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Pneumococcal Triggered Recurrent CNS Vasculitis

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Introduction

Streptococcus pneumoniae is one of the leading agents causing meningitis in the pediatric population. It is associated with neurological sequalae in at least 30% of the patients [1]. Pediatric patients form a special cohort in terms of vulnerability to infection and response to treatment. We will discuss the case of a 9-year-old boy with treatment resistant pneumococcal meningitis with recurrent CNS vasculitis presenting in a tertiary care setting in North India.

Case

A 9-year-old male child presented with fever for 4 days, headache, vomiting, chills and neck pain since 2 days followed by abnormal body movement, frothing from the mouth for 3-4 minutes. On arrival to the emergency, child was intubated in view of poor GCS, measures for raised ICP were started, 3% NaCl(2ml/hr) and antiepileptics (Levetiracetam 40mg/k). Child admitted to pediatric intensive care unit, started on broad-spectrum antibiotics (meropenem 120 mg/kg/day, vancomycin 60 mg/kg/day) acyclovir and dexamethasone. Blood parameters revealed leucocytosis with thrombocytopenia (Table 1) along with features of acute kidney injury (BUN:28 Creatinine:1.06) and hepatic injury (Total/Direct Bilirubin:2.5/1.28), with raised inflammatory markers (CRP:194mg/dl). ANA profile was negative, systemic vasculitis ruled out.

After 11 days, Injectable Clindamycin (30mg/k) added for persistent fever spikes. Lumbar CSF examination revealed Streptococcus pneumoniae on rapid biofire panel. MRI brain revealed diffuse leptomeningeal enhancement along with areas of cytotoxic edema in vermis and subcortical white matter of frontal, temporal lobes. Fundus was normal. Acyclovir was discontinued as no viral etiology was isolated. Fever spikes persisted, repeat neuroimaging CT scan done on Day 7 revealed right maxillary sinusitis with frontal encephalocele. Sinus drainage performed, tissue samples revealed Acinetobacter baumanii, minocycline and colistin added, meropenem withdrawn.

Table 1: Complete Blood Count

Date	DOA 1	DOA3	DOA7	DOA21
Haemoglobin (g/dl)	11.8	8.5	7.7	9.3
TLC (cu mm)	10,200	14,450	17,130	4660
DLC	N85 L12 E0M3	N84 L14 E0 M2	N52 L29 E2 M10	N57 L35 E0 M8
Platelets (/ul)	16,000	1,37,000	2,55,000	5,11,000

He was extubated on Day 10 of admission. He underwent repeat lumbar puncture (table 2), which showed partial resolution (improvement in leucocytosis). However, he continued to have neck rigidity, intermittent headaches but sensorium improved, following commands and acceptance of oral feeds.

After 11 days, Injection Clindamycin (30mg/kg/day) added for persistent fever spikes. Lumbar puncture (Day14, table 2), showed decrease in leucocytosis without absence of complete resolution. He then had sudden onset bilateral blurring of vision with right medial rectal palsy. Rescue doses of methylprednisolone 10mg/kg/day given and Rifampicin started. He was shifted to critical care unit for observation. Repeat MRI brain with contrast with MR angiography (Figure 1) revealed right middle cerebral artery infarct involving upper capsuloganglionic lesion. Child showed improvement over the course of next 2-3 days, shifted to oral steroids (1.5mg/kg/day). This was followed by sudden onset left sided hemiparesis along with decreased reflexes in B/L lower limbs for which NCV was done and was normal. Dose of oral steroids increased to 2mg/kg/day. Child showed reduction in fever spikes, improvement in neck rigidity and ability to sit without support. However, repeat CSF done on Repeat CSF (D21) showed increase in cell number (Table 2). Started on Aspirin. Vancomycin stopped after a course of 21 days, cefotaxime started at a meningitic dose. On Day 25, child had development of left sided hemiparesis with slurring of speech and severe headache. MRI brain showed development of infarct in right cerebral peduncle. Child given IVIG 2gm/kg/day for 5 days. Clinical improvement seen; shifted on oral steroids. Lumbar puncture showed improving trend. Cefotaxime stopped after completing a course of 14 days. The child could sit without support, had improving grip in left upper limb and could abduct shoulders. At 2 week follow up, child had 5/5 power in upper limbs and was able to walk without support. He was continued on low dose steroids with tapering over 2 months. Last follow up after 6 months, child is clinically stable with no new infarcts.

