Recurrent CSF Shunt Meningitis

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Abstract

Cerebrospinal fluid shunt infections are hard to treat especially in a limited resource country. We report a child with 6 episodes of cerebrospinal fluid shunt reinfections. At the age of 9 months, the neurosurgeon implanted ventriculoperitoneal shunt to the child based on ventriculomegaly following the first episode of bacterial meningitis. After the initial shunt infection, the new episodes of shunt infections occurred after 14, 6, 7, 10 and 3 months. First five episodes of shunt infections are treated with conservative treatment without removing cerebrospinal fluid shunt. Also in each new episode of meningitis, we recorded tenderness and redness of the abdominal part of shunt derivation. In the last episode, the right ventriculoperitoneal shunt was removed and a left external ventricular drain was implanted. The child suffered a severe form of meningoencephalitis caused by E. coli, was in coma, manifested recurrent seizures and right haemiparesis. He was treated with imipenem given parenterally and amikacin injected intraventriculary. Our patient is now 17 years old with no cerebrospinal fluid device implanted, attending high school having intellectual impairment. The magnetic resonance imaging showed ventriculomegaly similar to the images seen on computed tomography scan during the last episode of meningitis.

Conclusion: Nonsurgical management of cerebrospinal fluid shunt infections lead to 5 new episodes of shunt reinfection. The decision for shunt implantation and removal should be carefully evaluated.

Keywords: Meningitis; Shunt Infections; Recurrent Meningitis; Bacterial Infections; Children

Abbreviations

CSF: Cerebrospinal Fluid; VP: Ventriculoperitoneal; ESR: Erythrocyte Sedimentation Rate; CT: Computed tomography; MRI: Magnetic Resonance Imaging; CNS: Central Nervous System; MIC: Minimal Inhibitory Concentration

Introduction

The case incidence of CSF shunt infection has ranged from 5 to 41% in various series [1] although the incidence is usually in the range of 4 to 15% [2,3]. There are many mechanisms by which CSF shunts may become infected such as retrograde infection from the distal end of the shunt, through the skin, hematogenous seeding from bacteremia and colonization of the shunt at the time of surgery [1]. Infection of tissues near the shunt site may also lead to direct inoculation of microorganisms.

Case Presentation

In this study, we present a child who underwent shunt surgery for hydrocephalus and experienced 6 episodes of CSF shunt infection. At the age of 9 months, the child was treated for bacterial meningitis at the Pediatric clinic. On head computed tomography scan was seen ventriculomegaly following the first episode of bacterial meningitis and neurosurgeon implanted the right ventriculoperitoneal (VP) shunt. After two days, the child was transferred to the Infectious diseases clinic for diagnosed shunt meningitis. After the initial shunt infection, the new episodes of shunt infections occurred after 14, 6, 7, 10 and 3 months. During 5 new episodes of shunt meningitis, the
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Child manifested clinical signs and symptoms of meningitis (e.g. fever, meningeal signs, change in mental status, seizures during second and last episode, neurological deficit in last episode) and changes in CSF: pleocytosis (> 10 000/mm³), decreased glucose and increased proteins in CSF. Also in each new episode of meningitis, we recorded tenderness and redness of the abdominal part of shunt derivation. During each episode of meningitis, the child was previously treated with ceftriaxone parenterally two doses and CSF cultures remained sterile except the CSF Gram stain was twice positive (gram-negative bacilli). The child was treated with ceftriaxone and chloramphenicol. The neurosurgeon was consulted during each episode of shunt meningitis and he didn’t remove the VP shunt. In the last episode, the patient was first admitted at the Neurosurgery clinic where the right VP shunt was removed and a left external ventricular drain was implanted. The second day, the child was transferred to our clinic. At the admission, the child was in soporous state which progressed to coma, manifested recurrent generalized seizures and right hemiparesis. Samples of CSF from external drainage and spinal tap revealed thick purulent CSF with pleocytosis (> 10 000/mm³), CSF/blood glucose ratio 2.5 vs. 2.5% (glycorachia 0.2 vs. 0.2 mmol/L) and increased proteins in CSF (6.4 vs. 7 g/L). Laboratory analyses at admission: ESR = 95, Le = 6.8, glucose = 7.9 mmol/L, urea = 4.5 mmol/L, creatinin = 44 mmol/L, liver enzymes normal values, chest x rays normal finding, blood cultures sterile, on computed tomography scan of the brain were seen ventriculomegaly with sign of vetriculitis and in left parietal lobe hipodenze zone suspected for cerebritis. On repeated CT scan after 45 days: ventriculomegaly was with same dimensions compared to previous images (Figure 1).

