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RESEARCH ARTICLE

AN UNCOMMON CAUSE OF INFECTIVE ENDOCARDITIS IN A HEALTHY 11-YEAR-OLDGIRL WITH NATIVE HEART VALVE

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Abstract

Infective endocarditis (IE) is a rare disease in children, and it can result in significant morbidity and mortality. The epidemiology of infective endocarditis in children has shifted in recent years with less rheumatic heart disease, more congenital heart disease survival, and increased use of central venous catheters in children with chronic illness. Less commonly, infective endocarditis occurs in children with no preexisting cardiac disease or other known risk factors (1).IE due to anaerobic bacteria is an uncommon event, accounting for 2—16% of all cases of IE over the past three decades (2).We present the case of an 11-year-old girl with no known cardiac disease or significant risk factors, who was diagnosed with infective endocarditis according to the modified Duke criteria. Her clinical presentation included a prolonged fever lasting 2.5 months, along with chest pain and easy fatiguability. Blood cultures identified Anaerococcus prevoti**,** a rare pathogen with limited available data and research.This case underscores the importance of maintaining a high index of suspicion for infective endocarditis in children with prolonged fever, even in the absence of obvious sources of infection or predisposing factors such as cardiac anomalies. Prompt diagnosis is essential for effective treatment and improved patient outcomes.

Introduction:-

Case report:

11-year-old previously healthy girl who presented to the emergency department of Al Jalila Children's Hospital(AJCH) in Dubai in Nov 2023with a history of fever for 2.5 months, with temperatures reaching as high as 39.5°C. The fever was intermittent. She also reported occasional central chest painwith poor weight gain.Her parents observed that she quickly became fatigued even with trivial exerciseFor example, after walking around a mall for a few minutes, she experiences breathlessness and needs to sit down to rest. On a few occasions, her mother noticed central cyanosis, though there were no episodes of syncope.For the past two months, her father mentioned that she has been complaining of lower back pain without any clear history of trauma or physical activity. They consulted several pediatricians, but symptomatic treatments were ineffective.Upon further questioning, the father recalled that 10 days before her illness began, she had visited a dentist for a dental extraction. She has had no exposure to pets, no recent travel history, and has not consumed unpasteurized milk. There was no changes in her appetite or any weight loss.

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She was initially treated with a 10-day course of oral amoxicillin and clavulanic acid at a private hospital, but her symptoms did not improve. Due to the persistence of symptoms, she returned to the same hospital, where an echocardiogram revealed vegetation on the mitral valve (MV) along with mitral regurgitation (MR).There was no family history of congenital heart disease. She was referred to AJCH for further management .On admissionto AJCH she had a temperature of 36.7°C, a pulse of 124 beats/minute, a blood pressure of 99/68 mmHg, a respiratory rate of 22 breaths/ minute, and an oxygen saturation 98% in room air. Her physical examination she looked tired and fatigued.She didn't have any obvious tooth decay. She had no skin rash or petechiae. She was also noted to have clubbing in hand and feet. She had a regular heart rate and rhythm but had systolic murmur 3/6 more at apical and axillary area and much less at bases,no thrill no gallop. Her lungs were clear to auscultation bilaterally, and had soft abdomenwithout hepatosplenomegaly. The rest of her examination was unremarkable.

