# Complicated Infective Endocarditis (IE) with Large Right Ventricle Vegetation in Congenital Heart Disease (CHD): Managed Conservatively

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***Abstract-*** Congenital cardiac diseases are recognized as a major predisposing substrate for infective endocarditis(IE) worldwide, due to wide spread epidemiological changes observed in last decades in children with infective endocarditis(IE), resulting from increased survival rate of children with congenital heart disease (CHD) due to advancement in surgical interventions together with decreasing incidences of Rheumatic valvular heart disease (RHD) in the pediatric age due to involvement of new modified antibiotics policy, surgical management and enhancement of public awareness. Around 70% of cases of infective endocarditis (IE) occur in children with congenital heart disease (CHD), in whom majority of cases are ventricular septal defect (VSD). Here endothelial damage followed by presence of bacteremia are two important determining factors for Infective endocarditis (IE). These children had some form of underlying hemodynamic instability, such as a pressure gradient or turbulent flow between two cardiac chambers or the great vessels that create shear forces of abnormal high-velocity jet. Thrombogenesis occurred at damaged site leading to formation of nonbacterial thrombotic endocarditis (NBTE). In the presence of bacteremia, bacteria adhere to the NBTE lesion, trapped within the vegetation, protected from phagocytic cells and ultimately forming more enlarging vegetations. Present case of infective endocarditis (IE) is a ventricular septal defect, complicated by infective endocarditis diagnosed on the basis of Duke criteria, had a large vegetation of more than 10 mm in right ventricle managed successfully upon conservative approach, regardless of having surgical indication according to recent American heart association guidelines, indicating surgical interventions for more than 10 mm vegetation. However, the most important measure is prevention, based on good oral hygiene and antibiotic prophylaxis prior to high-risk invasive procedures in susceptible populations.

***Index Terms-*** Congenital heart disease (CHD), Infective endocarditis (IE) ventricular septal defect (VSD), Rheumatic valvular heart disease (RHD), Nonbacterial thrombotic endocarditis (NBTE).

## I. Introduction

Congenital cardiac diseases are still recognized as a major predisposing substrate for infective endocarditis(IE) worldwide. In spite that Infection, Poverty, malnutrition, poor general hygiene and inadequate health care are leading contributors for high prevalence of infective endocarditis (IE) within pediatric age groups specially in developing countries. However wide spread epidemiological changes had been observed during the last decades in children with infective endocarditis(IE), it was happened because of the increased survival rate of children with congenital heart disease (CHD) due to incorporation of advanced surgical interventions as well as decreasing incidences of rheumatic valvular heart disease (RHD) in the pediatric age group with involvement new modified antibiotics policy, surgical management and enhancement of public awareness.

Over all incidence of Infective endocarditis (IE) accounts for 0.5–1 of every 1000 hospital admissions including all congenital heart disease (CHD) patients (excluding postoperative endocarditis)[(1)]. Amongst these ventricular septal defects (VSD), aortic valve abnormalities, patent ductus arteriosus (PDA), or tetrology of Fallot (TOF) continued to be affected in majority of cases[(2)]. Around 70% of cases of infective endocarditis (IE) at pediatric ages occur in children with congenital heart disease (CHD), in whom majority of cases of belongs to ventricular septal defect (VSD)[(3)]. The incidence of infective endocarditis (IE) in children with ventricular septal defect (VSD) was 1–2.4 per 1000 reported[(4)].

Here we report a case of a child presenting with acyanotic CHD [ventricular septal defects] complicated with infective endocarditis with large vegetation of more than 10 mm requiring surgical excision but treated conservatively without any surgical intervention.

## II. Case report

We describe the case of a 5 year-old boy, of lower socioeconomic status, without any known history (there was no record of medical check-ups) or previous diagnosis of a heart defect. He was brought to the emergency department with prolonged history of intermittent fever, coughing, dyspnea, weakness in left side of body and weight loss, with other

symptoms as vomiting and difficulty in walking. General Physical examination showed multiple untreated dental caries, with pallorness, while on systemic examination hepatosplenomegaly and signs of congestive heart failure (CHF) with left sided hemiparesis were observed. On cardiac auscultation revealed a harsh pan systolic murmur at the left sternal border and precordial systolic thrill on bulged precordium. Laboratory tests showed anaemia Hb 5.8 mg/dl and neutrophilic leucocytosis: 22 103/µl, 79% neutrophils, and C-reactive protein 5.6 mg/dl). Transthoracic echocardiography with Doppler study revealed a 6 mm peri-membranous restrictive VSD with moderate regurgitation and a vegetation of more than 10 mm in Right ventricle along the jet of VSD flow with preserved Left ventricular function(figure 1). Presentation of left sided hemiparesis to be showed as suspiciously involvement of central nervous system for which CT angiography of brain was performed, finding was with in normal limit.

