

A Rare Presentation of Infective Endocarditis in a Child: A Case Report

*Amar Taksande¹, Gnanvelu Injeti², Rewat Meshram³, Amol Lohakare⁴

¹MD, FIAE, Fellow in Ped Card., Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi Meghe, Wardha, Maharashtra: 442004, India.

²MBBS, Resident, Department of Pediatrics, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi Meghe, Wardha, Maharashtra: 442004, India.

³MD, Associate Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi Meghe, Wardha, Maharashtra: 442004, India.

⁴MBBS, DCH, Senior Resident, Department of Pediatrics, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi Meghe, Wardha, Maharashtra: 442004, India.

Abstract

Infective endocarditis is a rare disease which can lead to serious morbidity and mortality in children if not managed timely. The clinical features of the disease could vary in children and most of its immunological and micro-embolic features are rarely seen. This case report describes the cutaneous and neurological manifestation of infective endocarditis in a child. The case is a 4.5-year-old male child admitted at AVBRH hospital in Sawangi, Central India, with high grade fever and hemiparesis and later diagnosed with infective endocarditis.

Key Words: Child, Fever, India, Infective endocarditis, Janeway lesion, Vegetation.

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*Corresponding Author:

Dr. Amar .M. Taksande, Department of Pediatrics, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi Meghe, Wardha, Maharashtra: 442004, India.

Email: amar.taksande@gmail.com

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1- INTRODUCTION

Infective endocarditis (IE) is an uncommon life-threatening condition in the paediatric community with an annual incidence rate ranging from 0.05 to 0.12 cases per 1000 paediatric admissions (1). Although most of the cases can be traced back to complications of congenital or rheumatic heart disease, the exact cause of IE is unknown. Roughly 10% of cases have no underlying cardiac condition. In children, the clinical features are varied and may include fever, weight loss, heart failure, or complications of septic embolization (2, 3). However, most of the immunological features found in adults are often absent in children. The neurological complications that develop in 20–40% of all patients with IE are mainly due to embolization of vegetations. The diagnosis of IE is crucial in preventing these neurological complications (4, 6). Here, a case of IE with neurocutaneous embolic manifestation is described.

2- CASE REPORTS

A 4.5-year-old male child was admitted to Acharya Vinoba Bhave Rural Hospital in Sawangi, Wardha district, in Central India. The patient presented with high grade, continuous fever without chills for ten days. He had a history of weakness on the left side of his body since 1 day which had been acute at onset, non-progressive, and associated with altered sensorium. The child had no history of convulsions, vomiting, or headache and also no chronic illness. He was born full-term with no complications at birth, and his growth and development were age-appropriate. On admission, he had a temperature of 38.5°C, a heart rate of 118 beats per minute, a blood pressure of 86/56 mmHg, a respiratory rate of 24 breaths/minute, and an oxygen saturation of 98% on room air. On general examination, a number of multiple, discrete, erythematous, haemorrhagic macules were observed on the plantar aspect of soles which were

suggestive of Janeway lesions (**Figure. 1**). On cardiovascular examination, a grade four harsh pansystolic murmur at the lower left sternal border was detected. At the left lower sternal border, a systolic thrill was palpable. On neurological examination, the patient showed ipsilateral supranuclear facial palsy with left-sided hemiplegia. There were no signs of meningeal irritation or sensory deficit. The fundoscopic examination was normal. Respiratory examination showed equal bilateral air entry. The abdomen was soft, non-distended, non-tender, and without hepatosplenomegaly (**Table. 1**).

The patient's chest X-ray showed cardiomegaly (a cardiothoracic ratio of 0.65). Sinus rhythm with right bundle branch block (RBBB) was observed upon electrocardiography (ECG). Sterile blood cultures were sent before starting antibiotics. Transthoracic echocardiogram showed mitral valve vegetation on the anterior leaflets (**Figure. 2**) with severe mitral regurgitation and normal ejection fraction. CT-scan of brain was suggestive of acute infarct in the right fronto-temporo-parietal region. There was no abscess seen on CT images of the brain. The diagnosis of infective endocarditis was confirmed by evidence of endocardial involvement with echocardiographic demonstration of vegetation attached to the mitral valve together with Janeway lesions, stroke, and fever. High-dose ceftriaxone (100 mg/kg/day) with amikacin were administered for four weeks.