A complete blood count revealed a normal white blood cell count of 8.4 10^3/uL (5.0 - 13.0 10^3/uL) , haemoglobin 11.6 g/dL (11.5 -15.5g/dl) with platelets 293 10^3/uL (170 - 450 10^3/uL). Electrolytes showed a sodium of 140 (136-145 mmol/L), a potassium of 4.2 (3.5-5.1 mmol/L), blood urea of 19 (15.6-40.6 mg/dL), a creatinine of 0.54 mg/dL (0.52-69 mg/dl), and a calcium of 9.3 mg/dL (8.8-10.8mg/dl). Her C-reactive protein (CRP) was elevated at 8.1 mg/L (normal 0 - 5 mg/L, ESR 36 (Normal 0 - 20 mm/1hr), Procalcitonin was 0.06 normal range < 0.5ng/ml, ASO< 20 (Normal <150 IU/ML), Troponin T was 4 (Normal < 11ng/l), CKMB < 0.3 (Normal 0-3.1), GAS antigen throat negative. Urine analysis was normal. Trans thoracic ECHO was done on the day of admission which showed large vegetation 8 x 14 mm mobile attached to posterior leaflet of the MV and posterior wall of LA. MV anterior leaflet full of Aschoff nodules, AM leaflet not prolapsing.Post leaflet looks prolapsing as vegetation is attached to it. Severe MV regurgitation. Dilated LA, and LVGood cardiac function EF 59%No pericardial effusion.
In the suspicion of IE three blood cultures were collected prior to starting antibiotics and patient was started on Lisinopril, Furosemide,Aspirin, Vancomycin and gentamicin. She became afebrile within 24 hours of admission.
Within 48 hours all 3 peripheral blood cultures came positive for Anaerococcus prevotii. After 48 hours of starting IV antibiotics blood culture was collected again which was also reported positive for Anaerococcus prevotii. Eye examination was done which was normal. After we got the culture sensitivity for the first culture the antibiotics were changed to IV ampicillin and clindamycin.Blood culture collected on day 5 of antibiotics which was reported negative and she was continued on total of IV clindamycin for 16 days and ampicillin for 7 weeks.

Table : 1

Susceptibility

	Anaerococcus prevotii AJCH SUSCEPTIBILITY	
Ampicillin	Susceptible	
Clindamycin	Susceptible	
PIPERA./TAZOBACTAM	Susceptible	
Vancomycin	Susceptible	

ECHO was repeated after 2 weeks of admission showed large vegetation -Thread like measures 25-9 mm in length and 3 mm in width, friable mobile attached to atrial surface of posterior leaflet of the MV -Part of it is Mitral valve leaflet and Chordae. There were also two perforations across posterior leaflets one was big and the other is smallish causing Moderate MR.Following ECHO almost after 3 weeks of admissionshowed persistence of large vegetation, mobile attached to posterior leaflet of the MV and posterior wall of LA. Post leaflet looked prolapsing as vegetation was attached to it and leaflet torn at the level of P2. Severe MV regurgitation, Mild PR, 6 mmhg dilated left atrium and left ventricle. In view of the ECHO findings cardiac surgeons decided to repair the mitral valve and excise the vegetations. Prior to surgery 2 peripheral blood cultures were noted to be negative and patient was given antibiotics for almost 3 weeks prior to surgery.During surgery it was found the main reason of the MV regurgitation was due to a floating segment of the MV posterior leaflet which was covered by an elongated vegetation. There was also a generalized picture of post IE changes on the rest of the mitral valve. These were mostly healed changes with good quality tissues. No vegetations were identified elsewhere. The posterior leaflet had an abnormal short secondary attachment which was also tethering segments of the valve leading to significant degree of regurgitation.The Vegetation was removed fully with the damaged scallop of the MV. Excision of all infected or suspected rims of

tissues from the LA side of the MV. The resultant quadrangular defect on the posterior leaflet was closed. The incised rims of tissue were sent for culture which was reported negative. Following day of surgery, ECHO was repeated which showed successful surgical results,MV well repaired, no MR,no vegetations, no effusion, good - improved cardiac function.She received almost 4 weeks of IV ampicillin after surgery and was continued on aspirin and lisinopril with follow up with pediatric cardiology team. She was discharged home in stable condition.

Figure 1 :

