

The detection value of CK-MB, Myo and cTnI in Patients with AMI and HF.

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Abstract

Objective: To investigate the diagnostic value of creatine kinase-myocardial band (CK-MB), myoglobin (Myo) and cardiac troponin T (cTnI) in acute myocardial infarction (AMI) and heart failure (HF).

Methods: Fifty patients with AMI, 50 HF patients and 50 normal subjects in hospital from January 2015 to December 2016 were enrolled. The levels of CK-MB, Myo and cTnI were measured for the analysis of heart failure NYHA classification. The levels of CK-MB, Myo and cTnI of AMI patients were measured at 2 h, 4 h and 6 h to analyze their specificity and sensitivity.

Results: The levels of CK-MB, Myo and cTnI in AMI group and HF group were significantly higher than those in normal group ($P=0.0027, 0.0004, 0.0014<0.05$). In AMI group, the sensitivity of Myo reached up to 65.54% and specificity 56.24% 2 h after onset. The sensitivity of CK-MB, Myo and cTnI 4 h after onset was 72.82%, 87.32% and 54.53% respectively, and the specificity was 75.62%, 82.48% and 58.43% respectively. The sensitivity of CK-MB, Myo and cTnI 6h after onset was 100% and specificity was 88.46%, 95.32% and 90.24%, respectively. There was a significant difference in CK-MB, Myo and cTnI between I, II, III and IV levels of HF patients.

Conclusion: CK-MB, Myo and cTnI have good application value in the diagnosis of heart failure and the early diagnosis of acute myocardial infarction.

Keywords: CK-MB, Myo, cTnI.

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Introduction

Acute myocardial infarction (AMI) refers partial acute myocardial necrosis caused by persistent and severe myocardial ischemia with clinical manifestations of persistent pain after the sternum, invalidation of nitric acid ester preparation, increased serum myocardial enzymes as well as corresponding ECG changes, finally leading to a high mortality [1,2]. Heart failure (HF) refers to the decline in cardiac function, which fails to meet the needs of peripheral blood circulation, with clinical manifestations of edema and hypoxia [3]. AMI and HF have become the most common kinds of vascular diseases endangering human health. However, the mortality and morbidity of patients will be greatly reduced if accurate diagnosis is made in time. At present, the diagnosis of myocardial enzymes for AMI has become a common means with less application of combined detection in the early diagnosis. Myocardial enzymes are also less applied for HF diagnosis and cardiac function classification prediction [4]. This study aims to explore the value of myocardial enzyme combined detection methods for the early diagnosis of AMI and HF.

Materials and Methods

Clinical data

Ethical approval was given by the medical ethics committee of Sixth People's Hospital of Qingdao with the following reference number: 2014012, CFifty AMI patients, 50 HF patients and 50 normal subjects were enrolled in hospital from January 2014 to December 2015. There were 20 female patients and 30 male patients in AMI group with the age range of 45-78 years (mean 62 ± 2.5 years). There were 22 female and 28 males in HF group with the age range of 42 to 77 years (mean 64.5 ± 3.5 years). There were 23 females and 27 males in normal group with the age range of 40 to 78 years (mean 62 ± 2.5 years).

Inclusion criteria

1) All AMI patients were diagnosed with the diagnostic criteria presented by the Chinese Medical Association Cardiovascular Society in 2001[5]; 2) All HF patients were diagnosed with the American Health Care Policy and Research Council (AHCPR) clinical guidelines [6]; 3) All selected cases had no live, kidney and central nervous system diseases; 4) This study was approved by the Council Ethics Committee.

Methods

1) **Sample collection:** Intravenous blood of all patients was taken before hospitalization. venous blood of AMI patients was taken at 2 h, 4 h and 6 h after onset; 2) **Detection method:** After collection of intravenous, the separation sample of serum was subjected to electrochemiluminescence immunoassay for the quantitative determination of Myo and cTnI. CK-MB was determined by immunosuppression using the isolated serum samples. All the procedures were performed in strict accordance with the standard experimental procedure.

