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Case report

Acute mitral insufficiency as a consequence of long-distance run

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ABSTRACT

The acute mitral insufficiency is a life-threatening condition that may be caused by heavy physical strain, especially during the simultaneous occurrence of the myxomatous degeneration of the mitral valve. The mortality of the untreated illness is 75% during the first 24 h after the occurrence; the perioperative mortality is also high.

The case study describes the story of a 57-year-old male, an active sportsman (long-distance runner), whose health condition was duly examined in an institute of sport medicine in Germany, and the results were reportedly always normal. Immediately after finishing a marathon run here, in Prague, he began to complain of severe dyspnea, NYHA III–IV. The physical examination revealed clearly audible strong systolic murmur with the amplitude on the heart apex with propagation into the left axilla, and the signs of pulmonary congestion were present. The transthoracic echocardiography confirmed the suspected acute mitral regurgitation, with a minor dilatation of the left atrium, and the hyperkinetic left ventricle with the preserved systolic function (EF 70%).

After the confirming examination using transoesophageal echocardiography, the surgical revision of the mitral valve was indicated. This revealed myxoid degeneration of both leaflets of the mitral valve, tendinous cord rupture, and the dilatation of its annulus. A successful preservation operation was performed, together with the mitral plastic surgery, annuloplasty, and tendinous cord replacement. Having recovered from the difficulties of the post-surgery period, the patient was released to the domestic care on the 14th day after the operation.

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Introduction

The mitral valve is a very complicated structure, consisting of many parts and elements. Its correct function depends entirely on the complex functionality of all elements of the valvular apparatus – i.e. mitral annulus, both leaflets of the valve, tendinous cords, papillary muscles, but also the myocardium of the left atrium and the left ventricle [1]. The correct function therefore depends on the correct functionality and coordination of all the parts, and the well-preserved geometry of the whole complex. An acute damage of any part may result in the life-threatening condition – acute mitral insufficiency. The trigger may also be heavy physical strain, such as the long-distance run.

The most frequent cause of the acute mitral insufficiency is the damage of the papillary muscle caused by ischemia, rupture of the fixation apparatus, or the perforation of the valve leaflets during the infectious endocarditis, or upon the myxomatous degeneration or fibroelastic deficiency, mostly affecting elderly patients [2].

The clinical image presents, above all, the symptoms of the severe lung congestion with the signs of the left-sided heart failure, which may result in a severe shock. The treatment of patients suffering from a*cute mitral regurgitation involves the use of* vasodilating drugs, and both pharmaceutical and mechanical support of the cardiovascular system, but the only life-saving method is immediate surgery.

The mortality of the untreated illness varies according to the cause of occurrence, scope of the valve damage, and also according to the function of the left ventricle and the findings on coronary arteries. The mortality can be high; in case of a severe damage to the valve and the bad condition of the left ventricle, it can reach up to 50% within the first 24 h, and up to 90% within 48 h following the occurrence. The perioperative mortality is also high (20–50%) [3].

Observation

Our workplace (Canadian Medical Care, Praha, CMC) was examined and treated a 57-year old male – a foreigner, active long-distance runner, who was regularly examined at a clinic of sport medicine in Germany, allegedly always with negative results. The CMC examined him due to major dyspnea (NYHA III–IV) that occurred after he had finished the marathon run.

The initial physical examination showed, above all, clearly audible strong holosystolic murmur above the mitral valve, with the amplitude on the heart apex (5/6) with propagation into the axilla. Also present were symptoms of pulmonary congestion and tachycardia 126/min, blood pressure 130/90. The ECG recorded the sinus rhythm with the borderline AV conduction, normal range of QRS complex and length of QT interval, and the elevation of ST segment in the leads V5, V6. The resting O_2 saturation was 88%.

Based on the history and physical examination, the patient was immediately sent for further examination to the cardiology ward of Hospital Na Homolce, with suspected acute mitral insufficiency.

At the ward, the transthoracic echocardiography confirmed the suspected acute mitral regurgitation, with a minor dilatation of the left atrium, and the hyperkinetic left ventricle with the fully preserved function of the left ventricle (EF LK 70%).

The subsequent transoesophageal echocardiography confirmed the occurrence of major mitral regurgitation with the myxomatous degeneration of the rear valve leaflet, P2-Barlow prolapse, and the ''flail leaflet''.

The chest X-ray describes the increased lung congestion on the X-ray located paracardially on the right, or almost diffusely in left lung. The cardiac shadow is extended to the left.

Excerpts from the available lab results: Troponin I 0.14 ng/l, Myoglobin 47.4 ng/l, CRP 51.90 mg/l, leukocytes 12.9 g/l, D-dimers 168.0 ng/ml.

The selective coronarography showed normal findings on coronary arteries, dilated left ventricle with good wall kinetics, and severe mitral regurgitation of the 4th degree.

The patient was stabilized using the intra-aortic balloon counterpulsation (IABK) with the optimized after load, and indicated for the emergency cardiac surgery.

Again, the perioperative transoesophageal echocardiography gave yet another evidence of the major mitral regurgitation of the 4th degree, with a broad jet to the whole left atrium, good systolic functionality and kinetics of the left ventricle, myxomatous degeneration of the rear valve leaflet with the P2 prolapse, and the ''flail leaflet''. The aortal and tricuspid valve does not show any signs of regurgitation, the findings were normal.

During the surgery, the revision of the mitral valve confirmed the diagnosis of myxomatous changes of both leaflets (morbus Barlow), ruptured tendinous cords to the whole P2 segment, and massive dilatation of the annulus. The plastic surgery of the mitral valve was performed with a partial resection, transposition and re-implantation of the rear leaflet of the mitral valve, together with the tendinous cord replacement, and annuloplasty.

In the early post-operational stage, the patient was stable in terms of both hemodynamics and ventilation; gradually, inotropic support and IABK were discontinued.

Further post-operative development was complicated by the paroxysmal atrial fibrillation with spontaneous termination, and the AV block of 2nd degree Wenckebach (the borderline AV conduction was present even before the hospitalization). Given the missing symptomatology, and upon the consultation with the patient, the implantation of the pacemaker was rejected; further decisions will be based on the findings from the examinations at the patient's residency. The patient was released to the domestic care on the 14th day after the operation.

Discussion

Biochemically, the myxomatous degeneration of the mitral valve is characterized by the thickening and proliferation of the spongiform layer of the valve, with accumulation of glycosaminoglycans. This damages the firm fibrous structure of the valve, resulting in the presence of cystic structures and less dense collagen [4]. The changes in