Right-sided Infective Endocarditis with Unknown Ventricular Septal Defect in Adult Complicated by Septic Pulmonary Emboli and Acute Renal Failure: Case Report

ABSTRACT

Introduction: Right-sided infective endocarditis (IE) accounts for 5-10% 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 present the case of a 21-year-old male patient with no previous medical history who was admitted to our department. The patient's admission was due to right-sided Staphylococcus aureus endocarditis, which was complicated by acute renal failure and haemoptysis resulting from multiple pulmonary emboli. Of note, there was an unidentified ventricular septal defect associated with the condition.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 another 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.

Conclusion: Patients with undiagnosed congenital ventricular septal defects are considered to be at a heightened risk for developing infective endocarditis. As a preventive measure, it is strongly recommended to administer prophylaxis for endocarditis following dental procedures and/or in cases of soft tissue infections. This approach aims to reduce the likelihood of bacterial infection reaching the heart and causing endocarditis.

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

ABBREVIATIONS

IE : Infective endocarditis
VSD : Ventricular septal defect
TTE : Transthoracic echocardiogram

1. INTRODUCTION

“Right-sided infective endocarditis (IE) comprises approximately 5% to 10% of all cases of IE. In contrast to left-sided IE, it is more commonly linked to factors such as intravenous drug use, intracardiac devices, and central venous catheters” [1].

“Patients with a congenital ventricular septal defect (VSD) are known to be at a heightened risk of developing infective endocarditis (IE)” [2,3]. Although the occurrence of right heart endocarditis in adults with ventricular septal defects is rare, it can lead to severe complications such as haemoptysis resulting from septic pulmonary embolisms and acute right-heart failure due to tricuspid regurgitation.

Additionally, Staphylococcus aureus infection can give rise to systemic complications, including acute diffuse glomerulonephritis characterized by the formation of immune complexes and the deposition of complement C3 in the glomeruli.

“In most cases, Staphylococcus aureus is determined as pathogens in blood culture, and managing specific antibiotic therapy is conservative” [1,4].

Its management has two components: that of congenital heart disease as well as infectious pathology. Its antibiotic treatment is more effective. Therefore, the timing of its surgical treatment is still controversial.

2. 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.

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

The transthoracic echocardiogram (TTE) showed three mobile structures adhering to the tricuspid valve leaflets, compatible with vegetations, 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 (Fig. 1).

Moderate tricuspid regurgitation was documented, as well as pulmonary hypertension, withpulmonary artery systolic pressure (PASP) of 34 mmHg (Fig. 2), The other valves presented no vegetation or severe regurgitation.A 9 mm sub-aortic VSD with a left-to-right shunt (Fig. 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.

The laboratory tests indicated negative rheumatoid factor, with circulating immune complexes and complement analysis within normal ranges. Serum protein electrophoresis revealed hypoalbuminemia and an inflammatory pattern. Other laboratory parameters, including cardiac markers, did not show any abnormalities.

Following admission, at least two sets of blood cultures confirmed the presence of 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 (Fig. 3).

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.
According to theaboveclinical, 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. Furthermore, the diagnostic urinalysis indicated the presence of macrohematuria, albuminuria, elevated levels of A1 microglobulin, and a high protein-to-creatinine ratio, pointing towards acute tubular injury. Autoantibody testing and C3 complement tests were conducted, revealing reduced C3 levels and negative ANCA titers.

Based on these results, we suspected glomerulonephritis linked to Staphylococcus aureus infection, we opted not to proceed with a renal biopsy. Instead, we focused on managing the patient's symptoms of generalized edema through diuretic therapy using amiloride and hydrochlorothiazide. The patient also exhibited proteinuria, as indicated by the 24-hour urine protein test. Consequently, we initiated corticosteroid therapy with prednisolone at a dosage of 1 mg/kg body weight for a duration of 4 weeks, followed by a gradual tapering on a weekly basis. This treatment approach resulted in progressive improvement of 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.