HF patients received cardiac functional grading based on I~IV grading method proposed by NYHA: 1) grade I: Patients had heart disease but not limited daily activity. The general physical activity would not cause excessive fatigue, palpitations, asthma or angina; 2) grade II: Physical activity of patients with heart disease is mildly limited. No subjective symptoms occurred during rest. General physical activity would cause fatigue, palpitations, asthma or angina; 3) grade III: Patients with heart disease were limited in physical activity. No symptoms occurred during rest. Light physical activity would cause fatigue, palpitations, asthma or angina; 4) grade IV: Heart disease patients could not engage in any physical activity. Symptoms occurred during rest and physical activity aggravated condition.

Evaluation methods

The levels of CK-MB, Myo and cTnI in patients were measured. The levels of CK-MB, Myo and cTnI were detected in patients with AMI at 2 h, 4 h and 6 h to analyze its specificity and sensitivity.

Specificity and sensitivity were calculated as follows: Sensitivity=true positive/(true positive+false negative), specificity=true negative (false positive+true negative).

Table 2. Sensitivity and Specificity of CK-MB, Myo, cTnI for Early Diagnosis of AMI (%).

Items	2h		4h		6h	
	Sensitivity	Specificity	Sensitivity	Specificity	Sensitivity	Specificity
Myo	65.54	56.24	87.32	82.48	100	95.32
CK-MB	0	0	72.82	75.62	100	88.46
cTnI	0	0	54.53	58.43	100	90.24
Myo+CK-MB	0	0	56.76	74.90	100	93.75
Myo+cTnI	0	0	42.46	84.66	100	92.05
CK-MB+cTnI	0	0	47.21	79.54	100	96.48
Myo+CK-MB+cTnI	0	0	34.38	92.14	100	100

Table 2 shows that in the early onset of AMI, combined detection of CK-MB, Myo and cTnI has a sensitivity of 100% to AMI.

Statistical methods

All the data of this study were analyzed by SPSS19.0 software. The data were measured by t value, and the difference between groups was tested. The test standard $\alpha=0.05$. $P<0.05$ was defined as a significant difference.

Results

CK-MB, Myo, cTnI levels

The levels of CK-MB, Myo, and cTnI in AMI patients, HF patients and normal subjects are shown in Table 1.

Table 1. CK-MB, Myo, cTnI levels.

Groups	Cases (n)	CK-MB (IU/L)	Myo ($\mu\text{g/L}$)	cTnI (ng/L)
AMI	50	46.53 \pm 10.67*	298.71 \pm 103.62*	8.25 \pm 1.74*
HF	50	47.26 \pm 12.93*	268.94 \pm 99.42*	9.46 \pm 1.37*
Normal	50	21.66 \pm 7.22	24.76 \pm 13.47	5.25 \pm 1.02
F value		5.249	12.932	4.046
P value		0.031	0.004	0.014

note: *Compared to normal group, $P<0.05$

The serum levels of CK-MB, Myo and cTnI in AMI patients and HF patients were significantly higher than those in normal subjects ($P<0.05$).

Sensitivity and specificity of CK-MB, Myo, cTnI for early diagnosis of AMI

The sensitivity and specificity of CK-MB, Myo and cTnI for early diagnosis of AMI are shown in Table 2.

Levels and CK-MB, Myo and cTnI between different NYHA of HF patients

According to NYHA classification, HF patients were divided into 16 patients with grade I, 14 patients with grade II, 13 patients with grade III and 7 patients with grade IV. The

differences of CK-MB, Myo and cTnI levels were found in Table 3.

Table 3. Levels and CK-MB, Myo and cTnI between different NYHA of HF patients.

