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Biomarker-Electrocardiography Synergy: Evaluating H-FABP and cTnI Serum Levels with ECG for Acute Myocardial Infarction Diagnosis

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Abstract: *Acute myocardial infarction (AMI), a life-threatening cardiovascular emergency, necessitates prompt diagnosis to optimize therapeutic outcomes. This prospective cohort study evaluated the diagnostic efficacy of serum heart-type fatty acid-binding protein (H-FABP), cardiac troponin I (cTnI), and electrocardiography (ECG) in 240 patients presenting with chest pain (150 AMI, 90 non-AMI) alongside 240 healthy controls. Coronary angiography served as the diagnostic gold standard. Serum biomarkers were quantified using immunoturbidimetric (H-FABP) and chemiluminescent (cTnI) assays, while ECG abnormalities were assessed per standardized criteria.Key findings revealed significantly elevated H-FABP and cTnI levels in AMI patients compared to non-AMI and control groups ($P < 0.05$), with concentrations correlating positively with coronary lesion severity (Gensini score: moderate-severe vs. mild group, $P < 0.05$). ECG demonstrated 82.7% sensitivity but limited specificity (46.7%) for AMI detection. ROC analysis identified optimal diagnostic thresholds at 8.78 μg/L (H-FABP, AUC=0.781) and 0.65 ng/mL (cTnI, AUC=0.912), while combined biomarker-ECG assessment achieved superior diagnostic accuracy (AUC=0.966, 95% CI: 0.934–0.986), significantly outperforming individual modalities ($P < 0.05$). These results underscore the complementary role of H-FABP (providing early ischemic signals) and cTnI (confirming myocardial injury) in AMI diagnosis. Multimodal integration with ECG enhances diagnostic precision during the critical therapeutic window, facilitating timely intervention to mitigate infarct expansion. This strategy addresses the limitations of isolated ECG interpretation and delayed cTnI elevation, offering a clinically actionable approach for rapid AMI triage.*

Keywords: *acute myocardial infarction, heart-type fatty acid-binding protein, cardiac troponin I, electrocardiography*

1. Introduction

Acute myocardial infarction (AMI) is a prevalent cardiovascular disease characterized by myocardial necrosis resulting from acute and persistent coronary ischemia and hypoxia, with a high mortality rate. In China, approximately one million patients die from AMI annually [1]. Currently, coronary intervention is routinely employed in clinical practice to promptly restore coronary circulation in AMI patients, salvage ischemic myocardium, reduce infarct size, and lower mortality and disability rates [2]. Studies have confirmed that administering appropriate treatment within 1 hour of AMI onset reduces the mortality rate to only 1%, whereas each hour of delay increases mortality by approximately 21‰ [3]. Therefore, early and accurate diagnosis during the initial phase of AMI is critical for reducing mortality and improving prognosis. At present, the primary diagnostic approach for early AMI involves electrocardiogram (ECG) combined with myocardial injury biomarkers. However, approximately 75% of AMI patients exhibit typical clinical manifestations of myocardial infarction, while 25% lack significant symptoms during the early stage and show no specific ST-T changes on ECG, necessitating greater reliance on biomarker detection for diagnosis. Cardiac troponin I (cTnI), recognized as the most specific biomarker for myocardial injury, becomes detectable only 3–6 hours after symptom onset [4]. Heart-type fatty acid-binding protein (H-FABP), a novel biomarker for myocardial injury, serves as a sensitive and highly specific indicator of myocardial ischemia and necrosis [5]. This study implemented ECG and serum H-FABP/cTnI testing in patients with chest pain suspected of AMI to evaluate their

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diagnostic value for early AMI detection, aiming to assist in the timely diagnosis of AMI within the effective therapeutic window.

