Case study

Right-sided infective endocarditis with unknown ventricular septal defect in adult complicated by septic pulmonary emboli and acute renal failure:CASE REPORT

ABSTRACT :

Background: Right-sided infective endocarditis (IE) accounts for 3-14% of all cases of IE. Compared with left sided IE and the association of right heart endocarditis with ventricular septal defect in adults is rare but may have serious complications such as pulmonary embolism. its antibiotic treatment is more effective. Therefore, the timing of its surgical treatment is still controversial.

Case presentation: We report a case of a 21-year-old man with right sided *Staphylococcus aureus* endocarditis with unknown ventricular septal defect complicated by acute renal failure and haemoptysis caused by multiple pulmonary emboli .TTE showed a large left-to-right shunt subaortic VSD and multiple vegetations were attached to the anterior leaflet, on the septal leaflet, and on the posterior leaflet and an other vegetation attached to the chordae tendineae.Despite continuous antibiotic treatment, the vegetations still remained we decided to perform surgical treatment,vegetectomy, tricuspid valve plasty and VSD patch closure.

Conclusions: Patients with undiagnosed congenital ventricular septal defects are at high risk for infective endocarditis. Therefore, prophylaxis for endocarditis after dental procedures and/or soft tissue infections is strongly recommended.

Keywords: Ventricular septal defect; right-sided endocarditis; septic pulmonary embolism, congenital heart disease,glomerulonephritis.

INTRODUCTION :

Right-sided Infective endocarditis (IE) accounts for 5% to 10% of all IE cases, and compared with left-sided IE, it is more often associated with intravenous drug use, intracardiac devices, and central venous catheters (1).

However, patients with a congenital ventricular septal defect (VSD) are at high risk of IE [2 ,3].

The association of right heart endocarditis with ventricular septal defect in adults is rare but may have serious complications such as pulmonary embolism and acute renal failure.

In most cases, Staphylococcus aureus is identified as the pathogen in blood cultures, and the management is conservative with specific antibiotic therapy [1, 4].

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CASE PRESENTATION:

A 21-year-old man admitted to our department for complaint of intermittent low-grade fever and dyspnea of one month duration

accompanied by palpitation and general weakness.He had loss of appetite, and weight loss over the same period.

He had no history of infective contacts, no history of tuberculosis and peripheral evidence of intravenous drug abuse.

Physical examination at admission, his vital signs showed a blood pressure of 100/60 mmHg, a heart rate of 130 bpm, oxygen saturation of 97% on room air and a febrile temperature 38 °C.Cardiac auscultation revealed aregular rhythm and a grade 4/6 holosystolic murmur over the tricuspid valve.

Examination revealed a paleconjunctivae, and hepatojugular reflux.The abdomenwas soft, with splenomegaly .No rash was noted ,there was no lower limb edema or signs compatible with deep vein thrombosis, or other alterations.

Electrocardiogramshowed sinus tachycardia at 130 bpm, without signs of underlying ischaemia or atrioventricular block and the chest X-ray revealed a right pleural effusion.

The transthoracic echocardiogram (TTE) showed three mobile structuresadhering to the tricuspid valve leaflets, compatible withvegetations, to be clarified by transesophageal echocardiography with the size of 20 mm × 7 mm on the anterior leaflet, 16mm × 11 mm on the septal leaflet, and 10 mm × 7 mm on the posterior leaflet and another vegetation attached to the chordae tendineae (Figure 1).

Moderate tricuspid regurgitation was documented, as well as pulmonary hypertension, withpulmonary artery systolic pressure (PASP) of 34 mmHg (Figure 2), The other valves presented no vegetation or severe regurgitation.A 9 mm sub-aortic VSD with a left-to-right shunt (Figure 3) and dilatation of the pulmonary trunk to 38 mm were identified.The left ventricular ejection fraction was normal, and intracardiac thrombi were not observed.The TTE showed a moderate pericardial effusion.