NYHA	Cases (n)	CK-MB (IU/L)	Myo (µg/L)	cTnI (ng/L)
I	16	31.26 ± 16.4	45 ± 8.52	3.13 ± 0.75
II	14	36.72 ± 14.9	61 ± 9.09	4.95 ± 0.87
III	13	53.12 ± 21.52	110 ± 11.23	6.01 ± 1.21
IV	7	82.94 ± 20.83	170 ± 7.98	7.97 ± 1.02
F test		6.03	5.96	6.93
P value		0.027	0.038	0.012

As shown in the above table, the levels of CK-MB, Myo and cTnI in HF patients were significantly different (P<0.05).

Discussion

Acute cardiac infarction (AMI) is absolute or relative stenosis of the coronary artery caused by local myocardial ischemia. In 2000, the European Heart Association (ESC) and the American Heart Association (ACC) presented new diagnostic criteria for AMI. The biochemical markers, troponin, showed a typical increase, or a rapid increase in troponin accompanied by the following manifestations: 1) Chest pain, chest tightness and other symptoms of myocardial ischemia; 2) Pathological Q wave in ECG; 3) ECG changes in ST segment elevation; 4) Effective percutaneous coronary intervention therapy [7]. Heart failure refers to the decline in cardiac function that cannot meet the needs of peripheral blood circulation. The clinical symptoms were mainly edema and hypoxia. Because of the long-term and chronic myocardial injury, myocardial necrosis markers also increased slowly. From the new diagnostic criteria, it can be seen that myocardial necrosis markers played a key role in AMI and HF diagnosis. At present, the wide application of myocardial necrosis markers included Myo, CK-MB and cTnI [8].

Myo, a new type of myocardial injury marker, is a small-molecular substance that is mainly distributed in the heart and skeletal muscle [9,10]. Myo could be released quickly into the blood after myocardial injury, and the level of Myo increased in HF patients due to long-term damage of the myocardium. In this study, the serum levels of Myo were significantly higher in AMI and HF patients compared with normal subjects. Increase in serum Myo level could be observed only 2 h after the onset of AMI, thus Myo could be used as a diagnosis basis of early AMI.

CK-MB is a classic marker of myocardial injury, but its sensitivity to early AMI diagnosis is not high according to the literature [11,12]. In the traditional diagnostic criteria, CK-MB still held about 5% misdiagnosis rate, showing obvious disadvantages over early diagnosis of AMI and HF [13,14]. In this study, immunosuppressive method was used for detection in order to improve specificity and sensitivity on early

diagnosis of AMI and HF. Besides, the serum levels of CK-MB in patients with AMI and HF were higher than those in normal subjects. CK-MB was not significantly increased when detected 2 h after AMI onset. For 4 h and 6 h after AMI onset, the sensitivity of CK-MB on AMI diagnosis increased to 72.82% and 100%, respectively. Thus, dynamic detection of CK-MB after onset did contribute to the diagnosis of AMI.

CTnI consisted of different amino acid sequence, coding genes and amino acid residues. Because of its widespread distribution in the myocardium, the specific sequence determined its status in the diagnosis of cardiac damage [15,16]. CTnI was widely applied in prediction and judgment of clinical processes, such as myocardial infarction thrombolytic therapy, heart failure treatment and acute myocardial infarction. There were obvious shortcomings in single enzyme diagnosis of myocardial injury. Therefore, CK-MB, Myo and cTnI were combined in this study for the diagnosis of AMI. The results showed that after 6 hours of onset, the specificity and sensitivity of the combined diagnosis were 100%, displaying early diagnostic value of combination therapy. On the other hand, CK-MB, Myo and cTnI levels were often positively correlated with the degree of myocardial injury. Therefore, the levels of CK-MB, Myo and cTnI in NYHA patients at different grades increased as heart failure level elevated, and the difference was statistically significant (P<0.05).

In summary, CK-MB, Myo and cTnI had favourable application value on early diagnosis of cardiac function classification for heart failure patients as well as on acute myocardial infarction.

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