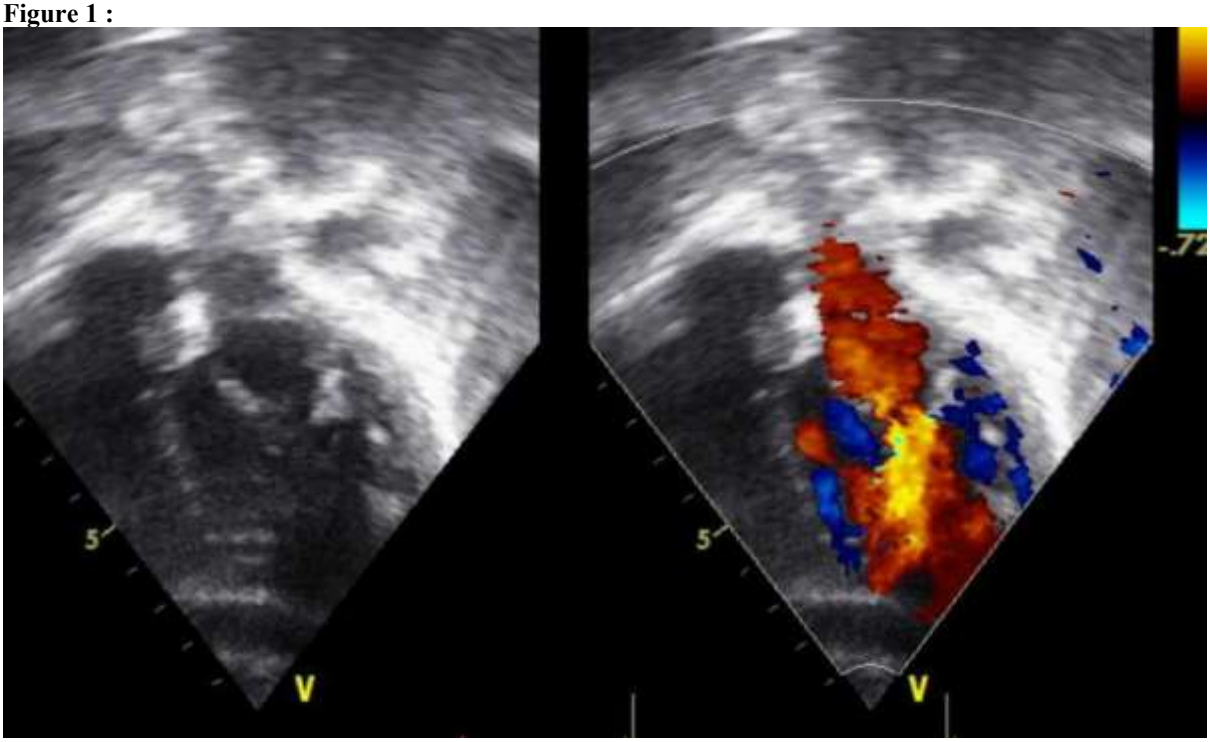

Figure 2:

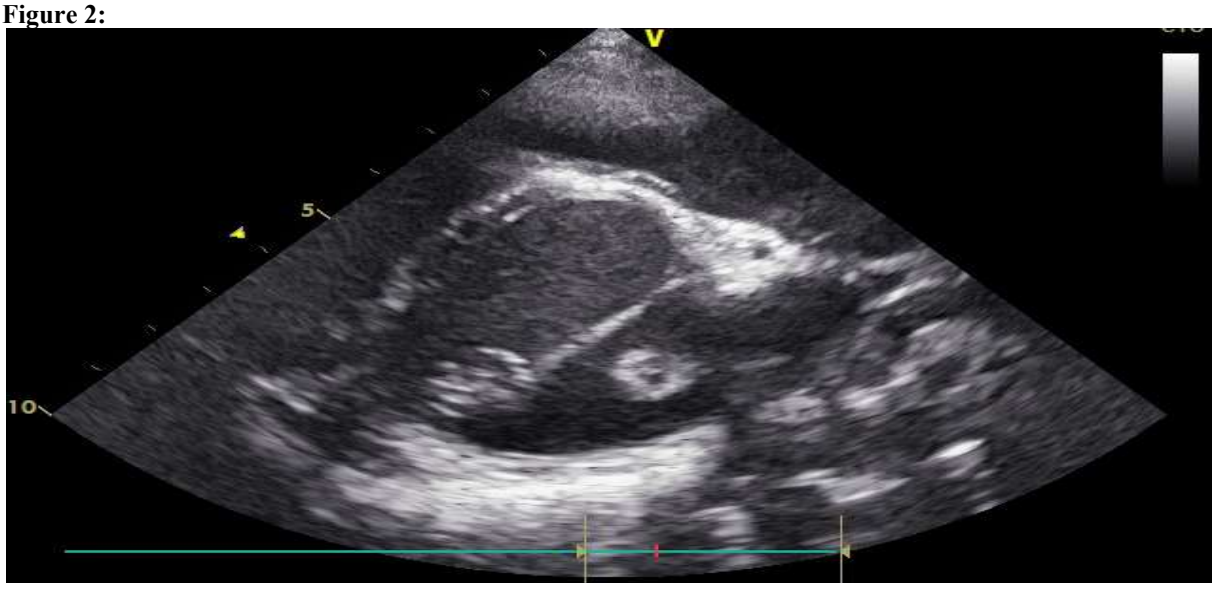

Figure 3:

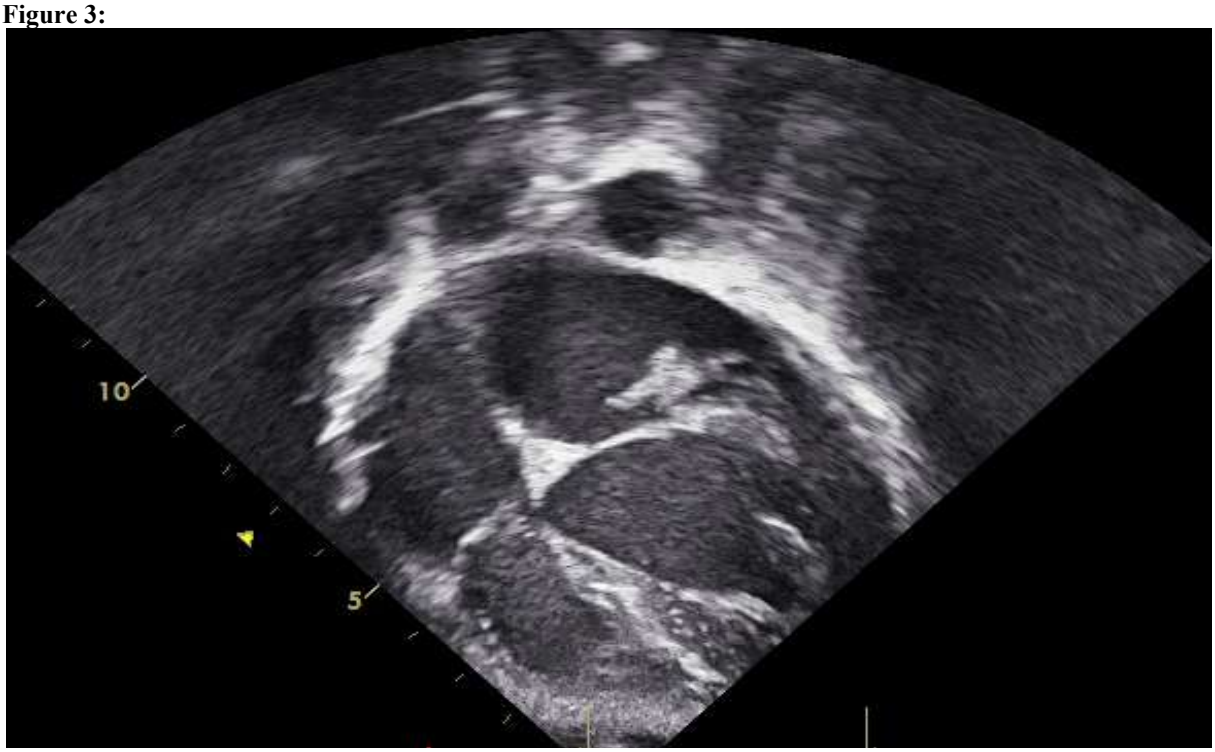

Figure 4:

