LETTERS TO EDITOR

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Myocardial Bridges

Mostki mięśniowe serca

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The presence of muscle fibers covering intracardiac segments of epicardial coronary arteries was first discovered in 1737. Their detailed morphological description was presented in 1951, but only coronarographic investigations and Doppler IVUS (intravascular ultrasound) have enabled an evaluation of their hemodynamic function and impact on heart circulation. Despite the common occurrence of myocardial bridges (MBs), few research reports on their clinical significance would be of interest to the readers of this journal.

Muscles overlying the intramyocardial segments of the epicardial coronary arteries are defined as myocardial bridges (MBs). In postmortem studies carried out in 2666 subjects, such MBs were found in approximately 39% of cases and also in 7% of 28,544 patients who underwent coronarography [1]. MB can occur in all the coronary arteries, but their presence was revealed most frequently, i.e. in about 80% of patients, in the left anterior descending coronary artery. The lengths of the MBs were usually from 10 to 30 mm, they were located at a depth of 1 to 10 mm [2], and in 75% of the subjects they ran perpendicularly to the coronary artery. They were occasionally found in veins. The greatest number of MBs in several arteries was demonstrated in patients with hypertrophic cardiomyopathy [3].

The presence of MBs causes an increase in systolic and diastolic pressure in that part of the artery which is proximal to the MB and a pressure decrease beyond the MB. The rise in arterial tension and acceleration of blood flow velocity in the region of the MB induced by this phenomenon predisposes to a rapid development of atherosclerotic changes. They may be caused not only by mechanical injury to the endothelium with the formation of a thrombus, but also by a decreased synthesis of nitric oxide, which has a vasodilatating effect, an increase in the synthesis of endothelin-1, with strong vasoconstricting and pro-aggregative effect on blood platelets, and also a local increase in the secretion of angiotensin I- to angiotensin II-converting enzyme [4, 5]. Cardiac ischemia in patients with MB(s) may result from both direct compression of the coronary artery by the bridge's muscle fibers and from its spasm caused by enhanced synthesis of endothelin-1 or a thrombus arising at the site of the damaged endothelium.

Clinically, ischemia of the heart is manifested as physical effort-induced angina pectoris. Such pain can also appear at rest due to an increase in emotional tension, which predisposes to contraction of the coronary artery or arteries. A thrombus arising in the coronary artery or prolonged spasm of the artery could be accompanied by symptoms of unstable angina pectoris, cardiac arrhythmia in the form of ventricular premature beats or ventricular tachycardia, transient atrioventricular blocks, and left bundle branch block, and in severe ischemia the patient may develop myocardial infarction. Recurrent myocardial ischemia could be the reason for stunning of the heart muscle and dysfunction of the left ventricle, both systolic and diastolic.

In the diagnosis of MBs, a resting electrocardiogram usually does not reveal changes unless it is being performed at the time of an ischemic episode, while an exercise electrocardiogram, often accompanied by cardiac arrhythmia, may be positive [1, 6, 7]. The gold standard for the detection of MBs includes coronarography, myocardial perfusion scintigraphy during exercise stimulation [8, 9], and Doppler IVUS [5, 10]. In the latter investigation, a short-lasting acceleration of coronary artery flow in the early stage of ventricular diastole followed by its deceleration in mid-diastole is a characteristic symptom of the presence of

a myocardial bridge [10]. Great hopes in the detection MB have been associated with Magnetic Resonance Angiography (MRA) and Multi-Slice Computed Tomography (MSCT) [11].

Generally it is estimated that prognoses regarding the survival of patients with MBs are good. Of 61 patients aged 50 with an MB in the left anterior descending coronary artery, 98% survived 11 years [8]. Another study demonstrated that from among 81 patients 46 years of age with an MB, it was the cause of death in only two patients [9]. The prevention and treatment of ischemia of the heart muscle caused by an MB is based on the administration of beta-adrenolytics, which not only display a negative inotropic effect, thus reducing cardiomyocyte tension in the MB, but also decrease the demand for oxygen in the heart muscle and the frequency of tachyarrhythmias [12]. These medications, however, as a consequence of blocking the beta-adrenergic receptors, cause a relative overbalance of alpha-adrenergic receptors, which may predispose to coronary artery contraction. Positive effects in the prevention and treatment of cardiac ischemia caused by the presence of an MB could be expected from treatment with calcium channel blockers, because apart from their negative inotropic effect, they dilate the lumen of the coronary arteries and can also restore the secretory function of the injured endothelium in patients with MB. The use of aspirin is also highly justified in these patients, as it can prevent the formation of a thrombus in the place of a functionally or constructively impaired coronary artery. Furthermore, aspirin diminishes the risk of spasm of the coronary artery. In patients with frequent relapse of cardiac ischemia in spite of mono- or polytherapy with the above-mentioned drugs, implantation of a stent to the bridged coronary artery segment [13, 14] or surgical myotomy on the MB should be considered [15].

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