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Clinical Case of Constrictive Pericarditis.

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ABSTRACT

This paper deals with analysis of clinical case of constrictive pericarditis (CP) having been manifested itself for two years only as recurrent swelling of the lower extremities. Based on the results of transesophageal echocardiography we diagnosed a congenital heart disease (CHD): right cor triatriatum complicated with chronic right ventricular failure (VF). The multi-slice spiral computed tomography revealed CP with massive calcification of the pericardium (up to 5 mm). A patient underwent the subtotal pericardectomy.
Keywords: constrictive pericarditis, surgical treatment

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INTRODUCTION

Constrictive pericarditis is a rare, severe disabling outcome of chronic inflammation in the pericardium [1]. A fibrinous change of parietal and visceral pericardium layers results from transition of the process to the phase of productive chronic inflammation. The layers may thicken up to 1.5-2.0 cm or more, forming a thick connective tissue armor, which further compresses the heart (“armored heart") due to the progressive shrinkage of fibrous tissue. The clinical signs are a right-ventricular type manifestation and progressive course of CP and often a permanent atrial fibrillation correlated with reversible mild mitral insufficiency in the echocardiography [2]. There are different causes of CP, while the etiology of the disease remains often unknown [3]. According to the literature, one of the reasons may be a strong hypothermia [4].

CASE REPORT

Female patient S., 27 years old, a worker of the cold shop at a cold storage facility, was admitted to the cardiac department of St. Joasaph Belgorod Regional Clinical Hospital on November 18, 2014, with complaints of dyspnea upon significant physical exertion, swelling of legs and feet intensifying in the evening.

The patient considers herself ill since October 2014, when according to the results of echocardiography she was diagnosed overdeveloped Eustachian valve and an additional linear formation in the RA cavity in the form of fenestrated membrane that was a reason to suspect a right triatrial heart, as well as the partial anomalous pulmonary veins drainage.

To clarify the diagnosis and decide on the therapeutic approach the patient was hospitalized to the cardiac department. Life history revealed no clinically significant pathology until disease. A burdened professional history was established: working in the cold shop of cold storage facility and contacting with chlorine-containing products used for disinfection of refrigerating plants.

Satisfactory general condition. Undernourished (height 172 cm, weight 58 kg), BWI 19.61. Skin and visible mucous membranes of normal color. Respiratory rate - 16 per minute. Vesicular breathing auscultated over lungs from both sides, no rales. The heart and large vessels region has no visual changes. Cardiac borders are extended 1 cm right. Slightly muffled heart sounds, regular rhythm, heart rate - 72 bpm. Blood pressure 110/70 mm. Hg. No pathological cardiac murmurs auscultated. The examination revealed swelling of legs and feet. Soft abdomen. The liver protrudes 4 cm from the right costal margin, painless on palpation, smooth edges; non-palpable spleen.

The general clinical laboratory studies revealed no significant abnormalities. Electrocardiography (ECG) at admission: sinus rhythm, 72 pulses per minute. Vertical electrical axis. Abnormalities in the left ventricle (LV), T-wave inversion in III, AVF, V_2-V_6.

According to the chest X-ray, the lungs have neither foci nor infiltration. The heart is expanded in diameter, calcified pericardium along virtually all cardiac alignments.

Transesophageal echocardiography revealed the increased RA and LA cavities, and dilatation of the inferior vena cava (IVC) and hepatic veins. RA cavity has a linear formation (membrane) located from IVC entry toward the base of the atrial septal with signs of fenestration (turbulent flow in the central part of the diaphragm). Echocardiography pattern of the right triatrial heart. LV ejection fraction - 61%. The assessment of diastolic function of both ventricles revealed a restrictive abnormality E/A 2.7, DT 110 msec, E/e 19.4, second degree tricuspid insufficiency, the IVC dilated up to 2.5 cm, inspiratory collapse less than 30%, moderate pulmonary hypertension (the pressure in the pulmonary artery (PA) - 35 mm Hg.), the pericardium within normal limits.

Ultrasonography (US) of the abdomen showed an enlarged liver by 4.0 cm with decreased echogenicity, IVC dilatation in diameter up to 28.0 cm, the hepatic veins - up to 12.0-13.0 cm, and free fluid in the abdominal cavity.

The results of contrast-enhanced multislice spiral computed tomography (MSCT) (bolus injection of 100 ml of Ultratvist-370) detected no focal or infiltrative changes of lungs. There is a massive calcification of

the pericardium along cardiac alignments and RA, max 5-6 mm thick, armored heart. These changes are accompanied by dilatation of the RA and LA cavity, expanded lumen of the IVC and superior vena cava (SVC) before the right atrium up to 42×34 mm and 27×19 mm, respectively. No visualizable enhancement defects of the heart chambers, PA, and its branches were detected. Pulmonary veins are connected typically to the LA.

The patient was made a clinical diagnosis: Chronic constrictive pericarditis. Calcification of the pericardium ("armored heart"). Congenital heart disease. Right triatrial heart. CHF II A, FC II.

The patient was sent to the cardiac surgery department, where she underwent the operation: Cardiolysis. Subtotal pericardectomy acc. D. Cooley's approach.