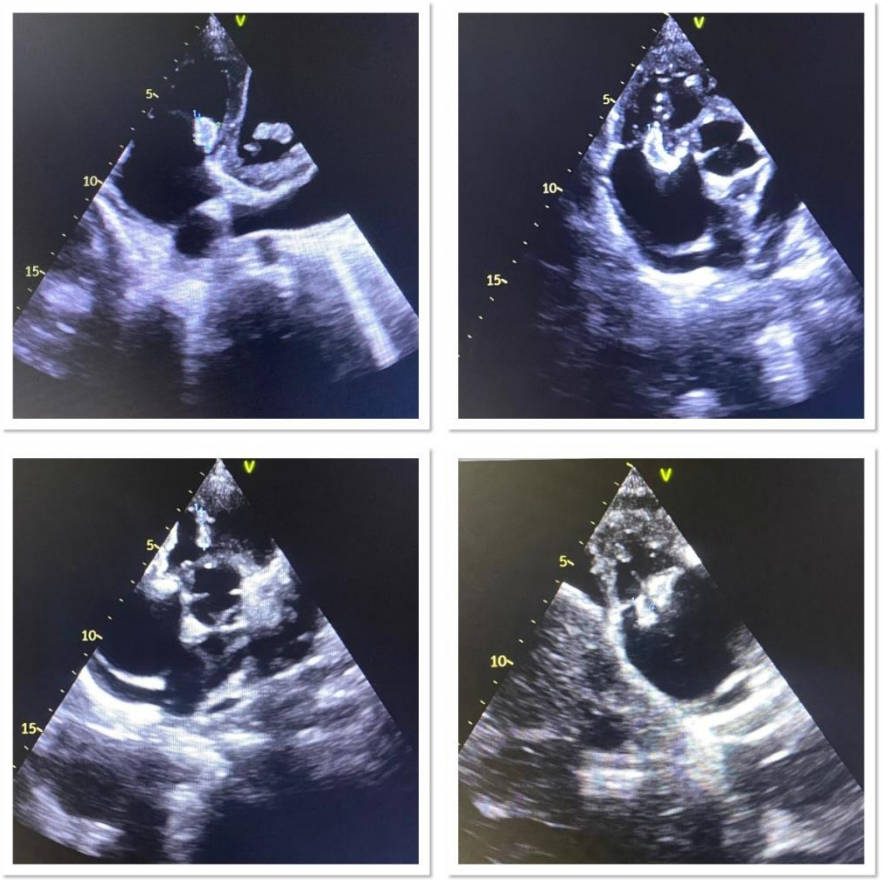

Fig. 1. Transthoracic echocardiography showing vegetations, on the anterior leaflet, on the septal leaflet, and on the posterior leaflet and an other vegetation attached to the chordae tendineae

