

The use of the ASET in the diagnosis of ventriculoatrial shunt infection

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Summary A 38-year-old man with a ventriculoatrial shunt presented with non-specific symptoms (headache, back pain, night sweats) and inconclusive laboratory results. He showed an extremely high titre of antibody to *Staphylococcus epidermidis* which proved diagnostic of shunt infection. This was confirmed on shunt removal and he was successfully treated.

BACKGROUND

Ventriculoatrial (VA) shunting has been superseded in popularity by the use of the peritoneal route (ventriculoperitoneal (VP)), mainly because of ease of insertion of the latter. However, a substantial number are still used, either as primary insertions or to replace a persistently failing VP shunt. ^{1 – 3} In addition, a large but unknown number of patients with VA shunts remain in the community, usually without neurosurgical follow-up, ⁴ and may require medical attention at any time. VA shunt infections can be particularly difficult to diagnose because unlike VP shunt infections they often present long after shunt insertion with non-specific symptoms, and often to a specialty other than neurosurgery. Also, while VP shunt infection is often associated with distal obstruction, VA shunt infection does not usually compromise shunt function. Though *Staphylococcus epidermidis* is the most common cause of VA shunt infection it is also a universal skin commensal. Blood cultures are often negative or inconclusive and the significance of a growth of *S epidermidis* can be difficult to determine. Ventricular cerebrospinal fluid (CSF) aspirated from the shunt reservoir can also show no evidence of infection when only the shunt hardware is colonised, and there remains a diagnostic problem.

Almost all VA shunt infections are contracted at the time of shunt insertion. *S epidermidis* is very adherent to shunt tubing, forming a biofilm inside the shunt catheter, ^{5 – 7} which periodically sheds into the bloodstream, repeatedly inoculating the patient. This causes a rising antibody titre to *S epidermidis*, which eventually leads to immune complex deposition and the problems that are associated with this, such as shunt nephritis. ⁸ If a VA shunt is infected for more than a year, immune complex disease can be the presenting problem, but it often manifests with non-specific or misleading symptoms such as vague joint or muscle pains, tiredness or skin rash. Here we present a patient with a VA shunt and non-specific symptoms and inconclusive laboratory results, where the diagnosis was eventually made with the assistance of serological testing.

CASE PRESENTATION

The patient, a 38-year-old man with a Dandy Walker cyst and shunted hydrocephalus, had required several shunt revisions for malfunction and for infection as a child, the last one before this episode being at the age of nine, since when he had had a VP shunt. He was well until the age of 35, when he presented with headaches and abdominal pain. He underwent several revisions over the next year, but he continued to have abdominal pain so his shunt was revised to a VA shunt. The procedure was straightforward. He was asymptomatic when reviewed 3 months postoperatively.

However, 7 months from the time of insertion he presented with severe headaches which were managed symptomatically. His symptoms resolved and he remained well for several months. After a further 4 months he presented with headaches and night sweats, and he was investigated for a possible shunt infection.

INVESTIGATIONS

CT scan, shunt series and inflammatory markers were all unremarkable at this time. In view of this, and because his night sweats resolved, a shunt infection was thought unlikely. He was admitted one month later, 17 months after VA insertion, with a short history of increased headache, back pain and night sweats. He now had raised inflammatory markers (C reactive protein 73.8 mg/l, erythrocyte sedimentation rate 33 mm/h), though white cell count ($7.6 \times 10^9/l$), blood urea (5.6 mmol/l) and creatinine (70 μ mol/l) remained normal. Renal function and serum complement levels were normal, excluding shunt nephritis. Blood cultures grew *S epidermidis* which was considered by the laboratory to be a contaminant. CSF obtained by lumbar puncture showed no growth but a raised protein level (949 mg/l). Despite this, the clinical suspicion of VA shunt infection was high but there was reluctance to explore his shunt without definite evidence of infection. Blood was taken for an anti *S epidermidis* titre (ASET) and he was started on intravenous antibiotics awaiting the result. The titre was >40 000 (normal adult range 160–640), supporting the clinical suspicion of shunt infection.

TREATMENT

He therefore proceeded to shunt removal and external ventricular drain insertion. Ventricular CSF taken perioperatively showed Gram positive cocci on microscopy but no growth on culture. However, the shunt hardware grew *S epidermidis*, confirming shunt infection. The patient was treated with a course of intravenous antibiotics, following which a new shunt was inserted. VA shunting was attempted but was unsuccessful so a ventriculo-pleural shunt was inserted instead.

OUTCOME AND FOLLOW-UP

He was discharged the following week and 1 year later remained well.

DISCUSSION

The ASET test, 59 available for almost four decades but not widely used, detects antibodies to *S epidermidis* that are formed as a result of inoculation from the distal catheter of the colonised VA shunt. The technology is simple, involving agglutination in microtitre trays. Serum ASET levels rise with age in a predictable manner, and normal ranges have been established. 10 The titre is usually <160 in children under 3 years of age. In adults, the normal range is 160–640. Titres in infected cases often rise to 2560–40 000, providing clear

diagnostic discrimination. In some cases, a rise in titre is more useful than the actual value itself.

Our case describes a typical course of a patient with VA shunt infection, and highlights the difficulties in obtaining a definite diagnosis of infection. The patient initially presented with non-specific symptoms 7 months after shunt insertion. These symptoms resolved without treatment, but recurred at 12-months from shunt insertion. Again, these symptoms greatly improved without treatment. The possibility of shunt infection was considered, but ruled out because the symptoms resolved without intervention. However, 17 months after shunt insertion the patient deteriorated. At this point, positive blood cultures were dismissed as contaminated and lumbar CSF cultures were negative. Only when a greatly elevated ASET result of >40,000 was obtained was it felt that there was sufficient evidence of infection to warrant shunt removal. Infection was confirmed when *S epidermidis* was grown from the shunt hardware following removal. The patient recovered fully after shunt removal and antibiotics. In this case, the diagnosis was established and the infection treated before the patient developed immune complex disease, which occurs after prolonged hyperimmunisation. 8 Immune complex diseases occur as a late complication of VA shunt infection. However, patients who develop immune complex disease may present with a wide variety of non-specific symptoms, including rash, cough, joint pains, anaemia or haematuria. 11 This presentation can result in a delay in diagnosis, partly because patients are often referred to the wrong specialty to investigate one of these symptoms. A positive ASET test can help establish the diagnosis long before immune complex disease occurs. 10

The ASET test can also be used as a screening tool for VA shunt infection. If the test is performed at the time of shunt insertion, this will establish a baseline level. If future tests show the level to have risen, this is indicative of shunt infection and prompt intervention can be carried out.

In summary, diagnosis of VA shunt infection was typically difficult in our patient but was aided by the ASET test, allowing treatment of the shunt infection and precluding development of immune complex disease. The ASET test is a useful diagnostic tool when VA shunt infection is suspected, and can also be used for screening and early detection of shunt infection.

- ▶ People with VA shunts still present with complications, sometimes years after shunting.
- ▶ Clinical features of VA shunt infection can be nonspecific and misleading.
- ▶ A high level of suspicion of shunt infection is essential.
- ▶ The ASET test can aid diagnosis.

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Competing interests None.

Patient consent Obtained.

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