

BIOLOGICAL MARKERS – PREDICTORS OF NO-REFLOW PHENOMENON AFTER PRIMARY PCI IN PATIENTS WITH ST - SEGMENT ELEVATION MYOCARDIAL INFARCTION

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Rezumat

Parametrii lipidici, împreună cu markerii biologici ai necrozei miocardice au un rol esențial în evidențierea pacientului cu risc crescut de a dezvolta fenomenul no-reflow în urma unui STEMI. Acest fenomen este dat, în urma efectuării PCI primar, de nereluarea fluxului sangvin la nivelul microcirculației coronariene, deși artera implicată în infarct a fost dezobstruată.

Cuvinte cheie: fenomenul no-reflow, markeri biologici.

Abstract

Lipid parameters along with the biological markers of myocardial necrosis play an essential role in highlighting the patient at an increased risk of developing the no-reflow phenomenon from an STEMI. This phenomenon is due to primary PCI failure of blood flow in the coronary microcirculation, although the artery involved in the infarction is disintegrated.

Keywords: no-reflow phenomenon, biological markers



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Introduction

Following an acute myocardial infarction due to the occlusion of an epicardial coronary artery accompanied by the absence of blood flow in the territory of the artery involved, the myocytes undergo a physiological process of ischemia, lesion and necrosis, with the release in the interstitial space of enzymes that can be revealed paraclinically using laboratory analyzes⁽¹⁾.

The atheromatous plaque actively contributes to thickening of the arterial endothelium by depositing lipids, especially cholesterol, increasing progressively in size and resulting in ischemic coronary artery disease, and with the breakdown of the atheromatous plaque arterial artery occlusion with the occurrence of acute myocardial infarction. Since cholesterol contributes directly to atherosclerosis, its dosing is essential in the prevention of ischemic heart disease, and LDL-cholesterol dosing is important to be known for over 50% of total plasma cholesterol is transported in this form⁽²⁾.

Plasma cardiac troponins are the standard in the evaluation of myocardial necrosis due to their high specificity and high sensitivity, and their detection after acute myocardial infarction in the bloodstream increases rapidly, three hours after symptom onset, reaching the maximum threshold after the 12-24 hours, and normalization occurs after 10-14 days^(1,3).

Because its values are specific for a myocardial infarction, it must exceed the 99th percentile of the characteristic of each individual test⁽⁴⁾.

If plasma troponin dosing is not feasible, creatine kinase-CK and its MB isoenzyme can be detected, which can be detected after 16 hours after onset of STEMI. Compared to CK that has specificity for skeletal muscles, CK-MB has an increased specificity for myocardial muscle^(1,5). Myoglobin plasma concentration increases rapidly after an acute myocardial infarction but its lack of specificity makes it more rarely used in clinical practice⁽¹⁾.

The no-reflow phenomenon occurs after an acute myocardial infarction, when after percutaneous reperfusion of the coronary artery-inducing infarct, the blood flow is not resumed in the coronary microcirculation⁽¹⁾. For the occurrence of the phenomenon it is considered to be very low intramedocular vessels obstructed, having incriminated mechanisms of microvascular dysfunction vasospasm, thrombus fragments migrating after myocardial revascularization, oxygen free radicals from endothelial injury, capillary obstruction caused by red blood cells and neutrophils and compressive edema⁽⁶⁾.

Objectives

The study wishes to demonstrate the correlations between the biological markers involved in the atherogenesis and

Dyslipidemia	Nr.	%
Col ↑ + LDLc ↑ + HDLc ↓ + Tg ↑	56	58,33
Col ↑ + LDLc ↑ + HDLc ↓	3	3,13
Col ↑ + LDLc ↑ + Tg ↑	9	9,38
Col ↑ + Tg ↑	4	4,17
Col ↑	5	5,21
Tg ↑	5	5,21
Lipid parameters within normal range	14	14,58