Table 2: CSF Analysis

CSF analysis	DOA1	DOA5	DOA10	DOA14	DOA25	DOA32
Cell count	7880	311	150	22	62	23
Differential	N82 L18	N50 L50	N70 l30	N30 L70	N55 L45	N20 L80
Protein	601	197	127	104	119	104
Glucose	102	50	63	54	50	52
Biofire	S.pneumoniae	Sterile	Sterile	Sterile	Sterile	Sterile

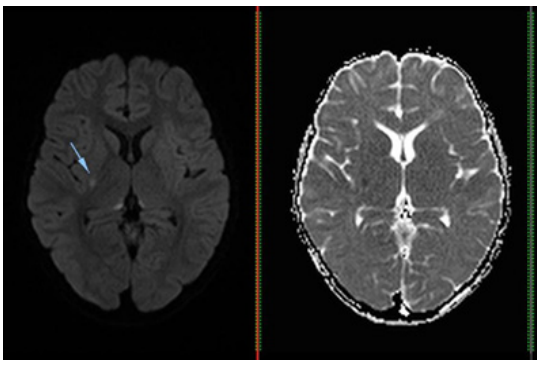

Figure 1: MRI Sequence: DWI/ADC Right Capsuloganglionic Infarct

Discussion

Most common organisms causing meningitis include Streptococcus pneumoniae, Haemophilus influenzae b and Neisseria meningitidis with specific age group dependent predilections. Vaccines have decreased incidence but dramatic change in case fatality rates has not been seen [2]. We report a case of a 9-year-old male with pneumococcal meningitis resistant to treatment as seen by delay in CSF clearance along with cerebrovascular complications and vasculitis. This diagnosis was made on a combination of clinical manifestations, neuroimaging, investigations, and response to immunosuppressants.

Pathogenesis

Commonly CNS infections occur after hematogenous invasion or through direct extension from the nasopharyngeal mucosa/ sinuses. Once the organism has invaded it initiates an inflammatory cascade resulting in bacterial lysis. The released bacterial particles trigger cytokine release (IL-1B/ CXCL1/2/5) [3]. Persistent inflammatory response leads to decreased cerebral perfusion, raised pressures, vasculitis, metabolic injury all of which leads to ischaemia and neurological damage.

Pneumococcus has evolved several strategies to evade the innate immune system. Firstly, the capsule of pneumococcus repulses sialic acid which is present in the mucus avoiding entrapment along with several exoglycosidases such as neuraminidase A (NanA), neuraminidase B (NanB), beta galactosidase A (BgaA) and beta-N-acetylglucosaminidase (StrH) causing cause deglycosylation of the mucus preventing entrapment. Invasive disease is more common in extremes of age group or those who are immunocompromised [4].

The bacterial products released lead to a heightened inflammatory response which further gets exacerbated with antibiotic use [4]. This in our case would explain the worsening of neurological symptoms despite adequate antibiotic use. Multiple experimental studies have been done to study the role of bacteriostatic agents such as rifampicin, clindamycin and daptomycin. It has been seen that rifampicin used before ceftriaxone led to less hippocampal damage but no decrease observed in mortality as seen in rat studies [5].

Cerebrovascular complications are common with an incidence of approximately 30% [6]. In adult patients, the incidence of arterial stroke is 30%, venous thrombosis 9% and intracerebral haemorrhage is 9%. Extensive angiographic studies have shown the most common inciting agent for stroke in pneumococcal meningitis is vasculitis [4].

Weisfelt et al. in their comparative study of CSF analysis between patients with viral meningitis and bacterial meningitis revealed that cases of bacterial meningitis had greater concentrations of CSF soluble tissue factor and prothrombin factor F1+ F2, along CSF plasminogen activating inhibitor factor-1 (PAI-1) [7]. The overall effect may lead to fibrin formation and hence, infarction. Use of steroids leads to decrease in neurological complications by attenuating the inflammatory response and preventing vasculitic changes [8]. A similar picture was seen in our child where use of steroids and immunomodulatory (IVIG) showed improvement.

Hence immunosuppression along with antibiotic use should become the mainstay of treatment, preventing neurological sequalae in the paediatric population. Consensus needs to be made on the dosing, duration as well as early recognition for need of steroid use [9].

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