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Case Report

A Rare Case of Tricuspid Valve Endocarditis in an Adolescent Boy with History of Drug Use

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Introduction

Since endocarditis was first described, both the spectrum of causative organisms and the patients affected have changed [1]. According to literature, right-sided infective endocarditis (IE) constitutes 5% to 10% of all cases of endocarditis [2]. It occurs predominantly in intravenous drug abusers (IVDAs) and patients with pacemakers, central venous lines or congenital heart disease. The vast majority of cases involve the tricuspid valve [3].

Reports of tricuspid valve endocarditis (TVIE) are growing because of the increasing frequency of injection drug use and poor hygiene associated with homelessness in many urban communities, [4] even among younger age groups.

We present the case of a 17 year old adolescent with history of intravenous drug use and subsequent TVIE.

Case Report

A 17-year-old adolescent male, with a history of intravenous drug use one year prior to hospitalization (started at age 16 years), attending a buprenorphine substitution program, presented with fever, rigors, night sweats, cough, and pleuritic chest pain of about one week duration.

On admission, he was toxic, with a temperature of 40°C, tachycardic with a heart rate of 110/min, and a respiratory rate of 20/min.

Physical examination showed mild jaundice. Lung auscultation revealed fine end-inspiratory crackles in the lower third of both lung fields. There was a grade 3/6 pansystolic murmur in the tricuspid area and mild pedal edema.

All other aspects of the comprehensive physical examination were normal. There was no skin or mucosal stigmata of endocarditis, venepuncture marks, lymphadenopathy, signs of meningism, or neurological symptoms.

Laboratory Work-Up

Laboratory analysis showed normocytic anaemia with hemoglobin 71 g/dl, a white cell count 8400 cells/µl, neutrophils were 75% and thrombocytopenia of 57000/µl.

C-reactive protein was elevated 170 mg/l (0-5), with an erythrocyte sedimentation rate of 90 mm/hr and procalcitonin (PCT) of 8.36.

Liver panel alterations with moderate cholestasis were revealed (total bilirubin: 61µmol/l; direct bilirubin: 33; aspartate transaminase (AST): 52 U/l; alanine transaminase (ALT): 55 U/l; µGT: 56 U/l; albumin: 23 g/l; proteins 53 g/l).

Mild electrolyte disbalace was found (Na: 129 mmol/l; K: 3.4 mmol/l; Mg: 0.63 mmol/l).

Troponin I (hsTnI) levels were normal (0.010) and BNP was 207 pg/ml.

HIV virus and hepatitis B antigen were negative, while hepatitis C virus was positive.

Three blood cultures were drawn aseptically from three separate sites, 1h apart. Methicillin-resistant Staphylococcus aureus *(MRSA)* was isolated from one sample.

Investigations

Chest X-ray showed bilateral basal pulmonary infiltrates which was consistent with the physical examination.

The electrocardiogram did not show any pathological changes.

Transthoracic echocardiography showed dilated right atrium (RA) and right ventricle (RV) with medium-sized, oscillating vegetation attached to the anterior tricuspid valve leaflet, 9.7 mm x 6.8 mm (Figure 1). There was severe tricuspid regurgitation with calculated right ventricular systolic pressure of 35 mmHg (Figure

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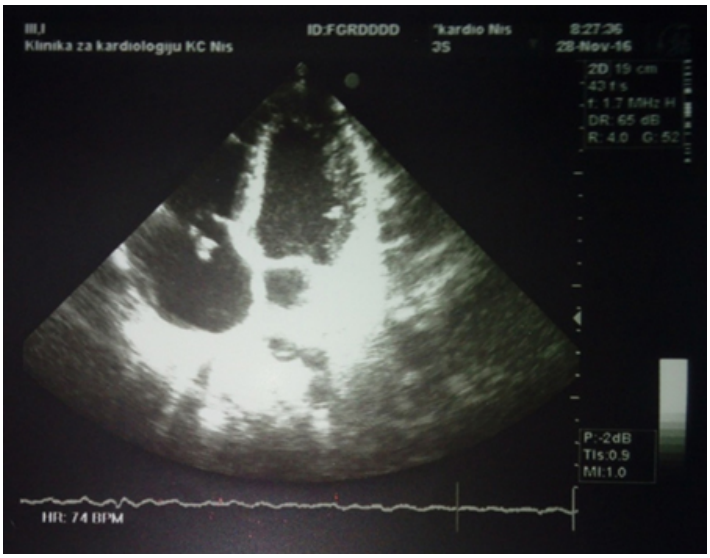

Figure 1. Transthoracic echocardiography showing mobile vegetation attatched to tricuspid valve leaflet.