2. Methods

2.1. Baseline Characteristics

A total of 240 patients with chest pain suspected of AMI, admitted between May 2022 and March 2024, were enrolled. Based on diagnostic criteria, they were divided into the AMI group (n = 150) and non-AMI group (n = 90). Additionally, 240 healthy individuals undergoing physical examinations during the same period were selected as the control group. Demographic characteristics were as follows: (1) AMI group: 82 males and 68 females; age range 43–70 years (56.28 ± 6.41); body mass index (BMI) 19–26 kg/m² (23.76 ± 1.67). (2) Non-AMI group: 50 males and 40 females; age range 45–73 years (57.96 ± 5.84); BMI 21–27 kg/m² (23.22 ± 1.32). (3) Control group: 138 males and 102 females; age range 43–71 years (56.43 ± 5.38); BMI 21–26 kg/m² (23.72 ± 1.13). No statistically significant differences were observed in baseline characteristics among the three groups ($P > 0.05$, Table 1). This study was approved by the Medical Ethics Committee of The Second Affiliated Hospital of Wannan Medical College (Ethics Approval No.: L20220323).

2.1.1. Selection of Healthy Population

The inclusion criteria comprised (1) absence of clinically significant abnormalities in routine examinations (including hematological profiles and imaging studies), (2) no documented history of surgical interventions, traumatic injuries, or acute/chronic comorbidities within the preceding 3 months, and (3) provision of written informed consent. The exclusion criteria encompassed (1) diagnosis of cardiovascular or cerebrovascular pathologies (e.g., coronary artery disease, ischemic stroke), (2) impaired renal/hepatic function (serum creatinine >1.5 mg/dL or ALT/AST >3×ULN), (3) active systemic infections or chronic inflammatory disorders (e.g., rheumatoid arthritis, inflammatory bowel disease), and (4) confirmed malignancy.

2.1.2. AMI Case Selection

The inclusion criteria comprised (1) diagnosis of AMI confirmed by coronary angiography, (2) onset time ≤3 hours, (3) signed informed consent form. The exclusion criteria encompassed (1) comorbid cardiac diseases (e.g., valvular heart disease, congenital anomalies), (2) renal or hepatic dysfunction, (3) myopathic disorders, (4) cerebrovascular diseases, (5) pulmonary diseases or active infections, (6) malignancy, (7) psychiatric disorders.

2.2. Assessment of Coronary Artery Lesion Severity

The severity of coronary artery lesions in AMI patients was quantitatively assessed using the Gensini scoring system, comprising two determinants: (1) a graded stenosis severity scoring system (0–25% stenosis: 1 point; 26–50%: 2 points; 51–75%: 4 points; 76–90%: 8 points; 91–98%: 16 points; 99–100% [total occlusion]: 32 points) multiplied by (2) segment-specific anatomical weighting coefficients reflecting hemodynamic significance. Coefficients were assigned per coronary anatomy: right coronary artery (proximal, mid, and distal segments; posterior descending branch) ×1; left main coronary artery ×5; left circumflex artery (proximal ×2.5; mid ×1.5; distal/obtuse marginal/posterior descending branches ×1; posterolateral branch ×0.5); left anterior descending artery (proximal ×2.5; mid ×1.5; distal/first diagonal branches ×1; second diagonal branch ×0.5).

2.3. Electrocardiographic Examination Methodology

Electrocardiographic (ECG) examinations were conducted using a CV200+ standard 12-lead electrocardiograph (Beijing Gushanfeng Biomedical Technology Co., Ltd.) with patients in a supine position under standardized relaxation protocols. During acquisition, the following parameters were rigorously maintained: stable baseline without artifacts, continuous tracing at 25 mm/s paper speed, and high-fidelity signal quality validated by dual independent analyses from certified ECG specialists. Diagnostic confirmation of AMI required fulfillment of three electrographic criteria: (1) pathological Q-wave morphology defined as duration ≥40 ms and/or amplitude exceeding 25% of the subsequent R-wave complex; (2) ST-segment elevation ≥1 mm in ≥2 contiguous leads (excluding V2-V3), indicative of transmural myocardial injury; (3) dynamically evolving T-wave inversions demonstrating ischemic

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progression patterns.

