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Case Report

Multivalvular Infective Endocarditis in Double-Chamber Right Ventricle and Ventricular Septal Defect

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ABSTRACT

Infective endocarditis (IE) involving both the aortic and pulmonary valves is uncommon. A young man presented to our department with fatigue, chest pain, and intermittent fevers. His echocardiogram showed multiple large vegetation pieces in the aortic and pulmonary valves, a perimembranous ventricular septal defect (VSD) with a left-to-right shunt, and a double-chamber right ventricle (DCRV). After 3 weeks of antibiotic therapy, surgery was indicated since there was no change in the vegetation size and high risk of embolism. Postoperative echocardiography showed post-repair DCRV with insignificant residual obstruction in mid-RV, no residual VSD shunt, and no residual vegetation. Multiple valvular involvement in IE is rare and is associated with a more complicated clinical course and a higher risk of complications. In this case, perimembranous VSD is a known predisposing factor for IE in the aortic valve, while DCRV is associated with IE development in the pulmonary valve. This case report also emphasizes the importance of early diagnosis, timely antibiotic treatment, and surgery in complicated cases.
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KEYWORDS: Infective endocarditis, Ventricular septal defect, Double-chamber right ventricle

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Infective endocarditis (IE) usually occurs on only 1 heart valve. IE of multiple valves is rare and has a greater risk of complications than that of a single valve. Multivalvular IE has a complicated clinical course with a higher incidence of heart failure and perivalvular complications and often requires surgery and more aggressive treatment. [1]

Congenital heart defects are predisposing factors for IE in the right heart. Among congenital heart diseases, ventricular septal defect (VSD) has a high risk of IE and is the most common congenital heart disease causing IE. The incidence of IE in adult patients with VSD is 1.7–2.7/1000 years, with a 20–30 times higher risk of developing IE than the general population. [2] Double-chamber right ventricle (DCRV) is a rare congenital heart defect (0.5–2.0% of congenital abnormalities) characterized by a mid-cavitary obstruction that divides RV into 2 parts: proximal, which has high pressure, and distal, which has low pressure. [3] DCRV is often accompanied by other cardiac anomalies, with VSD (90% of cases) being the most common. [4] This case report emphasized IE of the aortic and pulmonary

valves in a 21-year-old man with VSD and DCRV.

CASE PRESENTATION

A 21-year-old man came to our hospital with a history of fatigue and intermittent fevers over the past month. The patient also had a history of intermittent chest pain on the right side of the chest 6 months before admission. He had no palpitations or breathing difficulties, nor did he report any dental issues, intravenous drug use, or skin rashes.

On physical examination, the patient was conscious with a blood pressure of 110/70 mm Hg, a heart rate of 80 beats per minute, a respiratory rate of 20 breaths per minute, and a body temperature of 36.1 °C. Oxygen saturation was 97% in the room air. The physical examination revealed a grade 3/6 pansystolic murmur at the lower left sternal border. The electrocardiogram showed a sinus rhythm, a heart rate of 68 beats per minute, a normal axis, and no ST-T changes. Laboratory tests revealed mild leukocytosis. Bacterial culture, performed 4 times, demonstrated no bacterial growth.

Chest X-ray illustrated cardiomegaly with a cardiothoracic index of 0.53 and normal bronchovascular markings. Echocardiography showed a perimembranous VSD with a left-to-right shunt with a defect diameter of 4 mm and a pressure gradient of 63 mm Hg (Fig. 1), turbulent flow in mid-apical RV (pressure gradient = 60-80 mm Hg), dilation in RV (Fig. 2), an oscillating mass on the aortic valve (the right coronary cusp [RCC]) = 14 mm × 12 mm) (Fig. 3), 2 oscillating masses on the pulmonary valve (7 mm × 13 mm and 9 mm × 12 mm) (Fig. 4), mild aortic regurgitation (pressure half-time = 578 ms), mild pulmonary regurgitation (pressure half-time = 208 ms and regurgitant jet width < 1/3 RVOT), and moderate tricuspid regurgitation (Vmax = 4.19 ms and pressure gradient = 70.11 mm Hg). The systolic function of LV and RV was normal, with an ejection fraction of 70% and a tricuspid annular plane systolic excursion value of 2.1 cm.