Keeping with all above facts in mind along with on the basis of modified Duke s criteria, diagnosis of Right ventricle infective endocarditis with 6 mm peri-membranous restrictive VSD with left sided hemiparesis with anaemia with hepatosplenomegaly with sign of congestive heart failure was made. Subsequently blood culture was taken and empirical intravenous antibiotic therapy was begun with ceftriaxone, vancomycin (30 mg/kg/day) and gentamicin (3 mg/kg/day), together with oral anti-congestive therapy with diuretics (furosemide 1 mg/kg and 25 mg spironolactone every 12 hours) and an angiotensin-converting enzyme inhibitor (enalapril 0.2 mg/kg every 12 hours). Streptococcus orallis was isolated in blood culture collected before antibiotics and it was susceptible to the penicillin, clindamycin, vancomycin, and ceftriaxone antibiotics. The initial clinical course was favourable during the first week of hospital stay with subsidence of fever, improvement in the hemiparesis and congestive heart failure(CHF). However fever was reoccurred during second weeks of hospital stay together with signs and symptoms of congestive heart failure(CHF) then Rifampicin (15 mg/kg/day) was added to the antibiotic regime and diuretic therapy was intensified. Transesophageal echocardiography was performed at this stage still showed right ventricle vegetation of more than 10 mm along the jet of VSD flow. Then child was discussed with cardiothoracic surgeon for surgically resection of vegetation of more than 10 mm, in spite of deterioration of clinical condition while child remained on optimized anti-congestive therapy with modified drugs. But intervention was deferred in view of unstable patient and features of septicaemia with persistent of fever.

Despite addition of Rifampicin in antibiotic regime, persistent of fever was not subsided, and signs of congestive heart failure(CHF) were not improved. Then antibiotics were changed to fourth generation cephalosporin (cefepime), the dosage and form of administration of other therapy was unchanged. Cultures of blood samples collected before the change in antibiotics were negative.

When child was shifted on second antibiotic regime he showed improvement in vitals with subsidence of fever. Repeat echocardiography was performed after 1 week of completion antibiotics still showed right ventricle vegetation but smaller then previously noticed (7mm) along the jet of VSD flow. Antibiotics were continued for total six weeks, (4 weeks with vancomycin gentamicin and five weeks with cefepime, and rifampicin). There was marked improvement in the changes of echocardiography performed after completion modified antibiotics showed disappearance of vegetation in Right ventricle along with improvement in LV function. At that time, the patient is clinically stable without sign of hemiparesis (CNS) and congestive heart failure. The last transthoracic echocardiogram showed absolute normal biventricular function with 6 mm peri-membranous ventricular septal defects (VSD).

## III. Discussion

Infective endocarditis (IE) is not rare but potentially fatal complication among congenital heart disease (CHD) specially in pediatric patients, because it Include 20% in-hospital mortality specifically higher in complicated cases[5]. Association between congenital heart disease (CHD) and infective endocarditis (IE) represents a lifetime risk, as all forms of CHD except ostium secundum atrial septal defect predispose to infective endocarditis (IE). The defects most commonly involved are tetralogy of Fallot, ventricular septal defects(VSD), aortic valve disease, transposition of the great arteries and systemic-pulmonary shunt[2].

There are two important factors in the pathogenesis of Infective endocarditis (IE) first is endothelial damage followed by presence of bacteremia, even if transient. In children with heart disease, have some form of underlying hemodynamic instability, such as a pressure gradient or turbulent flow between two cardiac chambers or the great vessels that create shear forces of abnormal high-velocity jet. This stream of blood (jet) damage the endothelium followed by Thrombogenesis at damaged site along with deposition of sterile clumps of platelets, fibrin, and occasionally red blood cells, leads to formation of nonbacterial thrombotic endocarditis (NBTE)[6]. In the presence of Bacteraemia, bacteria adhere to the NBTE lesion, together with platelets and fibrin results in enlargement of the vegetation. The organisms trapped within the vegetation are protected from phagocytic cells and other host defence mechanisms. As in our patient, one cause of transient bacteraemia is poor oral hygiene and untreated dental caries, whether or not dental procedures are performed along with 6 mm peri-membranous ventricular septal defects (VSD).

