

A Case of Meningitis Caused by *Streptococcus pyogenes* in a Child with Ventriculoperitoneal Shunt and Ommaya Reservoir

Ventriküloperitoneal Şantı ve Ommaya Rezervuarı Olan Çocukta *Streptokokkus pyogenes* Menenjit

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Abstract

Streptococcus pyogenes is a well-known cause of invasive infections. However, acute bacterial meningitis caused by this pathogen is unusual. Herein, we presented a child with ventriculoperitoneal (VP) shunt and bilateral ommaya reservoir who developed group A streptococcal (GAS) meningitis a month after the last neurosurgery operation. The patient was treated with removal of VP shunt and ommaya reservoir and antibiotic therapy including vancomycin, ceftriaxone and clindamycin for 21 days. Although many predisposing factors have been described for GAS meningitis, this is the first report of an association between ommaya reservoir and VP shunt due to *S. pyogenes* meningitis. Clinicians should be aware that sporadic cases can occur and if it is treated promptly, the outcome tends to be favourable. (*J Pediatr Inf 2013; 7: 35-8*)

Key words: Meningitis, *Streptococcus pyogenes*, ventriculoperitoneal shunt, ommaya reservoir

Özet

Streptococcus pyogenes az rastlanan enfeksiyonların iyi bilinen bir nedenidir. Akut bakteriyel menenjit nedeni olarak bu patojen nadirdir. Biz bu makalede ventriküloperitoneal (VP) şantı ve bilateral Ommaya rezervuarı olan ve son ameliyatından sonra grup A streptokok (GAS) menenjit gelişen bir çocuğu sunduk. Hasta VP şant ve Ommaya rezervuarlarının çıkarılması ve 21 gün süreyle vankomisin, seftriakson ve klindamisin verilerek tedavi edildi. GAS menenjit için birçok predispozan faktör rapor edilmiş olsa da bu Ommaya rezervuarı ve VP şant ilişkili ilk rapor edilen makaledir. Klinisyenler sporadik vakaların olabileceği konusunda uyanık olmalıdır ve hızla tedavi uygulanması durumunda daha olumlu sonuçlar elde edilecektir. (*J Pediatr Inf 2013; 7: 35-8*)

Anahtar kelimeler: Menenjit, *Streptokok pyogenes*, ventriküloperitoneal şant, ommaya rezervuar

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Introduction

Invasive infections due to group A beta hemolytic streptococcus (GAS) include bacteremia, pneumonia, skin and soft tissue infections, meningitis and toxic shock syndrome. Meningitis represents 2% of all cases of GAS invasive disease (1). GAS is usually not mentioned as the causative agent of bacterial meningitis beyond the neonatal period (2-4). Although GAS has the ability to invade soft tissue, CNS infections remain a rare event and account for less than 0.2% of all bacterial meningitis (5, 6).

In a MEDLINE search of English literature, we found only 12 cases of GAS meningitis reported

in the pediatric age group since year 2000 but no cases of GAS meningitis in a child with Ommaya reservoir. Herein we report the case of a child with ventriculoperitoneal shunt and bilateral Ommaya reservoirs who developed GAS reservoir poche abscess, meningitis and bacteremia and was treated with removal of VP shunt-ommaye reservoir and antimicrobial therapy.

Case Report

A 8-year old boy was operated for multicystic craniopharyngeoma three years previously and after this operation he progressively lost vision and hearing ability and developed hydro-



cephalus (Figure 1). VP shunt was implanted after craniopharyngeoma surgery and bilateral ommaya reservoir implantation was carried out because of his hydrocephalus. The last ommaya reservoir was implanted one month previously. This child was admitted to our clinic because of erythema, warmth and tenderness over the reservoir that has been implanted one month previously.

On physical examination the child was drowsy with a poor general condition. The temperature was 39.9°C and he had severe headache. His pulse rate was 94/min, respiratory rate was 24/min and blood pressure was 118/77 mmHg. His pupils were unequal in size and slowly reactive to light. All the signs of meningeal irritation (neck rigidity, Kernig's and Brudzinkin's sign) were present. Examination of the respiratory system, cardiovascular system and abdomen were found to be normal and no rashes were observed.

