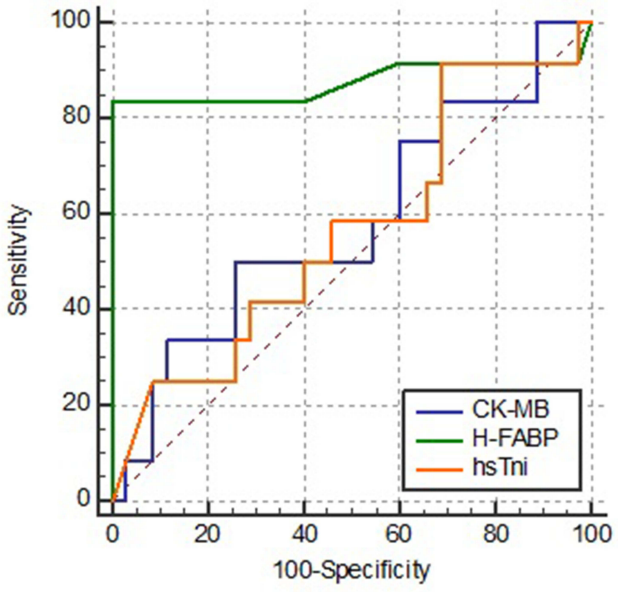


**Figure 3** Pairwise comparison of ROC curves for patients included in group 2.

**Table 8** Sensitivity and Specificity of CK-MB, hsTnI, and H-FABP in Myocardial Infarction Patients

| Cardiac Marker | Group 1 (0–4 hrs) | | Group 2 (4–24 hrs) | |
|---|---|---|---|---|
| | Sensitivity | Specificity | Sensitivity | Specificity |
| CK-MB | 80.00 | 79.88 | 75.25 | 37.50 |
| hsTnI | 75.11 | 60.44 | 37.33 | 69.98 |
| H-FABP | 89.67 | 95.65 | 86.73 | 49.84 |

of coronavirus. In such instances, tests to rapidly exclude a cardiac cause, such as myocardial infarction or a thromboembolic event causing coronary blockage, which result in myocardial injury, can help physicians to a great extent. Rosca et al reported cases of rhythm disturbance without previous cardiac pathologies in healthy young men who presented with chest pain and syncopal episodes.[31] Although the culprit was coronavirus infection leading to arrhythmias, a test like H-FABP can make it easier for the physician to work in critical periods such as the pandemic and rule out the need for the patient to be transferred toa cardiac catheterization lab immediately. Moreover, the use of bioassays with increased sensitivity for myocardial injury will decrease the need for invasive coronary investigations, thus limiting the iatrogenic risk in these patients. Multimerin-2 levels and a rise in interim blood pressure difference are shown to indicate the presence of collateral coronary circulation in MI patients and post-interventions,[32,33] but they are not commonly taken into consideration and standard biomarkers are used extensively. The rise in troponin levels and CK-MB due to noncardiac causes increase the difficulty of diagnosis, especially when the patient is presenting with nonspecific chest pain and stroke.[34] One rare association, but which is burdened by increased mortality, is that of the overlap between myocardial infarction and stroke.[35] This clinical situation is all the more therapeutically exciting because the two ischemic events have different therapeutic windows.[9,10,36] The limitations of cardiac markers of myocardial necrosis are mainly represented by the time elapsed from the occurrence of the acute coronary event to their increase, as well as their existence from non-cardiac sources.[10,36] It should also not be forgotten that there are non-cardiac conditions that can induce electrocardiographic changes that can mimic acute myocardial infarction, including stroke.[37] In these situations, the existence of markers of myocardial necrosis that have an increase in plasma concentration as close as possible to the moment of its installation is mandatory. One of the possible variants is the dosage of H-FABP whose origin is predominantly cardiac.[38] With the improvement in laboratory methods for H-FABP dosage, their sensitivity and specificity has increased.[38,39]

## Conclusion

H-FABP can be considered an important tool due to its accuracy and early rise in patients suffering from myocardial injury. Standard procedure protocols for all patients presenting to the emergency units with a suspicion of acute myocardial infarction should be evaluated for H-FABP, in addition to the pre-existing testing plans, as it can save time and the financial burden of performing serial tests, especially in the case of NSTEMI. Ruling out the possibility of myocardial infarction by performing a single test like H-FABP can reduce not only the financial burden of a series of investigations but also the anxiety levels of the patient and their relatives. Upon ruling out the possibility of a heart attack, the physicians can search for the underlying cause that mimicked the symptoms of a possible MI. This study also reiterates the importance of prompt recognition of non-STEMI myocardial infarction, knowing that "time is myocardium" and the more prompt and correct the therapeutic sanctions are, the greater is the amount of saved myocardium.

## Data Sharing Statement

All data and materials will be made available on written request to the corresponding author.

## Ethics Approval

Before the commencement of the study, ethical approval and written consent was obtained from all patients/individuals and both ethical committees based on the tenets of the Declaration of Helsinki. The study was approved by the "Comisia de Etică a Cercetării Stiintifice" (Ethics Committee for Scientific Research) of the Dr. Constantin Opriș County Emergency Baia Mare Hospital, Romania (24406, 27.08.2021) and the ethics committee of Victor Babes University of Medicine and Pharmacy, Timisoara (approval reg. nr. 74/2020, 22/2020 rev. 01.04.2024).

## Approval Statement

All authors have read and approved the final version of the manuscript.

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## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

The authors declare that there is no conflict of interest.

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