Staphylococcus aureus is the most common causal agent in acute infective endocarditis (IE) in developed countries. But Infection by viridans group or alpha-haemolytic streptococci (which includes S. mitis) is also common, particularly in children with dental disease, leading to sub- acute disease, as presented in our case[7]. More recently, there has been a marked increase in the incidence of infective endocarditis (IE) caused by fungi or microorganisms of the HACEK group (Haemophilus, Actinocacillus, cardiobacterium, Eikenella and Kingella) in newborns and immunocompromised patients[2]. Increased incidence of these organisms in this sub population determined by enhancement of survivability due to involvement of advanced technology in their medical as well as in surgical management.

The modified Duke criteria are now the most widely used to diagnosis (infective endocarditis) IE, and it is based on the patient's medical history, physical examination and complementary diagnostic exams, including two or more blood

cultures positive for the microorganisms typical of infective endocarditis (IE) and echocardiographic evidence of endocardial involvement[8].

Initial empirical treatment is anti-staphylococcal penicillin together with an aminoglycoside, effective against the most common microorganisms (S. viridians, S. aureus and Gram-negative bacteria). Antibiotic regime and duration of therapy depends on the etiological agent isolated. Average treatment duration ranges between four and eight weeks [9].

Surgery plays a crucial role in more severe cases, notably when there is congestive heart failure (CHF) refractory to medical therapy or secondary to valve dysfunction, perivalvular abscess or vegetation's larger than 10 mm according to Recent guidelines [9,10,11].

Our patient was discussed with surgical intervention for resection of this large vegetation on Right ventricle but decision was deferred due to unstable patient and features of septicaemia with persistent of fever, later on addition of new antibacterial strategy vegetation was disappeared as own as without any intervention.

Finally the prevention of IE is as important as diagnosis and treatment, because even transient bacteraemia is very important determining factor for infective endocarditis (IE), for which good oral hygiene and regular dental check-ups are essential. The latest guidelines recommend a more rational use of prophylactic antibiotic therapy prior to interventional procedures, limiting their use to patients with predisposing cardiac conditions [8,11].

## IV. Conclusion

Infective endocarditis (IE) at pediatric ages is generally associated with congestive heart disease (CHD). The Duke criteria help in the diagnosis of endocarditis. Antibiotic therapy is the cornerstone of treatment, Depending on the clinical and laboratory responses, antibiotic therapy may require modification and should last for four to eight weeks and be administered intravenously. However, the most important measure is prevention, based on good oral hygiene and antibiotic prophylaxis prior to high-risk invasive procedures.

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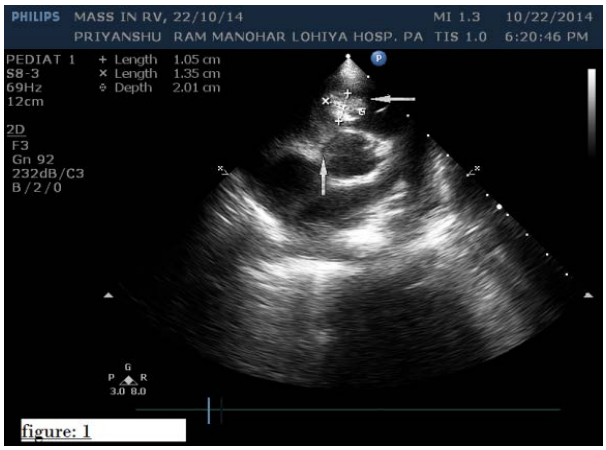
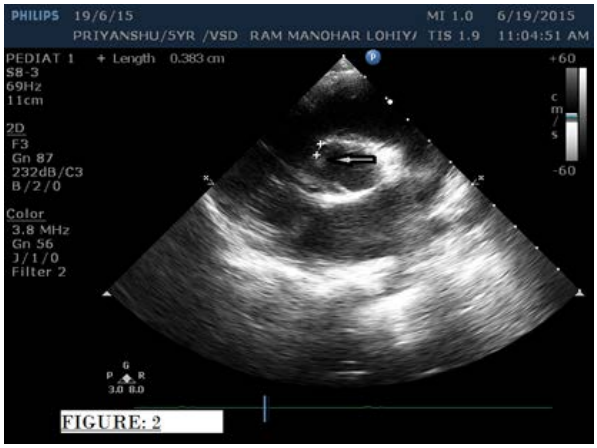
FIGURE




Transesophageal echocardiogram in short-axis view, showing peri-membranous VSD (arrow) with large vegetation (arrow) in a Right ventricle (FIGURE:1) and disappearance of vegetation after treatment (FIGURE:2).

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