Laboratory investigation revealed microcytic hypo chromic anemia with hemoglobin level of 6.5 g/dl and no ferritin deficiency explained by inflammatory systemic disease, leukocyte count was 9820 /µL, urea 1.2 g/l, creatinine 22 mg/l, and elevated inflammatory markers(C-reactive protein [CRP] 226 mg/dl and erythrocyte sedimentation rate [ESR] 60 mm in 1st hour); proteinuria was positive and viral serology was negative.

Other laboratory testsshowed normal thyroid function, negative rheumatoid factor, and circulating immune complexes and complement analysis within references values. Serum protein electrophoresis revealed hypoalbuminemia and an inflammatory pattern. no abnormalities in other laboratory parameters, including cardiac markers.

After admission, at least two blood culture sets were reported to be positive for methicillin-sensitive Staphylococcus aureus (MSSA).

In addition, a CT scan of the thorax demonstrated septic pulmonary emboli, segmental and lobar, upper and lower right complicated with pulmonary infarction as well as mild right pleural effusion and moderate pericardial effusion (Figure 4).

On the CT abdomen scan, we found a splenomegaly and no additional organ emboli or abscess formations.

Although this patient did not present neurological symptoms, brain magnetic resonance imaging indicated no evidence of a stroke or intracranial aneurysm,

In the light of the above clinical, laboratory and echocardiographic findings, a diagnosis was made of subacuteinfective endocarditis Empirical antibiotic therapy withintravenous Ceftriaxone and gentamicin according to the current European Guidelines, was administered. After results of the antibiogram, we switched to Vancomycin and Gentamycin adjusted to the renal function.

During hospitalization, the patient gained weight, increased peripheral edema, and generalized edema, and laboratory tests showed elevated creatinine, BUN, and hypoalbuminemia values. In addition, diagnostic urinalysis revealed macrohematuria, albuminuria, high levels of A1 microglobulin, and high protein-to-creatinine ratios, suggesting acute tubular injury. Autoantibodies and C3 complement tests were performed, which showed low C3 levels and negative ANCA titers.

Based on these findings, we suspected glomerulonephritis associated with S. aureus infection and decided not to perform a renal biopsy. Instead, we treated symptoms of generalized edema with diuretic therapy with amiloride and hydrochlorothiazide. Associated proteinuria was also observed on his 24-hour urine protein test. Therefore, we started corticosteroid therapy with prednisolone (1 mg/kg body weight) for 4 weeks, followed by weekly tapering, showing gradual improvement in the patient's proteinuria and edema. Follow-up TTE was performed two weeks later. Despite continued antibiotic treatment, vegetation remained and the size of the vegetation had not changed since the initial inspection. Although he had no fever and repeated negative blood cultures, Vegetation did not respond to antibiotic treatment. He also has unrepaired congenital heart defects.

Therefore,in consensus with our endocarditis team, including a cardiac surgery evaluation, we decided to perform surgical

treatment,vegetectomy, tricuspid valve plasty and VSD patch closure.

The patient’s postoperative course was uneventful and he was discharged 2 weeks after surgery.

DISCUSSION :

Infective endocarditis is a serious risk in patients with congenital heart disease.Mortality measures are considerable despite improvements in antibiotic and prophylactic treatments,. The advent of cross-sectional echocardiography is a major advance. In our patient it confirmed the clinical diagnosis of an interventricular communication and showed right sided vegetation.

Infective endocarditis, caused by S. aureus, has been associated with other common complications, such as nephrotic syndrome and glomerulonephritis [5-6]. Its pathological mechanism is immune-mediated due to the formation of immune complexes and glomerular deposition of C3 complement [5, 7, 6]. For treatment, we decided to administer corticosteroid therapy to treat acute diffuse glomerulonephritis associated with antibiotic therapy with satisfactory results, including reduced proteinuria and progressive improvement in the patient's renal function.

