SMALL MYOCARDIAL INJURY IN PATIENTS UNDERGOING PERCUTANEOUS CORONARY INTERVENTION

Gayrabekova F.R.
Polyclinic № 7, Grozny, e-mail: miss-orstho@mail.ru

The article presents the relevance of coronary heart disease and cardiac surgery technique of its treating – percutaneous coronary intervention. It has been described complication arising after the planned percutaneous coronary intervention: a small myocardial injury, manifested by increased serum levels of cardiac markers such as myoglobin, troponin T, creatine phosphokinase-MB.

Keywords: myoglobin, troponin T, creatine phosphokinase-MB, stenting, damage to the myocardium, coronary artery

In modern cardiology, coronary heart disease (CHD) is one of the actual problems because it is the most common disease, despite the high level of development of world medicine. In 2012, CHD mortality was 13,2% (7,4 million). In 2011 the total mortality of coronary heart disease in the Russian Federation – 52,8% [12]. CHD mortality in Russia is higher than in the USA, three times or higher than that in Japan – 9 times in [5].

Thus, the treatment of coronary artery disease in patients – the actual problem now. Annually it has been taken more than 3,0 million of revascularization procedures in the world. In recent years, the ratio of percutaneous coronary intervention (PCI) (angioplasty and stenting of the coronary arteries) and coronary artery bypass grafting is 2:1 in Europe, and 6:1 – USA, Japan [5].

The concept of using endovascular prostheses for the affected vessel and the preservation of its lumen was first proposed by C. Doner in 1964 [1]. The gained experience in 1987 of H. Rousseau on implantation of self-expanding stents in the coronary arteries of animals served as the basis for the application of stents for the treatment of coronary artery disease patients [13]. First self-expanding stent implantation in human coronary arteries was performed in 1986 by J. Puel [6].

In 2012, the Russian Federation there have been carried out 75,000 coronary stenting [5]. Observational study involving a large number of patients, confirm that PCI procedure is highly effective with a low rate of complications compared with other methods of myocardial revascularization. Nevertheless, the problem of PCI still has many open questions.

Today there have been thoroughly studied and described, according to the classification of ACC/AHA, such serious complications after PCI as death, myocardial infarction, stroke. Minor complications include transient ischemic attack, complications at the puncture site, renal failure, allergic reaction to the contrast agent; and specific complication – thrombosis, coronary artery disease, coronary artery perforation, arrhythmias and tamponade [13]. However, the term "minor myocardial damage" (MMD) has been recently appeared in the literature, found in 8–15% of cases after a routine PCI and manifested only by increased levels of cardiac markers without clinical and electrocardiographic signs of myocardial damage [4].

Cardiac markers are highly sensitive and specific in the diagnosis of MMD, but relatively rarely used for their diagnosis after the planned PCI.

Changes occurring in the myocardium depends on the duration of ischemia duration – less than 30 minutes in the myocardium appear small foci of necrosis, which are subsequently replaced by connective tissue, leading to an additional coronary risk [15]. On 30 min time increase of ischemia there occur irreversible damage to the myocardial cells, by activation of lipid peroxidation and release of lysosomal enzymes into the extracellular space with microcirculatory occlusion of the coronary vessels. Persistent ischemia for 40–60 min leads to structural changes in the heart muscle, and ischemia lasting 60–120 minutes – to the death of cardiomyocytes. Thus 80% of the cells are killed for 3 hours, and nearly 100% – within 6 hours of myocardial ischaemia [7].

Cardiac markers released from damaged cardiomyocytes: myoglobin, troponin T, CK-MB [8] are secreted into blood during myocardial necrosis.

Myoglobin – heme-containing protein found in all muscle cells. Its molecular weight is 18 kDa [8]. Myoglobin functions are transporting oxygen from the hemoglobin in the blood to the muscle cells, passing his muscle mitochondrial cytochrome oxidase.

During myocardial necrosis myoglobin easily penetrates through the membranes of damaged muscle cells, as it is quite a low molecular weight protein. Thus, myoglobin is early enough sensitive marker, but not sufficiently specific.

Troponin – the structure of a protein nature, located on the thin myofilaments contractile

apparatus, universal for striated muscle. There is a troponin complex consisting of three subunits: T – is associated with tropomyosin, I – which is an inhibitory protein and C, which is connected with calcium ions. Troponin C is the same for all types of muscles, therefore is not used as a heart biomarker. Troponin isoform – T and I, specific for myocardium [11]. The molecular weight of T troponin is 37 kJ, I troponin – 23,8 kDa [8]. Blood contains in the cytoplasm 6–8 % of troponin T and 2,8–4,1 % of troponin I that indicates that the concentration of troponin T in the blood rises more quickly than of troponin I [10]. In this regard, troponin T is used in clinical practice.

After success of PCI and coronary artery stenting it is noted a slight increase in the level of troponin by 24–40 % of patients with coronary artery disease [16]. The main reason of troponins increasing in such situations is transient ischemia caused by intracoronary balloon blowing up, intervention in the arteries, coronary artery dissection, microembolization of distal channel by plaques material. This is due to their high sensitivity and specificity; they are also used for the diagnosis of MMD. The results of these studies indicate poor prognosis of troponin-positive patients in terms of cardiovascular events risk [16].

Creatine phosphokinase (CK) – an enzyme contained in cardiomyocytes in skeletal muscle cells, brain, thyroid and lungs. Determination of total creatine phosphokinase in blood serums for the diagnosis of myocardial ischemia is currently impractical since it is contained in a large amount in the skeletal muscle and has low specificity for the myocardial necrosis [3].

CK is composed of two sub – M (muscle type) and B (brain type) which form three isoforms: CK-MB – heart isoenzyme, the concentration of which changes in cardiomyocyte injury; CK-BB – brain isoenzyme, reflecting the pathology of brain cells; CK-MM – muscle isoenzyme, located in skeletal muscle.

CK-MB isoform – heterodimer with a molecular weight of 86 kDa, which is used as the "gold standard" in immunochemical diagnosis of ischemic myocardial injury.

There are data in the literature that for the detection of myocardial damage in patients in the performance of PCI and coronary artery stenting CK-MB is a specific and sensitive marker of myocardial necrosis [2].

Thus, for the diagnosis of small myocardial damage in patients undergoing percutaneous coronary intervention and stenting of the coronary arteries, one can use cardiac markers, such as myoglobin, troponin-T, creatine phosphokinase-MB.

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