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Veillonella parvula Discitis and Secondary Bacteremia: a Rare Infection Complicating Endoscopy and Colonoscopy?▽

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We report a case of *Veillonella parvula* lumbar discitis and secondary bacteremia confirmed by molecular characterization of the 16S rRNA genes. Identification of the organism was essential for an appropriate choice of antimicrobial therapy following the failure of empirical flucloxacillin. *Veillonella* spp. are normal flora of the gastrointestinal tract, raising the possibility that an endoscopy and colonoscopy performed 8 weeks prior to presentation, during which small intestinal and rectal biopsies were obtained, was the portal of entry. This case highlights the importance of obtaining a microbiologic diagnosis, particularly in patients who previously have had procedures involving instrumentation.

CASE REPORT

A 55-year-old school headmaster presented to the Emergency Department of St. Vincent's Hospital, Sydney, Australia, with a 48-h history beginning with the sudden onset of severe lower back pain. There was no history of injury or trauma. The pain was associated with night sweats, and he described an episode of shaking consistent with rigors. There was no significant past medical history and no recent dental procedure was reported. He had undergone a routine endoscopy and colonoscopy for a slight change in bowel habits 2 months prior to presentation. There were no abnormal findings, but a small bowel biopsy and a rectal biopsy were obtained to exclude celiac disease and histologic colitis.

On examination, the patient was initially afebrile, although his temperature rose to 38°C that night. No heart murmurs were present, and there were no peripheral stigmata of infective endocarditis. Dentition was normal, and no gingivitis was noted. Severe back pain was precipitated by minimal movement of the legs and trunk.

On examination, moderate tenderness was noted over the lumbar spine and left lumbar paravertebral region. The neurological examination was limited by pain but was considered to be normal. The neutrophil count was 4.6×10^9/liter, but the erythrocyte sedimentation rate was 68 mm/h and the C-reactive protein (CRP) level was 211 mg/liter. An X-ray examination of the lumbar spine revealed degenerative changes at L3, L4, and L5. During the next 12 h the pain worsened, requiring narcotic analgesia, and urinary retention developed, necessitating bladder catheterization and an indwelling catheter. Two sets of blood cultures were collected, and antimicrobial therapy was withheld pending a magnetic resonance imaging scan, which was performed 18 h after admission. The magnetic resonance imaging scan revealed multilevel disc destruction with posterior disc extrusions most severe at the L2/3 level, resulting in compression of the thecal sac and central canal stenosis at this level. No bony destruction was noted.

Blood cultures remained negative 48 h after admission. A computed tomography-guided biopsy of the L2/3 disc was undertaken, with three fine-needle aspirate biopsies obtained using a 22-gauge needle. Following the biopsy, the patient was started on flucloxacillin and given a single 1-g dose of ceftriaxone. Within 24 h, fever and pain began to improve and the indwelling urinary catheter was removed. However, during the next 72 h, fever and pain again worsened and reduced oxygen saturation in conjunction with bibasal pulmonary collapse/consolidation developed. Six days after admission, a gram-negative anaerobic coccus was isolated from the L2/3 disc aspirate.

The antimicrobial therapy was changed to cefotaxime, which resulted in rapid defervescence. A dental examination performed at this time was unremarkable. The patient was discharged on day 10 to continue ceftriaxone at 2 g/day as an outpatient. On day 20 he was progressing well, and the CRP level had fallen to 80 mg/liter. By day 42 the CRP had fallen to 8.8 mg/liter, and intravenous antibiotics were ceased. Amoxicillin-clavulanic acid (Augmentin duo forte) was administered to complete a total of 3 months of antimicrobial therapy. He was clinically cured on follow-up 6 weeks later, and the CRP level was 2.3 mg/liter.

Microbiology. Three fine-needle aspirate specimens from the L2/3 disc space were forwarded to the Microbiology Department for routine microbiological analysis. The Gram stain of the disc aspirate revealed polymorphonuclear leukocytes, but no organisms were seen. Horse blood agar (Oxoid, Australia) and chocolate agar (Oxoid, Australia) were inoculated and incubated for 7 days at 35°C under 5% CO_2 atmospheric conditions. A brain heart infusion agar plate (Oxoid, Australia) was incubated at 35°C anaerobically for 7 days. On day 3 a heavy pure growth of a tiny gram-negative coccus was observed on the anaerobic culture plate. No growth occurred on the aerobic plates, and no other pathogens were isolated despite prolonged incubation.

Blood cultures were processed using the BacTAlert system (Biomerieux, Marcy, France). On day 4, the anaerobic blood culture yielded a slow-growing, gram-negative, nonmotile coccus. The blood culture was subcultured onto brain heart infu-

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TABLE 1. Previously reported cases of discitis or vertebral osteomyelitis caused by *Veillonella* species

No. of patients (source or reference)	Age (yr)/sex[a]	Culture result		Underlying disease/risk factors	Antibiotic therapy	Outcome
		Blood	Bone/disc			
1 (this study)	55/M	*V. parvula*	*V. parvula*	Colonoscopy and endoscopy	Ceftriaxone	Cure
1 (20)	61/F	*V. parvula*	*V. parvula*	Sjogren's syndrome, xerostomia	Ceftriaxone	Cure
1 (2)	74/M	Not reported	*V. parvula*	None	Penicillin	Cure
1 (7)	70/M	Not reported	*V. parvula*	None	Unspecified	Cure
1 (9)	27/M	Not reported	*Veillonella* sp.	None	Amoxicillin	Cure

[a] M, male; F, female.

sion agar and incubated under anaerobic conditions. Small colonies identical to those from the disc aspirate grew after 48 h and were presumptively identified as a *Veillonella* species by anaerobic growth requirements. Further identification and susceptibility testing were unable to be performed, as the subcultured isolates failed to grow and the significant time delay before attempting to reisolate organisms from the blood culture bottle rendered them nonviable.

