Ventriculoperitoneal shunt infections in children

A 6-year study

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Summary

In a study of ventriculoperitoneal shunt infections conducted retrospectively between 1983 and 1987 and prospectively in 1988 39 infections from 372 shunt procedures (incidence 10,5%) were identified. The most common organism isolated was *Staphylococcus aureus* (18; 47%) followed by *S. epidermidis* (10; 26%). Forty-two per cent of staphylococci were methicillin-resistant. Gram-negative infections were associated with myelomeningoceles and Gram-positive infections with other forms of hydrocephalus ($P = 0,048$). Lymphocyte predominance was found more frequently than polymorphonuclear predominance in cerebrospinal fluid.

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Ventriculoperitoneal (VP) shunts have been the preferred form of treatment for hydrocephalus since the late 1960s.[1] Shunt infections are an important cause of morbidity and mortality in these patients.[1,2] Although a number of surveys of shunt infections in children have been published from developed countries,[3,6] there is little information available from developing countries. We are unaware of previous studies from the African continent.

The purpose of our study was to document the incidence, aetiology, clinical presentation, cerebrospinal fluid (CSF) findings, modes of treatment and outcome of shunt infections at Tygerberg Hospital, a 2 000-bed referral hospital in the south-western Cape Province.

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Subjects and methods

Patients < 13 years of age with shunt infections were identified from a register of abnormal CSF findings maintained jointly by the Departments of Paediatrics and Child Health and Medical Microbiology and from a review of all VP shunt procedures performed by the Department of Neurosurgery. Information on shunt infections was collected retrospectively from 1983 to 1987 and prospectively in 1988. From 1984 all patients undergoing a shunt procedure were given prophylactic cefamandole.

The following criteria were used to diagnose shunt infections: (*i*) microbiological — positive bacterial culture from either CSF or the wound site; and (*ii*) clinical — signs of inflammation over the reservoir or shunt tract.

A relapse was regarded as the reappearance of an organism of the same genus and species within 3 months of cessation of treatment.

The following information was extracted from the bed-letters of patients identified as having shunt infections: age; sex; primary diagnosis; preceding operative procedure (primary insertion or revision of an existing shunt); time interval between operation and diagnosis of infection; CSF findings; and site of positive culture and outcome.

Patients with an axillary temperature $\geq 38°C$ at the time of diagnosis of shunt infection were regarded as being febrile. The presence of shunt malfunction, meningism and abdominal signs (distension, tenderness and peritonitis) was also noted. All patients received antimicrobial therapy. Treatment was divided into the following: group 1 — medical treatment only; group 2 — removal of shunt with intermittent ventricular drainage and later replacement; group 3 — revision of the existing shunt; and group 4 — primary external ventricular drainage with later shunt replacement.

Bacteria were identified by standard laboratory techniques and antibiotic-sensitivity patterns evaluated by the disc method of Stokes.[7] Anaerobic cultures were done routinely for pus swabs but not for CSF specimens.

Results

Over the 6-year period 372 shunt procedures were performed. Thirty-nine episodes of shunt infection occurred in 35 patients. Shunt infection occurred after 25 of 238 primary shunts (10,4%) and after 14 of 134 revisions of existing shunts (10,5%). The case fatality rate was 31,4% ($N = 11$). The median age for patients with shunt infection was 10 months and mean age 24,2 ± 28,6 months. The primary diagnoses are shown in Table I. The male:female ratio was 0,95.

TABLE I. PRIMARY DIAGNOSIS OF PATIENTS WITH VENTRICULOPERITONEAL SHUNT INFECTIONS

Diagnosis	No. of patients
Myelomeningocele*	**6**
Tuberculous meningitis	**8**
Other bacterial meningitis	**8**
Congenital hydrocephalus†	**9**
Post intraventricular haemorrhage	**4**

* 1 patient developed 2 episodes of shunt infection.
† 1 patient developed 3 and another 2 episodes of shunt infection.

