

CARDIOMARKERS IN THE FIRST HOURS OF ACUTE MYOCARDIAL INFARCTION

Dadabayeva Nailya Akramovna

PhD, Associate Professor

Mahmudova Munira Safiyevna

PhD, Senior Lecturer, Tashkent Medical Academy

maxmudovamunira44@gmail.com

Kim Andrey Rudolfovich

Doctoral Student, Republican Specialized Scientific and
Practical Medical Center of Cardiology. Tashkent, Uzbekistan

kimandrey266@gmail.com.

ABSTRACT

In recent years, the definition of the components of the troponin complex of cardiomyocytes — troponin T and I - has been widely used in clinical practice. Their high specificity attracted the attention of cardiologists to them and in many ways changed the principles of modern diagnosis of MI. The myocardial protein troponin I (ThI) is found only in cardiomyocytes, it is not present in striated and smooth muscles.

The reason for Tnl's success among cardiologists was its exceptional cardio specificity. At the moment, it has been shown that the concentration of this protein in the blood exceeds the values characteristic of the norm only in the case of a necrotizing site of heart muscle cells (Bertinchant et al., 1996; Altinier S. et al., 1997). Unlike creatine kinase and all other previously used MI marker proteins, the concentration of troponin I in the blood does not increase in the case of acute or chronic skeletal muscle lesions.

However, before troponins, myoglobin appears in the blood, which is highly sensitive to myocardial damage. Myoglobin appears in the peripheral blood 2-3 hours after the onset of pain and reaches a pathological level 3-6 hours earlier than CK-MB.

Objective: Comparative evaluation of troponin I transaminase and myoglobin in AMI.

Research materials and methods: 95 patients diagnosed with acute myocardial infarction were examined. AST was examined in all patients, troponin was determined in 60 patients, and myoglobin level was determined in 35 patients. Troponin and myoglobin were examined in the first hours after admission to the hospital. The patients were divided into two groups: patients with AMI with a Q wave and without a Q wave.

Table 1. Characteristics of patients with AMI with a Q wave by gender and age

Indicator	Age from 40 to 59	60 and older	Total amount
Men	18	14	32
Women	13	15	28
Overall	31%	29%	60%

At the age of 40-59 years, there were more men than women, and after 60 years their number did not differ much.

Table 2. Characteristics of patients with AMI without Q wave by gender and age

Indicator	Age from 40 to 59	60 and older	Total amount
Men	10	9	19
Women	7	9	16
Overall	17	18	35

The ratio of patients by sex and age corresponded to patients with AMI with the Q wave. AMI over the age of 40, men and women get sick equally often. There were almost 2 times more patients with AMI with a Q wave than without a Q wave.

Table 3. The content of AST , troponin I and myoglobin in patients with AMI with a Q wave the effect of various clinical indicators

Enzymes	Pain		BP		HR	
	With pain	Pain free	SBP> 140 mmHg	DBP< 80 mmHg	>90 per minute	<60 per minute
AST	23%	7,3%	12%	21%	12%	14%
ThI	82%	80%	76%	87%	75%	60%
Myoglobin	86%	75%	82%	88%	78%	80%

In the presence of pain, there was an increase in all enzymes, but troponin I and myoglobin increased significantly than AST. With pain-free AMI, there was also an increase in enzymes, but very little. In the latter case, the diagnosis was made based on ECG data and an increase in specific cardiac markers. With an increase in blood pressure, the ratio was the same. In hypotension, all enzymes were elevated. In tachycardia and bradycardia, AST was also inferior in sensitivity to troponin I and myoglobin. The heart rate was accompanied by the same dynamics as with other symptoms. The content of transaminase, troponin I and myoglobin does not depend on clinical symptoms (pain, blood pressure, heart rate) and the depth of AMI.

Table 4. The content of AST, Troponin I and myoglobin in patients with AMI without a Q wave

Enzymes	Pain		BP		HR	
	With pain	Pain free	SBP> 140 mmHg	DBP< 80 mmHg	>90 per minute	<60 per minute
AST	5.2%	2.7%	6.5%	9.3%	8.1%	9.3%
ThI	80%	81%	84%	82%	82%	74%
Myoglobin	89%	79%	86%	88%	76%	80%

In AMI without a Q wave, the AST content increased very slightly everywhere, the level of cardiac markers was also high as in patients with AMI with a Q wave. The sensitivity of AST decreased with AMI without a Q wave, and the sensitivity of cardiac markers remained high.

Table 5. The effect of AMI localization on the sensitivity of ALT, ThI and myoglobin in patients with AMI

Localization	AMI with Q wave			AMI without Q wave		
	AST	ThI	myoglobin	AST	ThI	myoglobin
Anterior wall		50%	66,7%		50%	28%
Anterior septal wall	33%	66.7%	84%	8%	66,7%	75%
Anteroapical area	16,7%	82%	88%	25%	80%	88%
The spread of AMI	20%	75%	75%	16.7%	66.7%	80%
Lateral wall	-	80%	82%	-	50%	68.7%
Posterior wall	23%	92,3%	80%	25%	75%	66.7%
total	15.45%	80.1%	87%	12.45%	64.73%	84.3%

AST, regardless of the localization of AMI, showed low sensitivity, slightly increasing with AMI with a Q wave. Specific cardiac markers showed high sensitivity in both AMI with and without a Q wave. It was not possible to establish the natural sensitivity of enzymes from the localization of AMI of the left ventricle.

CONCLUSIONS

In order to diagnose AMI, clinical indicators must be combined with the content of biomarkers and ECG data. Of the enzymes, troponin and myoglobin have the greatest cardio specificity. ThI and myoglobin in AMI showed higher sensitivity compared to transaminase. The sensitivity of both ThI and myoglobin in AMI with a Q wave is the same. In AMI without the Q wave, myoglobin was more sensitive.