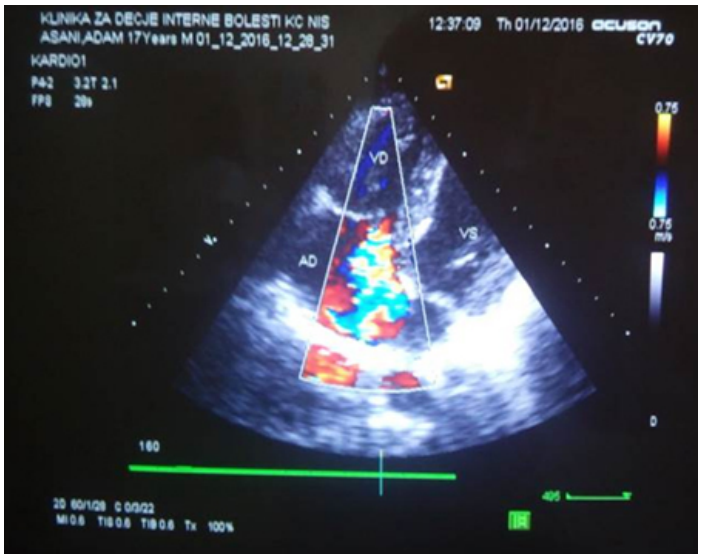

Figure 2. Transthoracic echocardiography showing severe tricuspid regurgitation

2), mild circumferential pericardial effusion, and right ventricular dysfunction. The inferior vena cava was dilated (1.9cm). There were no signs of vegetations on the mitral, aortic or pulmonary valves, which were all normal. The left side of heart was also normal.

Abdominal ultrasonography revealed mild hepatosplenomegaly with signs of evident liver parenchymal damage, explained by the diagnosed hepatitis C with perihepatic ascites. Additionally there was evidence of borderline congestion of the liver veins and the inferior vena cava, due to right heart failure.

Treatment

Antibiotic therapy with intravenous cefuroxime and gentamycin was started empirically as sepsis was suspected on admission, and changed to vancomycin alone following blood culture reports. Vancomycin was continued for 4 weeks until a negative blood culture was obtained. Intensive diuretic therapy was commenced for right-sided cardiac decompenstation.

After two weeks of antibiotic therapy, he was afebrile and clinically stable, but repeated echocardiograms showed persistence of vegetation with severe tricuspid regurgitation, and mild right ventricular dysfunction.

These findings were corroborated in the follow-up TTE studies performed during hospitalization

The endocarditis was complicated by symptomatic pulmonary involvement. Following 4 weeks of vancomycin therapy he complained of fever of 39°C, and chest pain. The C-reactive protein was 143 mg/l. Chest X-ray confirmed pleuritis and pulmonary infiltrates (Figures 3 and 4), probably due to septic pulmonary embolism. Antibiotic therapy was changed to ceftriaxone and amikacin. Blood cultures were repeated. Even though blood culture results were negative, due to persistent febrile state, elevated

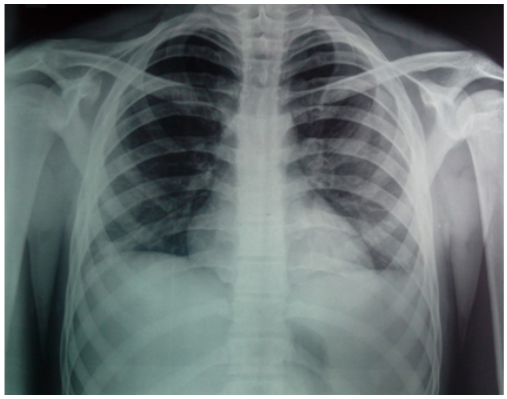

Figure 3. Chest X-ray showing bilateral lung infiltrates and pleuritis.