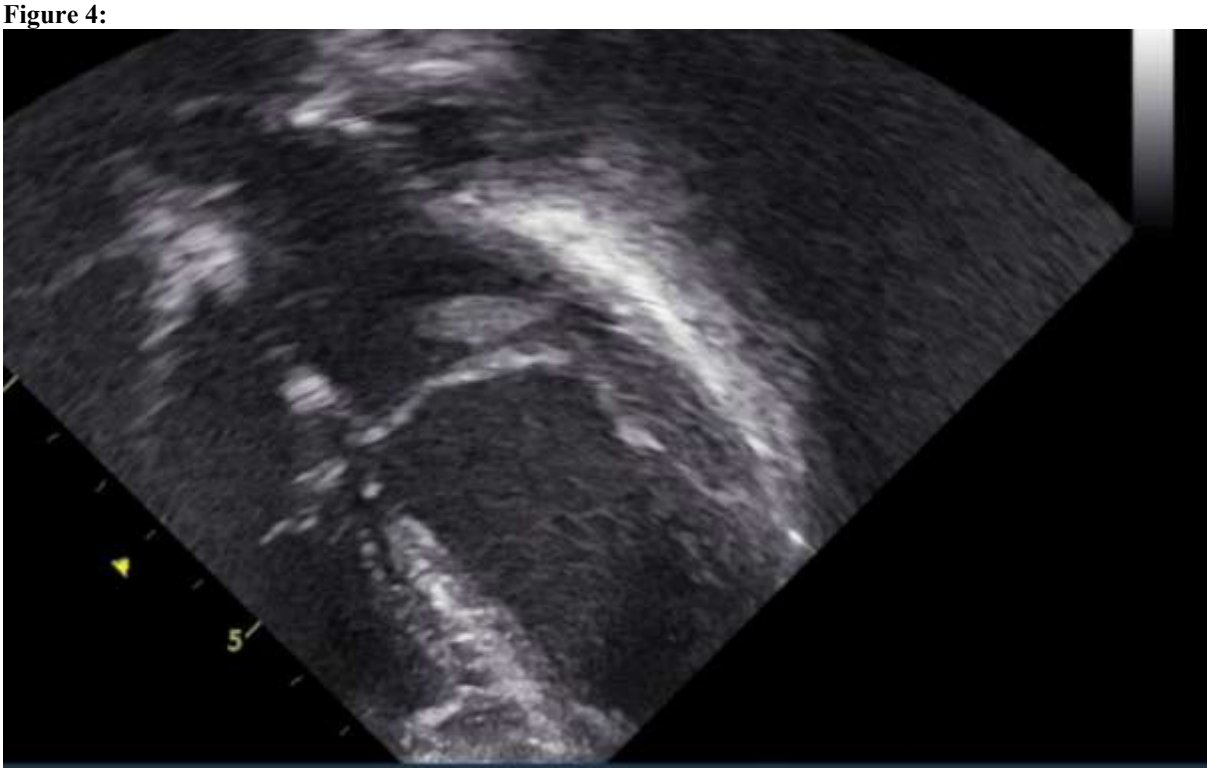

Figure 1- 4 :It shows the echocardiography before the surgery. Showing Large vegetation. It is mobile attached to posterior leaflet of the mitral valve and posterior wall of Left atrium .Mitral valve anterior leaflet full of Aschoff nodules,Post leaflet looks prolapsing as vegetation is attached to it. Severe mitral valve regurgitation.

Figure 5:

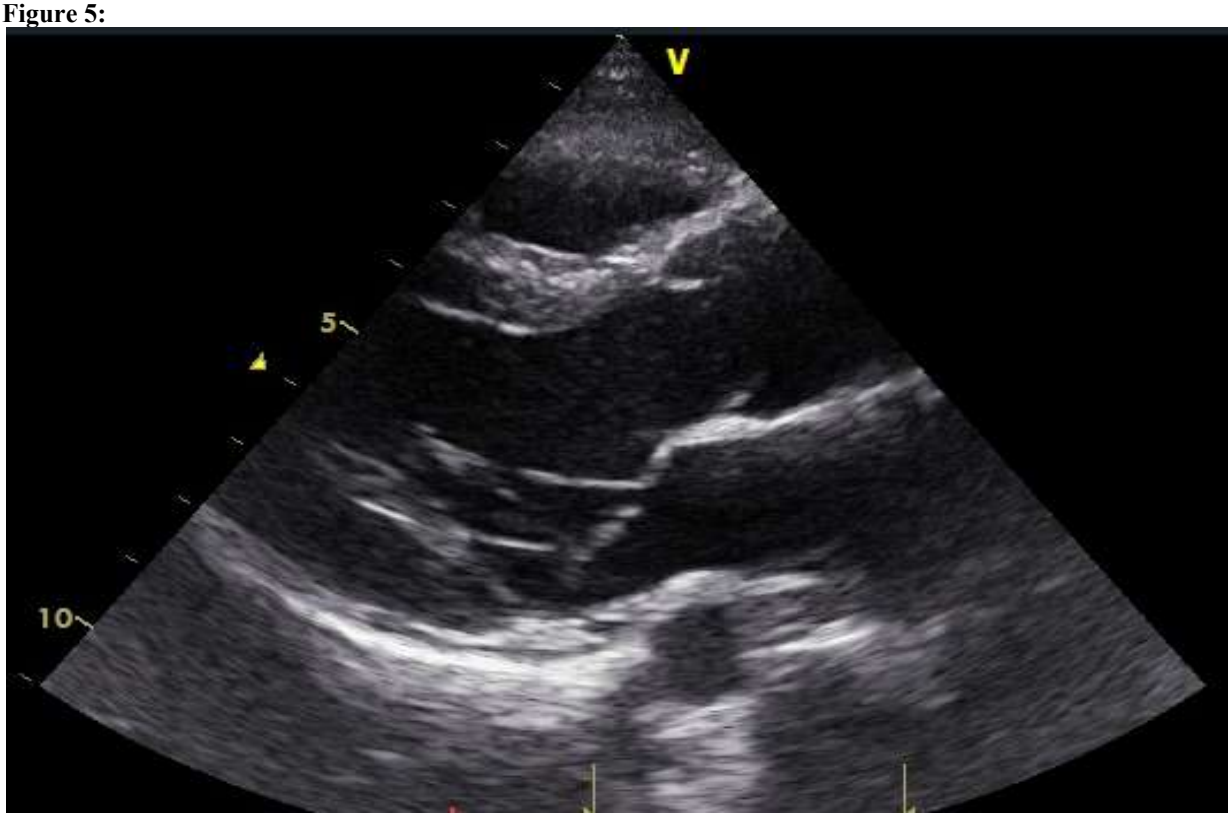

Figure 6:

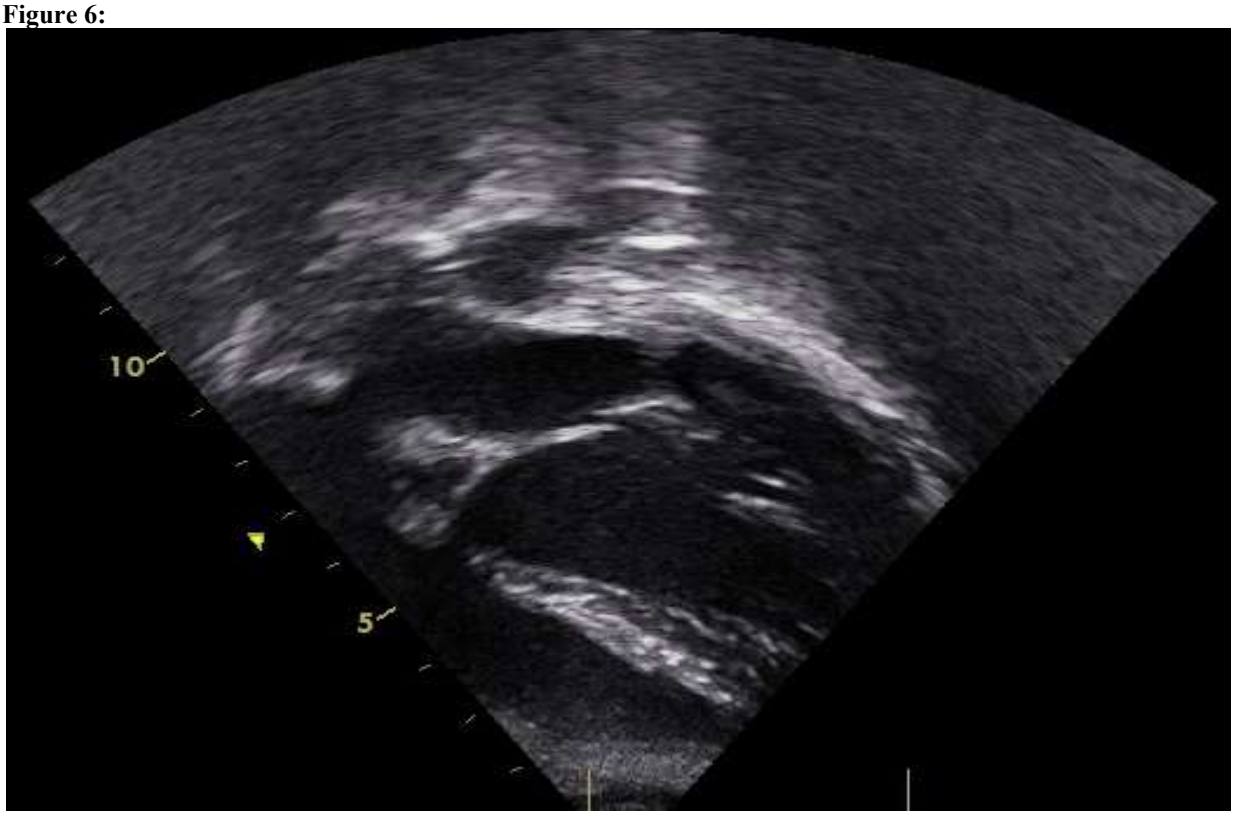

Figure 5-6 shows echocardiography post surgery . It shows mitral valve well repaired, no mitral regurgitation no mitral stenosis, no vegetations,no effusion

