LESSON OF THE MONTH

Intervertebral discitis presenting as oligoarthritis

A T Marshall, J K Gaffney, T J Marshall, H M S Williams

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The symptoms of intervertebral discitis may be indistinct, making diagnosis difficult. The onset may be acute or insidious, and radicular or spinal compressive symptoms and signs may be present. Purulent infection may spread to surrounding soft tissues, and epidural abscess formation is recognised. Long term neurological sequelae may result, and mortality of between 7% and 18% has been reported.^{1 2} Vigilance is required if the diagnosis is to be made early, and treatment should be of adequate duration to reduce the rate of recurrent infection. To our knowledge, this is the first report of a case of intervertebral discitis in a patient presenting with an oligoarthritis.

CASE REPORT

A 62 year old woman was referred with a 2 week history of oligoarthritis and fever. She had developed acute back pain with fever 12 hours after visiting a spa in Italy. On returning home, her back symptoms resolved, but she developed persistent fever with pain and swelling of both knees, left ankle, and left hip. There was no significant past medical history. On examination, her temperature was 39°C. She had a full range of spinal movements and no spinal tenderness, but had synovitis of both knees, left hip, and left ankle. Her throat was hyperaemic. Examination was otherwise unremarkable.

Investigations on admission showed a white cell count of $17.9 \times 10^9/l$ (4.0 – $11.0 \times 10^9/l$), haemoglobin 91 g/l (115–165 g/l), platelet count $932 \times 10^9/l$ (150 – $400 \times 10^9/l$), erythrocyte sedimentation rate >140 mm/1st h (1 – 15 mm/1st h), C reactive protein 2230 g/l (<100 g/l), γ -glutamyl transaminase 248 U/l (0 – 60 U/l), alkaline phosphatase 323 U/l (38 – 126 U/l), and uric acid 254 μ mol/l (0 – 450 μ mol/l). Blood cultures grew group B streptococcus. Twenty millilitres of straw coloured, turbid, synovial fluid was aspirated from her right knee. This was negative on Gram stain, sterile on culture, and negative for crystals. Throat swabs were negative.

Plain lumbar radiographs showed moderate degenerative change, with more severe narrowing at the L1–2 disc space (fig 1). A chest x ray examination, abdominal and left hip ultrasound were normal. An isotope bone scintigram showed increased uptake in a variety of upper and lower limb joints, in keeping with degenerative change. Focal increased uptake was noted in the region of L1–2. She remained pyrexial despite 5 days' treatment with broad spectrum antibiotics (gentamicin and benzylpenicillin), and her inflammatory markers were unchanged. Magnetic resonance imaging of the lumbosacral spine confirmed an increased signal and the presence of fluid within the L1–2 disc space, with a diffuse increase in signal in the adjacent end plates (fig 2), consistent with bone oedema, which was seen to be enhanced on the post-gadolinium images. No significant pre-vertebral paraspinal abscess formation was seen. The findings were consistent with intervertebral discitis.

Her antibiotic regimen was changed to vancomycin, as her continuing fever was considered to be partially penicillin induced. After 6 weeks of antibiotic treatment, she was asymptomatic, and the C reactive protein returned to normal.

Her peripheral arthritis settled within the first week of admission.

DISCUSSION

Intervertebral discitis is difficult to diagnose, as symptoms may be minimal and poorly localised. Two weeks before admission this woman had severe back pain, which had resolved by admission. The diagnosis of discitis is often delayed because of a lack of localising signs and symptoms, and because it is an uncommon condition. Intervertebral discitis must be included in the differential diagnosis of patients presenting with a pyrexia of unknown origin with raised inflammatory markers, or when there is an inadequate response to antibiotic treatment. Infection of the disc space usually arises from haematogenous spread, unless infection is directly introduced—for example, by instrumentation. The microbiological yield from a disc biopsy reportedly varies between 47% and 90%.^{3–5} In view of the positive blood cultures and the typical radiological appearances, we did not proceed to biopsy. A site of primary infection may be found in 78% of patients.⁶ The choice of antibiotic should take account of the drug's ability to penetrate bone and intervertebral disc. Antibiotic treatment was changed from benzylpenicillin and gentamicin to vancomycin following microbiological advice as group B streptococcal infection would have been sensitive to this regimen, and the persisting fever might have been penicillin induced. Surgical debridement of the disc should be considered if conservative management is inadequate to treat the fever and symptoms, and must be considered urgently if there is neurological deterioration, or the patient is immunocompromised.⁷

Figure 1 Plain films show reduced height at L1–2 but no convincing evidence of disc infection.

Figure 2 Magnetic resonance imaging demonstrates an increased signal from within the disc, and a diffuse increase in signal from within the adjacent end plates.

The cause of this patient's oligoarthritis remains unclear. The right knee synovial fluid was sterile, and although this does not exclude infection, raises the possibility of a postinfectious manifestation of septic discitis. The admitting team did not attempt to aspirate the left knee or left ankle before the administration of antibiotics. Synovial fluid from these joints might have helped to exclude sepsis in these joints, or might have strengthened the argument for a postinfective manifestation of discitis. Although urogenital and gastrointestinal infection are the classical triggers for postinfectious non-septic oligoarthritis, streptococcus may also be a trigger. Streptococcus A, C, and G are the most commonly reported infectious agents, but group B streptococcus (*Streptococcus agalactiae*) is an important cause of septicaemia and meningitis in newborn infants, and of pregnancy related morbidity in women. It also causes substantial morbidity and mortality in non-pregnant adults.⁸ A range of underlying disease predisposes to invasive group B streptococcus, including diabetes, cancer, and HIV, and age poses an increasing risk.⁸⁻⁹

THE LESSONS

- A careful history may disclose a history of back pain, although this may not be the predominant symptom in a patient with intervertebral discitis
- Back pain may be poorly localised and there may be no abnormal signs on examination of the spine.
- Intervertebral discitis should be included in the differential diagnosis of a patient presenting with a pyrexia of unknown origin and raised inflammatory markers or if a patient fails to respond adequately to antibiotics.
- Oligoarthritis may be the presenting feature of intervertebral discitis, although whether this is a septic or a postinfectious manifestation is not clear.

Authors' affiliations

A T Marshall, J K Gaffney, Department of Rheumatology, Norfolk and Norwich University Hospital NHS Trust, Colney Lane, Norwich, Norfolk NR4 7UY, UK

T J Marshall, Department of Radiology, Norfolk and Norwich University Hospital NHS Trust, Colney Lane, Norwich, Norfolk NR4 7UY, UK

H M S Williams, Public Health Laboratory Service, Norfolk and Norwich University Health Care Trust, Bowthorpe Road, Norwich, Norfolk NR2 3TX, UK

Correspondence to: Dr A T Marshall; t.marshall@uea.ac.uk

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