The patient was diagnosed with possible IE, perimembranous VSD with a left-to-right shunt, and DCRV. He was treated with intravenous ceftriaxone and gentamicin. After 3 weeks of antibiotic therapy, the vegetation size showed no change; therefore, surgery was indicated. Postoperative echocardiography showed post-repair DCRV with insignificant residual obstruction in mid-RV (pressure gradient = 30 mm Hg), no residual VSD shunt, and no vegetation.

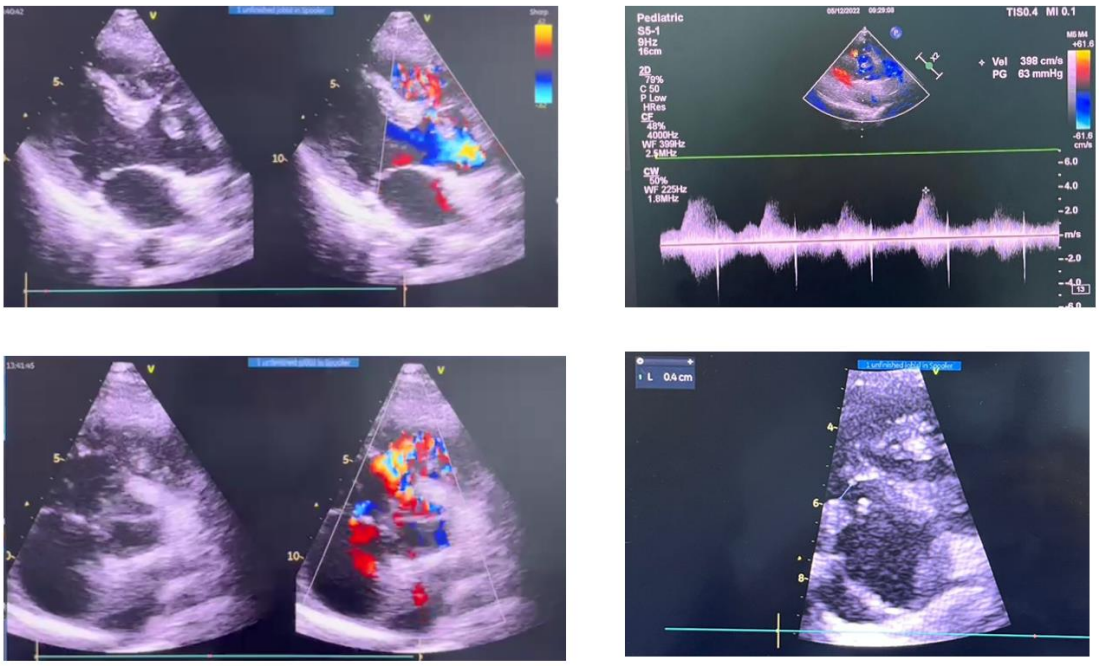

Figure 1: Transthoracic color Doppler echocardiography in the parasternal long- and short-axis views at the aortic level reveal a perimembranous ventricular septal defect with a left-to-right shunt (4 mm) and a pressure gradient of 63 mm Hg.

