

Bacterial Endocarditis of Systemic Atrioventricular Valve (Tricuspid Valve) in Corrected Transposition and its Surgical Treatment

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We describe a case of congenitally corrected transposition of great arteries (CCTGA). Tricuspid valve replacement was performed due to valve dysfunction following bacterial endocarditis. After two weeks' antibiotic therapy haemodynamic stabilisation was obtained and the patient was operated in the third week. On cardiopulmonary bypass with 28°C degree systemic hypothermia, the left atrium was approached transeptally. At exploration, the systemic atrioventricular valve was tricuspid valve and pulmonary atrioventricular valve was in shape of a mitral valve. The posterior leaflet of the tricuspid valve was ruptured and vegetations above it were observed. The valve was excised and a 29 mm St-Jude mechanical heart valve prosthesis implanted using a teflon reinforced separated suture technique. After operation the patient recovered rapidly and following six weeks' antibiotic therapy, the patient was discharged. (*Ann Thorac Cardiovasc Surg* 2005; 11: 201–3)

Key words: corrected transposition, bacterial endocarditis, tricuspid insufficiency

Introduction

Corrected transposition which was first described by Rokitansky, is a rarely seen congenital heart disease. Newborns with this abnormality, if no other abnormality is associated, may not develop any symptoms because of normal physiology. In this abnormality transposition of large vessels, inversion in ventricles and atrioventricular valves (A-V) persist.

Here we present a case of congenitally corrected transposition of great arteries (CCTGA) in which tricuspid valve replacement was performed because of valve dysfunction following bacterial endocarditis.

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Case Report

A 29-year-old woman presented with hemoptysis, dyspnea, bloody sputum, fatigue weakness and tachyarrhythmia for more than four months'. On physical examination, she was orthopneic, and had persistent sinus tachycardia (102 bpm) with the classical murmurs of mitral insufficiency. On lung auscultation there were bilateral crepitant rales. She had hepatosplenomegaly and ascites on her abdominal examination. Pretibial three positivity edema was also present. Her blood pressure was 130/90 mmHg. In her electrocardiogram Q waves were present in right precordial leads but were absent in the left precordial leads. Telecardiogram was showing cardiomegaly and congestion of lungs. Laboratory investigations revealed increased blood urea nitrogen (38 mg/dl) and creatinine (2.8 mg/dl) levels, anemia (haemoglobin 8.4 g/dl), leucocytosis (white blood cell count 15,000/mm³) and a decreased total serum protein/albumin ratio (5.2/2.8 g/dl). Latex agglutination was higher than normal limits (48 mg/dl) and erythrocyte sedimentation rate (ESR) was 68 mm/h. Two-dimensional and Doppler echocardiography demonstrated corrected transposition

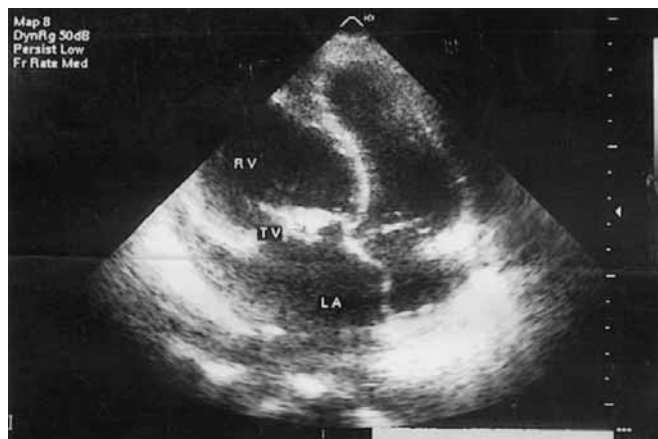


Fig. 1. Preoperative echocardiography shows abnormally high echo-density formed on the anterior leaflet of the systemic atrioventricular (Tricuspid) valve.

of great arteries with left atrioventricular (tricuspid) valve insufficiency (3/4 degree) and vegetations above it, and a patent foramen ovale (Fig. 1). Blood culture was positive (coagulase (-) staphylococcus aureus) and she was started on nafsilin (8 g/day). After two weeks' medical and antibiotic therapy haemodynamic stabilisation was obtained and the patient was operated in the third week.

