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Purulent Meningitis as an Unusual Presentation of Staphylococcus Aureus Endocarditis in A 10 Year Child : A Case Report

KEYWORDS: Endocarditis, cutaneous vasculitis, meningococcal septicemia, septic shock

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ABSTRACT *On presentation of Staphylococcus aureus endocarditis, unusual manifestations may represent the main clinical features of the disease. Isolated bacterial meningitis as the first manifestation of endocarditis is considered to be an unusual neurological complication. Here, we describe a case S. aureus endocarditis presenting as isolated meningitis and mimicking meningococcal septicaemia with cutaneous vasculitus with septic shock .This case is reported because of diagnostic challenge and how recognition of this infectious syndrome is of crucial importance for the correct management of patients.*

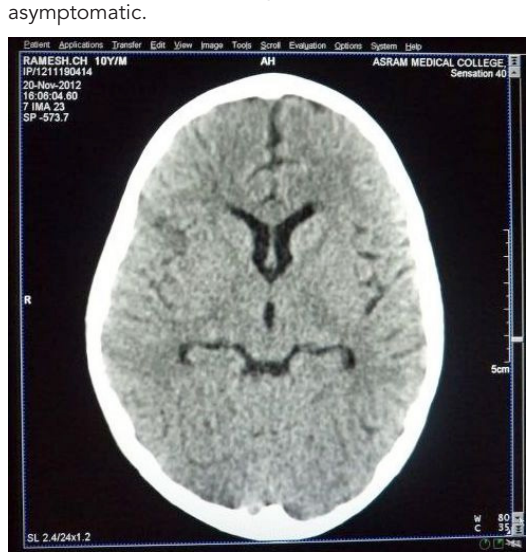
Introduction:

Staphylococcus aureus is a leading cause of bacteraemia and endocarditis, but also a rare cause of bacterial meningitis. The mortality rate of S. aureus endocarditis is approximately 20–40%, depending on the extreme variability in clinical presentation which may delay the early diagnosis and treatment of the disease[1-3]. Therefore, early diagnosis and adequate monitoring of the various complications due to S. aureus endocarditis are important. In this paper, we describe a case of S. aureus endocarditis presenting as isolated meningitis and mimicking meningococcal septicaemia

Case report:

10yr male child came to hospital with complaints of fever for 7 days, head ache and altered sensorium 2days.There was no h/o seizure.At admission child was in septic shock, so managed with fluid bolus followed by dopamine infusion@ 10microgm/kg/min. On examination child was irritable with GCS of 13/15,pupils were bilateral equal and reacting to light, neck stiffness was present, no cranial nerve deficit, no motor deficit were present ,bowel, bladder were normal. Eyes were congested, bluish black discolouration of right lower limb great toe and both palmar aspect of hands. So possibility of meningococcal meningitis with septic shock was kept and treated with inj ceftriaxone1,2gm iv bd,inj vancomycin 350mg iv 6th hrly,dexamethasone3.6mg iv 6th hrly for 2 days. Initial investigations were Hb- 8.9,TLC- 18200,N86%,L7%,total platelet count-75000,peripheral smear showing microcytic hypochromic anemia with neutrophilic leukocytosis with thrombocytopenia, MP smear negative,PT 19/14 with INR 1.5,APTT 45 s (normal 20-34),D-dimer was positive,urea 23mg/dl,creatinine 0.5mg/dl,bilirubin 0.8mg/dl,SGOT 105 IU/L,SGPT 60 IU/L,ALP 320IU/L,Serum Na 140meq/L,K 4.9 meq/L.Ca8.9mg/dl,RBS 98mg/dl.Urine r/m showed microscopic hematuria. After stabilization and recovery of shock with correction of coagulopathy CSF study was done. CS-Freport was 04 cells ,all are lymphocytes, sugar 61mg/dl, protein 76mg/dl ADA 2.5IU/,csf c/s sterile .CT scan brain reported as subtle hypodense lesion in left lateral thalamus. EEG was normal. Doppler USG was ordered for peripheral vasculitis,which suggested thrombosis of right cephalic vein with upper limb lower limb Doppler being normal.

On day 3 of illness, his sensorium improved but fever persisted and physical examinations revealed splinter hemorrhage in lower limb great toes. On CVS examination ejection systolic murmur of grade 3/5 was heard over mitral area.On reviewing history there was no past h/o breathlessness,joint pain ,sorethroat or any abnormal movement. With doubt of infective endocarditis 2D-Echo was done which revealed vegetation over AML(size 0.9x0.7cm) with moderate Mitral regurgitation with normal biventricular function .Three set of Blood c/s were sent. Out of 3 c/s ,one blood c/s suggested of staphylococcus aureus sensitive to erythromycin, clindamycin, linezolid and resistant to vancomycin. According to report linezolid was added and continued for 6weeks.ASO was negative, CRP was positive, ESR 60mm/1hr.OPthalmological examination revealed roth spot in both eyes.Final diagnosis was kept as native valve endocarditis with Mitral regurgitation with embolic phenomenon presenting as meningitis with septic shock and DIC .Child became afebrile after 4 days of linezolid therapy. On discharge and follow up child there was resolution of vegetations and child was clinically asymptomatic.



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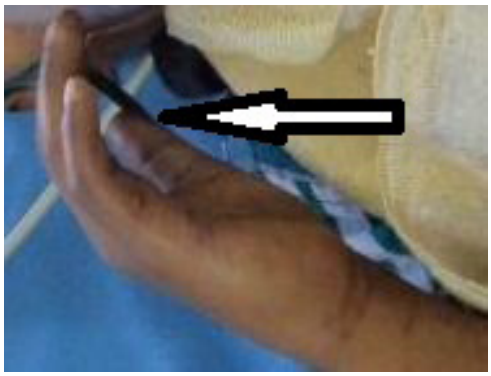

Fig 1: CT scan brain showing subtle hypodense lesion in left lateral thalamus

Discussion

Serious S. aureus infections caused by both resistant and susceptible strains are increasingly reported in community and hospital settings [4]. In particular, infective endocarditis represents the most serious S. aureus bacteraemia complication. S. aureus is a unique pathogen because of its ability to cause endocarditis often involving previously architecturally normal cardiac valves. S. aureus endocarditis also can have profound and devastating neurologic consequences. The frequency of neurological complications due to endocarditis was found to remain constant despite therapeutic advances and profound epidemiological changes[5]. The incidence of neurologic complications of infective endocarditis is dependent on the organism and valvular location; the highest incidence is 87% with S. aureus vegetations on the mitral valve. Jorge et al. described that patients with concomitant S. aureus meningitis and endocarditis had a higher mortality (56%) related to age, shock, and infection with phage type 95 strains [6]. Fong and Ranalli reported that meningitis associated with endocarditis caused by S. aureus showed a more fulminant course [7].

Spontaneous S. aureus meningitis as a consequence of endocarditis may pass unnoticed initially, because a murmur is absent at presentation in 30% of cases [3]. In our patient, murmur was found during the third day of hospitalization; moreover, skin lesions observed in meningococcal sepsis and in S. aureus sepsis are similar. For that reason, it is important to search for an underlying cardiac cause in patients who present with meningitis and skin lesions in a clinical course that may mimic the classical presentation of meningococcal sepsis. The presence of pustular skin lesions in a patient with sepsis could be suggestive of S. aureus as the pathogenic agent. In fact, in staphylococcal endocarditis purpuric lesions may progress into cutaneous gangrene or may be pustular, with organisms present on Gram stain [8].

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