Case report

A 38-year-old pre-menopausal woman presented to the Accident and Emergency Department, Leeds General Infirmary Teaching Hospitals, Leeds, with a 5-day history of an increasingly severe frontal headache. Lumbar puncture and a computed tomography scan of her head were normal, and the patient was sent home with analgesic medication and advice to visit her general practitioner if symptoms persisted. The following day the patient represented to the Accident and Emergency Department with additional symptoms of photophobia and vomiting in combination with persistence of the original headache. She denied any history of fever, rigors, back pain, back surgery, trauma, infection (including urinary), recent gynaecological procedures, use of antimicrobials or use of illicit drugs. She had no significant past medical history, and two normal vaginal births, the last of which was 10 years ago.

Physical examination of her respiratory, cardiac and abdominal systems revealed no abnormalities. Neurological and cranial nerve examination results were completely normal. There was some tenderness in the lumbosacral region and restriction in forward flexion of the lumbar spine.

She had a peripheral white blood cell count of 7.01 × 10⁹ cells l⁻¹, a haemoglobin level of 12.9 g dl⁻¹ and a platelet count of 2.19 × 10¹¹ platelets l⁻¹. Her C-reactive protein (CRP) level was 6.1 mg l⁻¹ and her renal function was normal. Urine analysis was unremarkable. Magnetic resonance imaging (MRI) of the spine (Fig. 1) revealed early signs of vertebral osteomyelitis and discitis at the level of L2/L3 vertebrae. A small epidural fluid collection at L2/L3, which showed marked rim enhancement with a small amount of fluid centrally, was effacing the lateral recess and was directly impinging upon the traversing left L3 as well as the exiting L2 nerve roots.

The patient was placed on bed rest, and blood and urine cultures were collected. A computed-tomography-guided biopsy of L2/L3 was performed, the biopsy specimen was sent for microbiological examination (but not histopathological examination) and empirical antimicrobial therapy (2 g flucloxacillin every 6 h intravenously) was started. Microscopy was performed on the small volume biopsy sample but showed no white blood cells or organisms. The biopsy was cultured aerobically [CLED (cystine lactose electrolyte deficient) agar, Sabaroud agar], anaerobically (fastidious anaerobe agar with and without neomycin, fastidious anaerobe broth) and in 5% CO₂ (blood and chocolate agar) for 5 days (all media were supplied by E&O Laboratories). Mycobacterial culture was also set up using an automated liquid-culture-based method (Bactec MIGIT960; Beckton Dickinson). Two colonies of a tiny Gram-negative bacillus grew at the original inoculation site on chocolate agar after 4 days incubation. Broth cultures remained negative, as did mycobacterial cultures.

The isolate subsequently grew only under anaerobic conditions (in an anaerobic cabinet), and was oxidase-negative, catalase-negative and indole-negative. The organism grew poorly on horse blood agar, but growth was better on chocolate agar. API NH and API anaerobe kits (bioMérieux) both gave doubtful profiles. Susceptibility was tested using a modified Stokes disc diffusion method with a 0.5 McFarland inoculum on fastidious anaerobe agar.
agar (E&O laboratories) with a *Bacteroides fragilis* NCTC 9343 control and the organism showed susceptibility to clindamycin (disc content 2 μg) and ciprofloxacin (disc content 1 μg) using this unvalidated test. Empirical flucloxacinillulin was discontinued and oral treatment with 450 mg clindamycin every 6 h and 500 mg ciprofloxacin every 12 h was commenced. (At this time the organism identity was not known and the significance of the isolate was unclear, so we wished to maintain broad cover for possible causes of vertebral osteomyelitis and discitis whilst giving activity against the organism we had grown.) We subsequently identified the organism by amplification of the 16S rRNA gene using primers RW01-F 5′-AACTGGAGGAAGTTGGGAT-3′ and DG74-R 5′-AGGAGGTGATCCACCGCA-3′ (Grijalva et al., 2003), followed by nucleotide sequence analysis of the 350 bp amplicon and comparison using the BLAST facility at the National Institutes of Health, USA. The submitted sequence had 99% homology to *Gardnerella vaginalis* (there were no sequences identified from any other species with this level of homology). Our usual 16S rRNA gene PCR primers, FD1 5′-AGATTTGATCCTG-GCTCAG-3′ (Kotilainen et al., 1998) and UR 5′-CTAC-GCATTTCCACCGCTACAC-3′ (Hendolin et al., 2000), did not give a product. The Laboratory for Hospital Infection, Colindale, London, confirmed the identity also using partial 16S rRNA gene PCR. No other organisms were identified from spinal biopsies, and blood cultures were negative.