At exploration via the median sternotomy, both ventricles were inverted, the aorta was left and anterior to the pulmonary artery. (Situs solitus) On cardiopulmonary bypass (CPB) with 28°C degree systemic hypothermia, the left atrium was approached transeptally. At exploration the systemic atrioventricular valve was in shape of a tricuspid valve and the pulmonary atrioventricular valve was in shape of a mitral valve. The right ventricle had usual coarse trabeculations, a septal band (Trabecula septomarginalis), and an infundibular septum. The valve was positioned in a sagittal plane and had three leaflets. The patient did not have an Ebstein-like abnormality of the tricuspid valve. The posterior leaflet of the tricuspid valve was ruptured and vegetations above it were observed (Fig. 2). The valve was not amenable to surgical repair. The valve was excised from tricuspid annulus and 29 mm St-Jude mechanical heart valve prosthesis implanted using a teflon reinforced separated suture technique. Valve replacement was the same as for a left sided mitral valve. Complete or incomplete heart block did not develop. Prophylactic permanent pacing wires were implanted to the right atrium and ventricle. No microorganisms were observed upon the microbiological examination of the resected tricuspid valve and cultures were negative. Patho-



Fig. 2. Intraoperative transatrial appearance (Posterior leaflet of tricuspid valve is ruptured and this figure shows vegetations above anterior and posterior leaflets of tricuspid valve).

logical examination was reported as degenerated and calcified valve material. After operation, the patient recovered rapidly and was administered six weeks' antibiotic therapy, then discharged. Examination of the telecardiogram and echocardiogram were undertaken on one month after discharge, and she was found in functional class I-II of the New York Heart Association (NYHA).

Discussion

In the CCTGA without additional cardiac defects systemic venous blood flow is from the right atrium to the transposed pulmonary artery through an inverted mitral valve and left ventricle and from the pulmonary venous system to the aorta through the left atrium, inverted tricuspid valve and right ventricle. In the presence of additional cardiac abnormalities the patient will be symptomatic and will need treatment. Because of normal physiology the patient may not be a symptomatic and there may be no need for treatment, in the absence of any additional cardiac abnormality. In this case, the patient had no problems for 28 years and had four pregnancies without any cardiac problems. But endocarditis of the systemic A-V valve had impaired physiological function.

The most common additional cardiac abnormality in CCTGA is ventricular septal defect (VSD). The VSD is generally wide and infracristal. Pulmonary stenosis, atrial septal defect, patent ductus arteriosus, and tricuspid insufficiency is other additional abnormalities that can accompany with CCTGA.

In cases without additional defects, cardiac complications that affect the survival and life quality are complete atrioventricular heart block, systemic (tricuspid) atrioventricular valve regurgitation, infective endocarditis, supraventricular tachycardia and congestive heart failure. The most common cause of death in these patients is congestive heart failure. The rate of appearance of congestive heart failure after the fourth decade is up to 67% in patients with additional cardiac abnormalities while it is 25% in simple cases.¹⁾

Tricuspid valve incompetence (TI) is frequently seen in patients with corrected transposition. There is a close relation between TI and systemic ventricular dysfunction leading to congestive heart failure, it is debatable which of them is the primary disorder, but RV dysfunction appears to be secondary to long-standing TI.²⁾

The incidence of endocarditis is increased in patients with cardiac lesions which cause turbulent flow.³⁾ Cyanotic congenital heart lesions, previous bacterial endocarditis, aortic valve disease, mitral regurgitation and uncorrected left-to-right shunt are high risk diseases for development of endocarditis.⁴⁾ CCTGA with or without additional cardiac defects is a high risk abnormality for endocarditis. Connelly and colleagues, in their review on 52 patients over 18 years old, reported the rate of endocarditis in CCTGA as 11.5%.⁵⁾

Surgical techniques for correction of systemic atrioventricular valvular pathologies in CCTGA do not differ from other valvular diseases. Reconstruction and repair techniques have better results than the valve replacement when hemodynamics, recurrence, mortality and long-term results are considered.⁶⁾ Occasionally, due to large tissue loss of leaflets secondary to infection or a degenerative process, or problems with subvalvular structures, it is impossible to perform a reconstruction and valve replacement is required.

Untreated, some cardiac complications like TI, rhythm disturbances especially atrioventricular heart blocks, pulmonary over circulation, and RV dysfunction dictate the outcome. Any surgical intervention towards additional lesions in corrected transposition increases the occurrence of these complications.⁷⁾

Van Son et al.⁸⁾ reported that immediate surgery is indicated for deteriorating heart failure from infectious

endocarditis in CCTGA. The patient was operated on three weeks after antibiotherapy administration. Surgical intervention after gaining control of infection is essential and safe, hence the delay in surgical intervention. The patient was assessed by serial clinical evaluation and echocardiography, to monitor ventricular function. Deterioration of the patient was not observed during medical treatment.

In conclusion, patients with CCTGA are generally asymptomatic if they have no any additional cardiac anomalies, but they are at risk for AV block, arrhythmias, congestive heart failure and rarely infective endocarditis. Surgery should be performed immediately when intrac-table heart failure appears due to infective endocarditis in patient with congenital heart disease. If clinical deterioration of the patient is not observed during medical treatment, cardiac operation may be safely delayed.

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