Short report

Ventriculitis from Acinetobacter calcoaceticus variant anitratus

R A M Y O G E V

From the Division of Infectious Disease, The Children's Memorial Hospital, Chicago, Illinois, USA

SUMMARY Ventriculitis from infection with Acinetobacter calcoaceticus variant anitratus occurred in an infant two weeks after surgical repair of a lumbosacral meningo(myelo)cele and ventriculoperitoneal shunting. Recovery took place on replacing the shunt with an extra-ventricular deviation device and giving gentamicin and ampicillin intravenously.

Acinetobacter calcoaceticus variant anitratus (syn B3W, Bacterium anitratum, Herellea vaginicola) is a short Gram-negative pleomorphic rod which is widely distributed in nature (Baumann, 1968) and frequently colonises various sites in humans (Rosenthal, 1974). Several clinical reviews (Robinson et al., 1964; Glew et al., 1977) describe the range of infections caused by this bacterium which include respiratory and urinary tract infections, septicaemia, endocarditis, and abscesses in different sites. Infections of the central nervous system are seldom reported. Most infections occur in old patients, patients with severe or chronic diseases, or after surgery, instrumentation, and antibiotic treatment. We recently saw a neonate who developed ventriculitis from A. calcoaceticus variant anitratus.

Case report

A baby girl (HK) was admitted to Children's Memorial Hospital at 7 hours of age for repair of a lumbosacral meningo(myelo)cele. The pregnancy and delivery had been normal. On admission, she was alert, with head circumference of 335 mm, and a soft anterior fontanelle (20×20 mm). There was a full range of passive movement in the lower extremities and anal hypotonia. An intact lumbosacral meningo(myelo)cele was noted. She underwent surgical reconstruction of the spinal cord and repair of the meningo(myelo)cele. A week after surgery, the anterior fontanelle was noted to be full, and some leakage from the repair site was found. Hydrocephalus was seen on angiography. A right ventriculoperitoneal (VP) shunt was placed, but had to be revised a week later because the peritoneal tip was plugged. A week later the child developed fever (38.5°C) and again the anterior fontanelle became full. The shunt was removed and a left extraventricular deviation (EVD) device was placed. The ventricular fluid had 16 WBC/mm³ (100% mononuclear cells), a protein level of 0.21 g/l and a glucose level of 2.4 mmol/l. Cultures of the CSF from the time of surgery and the first two days after operation were negative. On the third day, the peripheral WBC increased from 12300 to 23000/mm³ (43% polymorphonuclear, 26% bands), the ventricular fluid became turbid demonstrating 2645 white blood cells per mm³ (75% polymorphonuclear leucocytes and 25% mononuclear leucocytes) with protein level of 2.58 g/l and glucose level of 2.3 mmol/l. However, she remained clinically well. Cultures grew A. calcoaceticus variant anitratus. With the Kirby-Bauer disc method, the bacterium was sensitive to polymyxin B, tetracycline, kanamycin, and gentamicin. Treatment was begun with intravenous gentamicin 7.5 mg/kg/day and ampicillin 300 mg/kg/day. For the next six days the cultures remained positive although there was marked improvement in the ventricular fluid with the protein level decreasing to 0.43 g/l and cell count to 8 WBC/mm³. After the EVD was replaced on the ninth day, the cultures became negative and remained so for the next 10 days. Antibiotics were stopped, and after three days of negative cultures, a new VP shunt was placed.

Address for reprint requests: Dr Ram Yogev, Division of Infectious Disease, The Children's Memorial Hospital, 2300 Children's Plaza, Chicago, Illinois 60614, USA.

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Discussion

*A. calcoaceticus* variant *anitratus* was first described by Debord in 1939, as *Herellea vaginicola*. This is a Gram-negative coccobacillus which grows well on most media aerobically, does not ferment 1% lactose, but forms acid without gas in 10% lactose. Nitrites are not reduced to nitrates and it is non-motile (Daly et al., 1962; Lothe and Griffin, 1965). On Gram-staining this bacterium is sometimes confused with *Neisseria meningitidis* when diplococcal forms predominate. The presence of long filaments or rods in association with Gram-negative diplococci suggest *A. calcoaceticus* variant *anitratus*.

In reviewing the English literature, 12 cases of infections of the CNS with *A. calcoaceticus* have been reported (Table). Ten cases of meningitis occurred in patients ranging in age from 4 days to 57 years. Four of them died. One patient had a cerebral abscess and one had ventriculitis.

