International Journal of Infectious Diseases (2009) 13, e101–e103

http://intl.elsevierhealth.com/journals/ijid

CASE REPORT

Successful antibiotic eradication of *Streptococcus pneumoniae* infection of a ventriculoatrial shunt

Katia Orvin, Efraim Bilavsky*, Eran Weiner, Dror S. Shouval, Jacob Amir

Department of Pediatrics C, Schneider Children's Medical Center of Israel, Petach Tikva and Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

Received 28 February 2008; accepted 14 July 2008
Corresponding Editor: William Cameron, Ottawa, Canada

KEYWORDS
Streptococcus pneumoniae meningitis;
Ventriculoatrial shunt

Summary A case of *Streptococcus pneumoniae* meningitis in a possibly immune-compromised child with a ventriculoatrial shunt is described. The infection was successfully eradicated by treatment with intravenous ceftriaxone and rifampicin, without removal of the shunt.

Introduction

The recommended treatment for cerebrospinal fluid (CSF) shunt infection is antibiotic administration with removal of the shunt.[1] The main pathogens of shunt infection are Staphylococcus species.[1] The pathogens that typically cause meningitis in children (*Haemophilus influenza* type B, *Streptococcus pneumoniae* and *Neisseria meningitides*) are rarely implicated in CSF shunt infections. We describe a child with a *S. pneumoniae* ventriculoatrial (VA) shunt infection and meningitis who was treated successfully by antibiotics alone, without the need to remove the shunt.

Case report

A 17-month-old female presented to our hospital with high fever, vomiting, lethargy and apathy for a period of 72 hours. Amoxicillin 70 mg/kg/day had been prescribed by her pediatrician two days before hospital admission, without clinical improvement. The parents also reported focal seizures on the day of admission.

Past medical history revealed a normal-term pregnancy and vaginal delivery. Two days after birth, a colonic perforation of suspected intrauterine origin was detected and treated by surgery with stoma placement. During hospitalization in the neonatal intensive care unit, the child contracted *Escherichia coli* sepsis followed by two episodes of *E. coli* meningitis. After a protracted illness, a noncommunicating hydrocephalus was diagnosed and a ventriculo-peritoneal (VP) shunt was inserted. At the age of three months, the patient was readmitted with bacteremia and skin abscesses due to *Pseudomonas aeruginosa* infection; this was successfully treated with intravenous antibiotics. One month later, a *H. influenza* VP shunt infection was diagnosed. Treatment consisted of intravenous antibiotics and shunt removal. Insertion of a new VP shunt failed due to abdominal adhesions and a VA shunt was inserted. Given the patient history, an extensive laboratory work-up was performed for suspected immunodeficiency. The results yielded normal-range levels of immunoglobulin, subtypes of IgG and complement, normal neutrophil function, and normal B and T

* Corresponding author. Department of Pediatrics C, Schneider Children's Medical Center of Israel, 14 Kaplan Street, Petach Tikva 49202, Israel. Tel.: +972 3 925 3775; fax: +972 3 925 3801.
E-mail address: yoji@netvision.net.il (E. Bilavsky).

1201-9712/$36.00
doi:10.1016/j.ijid.2008.07.008

lymphocyte levels and function. Serology for HIV was negative. Colonoscopy ruled out the presence of a colonic fistula with bacterial seeding.

At the most recent admission, notable findings upon physical examination were a high temperature of 39.1 °C and focal seizures. The seizures were controlled with phenytoin and the patient was referred to the pediatric neurosurgery department for a shunt puncture. Because of the small volume, the shunt fluid was sent only for bacterial culture. Brain computed tomography showed normal-sized ventricles. Treatment with vancomycin (50 mg/kg/day) and ceftazidime (120 mg/kg/day) was initiated, and the patient was transferred to our department of pediatrics.

