Pseudomonas aeruginosa as an Unexpected Cause of Early Neonatal Meningitis: Two Case Reports

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ABSTRACT

Pseudomonas aeruginosa is a rare cause of neonatal meningitis, usually associated with nosocomial infection, prematurity, or immunodeficiency. We report two full-term neonates, one without identifiable risk factors and another in whom a health care–associated infection could not be excluded. The first presented with hematemesis and hypoglycemia, and the second with fever, jaundice, and hypotonia. Cerebrospinal fluid analysis in both cases showed pleocytosis, elevated protein, and low glucose. Cultures grew Pseudomonas aeruginosa. Both infants received targeted therapy including ceftazidime and ciprofloxacin. Head ultrasound revealed ventriculitis, subependymal hemorrhage, and white matter hyperechogenicity in one patient. Extensive investigations including immunological workup and sweat testing were normal. Both infants had favorable short-term outcomes.

Keywords : Pseudomonas aeruginosa, Neonatal meningitis, Cerebrospinal fluid, Antibiotics

RÉSUMÉ

La méningite néonatale due à Pseudomonas aeruginosa est exceptionnelle et survient le plus souvent dans un contexte d'infection nosocomiale, de prématurité ou d'immunodépression. Nous rapportons deux nouveau-nés à terme sans facteurs de risque identifiables. Le premier a présenté des hématémèses et une hypoglycémie, et le second, fièvre, ictère et hypotonie axiale. L'analyse du liquide céphalorachidien a révélé une pléocytose, une hyperprotéinorrachie et une hypoglycorrachie. Les cultures ont isolé Pseudomonas aeruginosa. Les deux nourrissons ont reçu un traitement ciblé comprenant céftazidime et ciprofloxacine. L'échographie transfontanellaire a montré une ventriculite, des hémorragies sous-épendymaires et une hyperechogénicité de la substance blanche chez un patient. Les investigations complémentaires, incluant le bilan immunologique et le test de la sueur, étaient normales. L'évolution à court terme a été favorable.

Mots-clés : Pseudomonas aeruginosa, Méningite néonatale, Liquide céphalorachidien, Antibiotiques

INTRODUCTION

Neonatal bacterial meningitis is a critical situation associated with high mortality and a significant risk of long-term neurological sequelae. In the early neonatal period, most cases result from hematogenous spread and are typically caused by Group B Streptococcus (GBS), Escherichia coli, or Listeria monocytogenes [1,2]. Pseudomonas aeruginosa, a Gram-negative bacillus, is an uncommon cause of neonatal meningitis. When identified, it is usually associated with nosocomial infections, prematurity, or immunosuppression [3,4]. Reports of early-onset P. aeruginosa meningitis in healthy, full-term newborns are exceedingly exceptional [1].

We report two cases of early-onset Pseudomonas aeruginosa meningitis in full-term neonates without evident risk factors. These cases underscore the diagnostic challenge posed by nonspecific clinical manifestations and highlight the importance of early recognition and prompt, appropriate treatment.

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CASES PRESENTATION:

Case 1:

A full-term male infant, born via vaginal delivery with appropriate birth weight and good neonatal adaptation, was hospitalized on the 4th day of life for fever, hypotonia, and jaundice. Perinatal history was unremarkable, and no infectious risk factors were identified.

On physical examination, his temperature was 38.1° C, and capillary glucose was 0.9 g/L. He had axial hypotonia. He had cutaneous and mucosal jaundice and no palpable hepatomegaly. Hemodynamic and respiratory parameters remained stable.

Laboratory tests showed a total bilirubin level of 329 µmol/L, with a conjugated fraction of 20 µmol/L, and a C-reactive protein (CRP) of 1 mg/L. The complete blood count revealed 11,750 white blood cells/µL, hemoglobin at 12.5 g/dL, and platelets at 411,000/µL. Cerebrospinal fluid (CSF) analysis demonstrated a turbid appearance with 800 white blood cells/µL (60% neutrophils), protein concentration of 1.78 g/L, and glucose concentration of 0.8 mmol/L.

He was initially started on empirical antibiotics (cefotaxime, gentamicin, and amoxicillin), intensive phototherapy, and intravenous albumin. CSF culture grew Pseudomonas aeruginosa, which was sensitive to most antipseudomonal β-lactams (piperacillin, ceftazidime, cefepime, aztreonam) and ciprofloxacin, but resistant to gentamicin and tobramycin, with intermediate sensitivity to carbapenems and amikacin. Antibiotic therapy was adapted accordingly, and the patient was switched on ciprofloxacin, and ceftazidime.

Cranial ultrasound (CUS) on day 10 revealed bilateral subependymal hemorrhage, ventriculitis, and increased periventricular echogenicity. Serial ultrasounds showed gradual resolution of the hemorrhage and partial regression of ventricular wall hyperechogenicity, although persistent signs of ventriculitis and white matter changes were noted. Overall, he received 10 days of ciprofloxacin, and a prolonged course of ceftazidime for 56 days. A repeat lumbar puncture at the end of treatment showed improvement, with CSF pleocytosis decreasing to 13 cells/µL.