Table 1. Distribution of cases depending on the type of dyslipidemia

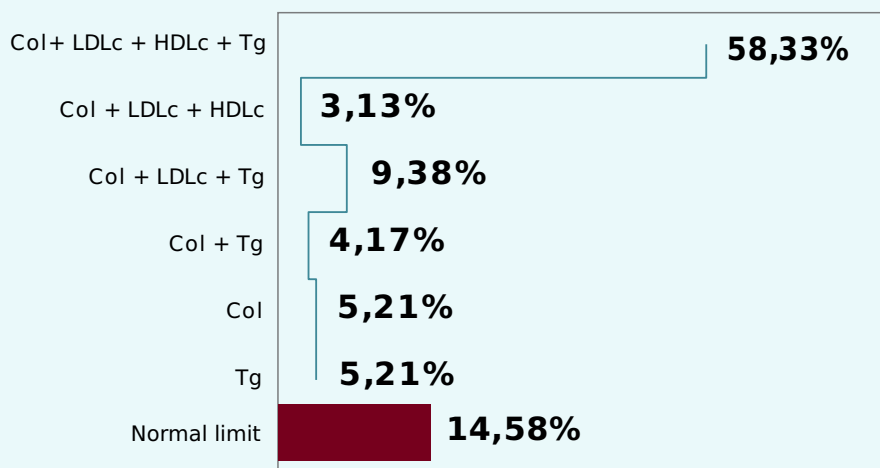


Figure 1. Distribution of no-reflow cases depending on the type of dyslipidemia

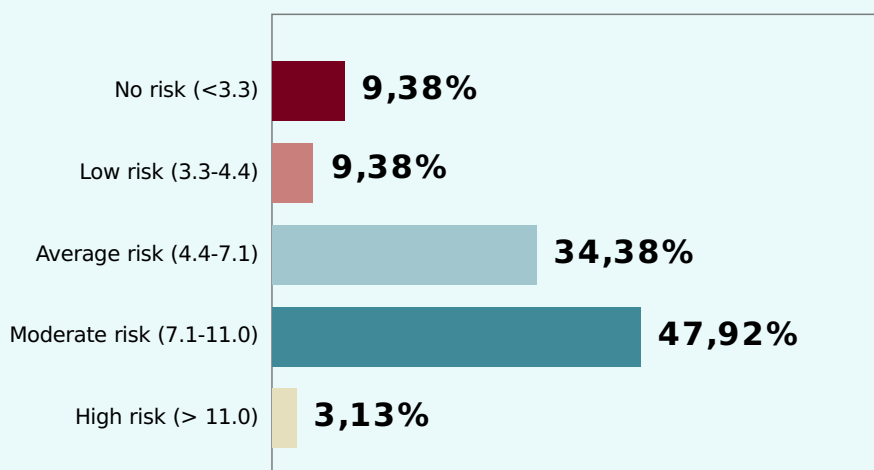


Figure 2. The cardiovascular risk assessed by the col / HD report



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myocardial necrosis enzymes and the no-reflow phenomenon, thus showing its biological predictors.

Material and method

A study was conducted on 656 patients admitted to an ST segment elevation acute myocardial infarction in the period 01.01.2016 - 31.03.2018 in the Cardiology Clinic of the Oradea County Emergency Clinical Hospital which was chosen as a method of myocardial revascularization primary PCI, and the procedure in 96 patients was no-reflow phenomenon. In this study the prevalence of these patients was predominant.

The patients were divided into several categories depending on the biological vectors involved in the atherogenesis, the welded myocardial necrosis enzymes and other biological parameters with potential for revealing a necrosis. Laboratory analyzes were performed by plasma spectrometry method for high sensitivity troponin I using the Architect ci4100 apparatus as well as for the determination of total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides, creatine kinase and its isoenzyme MB using the ARCHC4000 analyzer. With respect to the dosing of troponin I and myoglobin, both were performed on the Pathfast Compact Immuno-Analyzer.

Normal values of biological parameters were in line with current guidelines. LDL-cholesterol reference values <100 mg/dl, HDL-cholesterol >60 mg/dl, total cholesterol <200 mg/dl, triglyceride <150 mg/dL⁽⁷⁾, troponin I myocardial necrosis enzymes and troponin I - high sensitivity enzymes were considered to have optimum values if they are below the upper 99th percentile⁽⁸⁾, respectively 29 ng/ml and 15.6 pg/ml according to the data in force in the median unit where the laboratory tests were performed and for the CK the normal values were 168 U/l and CK-MB 24 U/L⁽⁸⁾.

