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Case report:

Infective Endocarditis in non-cardiac patients

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Abstract:

Infective endocarditis (IE) is an uncommon but life-threatening infection and features of bacterial endocarditis are due to bacteremia, local cardiac invasion by organisms, peripheral embolization, and the formation of immune complexes. The diagnosis is done based on Duke criteria. Patients who affected by Staph Aureus usually had high mortality rate.

Key words: Infective endocarditis, King Fahad Hospital

THE CASE:

Present illness;

- 5 months old Saudi girl presented to ER with history of lethargy &fever & irritability for 7 days. The fever is high grade intermittent not associated with rigor or sweating , not responded to antipyretic Patient parents sought medical advice in nearby PHC and private clinic, started on PO & IM antibiotics for 5 days but still have

persistent irritability , & fever , so presented to our ER with same complaint at day 7 of illness , with decreased oral intake , and decreased activity. She had history of diarrhea, 2-4 time/day, small in amount, semiliquid, and no blood or mucous, no vomiting. She have disturbed sleep for the last 7 days. No urinary symptoms. No cough – apnea or cyanosis, no history of contact to sick patient (respiratory infection). No history of skin rash or abnormal movement. No history of drinking raw milk. No recent travel.

Mother had history of left axillary abscess drained surgically with antibiotics 2 weeks back prior to the infant illness.

PRENATAL/NATAL /POST NATAL HISTORY

- Baby girl outcome of full term gestation by SVD after uneventful pregnancy to G3P2 mother. Uneventful postnatal period. No history of NICU admission.

PAST MEDICAL HISTORY

- No history of any previous medical or surgical illness. No history of any admission.
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NUTRITION HISTORY

- Breast-fed only started weaning food 2 weeks back & good appetite, & thriving well.

VACCINATION History: up to date.

DEVELOPMENT History*:* Sitting with support, no concern regarding hearing or vision.

FAMILY History: Second-degree consanguinity, other two older siblings are healthy. Educated parents, father is a teacher, mother homemaker both in good health. No history of

early infant deaths in the family or in the relatives. No history of serious infections in siblings or relatives at younger ages. No history of metabolic disorders in relatives.

CLINICAL EXAMINATION:

General Examination; Patient looks unwell, no obvious dysmorphic features, good body built; irritable, uncomfortable on bed, skin mottling, not pale , Not cyanosed or jaundiced , no skin rash.

Vital signs; Temp: 38.9 C°, RR; 40 breath /min., HR: 185 beat/min, BP: 109 /60, O2 Sat: 92% room air; Weight.:6.3 kg RBS; 88 mg/dl.

INITIAL SYSTEMIC EXAMINATION;

- Chest: Equal bilateral air entry , no added sound
- CVS: Warm extremities, hemodynamically stable, Normal S1 + S2
- Abdomen: Soft , Lax , liver 2 cm below costal margin, No hepatosplenomegaly
- CNS: Fully Conscious , alert ,irritable, no abnormal movement, anterior fontanel at level, GCS 15/15, intact cranial nerves ,normal motor system.
- Genital; normal. Female genital.

INITIAL INVESTIGATIONS;

- CBC: Initial WBC: 13000 (Neutrophil: 55. 7 %, lymphocyte: 23. 5 Hb : 12.3gm/l /, PLT : 60,000 , PT : 17 sec., /PTT: 35.7 sec. / INR 1.5 , RETICS: 1.5, ESR =26/1st hour, CRP was positive
- 6 hours after admission PLT. Count drops to 34000 Peripheral smear showed Toxic granulation, scanty plat.; sepsis picture.
- Chemistry: Na: 143 mmol/l, K: 4 mmol/l, Ca: 2 mmol/l PO4: 1.7mmol/l, Urea: 5.9 mmol/l, Creatinine: 21 mmol/l,

- Liver Function test: AST: 52.2 mmol/ l, ALT ; 27 mmol/ l, Serum Albumen: 28.5gm/l
- Complete Septic screen done except LP, which postponed because low platelets count.
- Urine analysis was normal.
- Immunoglobulin assay showed low IgA & IgM but IgG was lower limit of normal

INITIAL TREATMENT

- Started IV Fluid D5%1/2 NS as maintenance with revaluation after 4 hours.
- Covered with IV antibiotics ; Vancomycin & ceftriaxone in meningitis doses

COURSE OF ILLNESS

- Patient on close observation, reexamined again at 9 AM, and discovered to have pan systolic murmur in left lower sternal area as new finding.
- Urgent Echocardiogram done by pediatric Cardiologist consultant On 21 \ 09 \ 2017 & showed ;

1. Situs solitus, levocardia.
2. AV\VA concordance, PFO, SVC, IVC >>RA , PV>>LA
3. Intact interventricular septum, good LV, RV function, EF = 72 %. no PDA , No PHE, normal aortic arch and pulmonary veins ,
4. 4 cm in size (appearance of vegetation by ECHO), Minimal anterior pericardial effusion.
5. Moderate tricuspid regurgitations, PG = 40 – 45 mmHg There is two hypodense masses in septal and anterior leaflets of tricuspid valves , mobile , o.5 cm- 5cm.

- Send another 2 blood C&S 12 hours apart.
- Started Vancomycin & Ceftriaxone in doses for Acute Endocarditis.