Laboratory tests revealed a white blood cell count of 32,000 leukocytes/mL with 87% neutrophils. CRP (C-reactive protein) was elevated to 327 mg/dL. (0-5 mg/dL.) Renal and liver function tests were within normal limits. The cerebrospinal fluid (CSF) was turbid, the CSF contained abundant leukocytes (43% neutrophils), protein of 144 mg/dL, glucose of 70 mg/dL against the blood glucose of 108 mg/dL.

He went to surgery for the removal of the ommaya reservoir. In the operation, purulent discharge from the left ommaya reservoir was noticed so it was taken out. The culture from the surgically debrided tissue yielded

S. pyogenes (sensitive to penicillin, clindamycin and erythromycin). Meanwhile CSF and the peripheral blood culture had grown the same organism. For this reason on the second day, the reservoir on the right side was also taken out and external drainage was applied to the VP shunt on the right side. The diagnosis of surgical site infection, bacteremia and meningitis was made and the treatment of vancomycin, ceftriaxone and clindamycin were initiated for 21 days. The cultures of the CSF and the blood were sterile after the beginning of the treatment. He was treated successfully and a new VP shunt was implanted.

Discussion

Invasive GAS infections are defined as bacteremia, pneumonia, or any other infection associated with the isolation of GAS from a normally sterile site (7).

Invasive group A streptococci (GAS) infections have become more prevalent since the mid 1980's (8, 9). Despite the increase, GAS bacterial meningitis remains uncommon and accounts for less than 0.2% of all cases of bacterial meningitis, with a total of 51 cases reported (5, 10, 11). Recent data from the Centers for Disease Control and Prevention in the United States revealed 5400 cases of invasive GAS disease over a four year period. Meningitis and central nervous system disease were seen in 52 cases (1% overall) (12). Despite the increase in invasive GAS diseases during the last decades, *S. pyogenes* meningitis incidence remains unchanged. Van de Beek et al reported 41 patients aged 16 years and older with GAS meningitis in the Netherlands (11). In this report, the mortality rate was 27%, which contrasts with data from the literature that describe a mortality rate of 5 to 10% (5, 10).

Meningitis due to *S. pyogenes* usually follows upper respiratory tract infection, otitis media, sinusitis or related to head injury or cranial surgery (13). Furthermore, some risk factors have been described, including neurosurgery, skull fractures, CSF leaks. Shetty et al. (14) documented 30 cases of GAS meningitis over a 25-year period from 1976 to 2001. Among these, 52% had a primary focus on infection in the ear, nose and throat area. Moreover Arnoni et al. (15) reported 2 cases of GAS meningitis and reviewed 15 children with GAS meningitis from 1996 to 2006. In this report, the risk factors for GAS meningitis were otitis for three cases, varicella for two cases, cochlear implantation for one case, infected BCG scar for one case, infected haemangioma for one case, submandibular abscess for one case, occipital skull fracture for one case and five cases didn't have any risk factors. Our patient had undergone cranial surgery one month before admission. Bilateral ommaya reservoirs were implanted and one of them was the source of the infection. Although

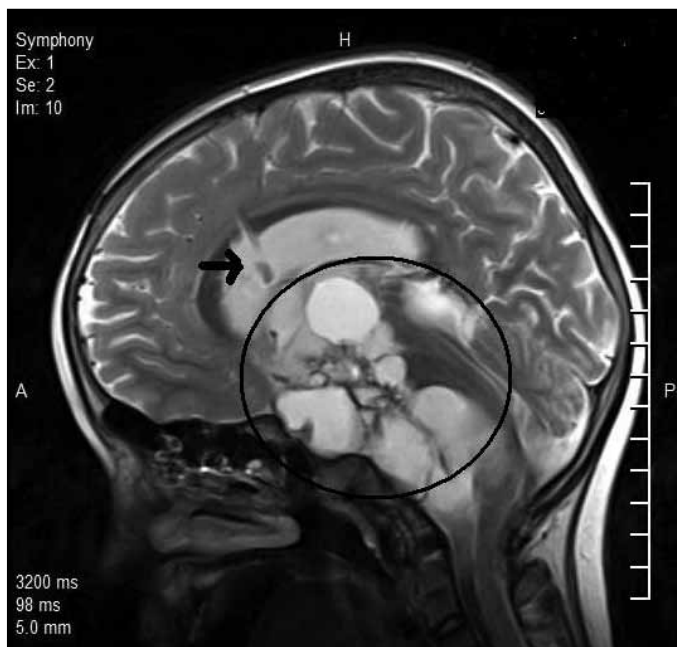


Figure 1. The shunt from the right frontal region to the left lateral ventricle is seen (arrow). The huge heterogenous cystic tumoral mass tissue occupies the third and the left lateral ventricle pushing the brain stem posteriorly

