Brain abscess due to *Wolinella recta* and *Streptococcus intermedius*

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ABSTRACT: The authors present a case of a 62-year-old female with a frontal lobe brain abscess caused by *Wolinella recta* and two strains of *Streptococcus intermedius*. One of the strains of *S intermedius* was resistant to penicillin. Resolution of the abscess required 60 days of antibiotic therapy and aspiration on four oc-

casions. The source of the brain abscess could not be determined. This is the first case in which *W recta* has been isolated from a brain abscess. **Can J Infect Dis 1990;1(1):31-34**

Key Words: Brain abscess, *Streptococcus*, *Wolinella*

BRAIN ABSCESS IS A RARE BUT SERIOUS CONDITION. And despite advances in antibiotic therapy and neurosurgical techniques, the mortality and morbidity associated with this illness remain high (1-3). Delay in diagnosis and, in some instances, difficulty in treating the infecting microorganisms, are factors leading to the high morbidity and mortality (3). In this paper a patient with brain abscess due to *Streptococcus intermedius* and *Wolinella recta* is described. One of the two strains of *S intermedius* was resistant to penicillin. This is the first case in which *W recta* has been implicated as a causative organism in a brain abscess.

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CASE REPORT

A 62-year-old female was admitted to the authors' institution on June 22, 1983 with a one week history of headache associated with nausea and vomiting, anorexia and chills. Two days prior to admission she was noted by her family to be apathetic and withdrawn, and one day later she was drowsy and disoriented. There was no history of sinusitis, otitis or trauma to the face or head.

On examination she was febrile with a temperature of 38°C, drowsy and had weakness of the right arm and leg. Her teeth were in good condition and there was no tenderness over the sinuses.

A computed axial tomography scan showed a contrast-enhancing ring lesion in the left frontal area (Figure 1). The radiologist considered a tumour to be the most likely cause of this lesion.

On the third hospital day a needle biopsy of this lesion yielded pus, Gram stain of which showed Gram-positive cocci in chains and Gram-negative rods. Penicillin G 2,000,000 units every 2 h, and

Figure 1) Computed axial tomographic scan of brain abscess done on the first hospital day. Note the large contrast-enhancing ring lesion in the left frontal lobe. Also note compression of the left lateral ventricle and shift of the midline structures to the right

intravenous chloramphenicol 1 g every 8 h was begun. The Gram-positive organisms were identified as *S. intermedius* strains I and II. Strain I was resistant to penicillin with a minimal inhibitory concentration of 2 µg/mL. The concentrations of other antibiotics required to inhibit this isolate were: ampicillin 1 µg/mL; clindamycin <0.12 µg/mL; vancomycin ≥32 mg/mL. Strain II was susceptible to penicillin. The Gram-negative rod was identified as *W. recta*.

Initially the patient improved and the abscess decreased in size. However by the 20th hospital day the white blood cell count fell from $13 \times 10^9/L$ on admission to $2.4 \times 10^9/L$. The chloramphenicol was discontinued. Six days later the abscess had increased in size. The lesion was aspirated for the third time on the 28th hospital day, and despite the increase in size, no organisms were recovered from the pus. Because of delayed resolution, intravenous clindamycin 600 mg every 6 h for 72 h and 300 mg every 6 h thereafter was begun. Repeat aspiration on the 37th hospital day yielded 25 mL of culture-negative pus. Both penicillin and clindamycin were continued until the 61st hospital day. A scan on the 54th hospital day showed almost complete resolution of the lesion.

Additional investigations performed to look for a source of the abscess included roentgenographic films of the sinuses, chest and teeth, all of which were normal. A ^{67}Ga scan showed uptake of the isotope in the brain abscess only.

Brain abscess pus was plated on blood agar plates (trypticase soy agar; BBL Microbiology Systems, Cockeysville, Maryland) containing 5% sheep blood and incubated aerobically at 37°C for 18 h. In addition, the specimens were inoculated onto a prereduced Brucella blood agar plate containing 10 µg of vitamin K₁/mL and 5% sheep blood, and onto a prereduced kanamycin-vancomycin-laked blood agar plate containing a Brucella agar base, 5% laked sheep blood, 10 µg of vitamin K₁/mL, 100 µg of kanamycin/mL and 7.5 µg of vancomycin/mL. These plates were incubated anaerobically in Gas Pak jars (BBL Microbiology Systems) for 48 h. Organisms growing anaerobically were inoculated into 5 mL chopped-meat carbohydrate medium (prereduced and anaerobically sterilized) containing 5 µg of hemin and 0.1 µg of vitamin K₁/mL for subsequent inoculation of biochemicals, and into peptone-yeast extract with 1% glucose for gas-liquid chromatography. All organisms were identified by reference to the Virginia Polytechnic Institute's anaerobe laboratory manual (4).

The anaerobic Gram-negative rod tentatively identified as *W. recta* was referred to the Laboratory Centre for Disease Control, Ottawa for confirmation of identification, and it was confirmed as *W. recta*. This organism was motile and hydrolyzed gelatin. Nitrate was reduced, benzidine was positive and end products consisted of hydrogen sulphide and succinic acid. Urease and oxidase tests were negative. The characteristics of this isolate were consistent with those of *W. recta* (5).

