Ventriculoperitoneal Shunt Infection Caused by \\

*Actinomyces neuii* subspecies *neuii*

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*Actinomyces neuii* ssp. *neuii* is a rare isolate in clinical specimens. The organism was previously designated CDC coryneform group 1 and was renamed in 1994. A case of a ventriculoperitoneal shunt infection caused by this organism is described.

A 64-year-old woman had a ventriculoperitoneal shunt (VP shunt) inserted in 1995 after a cerebral aneurysm. She presented in October 2007 with one month of vomiting and increasing difficulty with balance. She had been having low-grade fevers, up to 37.8°C. She denied headaches, blurry vision or neck stiffness. One week before admission she had a computed tomography (CT) scan of the head done which showed ventriculomegaly and the VP shunt in good position. The day prior to admission she developed worsening nausea and fell. No other symptoms of note were elicited. There was a history of left-sided hemiplegia following the cerebral aneurysm, hypothyroidism, depression, hypertension, mitral valve prolapse, and cataracts. Her home medications included anti-hypertensives, thyroid replacement and an antidepressant. She was a
On examination, she was afebrile (36.6°C) and non-toxic appearing. Her dentition was good and there were multiple fillings. There was no tenderness over the VP shunt. Her neck was supple with full range of motion. She was flaccid on the left side. Initial hematological investigations showed a white blood cell count of 10.5 X, 10⁹/liter and a platelet count of 296 X 10⁹/liter. Two sets of blood cultures were obtained. She underwent lumbar puncture in the emergency department. The first tube of cerebral spinal fluid showed 154 white blood cells, 14% segmented neutrophils and 80% lymphocytes. The Gram stain was initially read as Gram variable bacilli. The next day growth was observed on the sheep blood agar plates. A repeat Gram stain of the isolate showed Gram positive rods that were catalase-positive and observed to be non-hemolytic. The organism was then sent to the reference section of the lab for further work-up.

At the reference lab, the following biochemical reactions were positive: catalase, nitrate, mannitol, maltose, trehalose, pyrazinamidase, CAMP test, ornithine decarboxylase, fructose, glucose, lactose, sucrose, alpha glucosidase, β galactosidase, and trehalose. The negative tests included alkaline phosphate, urease, β glucuronidase, salicin, rhamnose, arabionose, 6% salt, citrate, motility, gelatin, methyl red, and indole. The organism grew poorly under anaerobic conditions but well in 6% CO₂. These results lead to the identification of the organism as *Actinomyces neuii* spp. *neuii*. API Coryne V3.0 confirmed at 99% the identification of *A. neuii* ssp *neuii*. A broth microdilution was performed using lysed horse blood and the organism was found to have very low MIC’s to the following β-lactam antibiotics: penicillin, ≤0.06 µg/ml,
amoxicillin, ≤ 1.0 µg/ml, ceftriaxone, ≤ 0.25 µg/ml, cefazolin, ≤ 4.0 µg/ml; to erythromycin, ≤ 0.12 µg/ml, to tetracycline, ≤ 1.0 µg/ml; ≤ 0.015 µg/ml to clindamycin; and ≤ 0.5 µg/ml to vancomycin. The MIC to levofloxacin was 1.0 µg/ml.

To our knowledge, this is the first reported case of the organism isolated from cerebral spinal fluid since it was renamed in 1994.

Based on the initial labs and gram stain, the patient was started on vancomycin, cefepime, ampicillin, and metronidazole. Her blood cultures remained sterile throughout her hospital course. On hospital day number three, her VP shunt was removed and a temporary ventriculostomy drain was placed. The culture of the shunt grew over one hundred colonies and a Gram stain of one of the colonies showed a Gram variable rod. The culture was similar to the initial CSF culture based on Gram stain, morphology and catalase and was not further evaluated. On hospital days number five and eleven repeat cerebral spinal fluid cultures were done and remained sterile. On hospital day number 15 a new VP shunt was inserted and the temporary shunt was removed. On hospital day number 18 the original isolate was identified as *Actinomyces neuii* spp. *neuii*. Her antibiotics were changed to penicillin G 24 million units daily by continuous infusion for six weeks. She was discharged to home and seen in follow up in the office. All of her symptoms had resolved and she remained afebrile. After completion of the intravenous therapy she was switched to oral penicillin for a planned six month course and has done well in follow-up.

Upon further questioning the patient recalled that she had a dental cleaning with teeth scraping about three months before her symptoms began. However, she took amoxicillin prior to the procedure for prophylaxis. We did not attempt to isolate the
organism from her mouth.

*Actinomyces neuii* spp. *neuii* and *Actinomyces neuii* spp. *anitratus* were previously known as CDC coryneform group 1 and group 1-like strains. In 1994 the 16S rRNA genes were amplified *in vitro*, and their nucleotide sequences were directly determined (5). Comparative sequence analyses showed that the CDC group 1 and group 1-like strains are members of the genus *Actinomyces*. *A. neui* was named in honor of Dr. Harold Neu, an infectious disease and antibiotic expert. The organism is a gram-positive bacilli, catalase-positive, and forms nonhemolytic colonies. The colonies appear on agar plates as circular, smooth, convex, and white with entire edges. It grows under both aerobic and facultative anaerobic conditions. It is nonspore-forming and does not branch nor form sulfur granules like other species of *Actinomyces*.

Members of the genus *Actinomyces* have been isolated from a number of clinical specimens including blood, wound, bone, abscesses, bronchial washes, gallbladder fluid, pleural fluid, and urine (2). Prior to its renaming in 1994 one report listed a shunt fluid culture with CDC group 1, although the age and clinical outcome of the case was not described (4). More recent reports describe *Actinomyces neuii* as a cause of neonatal sepsis (8), endophthalmitis (6,9,10), infective endocarditis (3), pericarditis (7), chronic osteomyelitis (11), and a mammary prosthesis infection (1).

Our patient had dental work done approximately 3 months prior to her symptoms, which is the most likely source for her infection. *Actinomyces* are part of the oral microflora of humans and animals. Several species are prevalent in plaque specimens from adults with periodontitis and gingivitis. Antibiotic susceptibility includes β-lactams, clindamycin, erythromycin, rifampin, tetracycline, and vancomycin.
Duration of therapy is individualized and prolonged, usually 3-6 months. As noted in the case report our patient had good dentition at the time of the diagnosis of the shunt infection. No information is available about the condition of her dentition prior to the dental work. It is important to understand that antibiotic prophylaxis is not 100% effective in preventing subsequent infection arising from the oral cavity but does decrease the risk. Our patient did not have any other identifiable source for the *Actinomyces naevis*.

In conclusion, *Actinomyces naevis* should be considered in the differential diagnosis of patients with CSF shunt infections, especially in the setting of recent dental procedures. Based on this case we recommend complete removal of VP shunt hardware followed by prolonged antibiotic therapy when *Actinomyces naevis* is isolated from CSF.

References