GAS frequently colonizes the oropharynx, in our patient we think that skin colonization over the ommaya reservoir was the source of the infection or, since infection developed in 30 days after ommaya reservoir implantation it may have been due to surgical site infection because of culture from the ommaya reservoir yielded *S. pyogenes*. The organisms most frequently causing infections of indwelling CNS prostheses are the coagulase negative staphylococci. The second most frequent pathogen is *Staphylococcus aureus* (16-19). As far as we know there was no previous report concerned with ommaya reservoir related GAS meningitis.

Treatment of GAS meningitis includes management of the complications of sepsis, aggressive surgical debridement, if an appropriate site of infection is identified and antibiotics for the underlying GAS infection. The antibiotic of choice for treatment is penicillin and there have been no reports of resistance of this agent to this drug (15). For patients who are allergic to penicillin, treatment with ceftriaxone may be an alternative (20).

Animal models and a review of treatment of 56 children with GAS bacteremia favor combination treatment with a beta-lactam plus clindamycin (21, 22). Our patient was treated by intravenous vancomycin (60 mg/kg/day divided every 6 hours), ceftriaxone (100 mg/kg/day divided every 12 hours) and clindamycin (40 mg/kg/day divided every 6 hours).

Uncomplicated GAS meningitis cases have been treated for 10-14 days (9, 13, 23, 24). Length of therapy depends on the clinical response to antibiotic treatment. Therapy is usually continued for 14 days from the last positive culture obtained during surgical debridement. Our patient was treated for 21 days.

Group A streptococci meningitis is associated with a low mortality if treated promptly. The case fatality rate in the review of Barlodes et al. (25) was 12%. The most frequent complications are neurologic, although in pediatric patients, neuroendocrinologic complications predominate (19, 20). Chow&Muder reported a global lethality of 5% and sequelae in 46% of the cases in a review conducted between 1981-1991 (5). A case series of 51 patients (mixed adult and pediatric patients) showed a 44% prevalence rate of sequelae in pediatric patients (the majority being neurological) compared with approximately 7% in adults (25).

Conclusion

Group A streptococcus is an uncommon cause of meningitis in children but has to be considered as a causative pathogen.

Conflict of Interest

No conflicts of interest were declared by the authors.