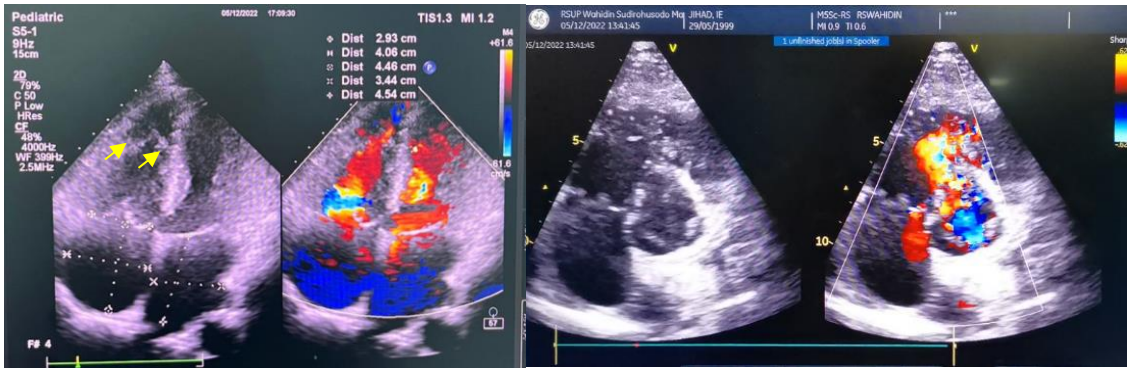

Figure 2: Transthoracic color Doppler echocardiography in the parasternal short-axis view at the aortic level and A4C show anomalous muscle bands (yellow arrows) in the right ventricle, causing obstruction with a pressure gradient of 60–80 mm Hg.

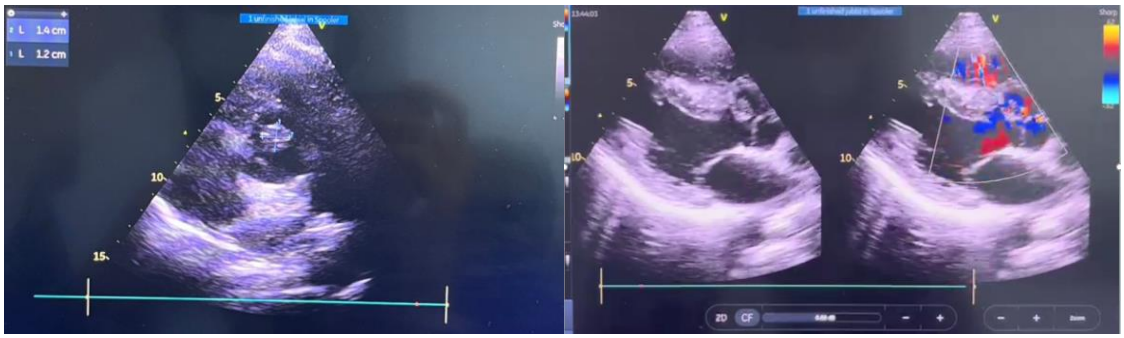

Figure 3: Transthoracic echocardiography in the parasternal long- and short-axis views at the aortic level demonstrate vegetation measuring 14 mm x 12 mm on the right coronary cusp.

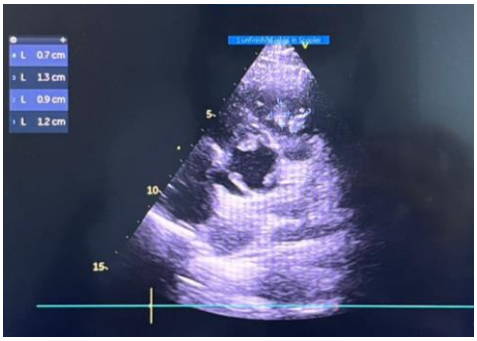

Figure 4: Transthoracic echocardiography in the parasternal short-axis view at the aortic level shows multiple vegetation pieces on the pulmonary valve and the right ventricular outflow tract with a diameter of 7 mm x 13 mm and 9 mm x 12 mm, respectively.