STEMI diagnosis was based on clinical data along with paraclinical data: the increase of myocardial necrosis markers over the 99th percentile at the upper reference limit, changes of ST-T segment or BRS new appearance on the EKG or the new appearance of Q waves, and imaging - echocardiographic detection of a territory with segmental kinetics disorders and acute coronary occlusion in the angiography⁽⁸⁾. The no-reflow phenomenon was detected by primary PCI if TIMI flow rate <3 and MGB <3, or if the values were optimal, but ST segment persistence on EKG was observed over 50-70% of baseline⁽⁹⁾. Coronarography was performed in the Angiography and Cardiac Catheterization Laboratory of the Oradea County Emergency Clinical Hospital, a procedure performed in accordance with current norms.

Cardiac markers	Values outside the normal range		Mediate
	No.	%	
CK (UI)	50	90,91	414,51±95,49
CK-MB (UI)	49	89,09	46,65±16,83
Myoglobin	39	100,00	493,58±113,14
Troponin I	54	100,00	3,232±1,609 ng/l
Troponin I - HS	50	100,00	28.635±14.528 pg/ml

Table 2. Distribution of cases according to cardiac markers

Other medical analyzes	Values outside the normal range		Mediate
	No.	%	
RFG (ml/min/1,73 m)	23	23,96	79,29±18,21
Platelets ($\times 10^3 \mu\text{L}$)	5	5,21	256,52±48,50
Neutrophil (mg/dl)	71	73,96	11,52±4,01

Table 3. Distribution of cases based on other medical analyzes



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Results

Dyslipidemia was present in one third of patients diagnosed with STEMI and, after primary PCI, developed the no-reflow phenomenon, and over half of the cases recorded values outside of normal interstitial at all lipid parameters. We note that only less than a quarter of patients had normal values of all lipid parameters (14.58%) (Table 1, Figure 1).

The cholesterol / HDL-cholesterol ratio had values ranging from 2.16-13.71. Moderate and increased cardiovascular risk (ratio col / HDLc > 7.1) was reported in half of the patients (51.04%) (Figure 2).

CK was performed in 55 patients, with values ranging from 228-777 IU, of which only 5 cases were normal, all of them being men (<308 IU).

CK-MB was also performed in 55 patients, with values ranging from 13-132 IU, of which normal values were recorded in 6 cases (<24 IU). Myoglobin was performed in 39 patients, with values ranging from 212-854 mg / l, with no case with normal values (<90 mg/l). Troponin I was performed in over half of the patients (56.25%), with values ranging from 0.443-36.500 ng/mL, with an average of 3.232 ± 1.609 ng/l.

Troponin I - High Sensitive was performed in half the patients (52.08%), with values ranging from 11,216.8-99,443.90, the

average being of $28,635 \pm 14,528$ (Table 2).

RFG, platelets and neutrophils were performed in all patients. RFG had values ranging from 19-114 ml/min/1.73 m², low values (<60 ml/min/1.73 m²) in 23 patients. Platelets had values ranging from 120.2-520.7 x 10³ µL. Thrombocytopenia occurred in 3 patients (3.13%) and thrombocytosis in 2 patients (2.08%). Neutrophil counts were between 3.40-38.15 mg/dl, normal neutrophil counts (<8 mg/dl) were recorded in 25 patients and values increased >15 mg/dl in 20 patients (Table 3).

Discussions

Dyslipidemia plays a major role in the formation of the atheromatous plaque, as observed in our study, one-third of patients with no-reflow phenomenon presenting elevated lipid factors, which again highlights the major risk of these patients developing the phenomenon, literature data showing this⁽¹⁰⁾.

Regarding myocardial necrosis enzymes, the elevated values predicted in all patients performed have shown their beneficial role in STEMI determination, and the high sensitivity Troponium I at very high values above 20,000 pg/ml can be a predictor of the no-reflow phenomenon, according to our study. In terms of platelet count, our study did not show clinically significant changes,

but RFG at low levels and the number of increased neutrophils may be a risk factor for the no-reflow phenomenon⁽¹¹⁾.

Conclusions

The no-reflow phenomenon is of particular importance among patients with this condition because of the short and long-term negative complications and the unfavorable prognosis of the patient.

Early detection and prompt establishment of treatment in dyslipidemic patients is an essential element in the prevention of the phenomenon. Increased lipid parameters accompanied by very high levels of myocardial necrosis enzymes in a patient diagnosed with acute myocardial infarction point to a possible development following the primary PCI of the no-reflow phenomenon.

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