References

- Zurawski CA, Barsdley MS, Beall B, et al. Invasive group A streptococcal disease in metropolitan Atlanta: a population-based assessment. *Clinical Infectious Diseases* 1998; 27: 150-7. [\[CrossRef\]](#)
- Feigin RD, Perlman E. Bacterial meningitis beyond the neonatal period. In: Feigin RD, Cherry JD (eds) *Textbook of pediatric infectious diseases*, 4th edn. Saunders, Philadelphia: 1998. pp.400-29.
- Prober CG. Infections of the central nervous system. In: Nelson WE (ed) *Textbook of pediatrics*, 15th edn. Saunders, Philadelphia: 1996. pp.707-16.
- Tunkel AR, Scheld WM. Acute meningitis. In: Mandell GL, Bennett JE, Dolin R(eds) *Principles and practice of infectious diseases*, 4 th edn. Churchill Livingstone, New York: 1995. pp.831-65.
- Chow JW, Muder RR. Group A Streptococcal meningitis. *Clin Infect Dis* 1992; 14: 418-21. [\[CrossRef\]](#)
- Schelech WF, Ward JI, Band JD, et al. Bacterial meningitis in the United States, 1978 through 1981. The national bacterial meningitis surveillance study. *J Am Med Assoc* 1985; 253: 1749-54. [\[CrossRef\]](#)
- Stevens DL. Invasive group A streptococcus. *Infectious Dis Clin Infect Dis* 1992; 14: 2-13. [\[CrossRef\]](#)
- Davies HD, Mc Geer A, Schwatz B. Invasive group A streptococcal infectious in Ontario, Canada. *N Engl J Med* 1996; 335: 547-54. [\[CrossRef\]](#)
- Moses A, Ziv A, Harari M, Rahav G, Shapira M, Engelhard D. Increased incidence and severity of streptococcus pyogenes bacteremia in young children. *Pediatr Infect Dis J* 1995; 14: 767-70. [\[CrossRef\]](#)
- Mathur P, Arora NK, Kapil A, Das BK. Streptococcus pyogenes meningitis. *Indian J Pediatr* 2004; 71: 423-6. [\[CrossRef\]](#)
- Van de Beek D, de Gans J, Spanjuard L, Sela S, Vermeulen M, Dankert J. Group A Streptococcal meningitis in adults, reports of 41 cases and a review of the literature. *Clin Infect Dis* 2002; 34: 32-6. [\[CrossRef\]](#)
- O'Loughlin RE, Roberson A, Cieslak PR, et al. Active Bacterial Care Surveillance Team. The epidemiology of invasive group A Streptococcal infection and potential vaccine implications. United States. 2000-2004. *Clin Infect Dis* 2007; 45: 853-62. [\[CrossRef\]](#)
- Asnis DS, Knez T. Group a Streptococcal meningitis. *Arch Intern Med* 1998; 58: 810-4. [\[CrossRef\]](#)
- Shetty AK, Frankel LR, Maldonado Y, Falco DA, Lewis DB. Group A Streptococcal meningitis: Report of a case and review of literature since 1976. *Pediatr Emerg Care* 2001; 17: 430-4. [\[CrossRef\]](#)
- Arnoni MV, Berezin EN, Safadi MA, Almeida FJ, Lopes CR. Streptococcus pyogenes meningitis in children: Report of Two Cases and Literature Review. *Braz J Infect Dis* 2007; 11: 375-7. [\[CrossRef\]](#)
- Bisno AL, Sternau L. Infections of central nervous system shunts. In: Bisno AL, Waldvogel FA, editor. *Infections Associated with Indwelling Medical Devices*. American Society for Microbiology, Washington; 1994. pp.91-109.
- Crnich CJ, Safdar N, Maki DG. Infections associated with implanted medical devices. In: Finch RG, Greenwood D, Norrby SR, Whitley RJ, editor. *Antibiotic and Chemotherapy: Antimicrobial Agents and Their Use in Therapy*. 8. Churchill Livingstone; 2003. pp.575-618.

18. Naradzay JFX, Browne BJ, Rolnick MA, Doherty RJ. Cerebral ventricular shunts. *J Emerg Med* 1999; 17: 311-22. [\[CrossRef\]](#)
19. Filka J, Huttova M, Tuharsky J, Sagat T, Kralinsky K, Kremery VJ. Nosocomial meningitis in children after ventriculoperitoneal shunt insertion. *Acta Pediatr* 1999; 88: 576-8. [\[CrossRef\]](#)
20. Lin HH, Liu YC, Chiou CC, Lheng DL. Group A Streptococcal meningitis. *J Formes Med Assoc* 1996; 95: 802-3.
21. Stevens DL, Gibbons AE, Bergstrom R, Winn V. The Eagle effect revisited: efficacy of clindamycin, erythromycin and penicillin in the treatment of streptococcal myositis. *J Infect Dis* 1998; 158: 23-8. [\[CrossRef\]](#)
22. Zimbelman J, Palmer A, Todd J. Improved outcome of clindamycin compared with betalactam antibiotic treatment for invasive streptococcus pyogenes infection. *Pediatr Infect Dis J* 1999; 18: 1096-100. [\[CrossRef\]](#)
23. Jagdis F. Group A Streptococcal meningitis and brain abscess. *Pediatr Infect Dis J* 1988; 7: 885-6. [\[CrossRef\]](#)
24. Campello MG, Miguel Diaz J. Meningitis por *Streptococcus pyogenes*, absceso cerebral y ependimitis. *Enferm Infecc Microbiol Clin* 1995; 13: 68.
25. Baraldes MA, Domingo P, Mauri A, et al. Group A Streptococcal meningitis in the antibiotic era. *Eur J Clin Microbial Infect Dis* 1999; 18: 572-8. [\[CrossRef\]](#)