Ventricular septal defects complicated by endocarditis are not hemodynamically important and are usually managed conservatively with prophylactic endocarditis to avoid further

episodes of endocarditis. Small ventricular septal defects of no hemodynamic significance following endocarditis have been reported successfully surgically in several cases [8, 9, 10].

Right-sided IE in patients with unknown ventricular septal defect has relevant clinical implications, from diagnosis to management, intervention, and prevention of further episodes of endocarditis. Acquired ventricular septal defect after tricuspid endocarditis is very rare but has been described as a possible complication [11-12, 13-14]. Patients with known congenital VSD should also be included in appropriate endocarditis prevention guidelines as high-risk patients. In addition, serious complications of right-sided endocarditis, including septic pulmonary embolism, frequently occur with hemoptysis and complicated pneumonia, and may require invasive intervention, intensive care, and mechanical ventilation. Nephrotic syndrome and glomerulonephritis due to acute S. aureus infection are common complications of infective endocarditis [5, 7, 6]. As we described in this case, concurrent antibiotic and corticosteroid therapy may be required to improve proteinuria and renal function.

CONCLUSIONS:

Patients with undiagnosed congenital ventricular septal defects are at high risk for infective endocarditis. Therefore, prophylaxis for endocarditis after dental procedures and/or soft tissue infections is strongly recommended.

Management of complications of right infective endocarditis requires a multidisciplinary approach. Echocardiographic approaches should include screening for VSD in patients without common risk factors for right heart endocarditis including intravenous drug use, intracardiac devices, and central venous

catheters. Surgical management of small ventricular septal defects of no hemodynamic importance remains controversial.

List of Abbreviations :

IE : Infective endocarditis

VSD : Ventricular septal defect

TTE : transthoracic echocardiogram

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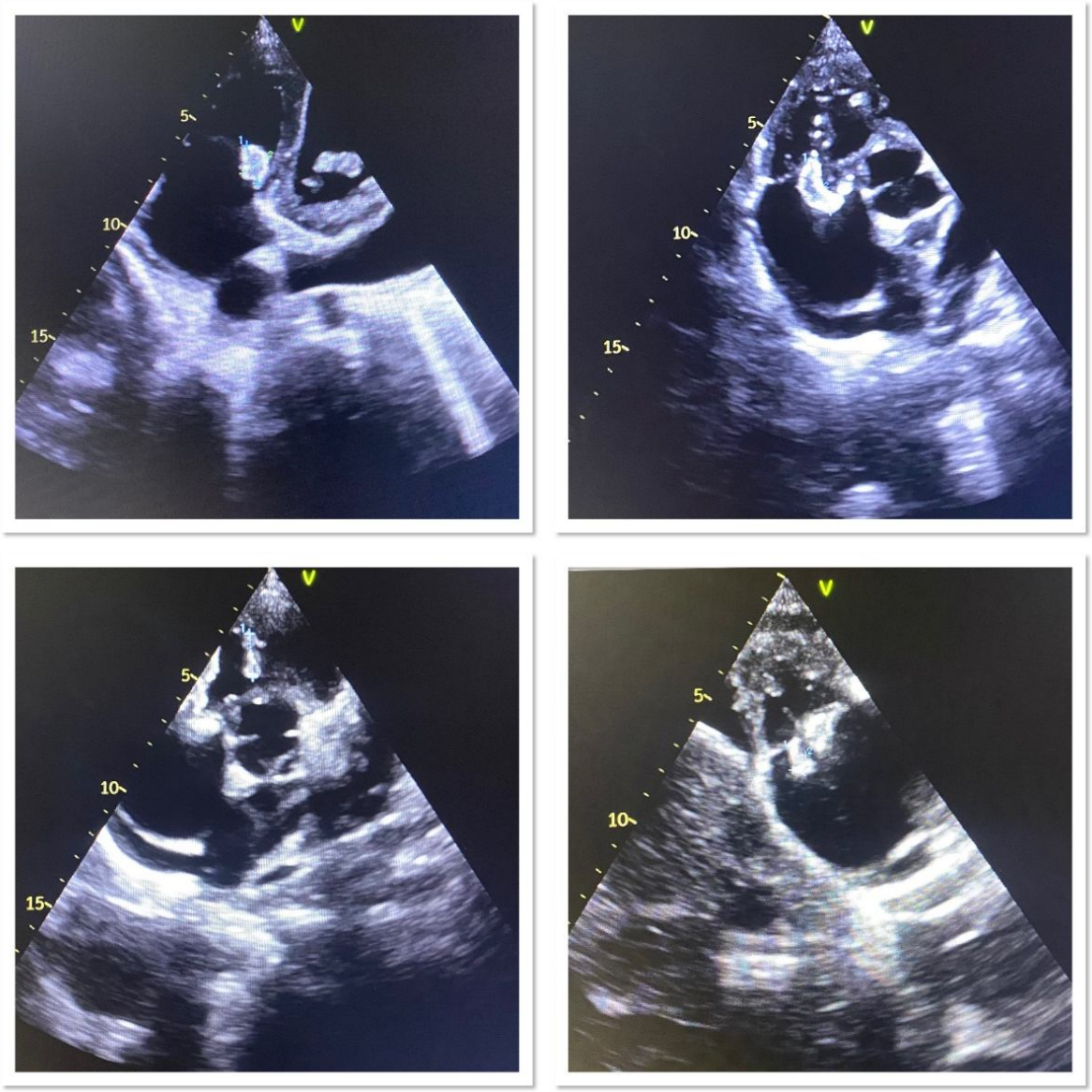