Additionally, since the patient presented with a relatively acute history and was sampled early during the clinical course of the illness, she had not been pre-treated with antimicrobials and she had no common pathogens associated with vertebral osteomyelitis (e.g. *Staphylococcus aureus*, coliforms); therefore, secondary infection of an already infected disc space seems unlikely but cannot be ruled out.

A repeat MRI of the spine 10 days into therapy showed improvement, with a reduction in size of the epidural fluid collection. The patient was discharged home and completed a 6 week course of oral antibiotic therapy. Eight months after stopping therapy she was well, still suffering some mechanical back pain but with no pain at rest nor nocturnal pain; her CRP level was normal and she was systemically well.

**Discussion**

The isolation of *G. vaginalis* as the sole organism from our patient’s disc-space biopsy was extremely unusual and laboratory evidence suggests that this result was unlikely to represent sample contamination. The only previously reported case of *G. vaginalis* spinal infection was seen in a 50-year-old post-menopausal woman, who presented to her medical practitioner with a 2-month history of an insidious onset of low back pain (Hodge et al., 1995). That case, published over 10 years ago, suggested that *G. vaginalis* was a potential cause of deep spinal infections, but it does not appear to have been reported since.

In an extensive review of extravaginal presentations of *G. vaginalis* infection there was only one instance of bone or joint infection reported (Catlin, 1992). Although the literature is extensive with regard to *G. vaginalis* maternal infection (intrauterine infections, intra-amniotic infections, chorioamnionitis, post-abortal pelvic inflammatory disease and post-partum endometritis following Caesarean section), there are no reported cases of osteomyelitis complicating such infection. Among the reports of neonatal infections there was one case of osteomyelitis reported (Nightingale et al., 1986).

Vertebral osteomyelitis and discitis typically have a slow, insidious onset with back pain and local tenderness being the usual presenting complaint (Luthy et al., 2008). The case described here was unusual in that headache is a rare presenting complaint in deep spinal infections. Systemic symptoms of infection, such as fever, chills and malaise, are not invariably present in deep spinal infections, and were absent in this case (Catlin, 1992). The most common physical signs are localized tenderness over the affected area, with concomitant paraspinal muscle spasm, as described in the only other previous reported case of *G. vaginalis* spinal infection (Hodge et al., 1995). The most consistent laboratory abnormalities seen are an elevated erythrocyte sedimentation rate and CRP level, but these were only minimally raised in this case (Barton et al., 2008). Positive blood cultures are seen in approximately one-half of cases, and so diagnosis must be made by aspiration and culture of disc fluid. Plain radiographic films can be useful in diagnosing vertebral osteomyelitis and discitis, but typical features such as disc space narrowing, erosion of endplates and calcification of the annulus around the affected disc usually only become visible several weeks into the course of the disease (Modic et al., 2008).
et al., 1986). MRI is the most sensitive and specific test for diagnosing discitis (Modic et al., 1986).

Factors predisposing to disc space infection include previous disc surgery, intravenous drug use, trauma and a recent history of urinary or respiratory tract infection (Fouquet et al., 1992); no clear predisposing factors were isolated in our described case. It is unusual for infection to originate in the disc space or vertebrae, and haematogenous spread is postulated as the most likely route of disease spread (Fouquet et al., 1992). Batson’s plexus in the epidural space has a close relationship between the venous drainage of the urogenital tract and the spinal system. It has been proposed that a urinary tract infection in the pelvic space can disseminate directly into the lumbar spine via Batson’s plexus without passing through the systemic circulation (Fouquet et al., 1992).

This case confirms that G. vaginalis can be a cause of deep spinal infection. From a clinical perspective, empirical antimicrobial therapy for deep spinal infections may not have activity against G. vaginalis. We highlight the need for appropriate microbiological sampling in this clinical situation.

References