Oral decongestive therapy with diuretics (furosemide 1 mg/kg) and an angiotensin-converting enzyme inhibitor (enalapril 0.1 mg/kg) were also added. Abdominal ultrasound was normal with no sign of hepatic, renal, or splenic abscess. A two-dimensional echocardiography was performed twice and showed a decrease in the size of vegetation with moderate mitral regurgitation. The patient recovered completely and skin lesions were totally resolved before discharge.



Fig.1: Non tender, erythematous lesion on the sole.

Table-1: The clinical characteristics.

Laboratory investigations	Values
Haemoglobin	12.3 g/dl
White blood count	31,200 /mm ³
Platelets	1.7lakh/mm ³
Erythrocyte sedimentation rate	130 mm/hour
C-reactive protein	51 mg/dL
Serum sodium	138 mmol/l
Serum potassium	3.5 mmol/l
Bicarbonate	27 mmol/l
Blood urea	14 mg/dL
Serum creatinine	0.7 mg/dL
Anti-Streptolysin O titre	Negative
Antinuclear antibody	0.40
Anti-Deoxyribonuclease B	0.46 (negative)
Urine analysis	Normal. No haematuria



Fig. 2: Long axis parasternal view on echo cardiogram shows a large vegetation on the anterior leaflet of mitral valve.

3- DISCUSSION

Endocarditis was first described by Sir William Osler, a Canadian physician, in 1885. In this disease, the mural endocardium is inflamed with either an infectious or non-infectious aetiology. The morbidity and mortality rate associated with the infection process is significantly high. Usually, rheumatic and/or congenital heart diseases are the complications contributing to infective endocarditis, but it can still be seen in a normal heart. The disease is uncommon in the paediatric community but its incidence has increased over the years. Among the children with a normal heart, it mostly occurs following the use of central venous catheters or in premature children with chronic diseases (6-8). Similar to our case here, about 10% of cases have no underlying heart disease (8). The most common pathogen in acute infective endocarditis is staphylococcus aureus. Subacute infective endocarditis can be present in children with dental problems who are commonly infected with viridans streptococci or alpha-haemolytic streptococci (*S. mitis*).

In immunocompromised people and in new-born babies, there is an increasing incidence of IE due to fungal infection and the HACEK group of bacteria (i.e., haemophilus, actinobacillus, cardiobacterium, eikenella, and kingella) (9). Endothelial damage occurs due to jet waves in the low pressure area adjacent to the valves. In the presence of transient bacteraemia, the region becomes infected which leads to thrombus formation, more bacterial adhesion, and formation of vegetations (8-9). The clinical presentation of IE has been classified as acute and subacute. Acute IE is presented with high fever and rapid deterioration if not recognized timely. In contrast, subacute IE is presented with prolonged low-grade fever for weeks or even months along with other symptoms such as fatigue, chills, myalgia, and weight loss. Development of

a new murmur or an increase in the intensity of the previous murmur in people who already have a heart condition along with cardiac failure can be the sign of endocarditis. Skin lesions characteristic of IE, such as Janeway lesions or Osler's nodes, are uncommon in children. In our patient, Janeway lesions were found in the form of painless, macular, haemorrhagic lesions occurring on the planter surface of the feet (6-7). Cerebral infarction, bacterial meningitis, intracerebral haemorrhage, and mycotic aneurysms are the most recurrent neurological complications seen in patients. Generally, dense hemiplegia with sensory dysfunction is attributed to thromboembolic complications of the middle cerebral artery involving the internal capsule. Thus, 20 to 40% of cases are due to the embolic complications causing ischemic or haemorrhagic cerebrovascular stroke, transient ischemic attacks, cerebral abscess, convulsions, and meningitis (9-10). Among all children with stroke, up to 60% may suffer permanent neurologic deficits and stroke may recur in up to 50% of cases (6-10).