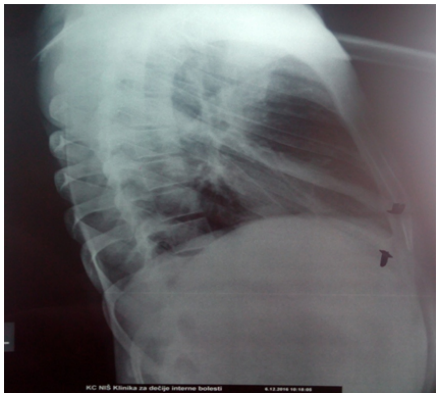

Figure 4. Chest X-ray showing left-sided pleuritis as a complication of septic pulmonary emboli

inflammatory parameters and high suspicion of vancomycin-resistant MRSA, he was started on linezolid. This treatment yielded clinical and laboratory improvement.

Follow-up TTE demonstrated persistent tricuspid regurgitation but no vegetations, while mild right heart dysfunction was noted.

Discussion

Herein, we report the case of a boy who unfortunately started drug abuse at a young age. He is a member of the minority Roma population in Serbia, with poor socioeconomic facilities. He developed tricuspid valve infective endocarditis (TVIE) with pulmonary septic emboli one year after quitting intravenous drug use. He was also diagnosed with hepatitis C. According to literature, 80% of cases of TVIE are found in drug addicts [5].

Diagnosis of endocarditis was made based on modified Duke Criteria. The major criterion was evidence of endocarditis on echocardiography (tricuspid vegetation and tricuspid regurgitation) along with three minor criteria: 1) fever, 2) pulmonary emboli, and 3) intravenous drug use.

It is known that vegetation on the tricuspid valve has a high risk of resulting in septic pulmonary emboli, causing various pulmonary complications such as pneumonia and pulmonary abscess [6]. Our patient had developed septic pulmonary emboli originating from the tricuspid vegetation before admission and most probably during the course of hospitalization as the tricuspid vegetation became smaller on echocardiography follow-up.

Common symptoms secondary to right-sided endocarditis are persistent fever, bacteremia, and multiple pulmonary emboli. Other findings include pleuritic chest pain or other pulmonary findings which may aggravate the clinical diagnosis. In exceptional circumstances, right-heart failure can arise, generated by the increase in pulmonary pressure, severe tricuspid valve regurgitation, or obstruction of pulmonary circulation through multiple pulmonary emboli [6], as was the case in our patient.

In our patient, methicillin-resistant Staphylococcus aureus was the identified causative organism. Right-sided endocarditis in IVDA is commonly caused by Staphylococcus aureus, Pseudomonas aeruginosa, and other gram-negative organisms, fungi, streptococci, and enterococci have also been implicated [6,7,8]. More than 90% of cases of infective endocarditis in intravenous drug users are caused by staphylococcus or streptococcus species [9]. Additionally, others have demonstrated a high infestation rate of methicillin-resistant Staphylococcus aureus (MRSA) in IVDA [10].

The main diagnostic tool in our study was echocardiography, by highlighting vegetations on the valves and valve regurgitation.

Conclusion

In summary, TVIE as a complication of IVDA occurs rarely in the pediatric population. The predisposing factors for this infection in our 17 year old patient were life in difficult socioeconomic circumstances, with which the Roma minority are faced, and an immune compromised state secondary to intravenous drug use and hepatitis C.

Optimum management of TVIE has not yet been defined. Uncomplicated tricuspid valve endocarditis can be successfully, medically treated in 80% of patients; in the remaining 20% with very large vegetations and expectably poor antibiotic penetration, surgical treatment is required [8,11]. Despite septic pulmonary embolism, recurring sepsis and right heart failure in our patient, medical treatment yielded very good results. We will closely monitor the patients' long term outcome.

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