Figure 1 :Transthoracic echocardiography showing vegetations, on the anterior leaflet, on the septal leaflet, and on the posterior leaflet and an other vegetation attached to the chordae tendineae.

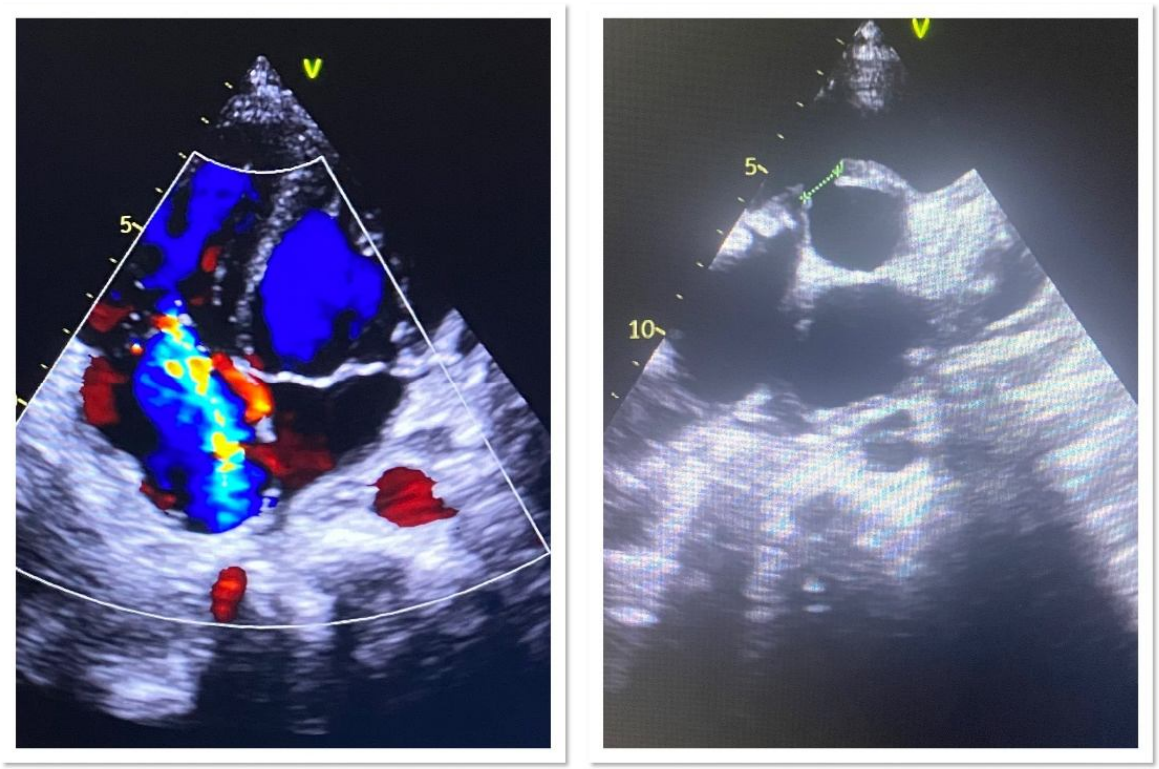