In our patient, the presence of fever, stroke, Janeway lesions, and previously undocumented heart murmur were clear signs suggestive of IE. The modified Duke criteria is used to classify the patients with infective endocarditis. Echocardiography is essential for the diagnosis and monitoring of vegetation size and cardiac function. Electrocardiography can demonstrate the rhythm and conduction disorders such as complete heart block. Prior administration of antibiotics or growth of fastidious organisms can result in a culture negative endocarditis (7). This embolus can drift to various parts of the body like the brain, lungs, spleen, kidneys, the retina or, less commonly, peripheral vessels, causing several complications. Patients usually require a prolonged course (4-6 weeks) of intravenous antibiotics. The antibiotic treatment depends on the

organism isolated and its antibiotic sensitivity. Native cardiac valve IE caused by streptococcus viridans is treated with IV penicillin or ceftriaxone combined with gentamicin. Staphylococcal IE is treated with semisynthetic β -lactamase-resistant penicillin (nafcillin, oxacillin, or methicillin). In cases resistant to medical management or in case of valve dysfunction, perivalvular abscess, vegetation size of more than 10mm, or fungal or Pseudomonas infection, surgical management may be required (10-11).

4- CONCLUSION

Infective endocarditis should be considered during treatment of febrile patients whose cause of fever is unknown. Management of IE in a normal child with a normal heart is different from that of an abnormal heart. Appropriate medical management and surgical intervention have to be undertaken to prevent complications and achieve a better recovery. It is important to suspect endocarditis in young children who present with stroke, peripheral vascular lesion, or transient ischemic attack. Antibiotic therapy is the cornerstone of treatment and should continue for at least four and up to six weeks. However, antibiotic prophylaxis before any procedure is indicated only in high risk populations. Thus, because of acute and complicated nature of the disease, the paediatricians must take great care in its timely diagnosis and proper management.

5- CONFLICT OF INTEREST: None.

6- REFERENCES

1. Baltimore RS, Gewitz M, Baddour LM, Beerman LB, Jackson MA, Lockhart PB. American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee of the Council on Cardiovascular Disease in the Young and the Council on Cardiovascular and Stroke Nursing. Infective Endocarditis in Childhood: 2015 Update: a scientific statement from the American Heart Association. *Circulation*. 2015;132:1487–1515.
2. Lin YT, Hsieh KS, Chen YS, Huang IF, Cheng MF. Infective endocarditis in children without underlying heart disease. *J Microbiol Immunol Infect*. 2013;46(2):121-28.
3. Saiman L, Prince A, Gersony WM. Pediatric infective endocarditis in the modern era. *J Pediatr*. 1993;122(6):847- 53.
4. Yoshinaga M, Niwa K, Niwa A, Ishiwada N, Takahashi H, Echigo S, et al. Risk factors for in hospital mortality during infective endocarditis in patients with congenital heart disease. *Am J Cardiol* 2008;101:114-8.
5. Kiefer TL, Bashore TM. Infective endocarditis: a comprehensive overview. *Rev Cardiovasc Med*. 2012;13(2-3):e105-e120.
6. Wei HH, Wu KG, Sy LB, Chen CJ, Tang RB. Infectious endocarditis in pediatric patients: analysis of 19 cases presenting at a medical center. *J Microbiol Immunol Infect*. 2010;43(5):430-37.
7. Salloum S, Bugnitz CJ. A case report of infective endocarditis in a 10-year-old girl. *Clin Pract*. 2018;8(3):1070.
8. Rosenthal LB, Feja KN, Levasseur SM, Alba LR, Gersony W, Saiman L. The changing epidemiology of pediatric endocarditis at a children's hospital over seven decades. *Pediatr Cardiol*. 2010;31(6):813-20.
9. Stockheim JA, Chadwick EG, Kessler S, et al. Are the Duke criteria superior to the Beth Israel criteria for the diagnosis of infective endocarditis in children?. *Clin Infect Dis*. 1998;27(6):1451-56.
10. Salgado AV. Central Nervous system complications of infective endocarditis. *Stroke*. 1991;26:19–22.
11. Nasser BA, Al Qwae A, Almesned AR, Akhfash A, Mohamad T, Chaikhouni et al. Infective endocarditis in children with normal heart: Indication for surgical intervention. *J Saudi Heart Assoc*. 2019;31(2):51-6.