Minimal inhibitory concentrations of penicillin, ampicillin, clindamycin and vancomycin were determined against the isolates of *S. intermedius* using commercially prepared microtitre plates containing increasing twofold concentrations of the above antimicrobials (Sensititer; Gibco Diagnostics, Burlington, Ontario). The organism was inoculated into Mueller-Hinton broth and incubated at 35°C for 18 h.

Turbidity was adjusted to that of a 0.5 McFarland standard. Then a 1:1000 dilution was prepared in Mueller-Hinton broth containing 0.5 mL horse blood. Fifty microlitres of this was added to the wells of the sensititre plate and incubated for 18 h at 38°C. The minimal inhibitory concentration was the well containing the lowest concentration of antibiotic that showed no visible growth. In addition, a well containing no antibiotic was inoculated to serve as a growth control and

Staphylococcus aureus ATTC 29213 and *Streptococcus fecalis* ATTC 29212 were used to ensure that the appropriate minimal inhibitory concentration of control organisms was obtained. *W. recta* could not be grown for susceptibility studies.

DISCUSSION

Wolinella is the name of a new genus which includes anaerobic, asaccharolytic, rod-shaped bacteria with a single polar flagellum (5). There are two species, *W. succinogenes* and *W. recta*, which have been isolated from humans with periodontal disease (5). Frontal lobe abscesses have been linked with covert dental sepsis (6,7), but the present patient had good dental hygiene, and roentgenographic studies of her teeth failed to show any abscesses. Spiegel and Telford (8) reported the first case of extraoral *W. recta* infection in a patient with a chest wall abscess due to *W. recta* and *Actinomyces viscosus*. This patient had poor oral hygiene.

The growth of *W. recta* is stimulated in peptone-yeast extract broth supplemented with formate and fumarate but not with 2% rabbit serum. It is oxidase-positive and motile, has a single polar flagellum and produces hydrogen sulphide (5,8). Tanner et al (5) tested six isolates and found that the minimal inhibitory concentrations of penicillin ranged from less than 0.025 µg to 4 µg/mL.

S. intermedius is an aerotolerant, slowly growing, nonhemolytic streptococcus often requiring either the addition of carbon dioxide or anaerobic conditions for its initial isolation (9). Lactose-positive strains are classified as *S. intermedius*, while lactose-negative strains are *Streptococcus anginosus-constellatus*. These two groups of organisms are synonymous with what has been termed *Streptococcus milleri* in the United Kingdom (9). *S. intermedius* (*S. milleri*) is a very common isolate from brain abscesses (2,9). These organisms are usually susceptible to penicillin (6), but not always, as exemplified by the present patient. Unfortunately the authors did not save the isolate of *S. intermedius* to confirm that it was resistant to penicillin and vancomycin.

Penicillin-resistant viridans streptococci may occur in patients who are receiving chronic penicillin therapy, or may arise de novo.

Penicillin-resistant isolates of viridans streptococci were recognized in the 1960s as part of the gingival flora of patients who were receiving penicillin prophylaxis for rheumatic fever (10-12). Such isolates have also been found to colonize the pharynx in South African children who were infected with multiply resistant pneumococci (13). Disease due to penicillin-resistant viridans streptococci remains uncommon; only five cases of

endocarditis have been reported due to these microorganisms (14-16). Recently Quinn et al (17) described a 50-year-old female with a subarachnoid hemorrhage and an epidural hematoma who required ventricular and subgaleal drains and developed meningitis due to a penicillin-resistant isolate of *S. intermedius* (minimal inhibitory concentration 4 µg/mL) (17). The mechanism of resistance in these strains is an alteration in penicillin-binding proteins – there is a diminished affinity for penicillin of the penicillin-binding proteins compared with those of susceptible strains (17). Generally these penicillin-resistant isolates of viridans streptococci have been susceptible to vancomycin – this was not so with the present isolate.

Vancomycin resistance among Gram-positive bacteria is rare (18). *Leuconostoc* species, some lactobacilli and *Pediococcus* species (19-21) are the most commonly resistant microorganisms. It has been stated that vancomycin-resistant isolates showing cell and colony morphology suggestive of viridans group streptococci are probably lactobacilli (19). It is possible that the authors' *S. intermedius* isolate was indeed a lactobacillus or a *Leuconostoc* strain; however plasmid-mediated vancomycin resistance in *Enterococcus faecium* has been transferred into *Streptococcus sanguis* (22). It is thus possible that we will see more isolates of viridans streptococci resistant to vancomycin.

Penetration of brain tissue and brain abscess pus by antibiotics is important in the treatment of brain abscesses. Penicillin (23, 24) and chloramphenicol (23,25) both achieve adequate levels in intracranial pus to treat susceptible pathogens. Chloramphenicol is primarily a bacteriostatic antibiotic which is suboptimally active in an anaerobic environment (26). The authors continued chloramphenicol in an attempt to treat penicillin-resistant *S. intermedius*. The abscess increased in size when chloramphenicol was discontinued because of leukopenia. Shortly thereafter therapy with clindamycin was instituted, to which the *S. intermedius* was very susceptible (minimal inhibitory concentration <0.12 µg/mL). Although clindamycin penetrates brain tissue poorly (27), the addition of this antibiotic combined with repeated aspiration resulted in cure.

De Louvois (28) has stated that cryptogenic brain abscesses are usually caused by penicillin-susceptible organisms, and that high dose penicillin is adequate pending culture results. This case illustrates the necessity for prompt identification and determination of antimicrobial susceptibility of all microorganisms isolated from a brain abscess.

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