2.4. Serum Biomarker Detection Methodology

Venous blood samples (5 mL) were collected after an 8-hour fasting period and centrifuged at 3000 rpm for 10 minutes (radius: 10 cm) to isolate serum. Qualified serum aliquots were cryopreserved at -80°C until analysis. Serum heart-type fatty acid-binding protein (H-FABP) levels were quantified using a latex-enhanced immunoturbidimetric assay on a Hitachi 7100 automated biochemical analyzer (Hitachi High-Tech Corporation, Japan), while cardiac troponin I (cTnI) concentrations were measured via a fluorescent immunoassay with a double-antibody sandwich technique on an Abbott i2000 automated chemiluminescence analyzer (Abbott Laboratories, USA). Reagents for H-FABP and cTnI assays were supplied by Randox Laboratories Ltd. (UK) and Abbott Laboratories, respectively, with internal quality controls validating all measurements. Reference thresholds were defined as H-FABP <5.0 ng/mL and cTnI <0.5 ng/mL. According to Gensini score, AMI patients were divided into Mild group (< 25 points, n = 68) and Moderate-Severe group (≥ 25 points , n = 82).

2.5. Statistical Analysis

Data were analyzed using SPSS 25.0 (IBM Corp., USA). Normally distributed continuous variables are presented as mean ± standard deviation (mean ± SD). Intergroup comparisons among multiple groups were performed using one-way analysis of variance (ANOVA), with pairwise comparisons conducted via Fisher's Least Significant Difference (LSD) post hoc test. Diagnostic performance of serum H-FABP, cTnI, and electrocardiographic indices individually or in combination for AMI was evaluated by constructing receiver operating characteristic (ROC) curves using MedCalc 9.3.0 (MedCalc Software Ltd., Belgium). Categorical variables are expressed as percentages and compared using Chi-square tests or Fisher's exact probability tests. A two-tailed $P < 0.05$ was considered statistically significant, with 95% confidence intervals reported for all diagnostic accuracy metrics.

3. Results

3.1. Baseline Characteristics

There were no significant differences in gender, age and body mass index (BMI) between three groups ($P > 0.05$, Table 1).

Table 1. Comparison for three groups of baseline characteristics.

Group	Gender		Age	BMI (kg / m^2)	Duration of chest pain (h)	History of diabetes (n)	History of hypertension (n)	History of smoking (n)
	Male	Femal						
AMI group	82	68	56.28 ± 6.41	23.76 ± 1.67	1.21 ± 0.41	10	22	14
Non-AMI group	50	40	57.96 ± 5.84	23.22 ± 1.32	1.25 ± 0.43	6	12	8
Control group	138	102	56.43 ± 5.38	23.72 ± 1.13	/	/	/	/
χ^2 (*F* /*t*) /*P*	0.161 / 0.923		0.152 / 0.859	1.056 / 0.349	-0.404 / 0.687	0.143 / 0.705	0.041 / 0.839	0.060 / 0.806

3.2. ECG Diagnostic Results

Taking coronary angiography as the gold standard, ECG showed that 124 cases were diagnosed as AMI and 42 cases as Non-AMI. The diagnostic accuracy, sensitivity and specificity were 69.2%, 82.7% and 46.7% respectively. (Table 2).

Table 2. ECG diagnostic results.

Group	Positive (n)	Negative (n)	Total (n)
AMI group	124	48	172
Non-AMI group	26	42	68
Total	150	90	240

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3.3. Comparison of Serum H-FABP and cTnI Levels among Non-AMI Group, AMI Group and Control Group

Compared with Control group, serum HFABP and cTnI levels in Non-AMI group and AMI group were significantly higher, and AMI group was significantly higher than Non-AMI group (P <0.05, Table 3).

Table 3. Comparison of serum H-FABP and cTnI levels among three groups (x ±s).

Group	H-FABP（μg/L）	cTnI (ng/ml)
AMI group	8.46 ± 1.52 [a, b]	0.58 ± 0.19 [a, b]
Non-AMI group	7.05 ± 1.02 [a]	0.30 ± 0.08 [a]
Control group	3.74 ± 0.46	0.10 ± 0.03
F* / *P	544.400 / <0.001	394.121 / <0.001

a vs Control group, $P < 0.05$. b vs Non-AMI group, $P < 0.05$.

