



INVASIVE PNEUMOCOCCAL INFECTION IN AN INFANT: A CASE REPORT

Microbiology

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ABSTRACT

Streptococcus pneumoniae is a major pathogen of humans, causing diseases such as pneumonia, bacteremia and meningitis. The authors report a case of pneumococcal septicemia with meningitis in an infant. The objective of reporting this case is to highlight the importance of blood culture for early diagnosis. Alertness and rapid diagnosis led to favorable outcome in our case.

KEYWORDS

INTRODUCTION

S. pneumoniae is a part of the commensal flora of the upper respiratory tract. Together with *Moraxella catarrhalis*, *Haemophilus influenzae*, *Neisseria meningitidis*, *Staphylococcus aureus* and various haemolytic streptococci, they colonise the nasopharyngeal niche. Though colonisation with pneumococci is mostly symptomless, it can progress to respiratory or even systemic disease. *Streptococcus pneumoniae* an important pathogen causing invasive diseases such as sepsis, meningitis and pneumonia.²

Risk groups for diseases caused by pneumococci, such as meningitis, sepsis, and pneumonia, include young children, elderly people, and patients with immunodeficiencies. Depending on age, 30–60% of survivors develop long-term sequelae including hearing loss, neurological deficits, and neuropsychological impairment.³ Because the highest frequency of pneumococcal colonisation and the highest crowding index are found in young children, this risk group is thought to be the most important vector for horizontal dissemination of pneumococcal strains within the community.⁴

Therefore, part of the strategy to prevent pneumococcal disease focuses on prevention of nasopharyngeal colonization, especially in children. Our ability to prevent pneumococcal infections in young children is quite limited. Available polysaccharide vaccines are not immunogenic in young infants, among whom the incidence is greatest.¹

The authors present a case of pneumococcal septicemia with meningitis in an infant highlighting the importance of blood culture for early diagnosis and favourable outcome.

CASE-REPORT

A 7 months old male infant was admitted in the Paediatric Intensive Care Unit (PICU) with cough, breathlessness and fever for 3 days. As stated by the patient's father, the child was apparently well 2 months back when he developed high grade fever with cough for which they consulted the local practitioner following which the symptoms subsided to some extent but did not recover completely. After few weeks, patient again had second episode of cough and fever which was more in severity, for which they were referred to our hospital. The mother also suffered from similar complaints at that time.

On examination, his general condition was poor. He was febrile (102°F). Heart rate (161 beats per minute) and respiratory rate (56/min) were high. He had respiratory distress and was hypotensive (80/51 mm

of Hg). He was initially stabilised with intravenous (I/V) fluids, oxygen inhalation and antipyretics. His haemoglobin was 8.0 g/dL, total leukocyte count (TLC)-21,500 cu/mm³ with predominance of polymorphs (75%). Blood culture and urine culture were sent to Microbiology laboratory for isolation of bacterial pathogen. Lumbar puncture (LP) was also carried out to drain the cerebrospinal fluid (CSF) and was sent for Gram stain, culture, cell counts, protein and sugar estimation. BacT/Alert 3D blood culture bottle was loaded in the machine.

After sending the samples for culture, empirically, I/V taxim 100mg x 12 hourly and amikacin 80 mg x 24 hourly was started. The CSF protein was 300 mg/dL, sugar 25 mg/dL, white blood cell count was 2800 cells/mm³ with 85% polymorphs and 15% lymphocytes. The test for malarial parasite (antigen), dengue NS1, IgM, IgG and *Salmonella* IgM, IgG: were all negative. However, CRP was raised (16.92 mg/L).

Gram stain of centrifuged deposit of CSF showed Gram positive lanceolate shaped diplococci with halo around suggestive of *Streptococcus pneumoniae* [Fig. 1] with moderate pus cells. On seeing the report of Gram, the clinician was alerted immediately and injection vancomycin was added to his treatment regimen. The CSF was cultured on Blood agar (BA), Chocolate agar (CA) and MacConkey agar (MAC) plates and incubated at 37°C in candle jar.

The positive growth signal was obtained in BacT/Alert 3D within few hours of incubation. Gram stain done from bottle also revealed Gram positive lanceolate shaped diplococci with halo around. Subculture from flagged blood culture bottle was also done on BA, CA and MAC and plates were incubated at 37°C in candle jar. The clinician was alerted again.