The pathogenesis of the illness in this child may be similar to shunt infections caused by *Staphylococcus epidermidis*. Bayston and Lari (1974) showed that *S. epidermidis* frequently colonises the scalp and may be found in the incision site despite meticulous preoperative skin cleansing. This observation suggests that *S. epidermidis* ventriculitis after shunt procedures results from transmission of bacteria from the skin into the ventricles during the operation. *A. calcoaceticus* is a frequent coloniser of the skin (Taplin et al., 1963; Greer et al., 1965) and an analogous sequence of events would explain the pathogenesis of our patient's ventriculitis.

The choice of antibiotic should be based on in vitro susceptibility tests and the ability of the drug to cross the blood-brain barrier. Sensitivity patterns have been variable with ampicillin, sulphonamides, tetracyclines, and streptomycin. Almost all strains of *A. calcoaceticus* have been found to be resistant to penicillin and chloramphenicol (Daly et al., 1962; Glew et al., 1977). Most studies have demonstrated susceptibility to polymyxin B, colistin, kanamycin, gentamicin, and minocyclin. Glew et al. (1977) reported that carbenicillin and an aminoglycoside are synergistic for most strains which were resistant to aminoglycosides alone. Therefore, it seems that the most reliable antimicrobial agents for *A. calcoaceticus* infections are aminoglycosides alone or in combination with carbenicillin.

*A. calcoaceticus* is a rare cause of central nervous system infections, but if this bacterium is growing from cerebrospinal fluid after head surgery or injury (Deacon, 1945) it should be considered as the pathogen and appropriate antibiotic treatment started.

I would like to acknowledge the Division of Neurosurgery of The Children’s Memorial Hospital for allowing me to study this patient.

Table 1  
*CNS infections caused by acinetobacter calcoaceticus variant anitratus*

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age</th>
<th>Sex</th>
<th>Disease</th>
<th>Outcome</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1 year</td>
<td>F</td>
<td>Meningitis</td>
<td>Recovered</td>
<td>Lothe and Griffin (1965)</td>
</tr>
<tr>
<td>2</td>
<td>1 month</td>
<td>M</td>
<td>Meningitis</td>
<td>Recovered</td>
<td>Lothe and Griffin (1965)</td>
</tr>
<tr>
<td>3</td>
<td>4 days</td>
<td>F</td>
<td>Meningitis</td>
<td>Recovered</td>
<td>Watson (1957)</td>
</tr>
<tr>
<td>4</td>
<td>6 days</td>
<td>M</td>
<td>Meningitis</td>
<td>Died</td>
<td>Watson (1957)</td>
</tr>
<tr>
<td>5</td>
<td>NR</td>
<td>NR</td>
<td>Meningitis</td>
<td>Died</td>
<td>Ray et al. (1951)</td>
</tr>
<tr>
<td>6</td>
<td>NR</td>
<td>NR</td>
<td>Meningitis</td>
<td>Died</td>
<td>Ray et al. (1951)</td>
</tr>
<tr>
<td>7</td>
<td>NR</td>
<td>NR</td>
<td>Meningitis</td>
<td>Recovered</td>
<td>Ray et al. (1951)</td>
</tr>
<tr>
<td>8</td>
<td>57 years</td>
<td>F</td>
<td>Meningitis</td>
<td>Recovered</td>
<td>Green (1960)</td>
</tr>
<tr>
<td>9</td>
<td>55 years</td>
<td>M</td>
<td>Meningitis</td>
<td>Recovered</td>
<td>Daly et al. (1962)</td>
</tr>
<tr>
<td>10</td>
<td>29 years</td>
<td>M</td>
<td>Meningitis</td>
<td>Died</td>
<td>Daly et al. (1962)</td>
</tr>
<tr>
<td>11</td>
<td>65 years</td>
<td>M</td>
<td>Ventriculitis</td>
<td>NR</td>
<td>Daly et al. (1962)</td>
</tr>
<tr>
<td>12</td>
<td>28 years</td>
<td>M</td>
<td>Cerebral abscess</td>
<td>Died</td>
<td>Watson (1953)</td>
</tr>
</tbody>
</table>

NR = not reported.

References


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Short report: Venticulitis from Acinetobacter calcoaceticus variant anitratus
Ram Yogev

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