Discussion:-

Infective endocarditis refers to the inflammation of the endocardium and the valves of the heart. It is mainly caused by bacterial infections and can present with a variety of symptoms and complications. If not diagnosed and treated promptly, numerous intracardiac and distant extracardiac issues can arise. Thus, a comprehensive evaluationincludin g an in-depth medical history,as pointed out in our caseand physical examination, is essential for diagnosing the condition and guiding treatment to reduce both mortality and morbidity.Infectious endocarditis is a rare condition with an estimated yearly incidence of 3 to 10 cases per 100,000 people.[3] Historically, this disease process has demonstrated a predilection for males, with a male to female ratio of nearly 2 to 1. The average age of infectious endocarditis patients is now greater than 65 years old. This preponderance for the elderly likely corresponds to the increased prevalence of predisposing factors such as prosthetic valves, indwelling cardiac devices, acquired valvular disease, hemodialysis, and diabetes mellitus within this demographic.[4] Although previously a major risk factor, rheumatic heart disease now underlies less than 5% of all cases in the modern antibiotic era. Recreational intravenous drug use represents a growing risk factor that now accounts for about 10% of all infectious endocarditis cases.[5]

Awareness among the family physician and primary physician about this entity is very crucial in early diagnosis and appropriate management.The vast majority of infectious endocarditis cases stem from gram-positive streptococci, staphylococci, and enterococci infection. Together, these three groups account for 80% to 90% of all cases, with Staphylococcus aureus specifically responsible for around 30% of cases in the developed world.[6]Infections due to anaerobic bacteria are common, and can be serious and life-threatening. The recent increase in the recovery of these organisms from all infectious sites [7], including bacteremia [8], has led to greater appreciation of the role anaerobes play in infections at all body sites, including infective endocarditis (IE). Most cases of anaerobic IE are caused by the anaerobic and microaerophilic streptococci, Propionibacterium acnes and B. fragilis [9], [10].

The mortality rate for patients with anaerobic IE is 21–43% [11], [12]Anaerococcus prevotii is a gram-positive, strictly anaerobic bacteria that belongs to genus Anaerococcus. It is part of the commensal human microbiota. However, it has been associated with various infections, including ovarian abscesses, chronic wounds, vaginal discharge, foot ulcers, knee arthritis, urinary tract infections, pleural empyema, blood infections, and soft tissue infections.This bacterium is non-motile and does not form spores.In adults there have been some case reports of Anaerococcus prevotii infections in various clinical cases, including injuries from road traffic accidents (RTAs) and odontogenic brain abscesses.There are no documented pediatric cases of IE due to Anaerococcus prevotii or any other Anaerococcus species in the medical literature. Our patient represents what appears to be the first reported case in a child, reinforcing the need to consider rare and unusual pathogens in febrile children without classic risk factors for endocarditis.

This case further highlights the importance of:

- Maintaining a high index of suspicion for IE in children with prolonged fever.
- Utilizing echocardiography early in the evaluation.
- Ensuring thorough microbiologic workup including multiple blood cultures.
- Considering anaerobic coverage when clinical suspicion is high.

Advanced molecular techniques such as metagenomic sequencing may further assist in diagnosing culture-negative IE, especially in the setting of fastidious or slow-growing organisms such as Anaerococcus spp.

Conclusion:-

This case highlights the complex presentation and management of infective endocarditis (IE) caused by Anaerococcus prevotii in a previously healthy pediatric patient. While Anaerococcus prevotii is part of the commensal microbiota, it is rarely reported as a causative agent of endocarditis, making this case particularly unusual. To our knowledge, there have been no reported cases, particularly in pediatric patients with native valves. Infective endocarditis should always be considered in febrile children, even in the absence of known heart disease or other obvious risk factors. It is important to remain vigilant for even the rarest causative organisms, such as Anaerococcus prevotii, which was identified after nearly 48 hours of incubation in our case.

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