On the second day, patient had jittery movements all over the body with frothing from mouth and uplifting of eye balls. CT scan brain showed multiple patchy area of hypodensity over bilateral frontal lobe suggestive of acute ischaemic insult. Injection lorazepam 60 mg I/V over 30 minutes was started and continued as 30 mg I/V 12hrly.

Pure growth of - pin-point, smooth, transparent, low convex, hemolytic colonies with draughtsman appearance was seen on BA plates in both the samples [Fig. 2]. The colonies were low convex with greenish discoloration underneath in CA. On identification the colonies were catalase negative, optochin-sensitive [Fig. 3], Bile esculine- negative, showed fermentative reaction in Hugh Leifson's Oxidation/Fermentation media for glucose and Bile soluble by both

tube and plate method [Fig. 4 and Fig.5].However , the urine sample of the infant was sterile after overnight incubation.

Drug susceptibility for isolates from both CSF and blood was performed by Kirby Bauer disc diffusion method in accordance with CLSI guidelines. 5The isolate was sensitive to-penicillin, cefotaxime, ceftriaxone, erythromycin, chloramphenicol, clindamycin ,ofloxacin, meropenem, vancomycin and linezolid and was resistant to tetracycline and co-trimoxazole.

The antimicrobial therapy was continued for 21 days. The patient responded significantly to the therapy and repeat blood culture after 21 days follow up was sterile.He was finally discharged after 30 days of stay in the hospital.

DISCUSSION:

Streptococcus pneumoniae remains an important bacterial cause of invasive infection in children worldwide.The World Health Organization (WHO) estimates that 1.6 million die every year from the disease, 0.7-1.0 million of which are children aged<5 years.6Vos T *et al*,from 1990-2013 reported more than 7.5 million cases of pneumococcal meningitis ,which is significantly higher than the prevalence of other bacterial meningitis. 7Abdullah A *et al*, studied invasive pneumococcal disease from March 2002 to November 2005 in which sepsis and pneumonia were the commonest manifestation, followed by meningitis. 8

In our case the child most probably contracted the respiratory infection from his mother as she was suffering from respiratory infection, which further progressed into bacteremia and meningitis.

Fatima T *et al*, studied the outcome of invasive pneumococcal disease and reported that comorbidities, immune status and focus of infection play an important role in predicting the outcomes of pneumococcal bacteremia. 9

Therefore, vigilant screening, rapid diagnosis and prompt institution of appropriate therapy are essential prerequisites for successful management of this pathogen.

CONCLUSION:

To conclude,in this case pneumonia was the underlying focus which led to pneumococcal bacteremia and progressed into meningitis. Hence, our case suggests that presence of sepsis warrants aggressive management as it is associated with poorer outcomes. *Streptococcus pneumoniae* causes invasive infections in infants,hence should be treated vigorously.Moreover, this case highlights the importance of direct microscopy (rapid reporting) and rapid communication with the clinician which led to timely initiation of appropriate treatment and favourable outcome.

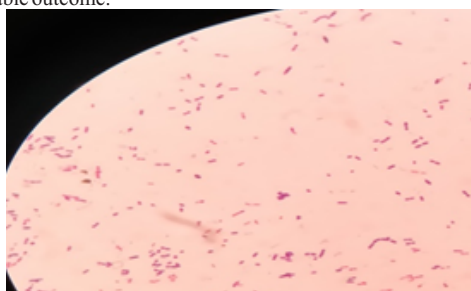


Fig. 1: Gram-positive diplococci ,lanceolate shaped with halo around

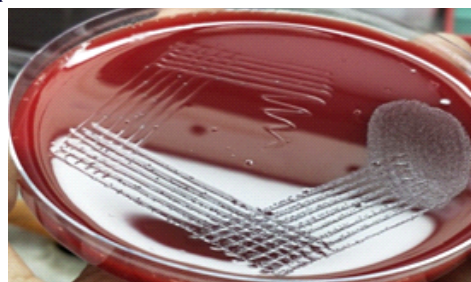


Fig. 2: Pin-point, hemolytic colonies with draughtsman appearance on Blood agar



BEFORE

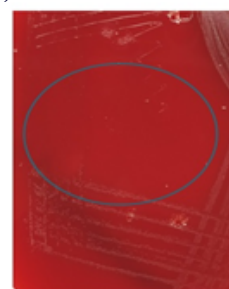


AFTER

Fig 4: Bile solubility (tube method)



BEFORE



AFTER

Fig 5: Bile solubility (plate method)

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