Genomic DNA was extracted from both of the clinical isolates by using a QIAamp DNA minikit (QIAGEN, Hilden, Germany), and DNA amplification of the 16S rRNA gene complex was followed by sequencing as previously described (17). The PCR products were purified using the QIAquick PCR purification kit (QIAGEN, Hilden, Germany) as per the manufacturer's instructions. The PCR products were sequenced directly in both directions on an ABI Prism 3730 automated sequencer at the SUPAMAC facility (Royal Prince Alfred Hospital, Sydney, Australia). The sequences were compared to those available in the GenBank databases by using the BLASTN program run on the National Center for Biotechnology Information server (http://www.ncbi.nlm.nih.gov/BLAST/).

The two 16S rRNA gene sequences were identical and demonstrated 100% homology with the 16S rRNA gene from *Veillonella parvula* (GenBank accession no. AF439640).

Discussion. *Veillonella* organisms are small, nonfermentative, strictly anaerobic, gram-negative cocci which form part of the normal flora of the oral, genitourinary, respiratory, and intestinal tracts of humans and animals (5). The genus *Veillonella* currently consists of eight species (10, 16).

Veillonella species are rare causes of serious infections such as meningitis, osteomyelitis, prosthetic joint infection, pleuropulmonary infection, endocarditis, and bacteremia (1, 2, 3, 4, 12, 15, 20). In most clinical reports of *Veillonella* infection, the isolates have not been identified to species level. There have been only three previous reports of confirmed *V. parvula* discitis or vertebral osteomyelitis (2, 7, 20) and one report of a case caused by an unidentified *Veillonella* species (9). All previous reports of discitis or vertebral osteomyelitis caused by *Veillonella* species are summarized in Table 1.

Risk factors for *Veillonella* infection include periodontal disease (4), immunodeficiency (1), intravenous drug use (4), and premature birth (3). The patient described in this case report had no apparent risk factors. However, he had undergone colonoscopy and endoscopy, with rectal and small intestinal biopsies, 2 months prior to presentation, raising the possibility of this procedure being the portal of entry for the organism. There is little recent literature on the incidence of bacteremia following colonoscopy or endoscopy. Much of the research dates from the 1970s and 1980s, when blood culture systems may not have been as effective as those used today. The incidence of bacteremia in those studies ranged from 0% to 4%, and potentially pathogenic organisms were rarely isolated (13, 14, 18). There have been no reports of *Veillonella* bacteremia following colonoscopy. However, as the organism is part of the normal gastrointestinal flora, the biopsy procedure is a potential port of entry.

The identification of *Veillonella* isolates to the species level remains problematic, as conventional phenotypic and biochemical testing does not provide adequate discrimination between species. Direct sequencing of the 16S rRNA gene has proven to be a stable and specific marker for bacterial identification (17) and has been described as the best method for identification of *Veillonella* strains at the species level. The sequence data generated for the 16S rRNA gene complex regions from both the aspirate and blood culture isolates showed 100% homology with *V. parvula* (GenBank accession no. AF439640).

Susceptibility testing could not be performed, as the isolates from both blood and tissue failed to remain viable on subculture. Due to the lack of adequate numbers of reports on *Veillonella* as a pathogen, there are little data in the literature on treatment strategies. Penicillin has traditionally been considered the antimicrobial agent of choice for the treatment of this organism. However, in a recent study *Veillonella* species isolated from the oral cavities of humans demonstrated a high level of resistance to penicillin G (MIC, >2 μg/ml) (19). These penicillin G-resistant isolates had reduced susceptibility to ampicillin or amoxicillin but were susceptible to the combination of amoxicillin and clavulanate. In general, *Veillonella* species are resistant to tetracycline, vancomycin, aminoglycosides, and ciprofloxacin and have intermediate susceptibility to erythromycin. Our patient had an excellent clinical and immunological response to expanded-spectrum cephalosporin therapy followed by oral amoxicillin-clavulanate.

The most common causative organisms isolated from patients with spontaneous discitis include *Staphylococcus aureus*, coagulase-negative staphylococci, and streptococci. These organisms generally account for over 80% of all positive cultures (6, 8). Although gram-positive organisms account for the majority of cases and will be appropriately treated by standard antistaphylococcal therapy, our patient highlights the need for a definitive diagnosis, by either conventional or molecular methods, so that appropriate antimicrobial therapy can be administered. A recent publication by Lecouvet et al. (11)

demonstrated the sensitivity of 16S rRNA universal target and *femA Staphylococcus*-specific target genes. Overall, a causative organism was isolated in 14 out of 19 patients, whereas molecular assay identified an organism in 19 out of 19 patients. Only two of the five organisms detected by molecular methods would have responded to antibiotic agents active against gram-positive cocci.

This is the fourth reported case of *Veillonella parvula* causing discitis. However, this report is unique in that molecular methods were used in the identification of the isolate and concomitant blood cultures yielded the same organism. This case report highlights the usefulness of molecular methods in identifying fastidious microorganisms which may be nonviable on repeated subculture, which in turn ensures appropriate antimicrobial therapy.

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