![Figure 1: CT scan of the brain (old neuroimages, year 2003).](image-url)

From CSF cultures taken from external drainage and spinal tap was isolated Escherichia coli multiresistant to antibiotics expect susceptible to imipenem and amikacin. CSF samples remained purulent with pleocytosis >10 000/mm³ for 14 days, CSF cultures were positive first 7 days and gram stain was positive for 10 days (seen gram-negative bacilli). Since at our hospital center we didn’t have imipenem, the parents were asked to by it, which they did but only after 8 days. From first day was given triple therapy with ceftriaxone and vancocymycin (the last one was discontinued from therapy when arrived results of CSF cultures – fourth day). Since the child was diagnosed also by neuroimaging for purulent ventriculitis, amikacin was injected intraventriculary (25 mg bid, 0.5 mg per ml of estimated CSF) through an external ventriculostomy for 6 days when child accidentally removed the external drainage. Amikacin was injected intrathecally at the same doses for additional 4 days. On 9th day was given imipenem (100 mg/kg 4 doses per day) during 4 weeks. The next two weeks the child was treated with chloramphenicol (80 mg/kg 4 doses per day) and with TMP-SMX orally for additional two weeks. CSF findings remained pathologic for 8 weeks (day 57 in lumbar tap was found clear CSF, 309 cells/mm³ (neutrofils = 30%), glucose = 2.1 mmol/L with CSF/blood glucose ratio 0.47 and proteins 3.17 g/L. Child was released cured after 10 weeks with normal CSF findings except increased proteins (1.65 g/L). Last neuroimaging done after ten years showed normotensive hydrocephalus and the child didn’t manifest any signs of intracranial hypertension. Our patient is attending high school but having intellectual impairment. On MRI, the images showed almost identical changes as those seen on last brain CT scan done during the last episode of meningitis (Figure 2).

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Figure 2: MRI of the brain (new neuroimages year 2013).

Discussion

The most common etiologic organism in infection of CNS medical devices is coagulase-negative staphylococcus, accounting for one-third to one-half of infections [4,5]. Approximately one-third of cases are caused by Staphylococcus aureus, another skin pathogen, with the remaining one-third produced by a wide variety of organisms, including aerobic Gram-negative bacilli [4]. Shunts terminating in the peritoneal cavity may have a greater risk of infection with gram-negative organisms [6,7].

When the infection arises from nosocomial acquisition, gram negative bacilli and antibiotic resistant organisms are more common as pathogens [8,9]. The most common isolated gram-negative species are Escherichia coli, Klebsiella, Proteus, and Pseudomonas; cases of nosocomial Acinetobacter meningitis have also been reported in patients with VP shunts [10-12].

Infection beginning in the proximal portion of the shunt (i.e., the catheter within the CSF space) results in meningitis or ventriculitis in approximately 30% of cases and may cause shunt obstruction or decreased function [1,6].

Our patient was hospitalized for the first time in 2000, first post war year in Kosovo, the poorest and undeveloped region of former Yugoslavia. Years of Serbian invasion and the war in Kosovo influenced the stagnation of health services and update of information in every field and especially in medicine. Our patient developed 5 new episodes of shunt meningitis since without removing the VP shunt.

Infected shunts that terminate in the peritoneal space may lead to an inflammatory response in the absorbing tissue (peritonitis). The child didn't manifest the symptoms of peritonitis which could be due to early antibiotic therapy started within 6 - 24 hours. On physical examination of abdomen were seen and palpated redness, swelling and tenderness from couple to 5 cm in length of peritoneal catheter with no local impairment of skin. On the echosonographic examination were not seen any pockets of fluid within the abdomen. The occlusion of an open-ended peritoneal catheter remained unexplained. The sudden pressure on the abdomen which might have been caused by other children, as his mother told us, could cause the penetration of the distal tip of the shunt into the bowel lumen and cause the infection. The other mechanism could be minor local skin erosions caused by scratching with fingers causing by patient himself. Infection from the distal end of the shunt or breakdown of the skin overlying the shunt remains the only reasonable mechanisms.

The agent selected for treatment of CSF shunt infections must penetrate the central nervous system and have bactericidal activity against the infecting pathogen. Intraventricular antibiotic therapy appears to be a useful treatment modality in those CSF infections in which systemic therapy may fail [13]. The concentration of the antibiotic in the CNS and the MIC for the suspected infecting organism cannot be measured in our hospital center. Most authors recommend starting with an empiric dosage of antibiotics. The recommended dosages of amikacin for daily intraventricular or intrathecal administration is 15 mg q24h (range 5 - 50 mg). Since the CSF was very purulent (thick pus), we injected amikacin intraventriculary at the highest doses of 25 mg twice per day for 6 days.

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The IDSA guidelines for management of bacterial meningitis recommend antibiotic therapy for gram-negative meningitis for 21 days [14]. No randomized trials have ever been done of different durations of therapy for Gram-negative meningitis. Clearly, administration of antibiotics should continue until CSF cultures are negative [15].

CSF shunt infections are hard to treat especially caused by multiresistant gram-negative bacilli and without needed antimicrobials in the ward. A decision to implant an internal shunt should be a multidisciplinary approach by neurosurgeon, infectious diseases specialist, neurologist and ophthalmologist. Dual therapy is recommended for patients with gram-negative bacillary shunt meningitis and ventriculitis using intravenous and intraventricular administration of antibiotics.

Conclusion

Nonsurgical management of cerebrospinal fluid shunt infections lead to 5 new episodes of shunt reinfection. The decision for shunt implantation and removal should be carefully evaluated.

Conflict of Interest

We declare that we have no conflict of interest.

Bibliography


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