Original Research Paper



Cardiology

INFECTIVE ENDOCARDITIS OVER THE GERBODE'S DEFECT

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KEYWORDS:

A 15-year-old male presented with symptoms of intermittent fever for past three months which was relieved by paracetamol and did not seek any medical help. Since last month he started having high grade fever which was continuous and without any diurnal variation associated with chills and rigors.

On further eliciting the history, the patient and his attendants revealed that he experienced fatigue on exertion for last 6 years which is relieved by taking rest. There is also history of recurrent respiratory tract infections in childhood. He was shown to a pediatrician and was diagnosed to have some congenital heart disease and was advised to follow up regularly. No medical records were available. No significant birth or maternal history and all developmental milestones achieved as per age.

On examination, he was found to be thin built, looking sick, febrile 101°F, pallor (+), Pulse 110/min, regular, low volume with all peripheral pulsation normal. BP 96/70 mm Hg.

On evaluation of CVS, S1& S2 were normal. There was aortic ejection click, pan systolic murmur of grade iv/iv that was prominent at the left parasternal region radiating to base and apex, medium frequency best heard with diaphragm of stethoscope in supine position. No other murmur heard.

Respiratory system examination revealed occasional fine crepts at both bases and scattered rhonchi.

Abdomen Examination revealed enlarged liver 2 cm, soft, non-tender and spleen palpable just below costal margin. Other system examinations were unremarkable.

Laboratory examinations revealed Hemoglobin-8.3g/dl, total leukocyte count -18100 ul, platelet value-2.26lakhs, ESR 100 mm/hour, urea-34mg/dl, creatinine-1.1mg/dl, sodium level-130mMol/lit, potassium-3.2 mMol/lit, bilirubin(T/D)-0.7/0.2; albumin-3.2mg/dl, alkaline phosphate-89u/l, aspartate transaminase =19u/l, alanine transaminase values = 15u/l, procalcitonin-2.96.

Electrocardiography showed sinus tachycardiawith normal axis, normal QRS duration, no signs of atrial abnormality or right or left ventricular hypertrophy.

Chest roentgenogram showed air space opacities in right middle and left lower zone with clear costophrenic angles bilaterally.

Transthoracic Echocardiography revealed a bicuspid aortic valve, mild AR, mild TR, mild PAH-RVSP=34mmHg, a small restrictive peri membranous VSD with left to right shunt.

Trans Esophageal Echocardiography was also done which revealed peri membranous VSD, 6mm with left-to-right shunt, jet directed into two by septal leaflets into right atrium (LV to RA suggestive of Gerbode's Defect), a freely moving mass/vegetation of size 9X6mm attached to the anterior leaflet of tricuspid valve on atrial side, echogenic appearance suggestive of healed vegetation; another small structure 5x4mm seen attached on the atrial side of septal leaflet of tricuspid valve.

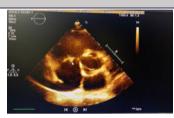


Fig 1: Vegetation at Tricuspid valve in Transesophageal Image

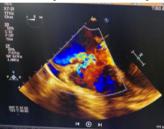


Fig 2: Transesophageal image showing regurgitate jets across VSD into RA $\,$

Following assessment, empirical antimicrobial therapy was initially started after sending blood cultures with the diagnosis of bacterial endocarditis and septic emboli. Further, patient had recurrent fever and breathing difficulty, HRCT chest (Figure 3) was done which revealed multiple randomly distributed nodules in both lungs few with central cavitation.



Fig 3: CT scan image showing multiple randomly distributed nodules in both lungs few with central cavitation and peripheral areas of consolidation in left lower lobe.

Two sets of Blood cultures were positive for methicillin sensitive Staphylococcus aureus. So the final diagnosis of S. aureus bacteremia and isolated right sided endocarditis was made according to modified Duke criteria for endocarditis. Patient was started on appropriate antibiotic based on culture & sensitive report and after 10 days there was complete resolution of fever and repeat Echocardiogram showed diminished size of vegetation 3/4 on the septal leaflet of tricuspid valve without any evidence of further valve lesion. The left to right shunt due to Gerbode defect was stable without hemodynamic significant.

Discussion

Infective endocarditis has a relevant clinical impact due to its high morbidity and mortality. Right sided endocarditis is often associated with several common risk factors such as intravenous drug abuse, a central venous catheters or infections due to implantable cardiac devices1. However, undiagnosed and uncorrected left to right shunt congenital heart defect is also a disposing factors for infective endocarditis especially right sided infective endocarditis which has different clinical manifestation and complication compared to left sided infective endocarditis².

Ventricular Septal Defects (VSDs) with left to right shunts are congenital lesion and are associated with a higher incidence of endocarditis. A congenital shunt from the LV to RA was first described in an autopsy report of a patient in 1838 by Thurnam J.3 In 1958, the defect was named Gerbode after the surgeon who performed a successful surgery on five patients with this anomaly4. The Gerbode defect is a perimembranous VSD with a secondary left ventricular to right atrial shunt. Though often it is congenital, it can be acquired also due to infective endocarditis or as a complication of surgical aortic and / or mitral valve replacement or blunt trauma to the chest5. The Gerbode defect could be a direct (supravalvular) or indirect (infravalvular) connection depending on the involvement of the tricuspid septal leaflet and the presence of additional communication between the left and right ventricle⁵.

Perry EL et al⁶ has classified the Gerbode defect into three types: a Type 1 Gerbode defect consists of a left ventricular to right atrial shunt localized supravalvular to the tricuspid valve. In type 2 Gerbode defects, there is a left ventricular to right ventricular shunt localized infravalvular to the septal leaflet of the tricuspid valve, and due to tricuspid regurgitation, an indirect left ventricular to right atrium communication develops. Type 3 Gerbode defect consist of a combination of both supra and infravalvular communication.

Around 80-90% of infective endocarditis cases results from staphylococcal, streptococcal and enterococcal infection. Staphylococcus aureus is the most frequent microorganism in the right sided infective endocarditis among intravenous drug users, whereas streptococcus viridans is the predominate microorganism in non-drug users⁷.

This case report reflects the importance of astute clinical and imaging examination in patients with IE as fever and septicemia may mask a new shunt formation, and the diagnosis of an acquired heart defects may be missed such as Gerbode's. In such patients, a shunt is created from the LV to RA due to a large pressure gradient that exists between these cardiac chambers. Asymptomatic patients with insignificant intracardiac shunt should be managed with antibiotic therapy as per culture and sensitivity. However, surgical management of IE with acquired Gerbode defect is preferred to ensure effective source control, prevention of embolism, and recurrence of IE.

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