3.4. Comparison of Serum H-FABP and cTnI Levels in AMI Patients with Different Coronary Artery Disease Degrees

Serum H-FABP and cTnI levels in Moderate-Severe group were significantly higher than those in Mild group (P <0.05, Table 4).

Table 4. Comparison of serum H-FABP and cTnI levels in AMI patients with different degrees of coronary artery disease (x ±s).

Group	H-FABP（μg/L）	cTnI (ng/ml)
Moderate-severe group	9.15 ± 1.39 [a]	0.72 ± 0.13 [a]
Mild group	7.63 ± 1.25	0.41±0.08
F* / *P	4.934 / <0.001	12.123 / <0.0011

a vs Mild group, $P < 0.05$.

3.5. Comparison of Diagnostic Value of ECG, H-FABP and cTnI in AMI

The accuracy, specificity and sensitivity of the three combined tests for diagnosing AMI were significantly higher than those of the single test (Z=4.575, 4.499, 2.954, P <0.05, Table 5). ROC curve analysis showed that the optimal cut-off values for H-FABP and cTnI alone for diagnosing AMI were 8.78μg/L and 0.65 ng/ml, respectively, and the AUC values for H-FABP, cTnI and ECG alone and in combination for diagnosing AMI were 0.781, 0.912, 0.680 and 0.966, respectively (Figure 1).

Table 5. Comparison of Diagnostic Value of ECG, H-FABP and cTnI in AMI.

Index	area under the curve (AUC)	95%CI	Sensitivity (%)	Specificity (%)	Youden index	cutoff value
ECG	0.680	0.589~0.762	82.67(62/75)	53.33(24/45)	0.360	
H-FABP	0.781	0.696~0.851	70.67(53/75)	80.00(36/45)	0.507	>7.850μg/L
cTnI	0.912	0.847~0.956	89.33(67/75)	75.56(34/45)	0.649	>0.35ng/ml
Combined	0.966	0.916~0.991	98.67(74/75)	84.44(38/45)	0.831	
$Z(\chi^2)$ / P_1	6.783/<0.001		11.345/<0.001	10.161/<0.001		
$Z(\chi^2)$ / P_2	4.499/<0.001		22.646/<0.001	0.304/0.581		
$Z(\chi^2)$ / P_3	2.954/0.003		4.255/0.039	1.111/0.292		

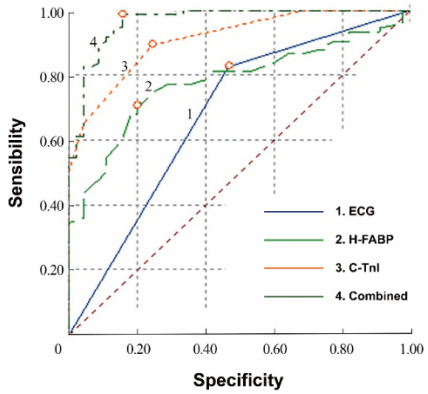

Figure 1. ROC Curves of Diagnostic Value of Serum H-FABP, cTnI and ECG.

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4. Disscussion

Acute onset of AMI, severe condition, myocardial cell ischemia, hypoxia necrosis or apoptosis in patients, resulting in rapid decline in cardiac function, leading to heart failure, is an independent risk factor for patient death. At present, there is no consensus on the pathogenesis of AMI in medical circles, but it is generally believed that it is related to plaque hemodynamic inflammatory reaction and lipid metabolism, and atherosclerotic plaque rupture leads to platelet aggregation and thrombosis, leading to AMI [6]. Clinical treatment principle is to maintain cardiac function and reduce the infarction area, and early recovery of coronary blood circulation is the key to reduce the infarction area [7]. Therefore, early diagnosis and identification of high-risk AMI patients is of great significance to improve the prognosis of patients.