- 2nd day the child deteriorate vitally so transferred to pediatric intensive unit
- VBG showed metabolic acidosis ; PH 7.26 , HCO3 16
- Blood culture was positive for Staph Aureus in the 1st 3 samples.
- Unfortunately the baby deteriorate more early morning 3rd day & expired.

INFECTIVE ENDOCARDITIS IN CHILDREN

Introduction

Infective endocarditis (IE) is an uncommon but life-threatening infection. Despite advances in diagnosis, antimicrobial therapy, surgical techniques, and management of complications, patients with IE still have high morbidity and mortality rates related to this condition.(1)

Features of bacterial endocarditis are due to bacteremia, local cardiac invasion by organisms, peripheral embolization, and the formation of immune complexes.

High-velocity flow through a stenotic or incompetent valve or an abnormal communication between systemic and pulmonary circulations causes turbulence at the valve, within the communication, or downstream where the flow eddies. This turbulence damages or denudes the endothelium, to which platelets and fibrin can adhere, and a small, sterile nonbacterial thrombotic endocardial lesion forms.

In addition, indwelling intravascular catheters may directly traumatize the endocardium or valvar endothelium. Circulating bacteria and inflammatory cells adhere to and grow in these thrombi, forming an infected vegetation. Once vegetation forms, the constant blood flow may result in embolization to virtually any organ in the body. A brisk immunologic response is produced. (2)

Features of bacterial endocarditis are due to bacteremia, local cardiac invasion by organisms, peripheral embolization, and the formation of immune complexes.[3]

Diagnostic criteria [4)]

Modified Duke criteria for diagnosis of IE: clinical criteria for definite IE requires two major criteria, one major and three minor criteria, or five minor criteria.

Major criteria;

- Positive blood culture for IE: typical microorganism consistent with IE from two separate blood cultures
- Evidence of endocardial involvement :
    - Positive echocardiogram for IE:
    - Oscillating intra cardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomical explanation; or
    - Abscess; or
    - New partial dehiscence of prosthetic valve); or
    - New valvar regurgitation (worsening or changing of pre-existing murmur not sufficient).

Minor criteria;

- Predisposition: predisposing heart condition or intravenous drug use.
- Fever: temperature >38°C.
- Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial haemorrhage, conjunctival haemorrhages and Janeway's lesions.
- Immunological phenomena: glomerulonephritis, Osler's nodes, Roth's spots and rheumatoid factor.
- Microbiological phenomena: positive blood culture but does not meet a major criterion as noted above or

serological evidence of active infection with organism consistent with IE.

- PCR: broad-range PCR of 16S (polymerase chain reaction using broad-range primers targeting the bacterial DNA that codes for the 16S ribosomal subunit).
- Echocardiographic findings consistent with IE but do not meet a major criterion as noted above.

Blood culture-negative IE (BCNIE) refers to IE in which no causative micro-organism can be grown using the usual blood culture methods. BCNIE can occur in up to 31% of all cases of IE and most commonly arises as a consequence of previous antibiotic administration[5].

Antibiotics remain the mainstay of treatment for IE.In the setting of acute IE, institute antibiotic therapy as soon as possible to minimize valvar damage. Three to 5 sets of blood cultures are obtained within 60-90 minutes, followed by the infusion of the appropriate antibiotic regimen. By necessity, the initial antibiotic choice is empiric in nature, determined by clinical history and physical examination findings.

Empiric antibiotic therapy is chosen based on the most likely infecting organisms.[6]

DISCUSSION

To our knowledge, this is the first reported case of definite infective endocarditis based on Duke Criteria in-patient with non-cardiac lesion and follow sever septicemia (i.e blood born septicemia).

Study done in Saudi Arabia (1995-2008) by Jaffar A. Al-Tewfik and Ismail Sufi [7] showed 65% of their patients had definite endocarditis and 35% were possible endocarditis based on Duke Criteria.

Our patient blood culture showed Staph Aureus growth in the three samples taken and ECHO cardiograph also shows vegetation.

The International Collaboration on Endocarditis-Merged Database (ICE-MD) also showed that of all patients with native valve endocarditis, 34% had *S aureus* infection.[8].in Jaffar A.AL-Tawfig Sufi# study Staph Aureus was found in 42.3% of the study group. However study from Kuwait in 1985-1988 where *Streptococcus Viridans* was the commonest infective organism.[9]. Another study from Lebanon showed that 51% of endocarditis cases were due to *Streptococcus* spp.[10]. However, the difference may related to the difference in the time of the study.

Mortality following Staph Aureus is high in most cases. In contrast, in a large study of *S aureus* endocarditis, patients with *S aureus* were more likely to die (20% vs. 12%) than other patients.[11]. In the International Collaboration on Endocarditis–Prospective Cohort Study, in-hospital mortality was 17.7 %.[12]. In another study, *S aureus* IE was associated with a higher 1-year mortality rate (43.9% vs. 32.5%; *P*=.04).[13]. Other studies showed a high mortality rate of *S aureus* endocarditis of at least 40 %.[14].However, most recent studies still show a high mortality rate, approaching 34%..

The limitation of this case that it's a one case report also this make us to put IE in our mind even no cardiac lesion in patients.

In CONCLUSION:

IE remains a very serious disease that follows sepsis and it carries very high mortality and morbidity rate.

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