The median time interval between shunt procedure and diagnosis of shunt infection was 24 days. Twenty-seven occurred within 30 days (69%) and 31 within 60 days (79%) of the shunt procedure. Three patients had particularly long time intervals between shunt insertion and onset of shunt infection. One patient, with a primary diagnosis of tuberculous meningitis, developed shunt infection due to *Staphylococcus aureus* 103 months after shunt insertion. A contributory factor may have been meningococcal septicaemia with extensive skin infarction 3 weeks before diagnosis of shunt infection. The second, a patient with a myelomeningocele, developed an *Escherichia coli* shunt infection 96 months after insertion of the shunt. She had a urinary tract infection due to a *Klebsiella* species 4 months before shunt infection. The third patient with a primary diagnosis of post-bacterial meningitis developed an *E. coli* shunt infection 56 months after insertion of the shunt. There were no obvious precipitating factors.

The clinical features of shunt infection were as follows. Fever occurred in 24 patients (61%), tract inflammation in 23 (58%), shunt malfunction in 17 (44%) and meningism in 9 (23%). Abdominal signs were noted in 3 patients (8%), of whom 1 had distension, 1 tenderness and 1 peritonitis.

CSF was evaluated in 30 cases. Our findings are shown in Fig. 1. Four specimens had ≤ 5 white cells × 10^6/l. Organisms were shown by Gram stain in 13 cases (45%) and culture in 29 (97%). Organisms were isolated from pus swabs in an additional 6 shunt infections. Of interest is that lymphocyte predominance was noted in 12 cases (40%) and was more common than polymorphonuclear cell predominance, which occurred in 7 (23%). Four of 8 patients with a primary diagnosis of tuberculous meningitis had lymphocyte predominance at diagnosis of shunt infection. Of these, only 1 had active tuberculosis at the time, having developed shunt infection 13 days after the start of antituberculosis therapy. Two of the remaining 3 patients developed shunt infections 2 years after having presented with tuberculous meningitis and 1 after 2 months. A CSF specimen from the latter patient taken 1 month before shunt infection showed 5 lymphocytes × 10^6/l and no polymorphonuclear cells. At diagnosis of shunt infection 85 lymphocytes and 45 polymorphonuclear cells × 10^6/l were documented.

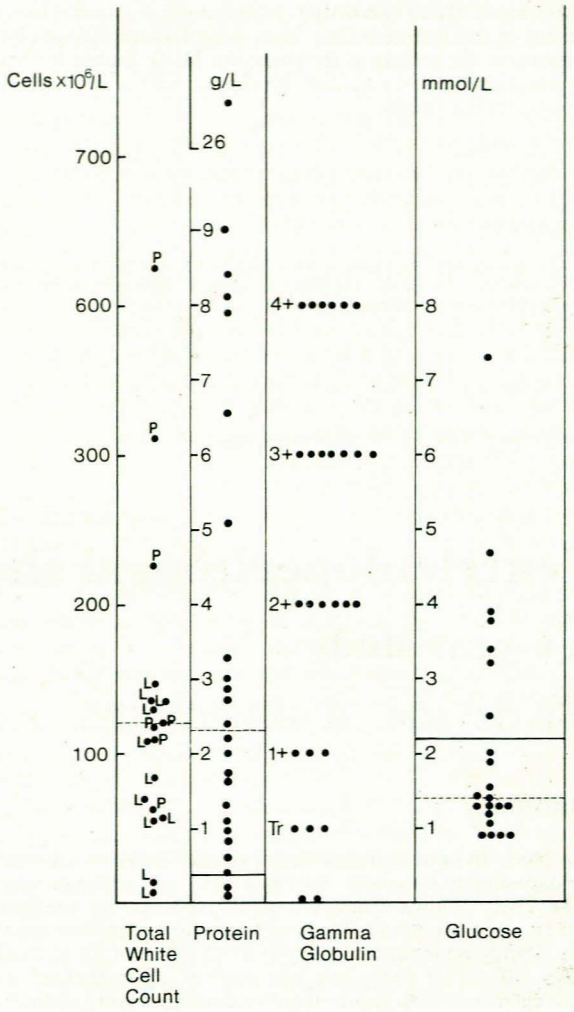

Fig. 1. Cerebrospinal fluid findings (P = polymorphonuclear cell predominance (> 50% cell count); L = lymphocyte predominance; Tr = trace; ——— = normal value; -------- = median value).