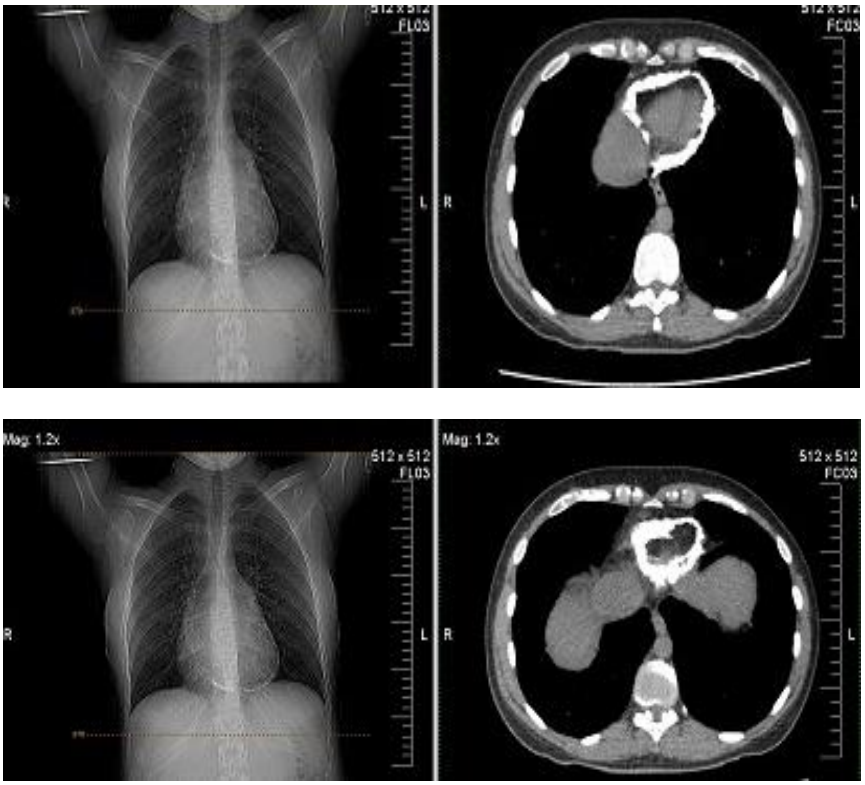

Figure 1: Chest X-ray and MSCT of a patient S., 27 years old Calcification and thickening of pericardium.

The myocardium was biopsied during operation. We conducted a carbon mapping of macroelement content in the tissue by using a scanning electron microscope FEI Quanta 600 (shooting mode: HighVac; 30.00 kV); X-ray fluorescence analysis (XRFA) found no local concentrations of Ca, K, Na, P, S, but revealed the pathological presence of chlorine atoms (0.07±0.03% At).

The patient was discharged 12 hours after surgery. February 2015, 3 months after the operation - the patient's condition is satisfactory: a significantly decreased dyspnea and leg swelling.

DISCUSSION

CP incidence is 0.005% [5]. Idiopathic pericarditis is 3.5% to >50% in the Marburg Pericarditis Registry (1988 - 2011). According to Russian scientists [6], the idiopathic pericarditis etiologically were 23.6% in patients undergoing pericardectomy in Ekaterinburg. The problem of toxic pericarditis at exogenous intoxication remains poorly explored [7].

Considering high heart failure incidence in the population and the low CP incidence, the diagnostics and differential diagnostics of CP can be a challenge. The vast majority of CP is observed in young and working age people who seek care at the stage of manifesting heart failure with severe complaints, which complicates greatly early detection of CP and complicates the differential diagnosis. Classically, diagnosing the CP is the

final exclusion of numerous cardiac and noncardiac trivial "masking" pathologies [8]. Despite the fact that the guidelines on imaging techniques refer to the ability of the echocardiographic study to identify productive inflammation of the pericardium by its layers thickening, there are no diagnostic techniques able to reliably detect the adhesive and constrictive pericarditis at different stages.

Our case report demonstrates non-typical clinical course of CP during the "clinical manifestations" (slight complaints of dyspnea upon significant physical exertion, legs and feet pastosity, no cardiac arrhythmia, tightness in the right hypochondrium). Echocardiography pattern non-typical of CP - the visualized pericardium is of normal size and free of calcification against the presence of congenital cardiac anomalies, which complicated the diagnosis. Triatrial heart incidence is 0.1-0.4% of total congenital heart diseases [9]. In 1981 there were about 200 cases described. Right triatrial heart develops because of pathological retention or excessive growth during embryogenesis the venous sinus valves.

Myocardium biopsy is indicated for the differential diagnosis of CP and restrictive cardiomyopathy [10]. Literature data on the content of chemical elements in the human myocardium is extremely limited. The presented case report involved mapping of spatial distribution of Na, Mg, P, S, K, Ca, Cl concentrations in the myocardium tissue in case of CP conducted for the first time, and the pathological Cl content determined. In our view, this may suggest a probable relation of CP with chlorine intoxication as etiologic factor in the development of CP against the congenital cardiac abnormality and repeated hypothermia.

CONCLUSION

The considered case report demonstrates the difficulty in diagnosing CP even at the stage of clinically significant symptoms of heart failure, in the presence of congenital cardiac abnormality. Using high-technology instrumental diagnostic techniques such as MSCT and studying nanoscale regions using transmission electron microscopy allow us to perform a differential diagnostics with cardiomyopathy, myocarditis, and to suggest etiological factors of the constrictive pericarditis such as chlorine intoxication against underweight or repeated hypothermia.

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