Case Report Pneumococcal Meningitis Complicated by Cerebral Vasculitis, Abscess, Hydrocephalus, and Hearing Loss

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Introduction

Intracranial abscesses, postinfectious vasculitis, and hydrocephalus are rare complications of Streptococcus pneumoniae (S. pneumoniae) meningitis, and to our knowledge, there have been no case reports where all these 3 complications occurred in a single patient with Streptococcus pneumoniae meningitis. Here, we report a case of a 48-year-old male who developed postinfectious vasculitis, abscess, hydrocephalus, and hearing loss after S. pneumoniae meningitis. Clinicians ought to be aware of the possible adverse outcomes of S. pneumoniae meningitis and the limitations of current treatment options.

Case description

A 48-year-old Indian male previously healthy, presented with fever, headache and dry cough associated with generalized body pain for 4 days duration and vomiting for 1 day duration. He was febrile, altered sensorium with GCS 12 x 15. CT scan head without contrast was unremarkable. He was diagnosed as pneumococcal meningitis based upon CSF analysis, CSF culture and blood culture. Intravenous ceftriaxone 2 g every 12 hour was started with high-dose dexamethasone.

Initially he showed clinical response. Dexamethasone was stopped after 3 days and ceftriaxone was continued. He started to develop altered sensorium with low-grade fever. MRI brain and repeat LP were done to rule out any complication of the disease.

MRI brain (Figures 1) showed meningoencephalitis, vasculitis, and extradural fluid collection. There was fluid in the mastoid cavity without bone destruction. Repeat CSF analysis showed down trending leukocyte count and proteins and negative CSF culture. Dexamethasone was restarted with continuation of IV ceftriaxone for a total of 6 weeks as pneumococcal meningitis complicated by infective vasculitis, mastoiditis, and subdural collection. Repeat MRI brain (Figure 3) showed significant improvement in leptomeningeal enhancement and resolution of epidural collection; however, there was a new communicating hydrocephalus. After completion of his IV ceftriaxone, the patient was repatriated to his home country. Although his condition improved on above treatment, he was discharged with mild disorientation to time and person.

Investigation	On presentation	Day 7
Peripheral WBC	19,000/micro ml	13,000/micro ml
CSF WBC	145 per microliter	11 per microliter
CSF protein	4.55 g/L	1.11g/L
CSF Glucose	< 0.3 mmol/L	5 mmol/L
CSF culture	Streptococcus pneumoniae	Negative
blood culture	Streptococcus pneumoniae	Negative
CRP	495	90
Pro-calcitonin	11 ng /mL	0.41 ng /mL
Serum creatinine	160 micromol/L	78 micromol/L

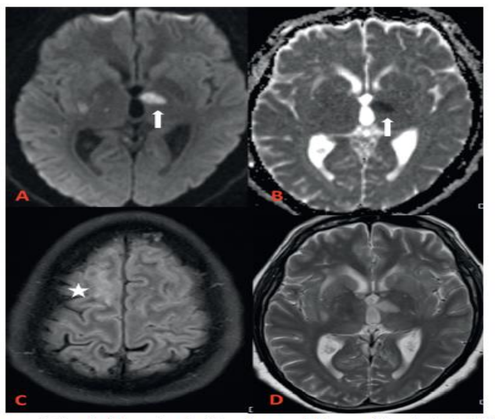

FIGURE 1: MRI brain examination of a 48-year-old male with meningoencephalitis, vasculitis, and extradural fluid collection. Axial DWI (A) and ADC (B) images show acute left thalamic (white arrow) and tiny right basal ganglia lacunar infarcts secondary to vasculitis. Axial FLAIR image (C) shows a cortical high signal in bilateral higher parietal lobes suggestive of epidural collection and underlying cerebritis (star). Axial T2-weighted image (D) shows a hyperintense signal in the left thalamus and basal ganglia secondary to vasculitis.

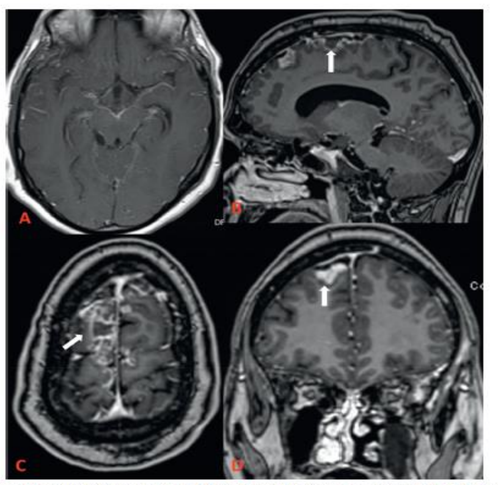

FIGURE 2: MRI brain examination of a 48-year-old male with meningoencephalitis, vasculitis, and extradural fluid collection. Postcontrast MRI axial T1 (A), GRE sagittal (B), GRE axial (C), and GRE coronal (D) images show diffuse leptomeningeal enhancement, enhancing basal exudates and high biparietal epidural fluid collection (white arrow) with cerebritis.

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Learning points

This complex case highlights the risk of intracranial complications in invasive pneumococcal infections. Invasive pneumococcal disease is defined as an infection caused by Streptococcus pneumoniae isolated from a normally sterile site such as blood, cerebrospinal fluid, and pleural, joint, or peritoneal fluid.

S. pneumoniae causes alteration in the host inflammatory response and results in the release of proinflammatory cytokines in the CSF which results in disruption of blood-brain barrier (BBB) . This results in increased neurological (74.7%) and systemic (37.9%) complications that include seizure (27.6%), diffuse brain swelling (28.7%), hydrocephalus (16.1%), hearing loss (19.7%), and ischemic or hemorrhagic brain damage (21.8%).

Infective vasculitis is a rare complication caused by S. pneumoniae. Classical 2 phase pattern of pneumococcal meningitis is described as Initial improvement followed by subsequent neurological deterioration with both cerebral thrombosis and inassociatedfective vasculitis. It is due to postinfectious inflammatory process rather than the infection itself.

Despite advancement in the medical field, only dexamethasone has shown to be effective adjunctive therapy. It has been shown that treatment with dexamethasone in patients with pneumococcal meningitis can decrease the neurological complications and mortality especially when given early during the disease course. Although treatment is usually given less than four days, some reports have shown a rebound vascular inflammation after steroid withdrawal. we have treated our patient with a prolonged taper of steroids to treat infective vasculitis as a complication of pneumococcal meningitis

Conclusion

Despite effective antibiotic treatment, mortality and morbidity are high in patients with bacterial meningitis. Since extremes of ages are predisposing conditions towards invasive pneumococcal disease, an effective vaccination program is very important to decrease the morbidity and mortality due to this condition. Morbidity like vasculitis after bacterial meningitis which can lead to brain injury is one of the known complications, but its treatment is unclear.

References

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