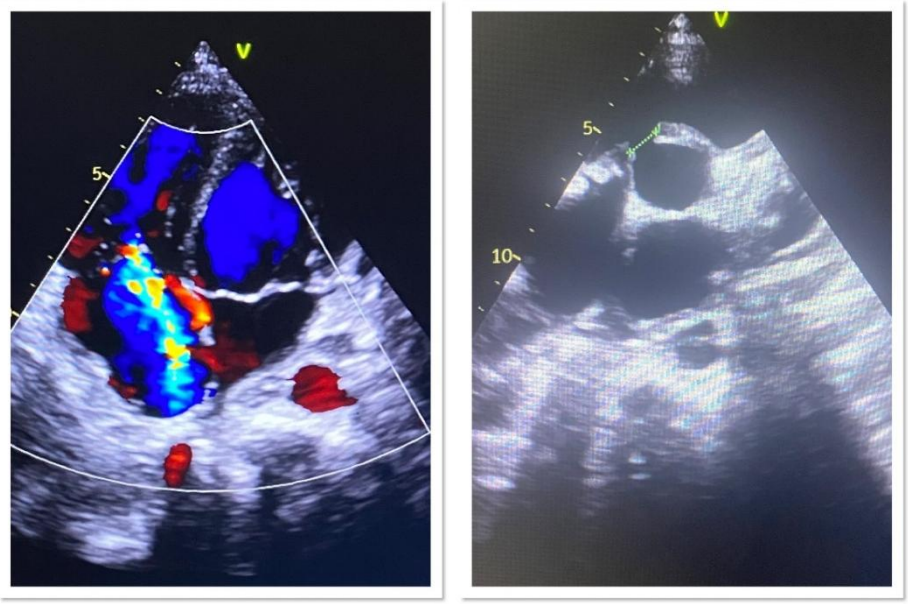

Fig. 2. Moderate tricuspid regurgitation -Sub Aortic ventricular septal defect

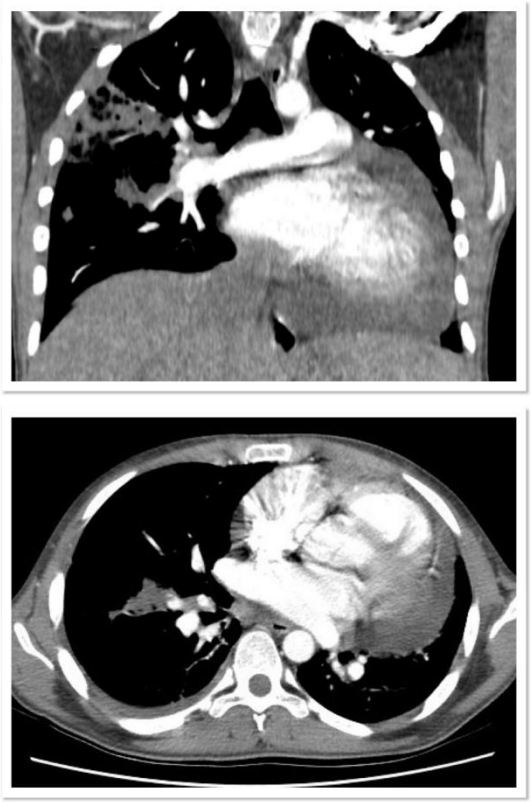

Fig. 3. CT pulmonary angiogram demonstrating septic emboli, and pulmonary infarction

3. 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.

Occurring on a VSD, the infection affects the edges of the VSD and often involves the pulmonary or tricuspid valves. IE of the right heart occurring in adults and sparing the pulmonary and tricuspid valves is a very rare event.

Patients with an unrepaired VSD are at increased risk of IE compared to the general population, this is independent of the size of the VSD, as the turbulence of blood flow through the shunt causes in situ endothelial damage forming by the following a favorable bed of constitution of vegetations. Compared to the attack of the systemic circulation, the IE with VSD affecting the pulmonary circulation is rare; it is generally associated with an attack of the aortic and tricuspid valves in addition to the VSD.
Complications related to infective endocarditis, which is caused by Staphylococcus aureus, may include glomerulonephritis and nephrotic syndrome [5-6]. The mechanism behind this pathology is immune-mediated and involves the formation of immune complexes, as well as the glomerular deposition of C3 complement [5,7,6]. In this case, corticosteroid therapy was chosen as a treatment option to address the acute diffuse glomerulonephritis, in conjunction with antibiotic therapy. This approach yielded satisfactory results, including a reduction in proteinuria and progressive improvement in the patient's renal function.

VSD complicated by endocarditis are not hemodynamically important and are usually managed conservatively with prophylactic endocarditis to avoid further episodes of endocarditis. Small VSD of no hemodynamic significance following endocarditis have been reported successfully surgically in several cases [8-10].

Acquired ventricular septal defect after tricuspid endocarditis is very rare but can be as a possible complication [11-14].
Therapeutic management is essentially based on antibiotic therapy, adapted to the antibiogram. The mortality rate associated with this condition is minimal, and surgical intervention is typically reserved for cases of refractory heart failure, recurrent embolization, or when the infection cannot be adequately controlled through appropriate antibiotic therapy [15]. The latest recommendations on endocarditis in congenital heart disease stipulate that surgical management is not systematic and should be considered in specialized centers[16]. The follow-up is ensured by close echocardiographic monitoring in order to monitor the evolution of the size of the vegetation and the early detection of a possible complication.