Follow-up cranial ultrasound showed complete regression of ventriculitis after 8 weeks of treatment. Additional work-up showed normal immunological profile and a negative sweat test (sweat chloride at 12 mmol/L).

The patient was discharged in good clinical condition and has been regularly followed in outpatient consultations. Psychomotor development has remained age appropriate. Complementary investigations showed normal auditory brainstem response (20 dB threshold), present visual evoked potentials and normal brain magnetic resonance imaging (MRI).

Case 2:

A male infant, born at 37 weeks and 4 days of gestation via vaginal delivery, was admitted at 24 hours of life for hematemesis. He was eutrophic, with good adaptation to extrauterine life. The pregnancy had been uneventful, with no obstetric complications or maternal infectious history. Vitamin K prophylaxis was administered at birth, and no maternal nipple fissures were noted.

Initial clinical examination showed no signs of hemorrhagic shock. Neurological as well as respiratory parameters remained stable. Laboratory investigations revealed a white blood cell count of 12,100/µL, hemoglobin level of 14.4 g/dL, and platelet count of 349,000/µL. CRP was 5 mg/L, and prothrombin time was 75%.

The patient was started on proton pump inhibitors, and no further episodes of bleeding occurred. However, on day 4 of life, he developed hypoglycemia and poor feeding during hospitalization. His temperature remained normal. Repeat laboratory evaluation revealed an elevated CRP level of 72 mg/L. A lumbar puncture was therefore performed. CSF was turbid, containing 8,625 white blood cells/µL (85% neutrophils, 15% lymphocytes). Glucose was undetectable (0 mmol/L), and protein was elevated at 5.6 g/L. Direct examination was negative, but culture grew Pseudomonas aeruginosa, which was sensitive to amikacin, ceftazidime, ciprofloxacin, and most other antipseudomonal agents.

The patient received intravenous ciprofloxacin (20 mg/kg/day for 10 days), amikacin (15 mg/kg/day for 3 days), and ceftazidime (50 mg/kg every 8 hours for 21 days). CUS was normal.

The clinical course was favorable under antibiotic therapy. A repeat lumbar puncture at the end of treatment demonstrated normal CSF findings. On monthly follow-up visits, the infant showed age-appropriate psychomotor development. Immunological work-up and sweat test were normal. Auditory and visual evoked potentials as well as brain MRI were requested and are pending.

DISCUSSION

Meningitis caused by Pseudomonas aeruginosa in neonates is rare but represents a serious medical emergency associated with high morbidity and mortality [1,2]. Nosocomial transmission is often reported, particularly in neonatal intensive care units, where recognized risk factors include prematurity, invasive procedures, prolonged hospitalization, and broad-spectrum antibiotic exposure [2,5]. Both of our patients were full-term and vaginally delivered. While no maternal or neonatal risk factors were identified in the first case, the second case developed meningitis after hospitalization, raising the possibility of a health care–associated acquisition. This raises the possibility of community-acquired or maternofetal transmission. Although transplacental infection with Pseudomonas aeruginosa is uncommon, vertical transmission during delivery has been described, particularly in cases

of maternal genital colonization or ascending intrauterine infection [6]. Unfortunately, maternal cultures were not available in our cases, which limits definitive conclusions. It should be noted that no maternal cultures were available in our cases, which limits interpretation of the source of infection. Nevertheless, the timing of presentation, within the first week of life, suggests that an early perinatal acquisition is plausible. Environmental sources of infection have also been reported, such as contaminated milk bottle brushes in domestic settings and contaminated feeding bottles in neonatal intensive care units [1,7].

The clinical presentation of early neonatal bacterial meningitis is often subtle and nonspecific, making early recognition particularly challenging. Reported signs include temperature instability, feeding difficulties, irritability, respiratory distress, seizures, or jaundice, but these findings are neither sensitive nor specific [1,4]. In our first patient, symptoms included fever, hypotonia, and jaundice, while our second patient had hypoglycemia and poor feeding but no fever. These presentations underscore the diagnostic difficulty in this age group. The absence of fever does not exclude meningitis, and lumbar puncture remains essential in neonates with unexplained symptoms.

CRP is widely used as a biomarker of neonatal infection, but its diagnostic value in meningitis is limited. CRP may remain normal during early disease or in localized infections [4]. In our first case, CSF culture was positive for P. aeruginosa despite a normal CRP on admission, underscoring that meningitis cannot be excluded on the basis of inflammatory markers alone. Serial measurements may increase sensitivity, as illustrated by the first case, where CRP rose sharply in parallel with clinical deterioration. This highlights the need for lumbar puncture in neonates with unexplained symptoms, regardless of CRP level [4,8].

CSF analysis remains the cornerstone for diagnosing neonatal meningitis. While blood markers such as CRP or leukocytosis can suggest infection, they are insufficiently sensitive to rule in or rule out central nervous system involvement [4,5]. In our cases, CSF abnormalities, marked pleocytosis with neutrophilic predominance, elevated protein levels, and reduced or absent glucose, were decisive for diagnosis, even when inflammatory markers in blood were normal or only modestly elevated. This emphasizes the essential role of lumbar puncture in neonates with suspected sepsis, unexplained systemic symptoms, or poor clinical evolution, regardless of blood test results. Early CSF analysis not only confirms the diagnosis but also guides antibiotic selection based on culture and sensitivity.

