CASE REPORTS

RARE VENTRICULOPERITONEAL SHUNT INFECTION BY GROUP B STREPTOCOCCUS IN CHILDHOOD

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ABSTRACT

Group B Streptococcus (GBS) is primarily recognized as a pathogen causing neonatal infections, but invasive disease can also occur in older children, particularly those with underlying risk factors. We report a case of a 9-year-old girl with a ventriculoperitoneal (VP) shunt who initially presented with status epilepticus. Although the patient had normal laboratory and neuroimaging findings, she subsequently developed fever, abdominal pain, and local signs of shunt infection. Ultrasound revealed a distal shunt abscess and cerebrospinal fluid (CSF) analysis showed hyperproteinorrachia. Cultures from the CSF and abscess drainage grew *Streptococcus agalactiae*. The patient underwent urgent surgical drainage, externalization of the shunt, and targeted antibiotic therapy, resulting in full recovery and successful reimplantation of the VP shunt. This case highlights the importance of considering GBS as a cause of late-onset VP shunt infection in children and underscores the challenges of early recognition.

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Introduction

Streptococcus agalactiae, or Group B Streptococcus (GBS), is a Gram-positive bacteria recognized as a leading cause of neonatal morbidity and mortality.1 It typically causes early-onset disease in newborns by vertical transmission, manifesting as sepsis, pneumonia, or meningitis.[1]

While primarily associated with neonates, GBS can also cause invasive infections in older children, though these cases are rare.[2] In this population, GBS infections are more likely to occur in patients with specific risk factors such as immunodeficiencies, congenital anomalies, or the presence of implanted medical devices, including ventriculoperitoneal (VP) shunts and cochlear implants.[3]

VP shunts are essential in the management of hydrocephalus, by redirecting excessive cerebrospinal fluid (CSF) from the ventricles to the peritoneal cavity, thus lowering intracranial pressure.[4] However, they are associated with significant complications, both mechanical and infectious. Infection accounts for approximately 10% of all VP shunt-related complications, and its clinical presentation can be nonspecific, thus the diagnostic threshold should be particularly low.[4] While most VP shunt infections are due to skin flora such as *Staphylococcus epidermidis* and *Staphylococcus aureus*, less common pathogens, including Gram-negative bacteria and GBS, must also be considered.[4]

We present a rare case of a GBS-related VP shunt infection in a 9-year-old child, highlighting the diagnostic complexity and management strategies for this uncommon, but serious condition.

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Case Report

A 9-year-old girl with a history of extreme prematurity (25 weeks' gestation), post-hemorrhagic hydrocephalus managed with a VP shunt, cerebral palsy and epilepsy, presented to the Emergency Department in status epilepticus. On arrival, she was febrile (38.9°C), unresponsive, and exhibited conjugated lateral gaze deviation, drooling, and repetitive oral automatisms, persisting for over one hour. Additional vital signs showed hypertension (138/69 mmHg), tachycardia (165 bpm), and hypoxemia (SpO_2 88% on room air).

The patient was admitted to the pediatric intensive care unit for refractory status epilepticus, unresponsive to both first and second-line anticonvulsants. Continuous midazolam perfusion (2.5 µg/kg/min) was initiated, resulting in rapid cessation of both clinical and electrographic seizures. Initial laboratory tests were unremarkable, with no evidence of metabolic or electrolyte imbalance or systemic infection. A non-contrast head CT scan revealed stable ventricular size and correct VP shunt positioning, with no signs of acute intracranial changes. Her maintenance antiepileptic medication - levetiracetam and carbamazepine - was optimized, and she was transferred to the paediatrics general ward on day 2, clinically stable and asymptomatic.

On day 5, the patient developed fever (38.5°C) and abdominal pain. Inflammatory markers were moderately elevated (CRP 89.1 mg/L; PCT 0.55 ng/mL), though abdominal ultrasound findings were unremarkable. Fever subsided within 24 hours, and no new neurological deficits emerged.

However, by day 12, she experienced clinical deterioration with fever (39.0°C), diffuse abdominal pain with guarding, vomiting, lethargy, and erythema along the VP shunt tract. Repeated blood work showed a significant rise in inflammatory markers (CRP 128.9 mg/L; PCT 8.78 ng/mL), leukocytosis (WBC 17,900/µL), and neutrophilia (89%). Abdominal ultrasound revealed

a pyogenic collection in the right iliac fossa near the distal shunt tip associated with abdominal wall cellulitis. A lumbar puncture was performed, revealing marked hyperproteinorrachia (523 mg/dL) without pleocytosis or hypoglycorrhachia. The patient underwent urgent surgical drainage of the abdominal abscess and externalization of the distal end of the VP shunt and was started on empiric intravenous antibiotics - cefotaxime (200 mg/kg/day) and vancomycin (50 mg/kg/day). Cultures from both the CSF and drained abscess fluid were positive for *Streptococcus agalactiae*. Based on the susceptibility test results, antibiotic therapy was narrowed to intravenous benzylpenicillin (450,000 IU/kg/day) for a total of 21 days. Clinical improvement was prompt, with resolution of fever, normalization of inflammatory markers, and negative follow-up CSF cultures. A new VP shunt was reimplanted on day 39. The patient was discharged two days later without further neurological deficits.

Discussion

GBS, though primarily a neonatal pathogen, should be considered a potential threat across all age groups, particularly in patients with certain risk factors.[1,5] Beyond the neonatal period, GBS infections present more commonly as sepsis, seconded by arthritis and skin infection.[5] Pediatric GBS meningitis remains rare, with only isolated cases reported beyond infancy.[5–7]

VP shunt placement dramatically improves outcomes in hydrocephalus but carries a notable risk of complications, infection being one of the most common (3-20%).[1,8] Risk factors for VP shunt infections include young age at insertion, prematurity, and postoperative CSF leakage.8 Although most infections are due to coagulase-negative *Staphylococci* and *Staphylococcus aureus*, atypical pathogens should not be overlooked, namely gram-negative bacteria and fungi, which may indicate a possible immunity impairment.[9] Infections typically occur within six months after shunt placement, often due to intraoperative contamination by skin flora pathogens.[9,10] Perioperative anti-bacterial prophylaxis and improvements in neurosurgical procedures are crucial to lower the incidence of VP shunt infections.8 Late-onset infections, as seen in this case, can also occur, potentially related to hematogenous seeding or abdominal contamination of the distal catheter.

The clinical manifestations of VP shunt infections are often subtle, heterogeneous, and vary according to patient's age, pathogen virulence and type of shunt, making early diagnosis challenging.[8] Infants and younger children may present with nonspecific symptoms such as fever, irritability, and vomiting, while older children may report headache or abdominal pain.[9] Less frequently, local signs may also be presented, namely as redness and induration along the subcutaneous course of the shunt.[10] Seizures, while not a classic feature of shunt infection, may result from increased intracranial pressure or local cortical irritation due to shunt malfunction or inflammation.[10]

In this case, the initial presentation with status epilepticus, absent elevated inflammatory markers, and normal imaging highlights the difficulty in early diagnosis.

Conclusion

This case underlines the importance of considering GBS as a potential pathogen in older children with VP shunts, especially in the presence of systemic symptoms and local signs of infection. While rare beyond infancy, GBS can lead to serious complications in patients with predisposing conditions such as prematurity and neurosurgical implants. Early recognition, thorough diagnostic evaluation, timely surgical management, and targeted antimicrobial therapy are vital to achieving optimal outcomes.

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