ECG is a commonly used imaging technique for diagnosing AMI, which can observe the changes of electrical activity produced by cardiac cycle from body surface, capture abnormal information at any time, and has the advantages of simple and convenient operation and repeatable examination [8]. This study found that 124 cases were diagnosed in 240 patients with chest pain suspected of AMI, and the diagnostic accuracy, sensitivity and specificity were 70.0%, 82.7% and 48.9% respectively. It is suggested that ECG has certain value in the diagnosis of AMI and can predict the progress of the disease. However, some AMI patients have no significant ECG changes due to small infarction area and various underlying diseases, and no abnormal Q wave is found. Only ST segment depression, T wave low or pseudo-normalization lead to low diagnostic accuracy, high missed diagnosis rate and misdiagnosis rate.

cTnI is a myocardial specific regulatory protein, belonging to one of the three subtypes of cardiac troponin, which is basically not expressed under normal conditions. Cardiomyocytes secrete a large amount of cTnI during myocardial injury, and invade the interstitial by damaged cell membrane. cTnI degrades slowly and continuously along with myocardial myofilament, and then diffuses into the blood, with high sensitivity and specificity [9]. Several authoritative organizations recommend cTnI as the first biochemical marker for the diagnosis of AMI. However, cTnI does not meet the early diagnosis required by reperfusion therapy, and is significantly affected by quantitative detection, improper use of anticoagulant and other factors, and the early missed diagnosis rate of AMI is relatively high [9]. H-FABP is one of the most abundant proteins in myocardial cells, which is a soluble protein with low relative molecular weight existing in myocardial cytoplasm. The concentration of H-FABP in serum begins to increase 20min after myocardial injury, which appears before cTnI, and has time advantage, which is beneficial to early diagnosis and treatment of AMI [10]. In this study, compared with the control group, the serum H-FABP and cTnI levels of non-AMI group and AMI group were significantly increased, and AMI group was significantly higher than non-AMI group. It is suggested that serum H-FABP and cTnI levels in patients with AMI are abnormally elevated, cTnI concentration in serum is low under normal conditions, and myocardial cells secrete a large amount of cTnI and disperse it in the blood during myocardial injury [9]. The content of H-FABP in blood under normal conditions is also very small. Myocardial cells in early AMI are highly sensitive to ischemia and hypoxia, the ability to mobilize fatty acids is improved, the expression of H-FABP in myocardial cells is rapidly increased, the permeability of cell membrane is enhanced, and H-FABP is rapidly released into blood [10]. It is speculated that serum H-FABP and cTnI levels play an important role in the occurrence and development of AMI through different pathological mechanisms. This study showed that serum H-FABP and cTnI levels in moderate and severe group were significantly higher than those in mild group, suggesting that serum H-FABP and cTnI levels were related to the severity of coronary artery disease in AMI patients, and the higher the expression, the more severe the severity of coronary artery disease in AMI patients, suggesting that serum H-FABP and cTnI levels were helpful to evaluate the severity of disease in patients [11].

In the review of H-FABP published by Ye et al, only cTnI and H-FABP have the advantages of sensitivity, specificity and diagnostic value at the same time [8]. Compared with cTnI,H-FABP has a good application prospect in rapid diagnosis or exclusion of AMI. ROC curve analysis shows that the best cut-off values of H-FABP and cTnI for the diagnosis of AMI are 7.850μg/L and 0.35ng/ml respectively, AUC is 0.781 and 0.912 respectively, suggesting that H-FABP and cTnI have diagnostic value for AMI. In addition, the accuracy, specificity and sensitivity of combined detection of H-FABP and cTnI were significantly higher than that of single detection, suggesting that ECG combined with H-FABP and cTnI detection had higher value in early diagnosis of AMI and was beneficial to early treatment of AMI patients. Otaki et al suggested that H-FABP and cTnT were an ideal combination for early application of AMI and could cover the entire diagnostic time window of myocardial infarction, which supported the results of this study to a certain extent [5].

To sum up, serum H-FABP and cTnI are related to the degree of coronary artery disease in AMI

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patients, and serum H-FABP and cTnI combined with ECG examination is beneficial to improve the accuracy of early diagnosis of AMI, and is conducive to early treatment of patients.

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