At admission to the pediatric department, vital signs were as follows: temperature 38.1 °C, heart rate 134 beats per minute and blood pressure 112/49 mmHg. The patient appeared pale, sleepy, irritable and apathetic. Neurological evaluation revealed 'sunset eyes' with defects in upward gaze and difficulty following objects. A complete blood count revealed a white blood cell count of 10.8×10^9/l, with 35.5% neutrophils and 47.2% lymphocytes. Hemoglobin measured 8.1 g/dl and the platelet count was 526 000/μl. Electrolyte levels, and kidney and liver function tests were within the normal range. A chest X-ray demonstrated a right upper lobe infiltrate that was consistent with lobar pneumonia.

Blood and urine cultures taken on admission to the neurosurgery department were sterile. The VA shunt fluid culture obtained on the day of admission grew *S. pneumoniae* with an intermediate susceptibility to penicillin (a minimum inhibitory concentration of 0.19 μg/ml) and susceptibility to ceftriaxone, vancomycin and rifampicin. Antibiotic treatment was therefore switched to ceftriaxone 100 mg/kg/day. A lumbar puncture performed on the third day after admission revealed a white blood cell count of 340 cells/mm^3 (50% segmented and 50% mononuclear), proteins 111.1 mg/dl and glucose 43 mg/dl. Gram stain of the fluid was negative, but culture grew *S. pneumoniae* with the same antibiogram as the culture from the shunt puncture. Rifampicin (20 mg/kg/day) was added to the ceftriaxone.

Two days later, the patient's condition deteriorated, manifesting as a high-pitched cry, right-hand weakness and worsening in gaze. Magnetic resonance imaging showed bilateral dilated ventricles. Echocardiography demonstrated a thrombus blocking the end of the VA shunt. Treatment with tissue plasminogen activator (0.8 mg/kg/hour) and enoxaparin (1 mg/kg/day) was initiated and led to substantial clinical improvement. Repeated shunt and lumber puncture cultures showed no bacterial growth. The patient was discharged home after 16 days.

The patient remained well for the next three months, when she was readmitted because of bacteremia and extended-spectrum β-lactamase *Klebsiella pneumoniae* shunt infection. She was treated with antibiotics, but this time, owing to an increase in intracranial pressure, the shunt was removed as well. A temporary shunt was inserted, which was replaced three weeks later with a new VA shunt.

Discussion

Typical meningitis pathogens in children (*H. influenza* type B, *S. pneumoniae* and *N. meningitides)* account for approximately 5% of all shunt infections[2,3] and the incidence of meningitis caused by these organisms appears to be higher in patients with a CSF shunt.[2] Shunt infections with typical meningitis pathogens have several unique characteristics. They can occur long after shunt placement, unlike staphylococcal infections, which usually occur within 2—6 months (80% of patients). The route of invasion to the meninges is usually hematogenous seeding (with retrograde infection), rather than wound colonization at the time of surgery.[3] Blood cultures are communally positive.[2]

In this present case, the infection occurred more than one year after shunt placement. Based on the early findings, we suspect that the bacteria probably caused lobar pneumonia initially and then, by hematogenous spread, meningitis. The blood culture might have been negative because of the amoxicillin treatment that was administered before the patient's admission to our department; the isolated pathogen was moderately resistant to penicillin and the oral treatment eradicated it from the blood. However, because of the low CSF penetrability of amoxicillin, the pathogen persisted in the meninges.

Of the various streptococcal species that cause meningitis, *S. pneumoniae* is rarely implicated in shunt infections in children. Our search of the English literature identified more than 700 cases of shunt infection in which an etiologic agent was identified, of which only 4 were due to *S. pneumoniae*.[4—10] However, uncommon culprits in shunt infection were included in only a few reports and, even then, they were categorized as 'miscellaneous'. Therefore, the true incidence could be somewhat higher. The specific characteristics of these cases were not discussed.