In 1 patient the site of isolation was not specified. No organisms were isolated in 3 patients with shunt infections, all of whom had tract inflammation. The organisms isolated and their antibiotic sensitivity profiles are shown in Table II. The most common organisms isolated were *S. aureus* in 18 cases (47%) and *S. epidermidis* in 10 (26%). Twelve (42%) of staphylococcal isolates were methicillin-resistant. In two episodes of shunt infection > 1 organism was isolated from pus swabs. In one a β-haemolytic *Streptococcus*, a clostridial species and *S. aureus* were isolated and in the other a *Proteus* species and *Streptococcus faecalis*. Seven patients had Gram-negative infections and all but 1 were sensitive to gentamicin. Four of 7 Gram-negative infections occurred in patients with myelomeningocele, 1 of whom, however, had a mixed Gram-positive and Gram-negative infection and was excluded from subsequent analysis. The relationship between Gram-negative infections and myelomeningocele was significant ($P = 0,048$; 2-tailed Fisher's exact test).

All patients received antimicrobial therapy. The agents most commonly used for methicillin-sensitive staphylococci were cloxacillin, sulphadiazine and co-trimoxazole and for methicillin-resistant organisms vancomycin, fusidic acid and rifampicin. Antimicrobial agents were given intrathecally through the shunt reservoir or through an Omaya reservoir in 5 patients (vancomycin in 3, gentamicin in 1 and amikacin in 1).

TABLE II. CAUSATIVE ORGANISMS AND THEIR ANTIBIOTIC SENSITIVITY PATTERNS

	Gram-positive bacteria					
	Total	Penicillin	Methicillin	Co-trimoxazole	Fusidic acid	Vancomycin
S. aureus	18	—	10	10	18	18
S. epidermidis	10	—	6	1	10	10
***Streptococcus* sp.**	3	3	—	1	—	—
	Gram-negative bacteria					
	Total	Gentamicin	Co-trimoxazole	Cefotaxime	Imipenem	
E. coli	3	3	1	2*	—	
***Acinetobacter* sp.**	2	0	0	1	1	
***Pseudomonas* sp.**	1	1	0	1	—	
***Proteus* sp.**	1	1	0	1	—	

* Not tested in 1 patient.
2 patients had polymicrobial infections (diagnosed from pus swabs).

TABLE III. SURGICAL MANAGEMENT AND OUTCOME

Group	No. of patients	Successful treatment		Relapse		Deaths	
		No.	%	No.	%	No.	%
1	8	3	37,5	3	37,5	2	25,0
2*	22	16	72,7	1	4,5	5†	22,7
3	6	2	33,3	0	—	4	66,7
4	3	2	66,7	1	33,3	0	—

* In 3 cases, shunts were not replaced as patients were found to be shunt-independent.
† Four of 5 deaths occurred before shunt replacement.

The results of the various forms of surgical treatment are shown in Table III. The most common procedure undertaken was shunt removal and later replacement, which was associated with the highest rate of cure (72,7%) and second lowest mortality rate (25%). Four of 5 deaths occurred before the insertion of a new shunt. Also of note is that 4 of 6 patients who had revisions of their shunts during medical therapy died. There were no deaths in the small group treated with primary external ventricular drainage. There were 5 relapses, 4 due to *S. aureus* and 1 due to *S. epidermidis*. Relapses occurred most commonly where medical treatment alone was attempted.

Discussion

The reported incidence of shunt infection ranges from 2% to 30%.[1-6] At present an infection rate > 5 - 7% is considered unacceptable in North America.[1] Our infection rate of 10,5% compares favourably with reported series, but is still too high.