Figure 2: -Moderate tricuspid regurgitation-Sub Aortic ventricular septal defect

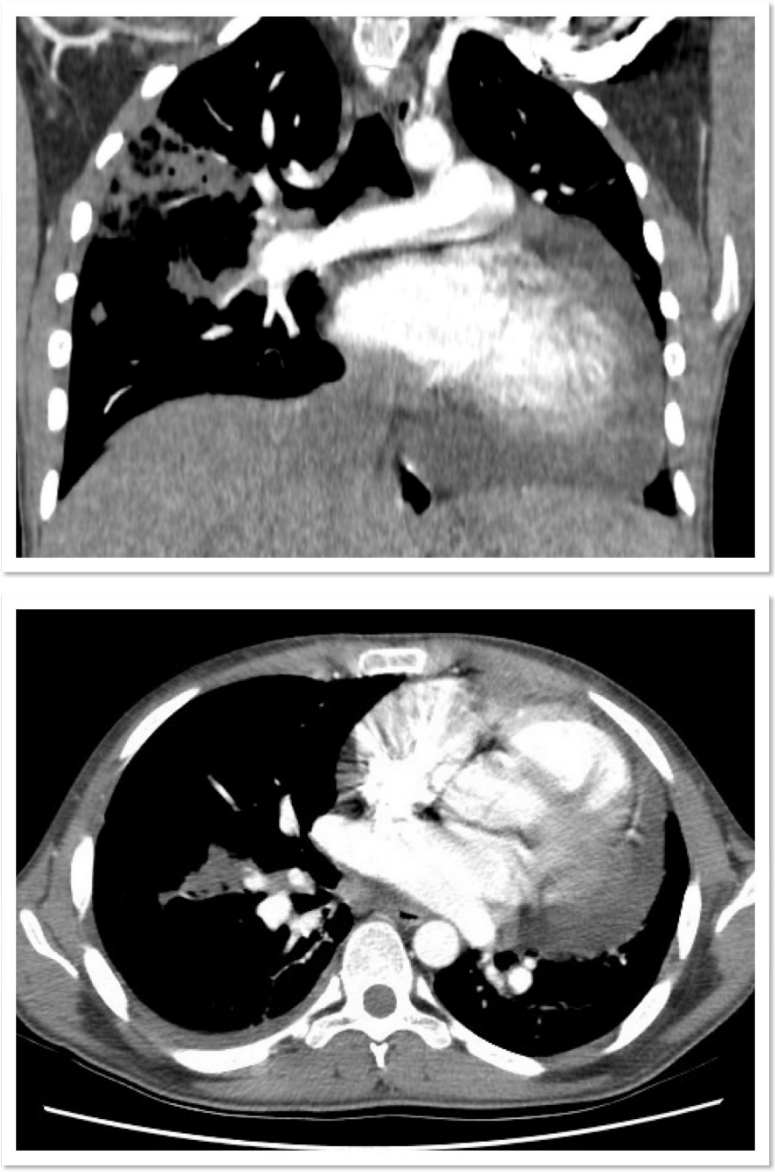

Figure 3 :CT pulmonary angiogram demonstrating septic emboli, and pulmonary infarction