Our patient had a significant history of serious bacterial infection, raising the suspicion of an underlying immunodeficiency disease. However, our extensive immunological investigation was noncontributory. We presume that an undiagnosed immunodeficiency contributed to this rare case of *S. pneumoniae* meningitis and to the other bacterial infections.

Shunt infections are traditionally managed by antibiotic administration and shunt removal. During the work-up in this case, shunt removal was considered. However, given the continuous positive response to ceftriaxone and rifampicin, and the negative findings from repeated lumbar puncture and shunt culture, we decided to keep the shunt in place. We found only one other report of a similar case in the English literature, wherein a six-month-old infant with *S. pneumoniae* meningitis and a VP shunt was successfully managed by antibiotic treatment without shunt removal.[11] Our decision to add rifampicin to the pharmacologic regimen was based on its high CSF penetration and favorable pharmacokinetics.[12] We propose that *S. pneumoniae* shunt infection is more amenable to antibiotic treatment alone because these pathogens are less likely to adhere to the shunt lumen than staphylococci.

In conclusion, although shunt removal should still be the 'gold standard' in most cases (regardless of organism) of shunt infection in children, we suggest that, for Gram-positive non-staphylococcal infections when shunt removal is difficult or impossible, clinicians could consider the use of antibiotic treatment alone, without shunt removal.

Conflict of interest: No conflict of interest to declare.
Funding source: None.
Ethical approval: None.

References

1. Schreffler RT, Schreffler AJ, Wittler RR. Treatment of cerebrospinal fluid shunt infections: a decision analysis. *Pediatr Infect Dis J* 2002;**21**:632—6.
2. Gardner P, Leipzig T, Phillips P. Infections of central nervous system shunts. *Med Clin North Am* 1985;**69**:297—314.
3. Tunkel AR, Kaufman BA. Cerebrospinal fluid shunt infection. In: Mandell GL, Bennetts JE, Dolen R, editors. *Principles and practice of infectious diseases*. 6th edition. New York: Churchill Livingstone; 2004. p. 1126—32.
4. Schoenbaum SC, Gardner P, Shillito J. Infections of cerebrospinal fluid shunts: epidemiology, clinical manifestations, and therapy. *J Infect Dis* 1975;**131**:543—52.
5. Kontny U, Hofling B, Gutjahr P, Voth D, Schwarz M, Schmitt HJ. CSF shunt infections in children. *Infection* 1993;**21**:89—92.
6. Forward KR, Fewer HD, Stiver HG. Cerebrospinal fluid shunt infections. A review of 35 infections in 32 patients. *J Neurosurg* 1983;**59**:389—94.
7. Vinchon M, Dhellemmes P. Cerebrospinal fluid shunt infection: risk factors and long-term follow-up. *Childs Nerv Syst* 2006;**22**:692—7.
8. Wang KW, Chang WN, Shih TY, Huang CR, Tsai NW, Chang CS, et al. Infection of cerebrospinal fluid shunts: causative pathogens, clinical features, and outcomes. *Jpn J Infect Dis* 2004;**57**:44—8.
9. Renier D, Lacombe J, Pierre-Kahn A, Sainte-Rose C, Hirsch JF. Factors causing acute shunt infection. Computer analysis of 1174 operations. *J Neurosurg* 1984;**61**:1072—8.
10. Walters BC, Hoffman HJ, Hendrick EB, Humphreys RP. Cerebrospinal fluid shunt infection. Influences on initial management and subsequent outcome. *J Neurosurg* 1984;**60**:1014—21.
11. O'Keeffe PT, Bayston R. Pneumococcal meningitis in a child with a ventriculo-peritoneal shunt. *J Infect* 1991;**22**:77—9.
12. Nau R, Prange HW, Menck S, Kolenda H, Visser K, Seydel JK. Penetration of rifampicin into the cerebrospinal fluid of adults with uninflamed meninges. *J Antimicrob Chemother* 1992;**29**:719—24.