Pseudomonas aeruginosa is a rare but highly aggressive cause of neonatal meningitis. Most reported cases are nosocomial, occurring in preterm infants with invasive devices or after prolonged antibiotic exposure [2,5]. Community-acquired infections in term neonates, as in our patients, are exceptional [1]. The organism's intrinsic resistance mechanisms and ability to form biofilms complicate treatment and often require prolonged courses of combination antipseudomonal therapy [2]. Reported mortality rates exceed those of meningitis caused by other gram-negative bacilli, and survivors remain at high risk of neurodevelopmental sequelae, including ventriculitis, hydrocephalus, and sensorineural deficits [3]. These features highlight the need for vigilance, early recognition, and aggressive management when Pseudomonas aeruginosa is isolated in neonatal CSF.

Treatment of Pseudomonas meningitis in neonates requires prolonged and carefully tailored antibiotic therapy. Ceftazidime, meropenem, and aminoglycosides remain the mainstay of therapy due to their activity against Pseudomonas aeruginosa and ability to penetrate the cerebrospinal fluid [2]. Combination therapy is often recommended initially to maximize bactericidal activity, with subsequent de-escalation based on susceptibility testing [9]. The use of ciprofloxacin in our patients was justified by its favorable susceptibility profile against Pseudomonas aeruginosa and its ability to achieve therapeutic CSF concentrations when combined with ceftazidime. Treatment duration is longer than for other gram-negative meningitides, often exceeding six weeks, particularly in the presence of ventriculitis or imaging abnormalities [5,6]. In our first patient, antibiotic therapy was extended beyond ten weeks because of persistent neurosonographic changes. In the first patient, antibiotic therapy was prolonged (total of 56 days of ceftazidime) due to persistent neurosonographic abnormalities consistent with ventriculitis, in line with recommendations suggesting extended treatment in complicated cases [6]. While such prolonged therapy increases the risk of toxicity and resistance, it may be justified in severe cases with ongoing central nervous system involvement.

Neuroimaging is a critical component in the evaluation and follow-up of neonatal meningitis, allowing early detection of structural complications that may influence management and prognosis. CUS is often the first-line modality due to its bedside availability, non-invasiveness, and ability to be repeated frequently without sedation or radiation exposure [10]. It is particularly effective in identifying ventriculitis, subependymal hemorrhage, hydrocephalus, and periventricular white matter abnormalities, all of which are prognostically significant. In more complex or ambiguous cases, brain MRI provides superior sensitivity for detecting parenchymal injury, abscesses, or ischemic changes [11]. In our second patient, serial CUS revealed evolving bilateral ventriculitis, subependymal hemorrhages, and persistent white matter hyperechogenicity, guiding both the duration of antibiotic therapy and the frequency of follow-up imaging. Regular neuroimaging, beginning with CUS and complemented by MRI when necessary, is therefore essential in monitoring disease progression and anticipating long-term neurodevelopmental outcomes.

Beyond acute management, comprehensive investigations are warranted in neonates with unusual pathogens such as Pseudomonas aeruginosa, to exclude

underlying susceptibility. In particular, immune deficiency screening (immunoglobulin levels, lymphocyte subsets, neutrophil function) is advised, as immunological disorders may predispose to severe gram-negative infections [12]. In addition, a sweat chloride test or genetic testing for cystic fibrosis should be considered, given the known association between Pseudomonas colonization and cystic fibrosis, even though neonatal meningitis is a rare initial presentation [13]. In both our patients, these investigations were normal, reducing the likelihood of an underlying immunodeficiency or cystic fibrosis. This strengthens the hypothesis of acute perinatal or early postnatal acquisition rather than an infection on the background of host vulnerability.

Our cases underscore various key points: the variable and atypical presentations of neonatal bacterial meningitis, the critical position of lumbar puncture even in the absence of classical symptoms, and the need for prolonged neuroimaging follow-up in cases complicated by ventriculitis. Regardless of the severity of the initial presentation, both patients demonstrated favorable developmental outcomes in the short term, though continued long-term follow-up remains essential [1,4].

CONCLUSION

Pseudomonas aeruginosa meningitis in neonates is rare but potentially severe, often associated with significant neurological complications. Our two cases highlight the diagnostic challenges posed by atypical or subtle initial signs, emphasizing the need for a high index of suspicion even in full-term newborns without recognized infectious risk factors. Early lumbar puncture, targeted antimicrobial therapy guided by sensitivity testing, and vigilant neuroimaging follow-up are crucial for optimizing outcomes. Despite the severity of infection, both infants demonstrated favorable short-term evolution, underscoring the value of timely diagnosis and individualized management. Nevertheless, given the risk of delayed neurodevelopmental sequelae, long-term follow-up remains essential.

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