DISCUSSION

IE involving the heart's left and right sides is rare. A retrospective study of the Spanish IE Registry (2008–2020) reported 4064 definite cases of IE, of which 577 (14.2%) had multivalvular IE. Among patients with multivalvular IE, the incidence of bilateral IE involving the left and right heart valves was 12% and associated with congenital heart disease, intracardiac devices, and repeated drug use injections. IE on the pulmonary valve is even rarer than that on the tricuspid valve, accounting for only 2% of endocarditis cases. [5]

The local hemodynamics underlying vegetation development on the aortic valve can be explained by the Bernoulli phenomenon. In small perimembranous VSDs, turbulent blood flow passes through the high-velocity flow, creating negative pressure beneath the RCC. Bacteria cross the aortic valve during the ventricular systolic phase and fall into the RCC due to the negative suction effect of the low-pressure zone created by the high-velocity turbulent jet passing through the restrictive perimembranous VSD. Subsequently, they form vegetation and develop into IE of the RCC, forming relatively larger colonies owing to the low-pressure zone and damaging the valve. The result is aortic regurgitation or even perforated or flail valves. [6]

The mechanism of multivalvular IE on the RVOT and pulmonary valve in our patient was related to intracardiac shunts caused by VSD and DCRV. The condition led to turbulent jet flow, causing shear stress and damaging the endothelial cells of the valve. Endothelial disruption causes fibrin deposition and, consequently, vegetation formation. Turbulent jet flow caused by the perimembranous VSD is associated with vegetation on the aortic valve. In contrast, turbulent jet flow caused by DCRV is associated with vegetation on the pulmonary valve.

Two principal factors in the pathogenesis of IE are damage to the endothelial area and bacteremia. Structural abnormalities in the heart or major arteries with significant pressure gradients or turbulence cause endothelial damage, triggering thrombus formation with sterile deposits consisting of platelets and fibrin (nonbacterial thrombi). Nonbacterial thrombi become the nidus for bacterial adhesion, which then forms infected vegetation. Platelets and fibrin accumulate on top of the organism, causing vegetation enlargement. Organisms trapped in vegetation are protected from phagocytic cells and other immune defense mechanisms and continue to proliferate. When the maximum density of bacteria is reached, most of them become metabolically inactive. [5] Our patient had VSD associated with vegetation on the aortic valve

and DCRV associated with vegetation on the pulmonary valve.

DCRV is a heart defect where RV is divided into 2 chambers, with high pressure in the proximal part and low pressure in the distal portion. The condition can be caused by anomalous muscle bands, endogenous trabeculae hypertrophy, or sometimes by an aberrant moderator band. DCRV is associated with VSD in 90% of cases. DCRV is characterized by an intraventricular pressure gradient exceeding 20 mm Hg and turbulent blood flow in the ventricle. [7, 8]

There are several hypotheses regarding the occurrence of RVOT muscle hypertrophy in VSD. It has been proposed that increased blood flow and pressure in RVOT could trigger hypertrophy in the supraventricular crest in VSD patients with significant pulmonary blood flow. However, some case reports have found infundibular stenosis in patients with VSD who have undergone VSD closure surgery, indicating that muscle hypertrophy may not be influenced by increased pulmonary blood flow. Another hypothesis suggests that infundibular stenosis might be a fundamental part of abnormal growth in VSD and not a secondary adaptation of the heart due to abnormal left-to-right hemodynamics. Infundibular stenosis is thought to occur due to different and complex hypertrophies involving the bulbar musculature. [9]

The position of VSD related to the muscle bundle causing obstruction affects the hemodynamic profile in DCRV. In our patient, a perimembranous VSD was proximal to the abnormal muscle bundle, with significant obstruction of more than 40 mm Hg, indicating the need for surgery.

Concerning IE, there are 3 principal indications for surgery: heart failure, uncontrolled infection, and embolism prevention (vegetation size > 10 mm). Emboli are common and constitute a life-threatening complication associated with vegetation migration (incidence = 20–50%). We considered surgery for our patient since the multiple and mobile vegetation exceeded 10 mm in size, and there was no improvement in clinical condition after antibiotic treatment. Therefore, the patient underwent surgery for vegetation evacuation, VSD closure, and DCRV resection.

CONCLUSIONS

The present case report highlights the complexity of multivalvular IE, particularly in patients with VSD and DCRV. Early diagnosis and prompt treatment with antibiotics and surgical intervention can improve the prognosis of these patients.

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