4. CONCLUSION

Right sided infective endocarditis with ventricular septal defect is a rare complication and requires a multidisciplinary approach for management of their complications. the indication for surgery is delicate and weighed according to clinical and evolutionary parameters.

Prophylaxis for endocarditis after dental procedures and/or soft tissue infections is recommended for the patients with congenital ventricular defects because they are at high risk for infective endocarditis.

CONSENT

As per international standard or university standard, patient (s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, Del Zotti F, Dulgheru R, El Khoury G, Erba PA, Iung B, et al. Group ESCSD . 2015 ESC guidelines for the management of infective endocarditis: The task force for the management of infective endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the

European Association of Nuclear Medicine (EANM). Eur Heart J. 2015;36:3075–3128.
2. Boils CL. Endocarditis-associated glomerulonephritis. In: Satoskar AA, Nadasdy T, editors. Bacterial infections and the kidney. Cham: Springer. 2017; 87–116.
3. Velebit V, Schöneberger A, Ciaroni S, Bloch A, Maurice J, Christenson JT, et al. Acquired left ventricular-to-right atrial shunt (Gerbode defect) after bacterial endocarditis. Tex Heart Inst J. 1995;22:100–102.
4. Baddour LM, Wilson WR, Bayer AS, Fowler VG Jr, Tleyjeh IM, Rybak MJ, et al. Infective endocarditis in adults: Diagnosis, antimicrobial therapy, and management of complications: A Scientific Statement for Health- care Professionals from the American Heart Association. Circulation. 2015;132:1435–86.
5. Boils CL. Endocarditis-associated glomerulonephritis. In: Satoskar AA, Nadasdy T, editors. Bacterial infections and the kidney. Cham: Springer. 2017; 87–116.
6. Pulik M, Lionnet F, Genet P, Petitdidier C, Vacher B. Immune-complex glomerulonephritis associated with *Staphylococcus aureus* infection of a totally implantable venous device. Support Care Cancer. 1995;3:324–6.
7. Neugarten J, Gallo GR, Baldwin DS. Glomerulonephritis in bacterial endo- carditis. Am J Kidney Dis. 1984;3:371–9.
8. Taskesen T, Prouse AF, Goldberg SL, Gill EA. Gerbode defect: another nail for the 3D transesophagel echo hammer? Int J Cardiovasc Imaging. 2015;31:753–64.
9. Tavakkoli Hosseini M, Quarto C, Bahrami T. Quadruple-valve infec- tive endocarditis and ventricular septal defect. Tex Heart Inst J. 2013;40:209–10.
10. Mellins RB, Cheng G, Ellis K, Jameson AG, Malm JR, Blumenthal S. Ventricular septal defect with shunt from left ventricle to right atrium. Bacterial endocarditis as a complication. Br Heart J. 1964;26:584–91.
11. Velebit V, Schöneberger A, Ciaroni S, Bloch A, Maurice J, Christenson JT, et al. “Acquired” left ventricular-to-right atrial shunt (Gerbode defect) after bacterial endocarditis. Tex Heart Inst J. 1995;22:100–2.
12. Gonçalves AM, Correia A, Falcão LM. Endocardite da válvulatricúspideemdoente com cardiopatiacongénita. Rev Port Cardiol. 2013;32:53–8.
13. Nayyar M, King B, Garg N. When guidelines fail, a case study in infective endocarditis and perimembranous ventral septal defect. J Am Coll Car- diol. 2016;67:1016.
14. Kretzer A, Amhaz H, Nicoara A, Kendall M, Glower D, Jones MM. A case of gerbode ventricular septal defect endocarditis. CASE (Phila). 2018;2:207–9.
15. li W, somerville J. infective endocarditis in the grownup congenital heart (guCH) population. Eur Heart J. 1998;19:166-173.
16. The esC guidelines for the managment of infective endocarditis.eur Heart J. 2015;36:3036-7.