There are no previous studies of VP shunt infection from Africa, although sepsis has been reported in patients with ventriculo-atrial shunts in Soweto, Johannesburg, and in the Sudan. The incidences of bacteraemia were 7,1% and 2% respectively.[8,9] Both studies also made note of a high incidence of hydrocephalus in the populations studied. The lack of difference between infection rate for new shunts and revisions has previously been noted.[5,6]

The time of diagnosis in relationship to the surgical procedure is also similar to that of previous studies.[3-6] The fact that 3 patients developed shunt infections between 56 months and 103 months after the shunt procedure suggests that as long as the VP shunt, a foreign body, is left *in situ*, it is at risk for becoming infected. It has been previously noted that while Gram-positive infections usually occur within 3 months of the shunt procedure, Gram-negative infections can occur at any time.[1,5] In contrast, the longest time interval in our series was, however, for an *S. aureus* shunt infection.

A CSF finding of interest in our study was that lymphocyte predominance was found in 40% of cases. Odio *et al.*[4] found that in a review of 139 CSF specimens from patients with shunt malfunction only or in CSF uninfected at the time of shunt placement there was a tendency to lymphocyte predominance. In contrast, in the presence of shunt infection, a predominance of segmented cells was noted. Frame and McLaurin[10] noted a mononuclear predominance in 1 of 7 CSF specimens associated with shunt infection. The reason for the lymphocyte predominance in our patients could be that they presented at a later stage than patients in other series. The CSF protein, glucose and white-cell estimations are similar to those reported by Odio *et al.*[4] and, in general, indicate a mild inflammatory response.

As in previous studies, we found that staphylococcal species were responsible for the majority of shunt infections. In contrast to other studies,[2-6] however, we found that *S. aureus* was responsible more often than *S. epidermidis*. This could reflect a higher skin carriage rate in children referred to our hospital. The finding that 42% of staphylococci were methicillin-resistant reflects the tendency for methicillin-resistance to be more common in hospital environments. Many of our patients were hospitalised pre-operatively for periods of up to 2 months, thus providing ample opportunity for colonisation by methicillin-resistant *S. aureus*. A survey of *S. aureus* infections at Tygerberg Hospital in 1985 showed that 48% were methicillin-resistant.[11] We were able to show a relationship between myelomeningocele and Gram-negative infections that was significant. Previous studies have shown conflicting results,

with a lack of association shown by most authors[3,6] and a positive correlation by others.[12]

Our management of shunt infection, consisting mainly of shunt removal, appropriate antimicrobial therapy and later replacement, is similar to that advocated by some authors.[1-4]

We have latterly been using external VP drainage more frequently, since this has the advantage of easy access for administration of antibiotics and for sampling of CSF for culture and determination of drug levels. Also, by allowing continuing CSF drainage, an acute increase in intracranial pressure is prevented. This mechanism may have been partly responsible for 4 of the 5 deaths that occurred in group 2 (initial shunt removal and later replacement).

A problem with intravenous administration of some antibiotics is unpredictable poor crossing of the blood-brain barrier by agents such as vancomycin and the aminoglycosides. Also, long-term intravenous cannulation, besides being technically difficult and painful, places the patient at risk for complications, such as suppurative thrombophlebitis.[13] A combination of oral rifampicin and co-trimoxazole with intrashunt vancomycin has been used successfully in a series of 11 children with shunt infection due to susceptible staphylococcal species.[10]

There is no unanimity regarding the role of prophylactic antibiotics for VP shunt surgery.[1,6] Nevertheless, the use of cefamandole at our hospital is clearly inappropriate, since it is not effective against methicillin-resistant staphylococci.[14]

The case fatality rate in our study was also similar to that reported elsewhere.[3,4] Schoenbaum *et al.*[3] found a case fatality rate of 40% in patients with shunt infection and 17% in patients with uninfected VP shunts. We did not determine the case fatality rate in this group, mainly because of poor follow-up records.

Conclusion

We have documented the incidence of VP shunt infection in children at Tygerberg Hospital. The most common organism is *S. aureus* in contrast to other reported studies where *S. epidermidis* was most common. Almost 50% of staphylococci were methicillin-resistant. These findings have bearing on appropriate antibiotic therapy for shunt infections, which should include cover for methicillin-resistant organisms until sensitivity patterns are available. In patients with myelomeningocele, Gram-negative cover should also be considered until culture results are available.

There is need for further study of both the prevention and optimal therapy of VP shunt infections.

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