Concise Report

Imaging does not predict the clinical outcome of bacterial vertebral osteomyelitis

V. Zarrouk, A. Feydy, F. Sallès, V. Dufour, P. Guigui, A. Redondo and B. Fantin

Objectives. Magnetic resonance imaging (MRI) and computed tomography (CT) are useful for initial assessment of bacterial spondylodiscitis. However, clinical relevance of imaging changes during treatment is less well-documented.

Methods. Between October 1997 and March 2005, 29 patients with documented bacterial spondylodiscitis were prospectively enrolled. They had clinical, biological and imaging examinations (MRI and/or CT) at M0 and M3, and in 22 cases, at M6.

Results. Mean age was 58 yrs. Antimicrobial chemotherapy lasted an average of 98 days. The median follow-up was 18 months, including 12 months after the completion of treatment. Infection was cured in every patient. Biological markers of inflammation returned to normal at M3. Six patients had painful and/or neurological sequelae.

Decreased disc height was a consistent and early sign, and remained stable during the follow-up. Vertebral oedema, present in 100% of cases initially, persisted in 67 and 15% of cases at M3 and M6, respectively. Discal abscesses and paravertebral abscesses, present in 65 and 39% of cases initially, persisted in, respectively, 42 and 9% of cases at M3 and in 18 and 3% of cases at M6. Epidural abscesses were present at diagnosis in 30% of cases, and had always disappeared by M3. Imaging abnormalities found at M0 and M3 did not differ between patients with and without late neurological or painful sequelae.

Conclusions. Imaging abnormalities often persist in patients with bacterial spondylodiscitis despite a favourable clinical and biological response to antibiotic treatment. They are not associated with relapses, neurological sequelae or persistent pain. Imaging controls are not necessary when bacterial spondylodiscitis responds favourably to treatment.

Key Words: Pyogenic spondylodiscitis, Vertebral osteomyelitis, Imaging, CT scanner, MRI.

Introduction

Computed tomography (CT) and magnetic resonance imaging (MRI) are examinations of choice for the diagnosis of infectious spondylodiscitis [1–4]. However, there are few data on the possible correlation between imaging and the clinical outcome of bacterial spondylodiscitis during and after the treatment.

Patients and methods

This was a prospective, single-centre study of patients admitted to a university hospital between October 1997 and March 2005. All the patients were hospitalized in the departments of internal medicine, neurosurgery or orthopaedics, and were managed by a specialist in internal medicine [5].

Patients

The following three criteria were required for inclusion: (i) compatible clinical signs (fever or other general signs, pain or spinal stiffness), (ii) initial CT and/or MRI diagnosis confirmation and (iii) bacterial isolation from a discovertebral sample, by blood smear, or from a portal of entry. The patients had to have at least one follow-up MR or CT examination within 3 months of diagnosis.

Collection of clinical and biological data

The following information was collected for each patient at baseline: (i) demographic data, (ii) comorbidity, (iii) clinical features at diagnosis, (iv) the portal of entry, (v) biological data [leucocyte and polymorphonuclear neutrophil counts, and C-reactive protein (CRP)], (vi) bacteriological data and (vii) analgesic consumption (WHO stepwise classification [6]).

At each follow-up visit (M3, and M6 and M12 in some cases), the following information was collected: (i) clinical data, (ii) biological data and (iii) analgesic consumption and the duration of antibiotic treatment. At the end of the study period, all the patients were re-assessed, either by telephone contact or by their usual physician.

Radiology

All the patients had CT and/or MRI at M0 and M3, and some were also examined at M6 and M12. All the images were reviewed by a senior musculoskeletal radiologist.

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CT was done using a multidetector device, with 1.5 mm slices, and tissue and bone windows. The images were reconstructed in the axial, frontal and sagittal planes.

MRI was done with a 1.5T device and the following pulse sequences: T1-weighted, T2-weighted, STIR-weighted and fat-saturated gadolinium-enhanced T1-weighted. Axial and sagittal views were obtained in every case.

The following criteria were analysed on CT and MRI images: (i) height reduction of the intervertebral disk relative to adjacent normal discs, (ii) discal abscess, corresponding to an intra-discal area with high signal on T2 or STIR-weighted MR images, with contrast enhancement of all or part of the disc space, (iii) vertebral signal on MRI with oedema signal defined as a T1 hyposignal and a T2 hypersignal with contrast enhancement, or a fatty signal, defined by a T1 hypersignal, (iv) vertebral destruction, and its type (endplate erosion, osteolysis or compression fracture) on CT and/or MRI and (v) intraosseous, paravertebral or epidural abscesses. Paravertebral abscesses appear as low-density areas with peripheral contrast uptake on CT, and as areas with low signal on T1-weighted and high signal on T2-weighted, with peripheral contrast intake on MRI. The size of paravertebral abscesses (measured in centimetres) was monitored during follow-up.

**Treatment**

All the patients received antibiotic treatment adapted to the bacterial isolate. The intravenous route was favoured for initial treatment. The recommended treatment period was 3 months. Corset immobilization was prescribed by the surgeons on the basis of spinal stability on standard radiography. All patients with signs of spinal compression were treated surgically.

**Statistical analysis.** We used Fisher’s exact test to compare qualitative data and the Mann–Whitney U-test for quantitative data. Results are reported as means ± s.d.

**Results**

**Clinical, biological and bacterial findings at diagnosis** (Table 1)

Twenty-nine patients met the inclusion criteria. Mean age at diagnosis was 58 yrs. Fifteen (52%) had a comorbidity. The median interval between initial symptoms and diagnosis of spondylodiscitis was 29 days. All the patients reported pain at diagnosis and were taking analgesics. Fifty-five per cent of patients had fever. Twelve patients (41%) had radicular compression at diagnosis and three had spinal cord compression requiring surgery. All but two patients had an elevated CRP or polymorphonuclear neutrophil count. The spondylodiscitis had a documented bacterial cause in every case. Blood cultures were positive in 14 patients (48%). Fifteen patients with negative blood cultures underwent radioguided needle biopsy, and of them 11 were positive. The remaining four patients had surgical biopsy that was positive in all cases. Bacterial isolates are listed in Table 1.

**Initial imaging findings**

Initial imaging studies showed two different sites of involvement in four patients. As the different sites of infection did not always show parallel imaging changes, we, therefore, analysed each of the 33 anatomical sites separately.

Decreased disc height was initially present at 31 (94%) of the 33 sites and MRI showed a discal abscess at 20 (65%) of the 31 sites concerned. Vertebral destruction was found at 29 (88%) of the 33 sites, but 11 sites (33%) showed only mirror erosions. Vertebral compression fracture was initially observed in one patient.

Vertebral oedema was detected at 29/33 sites overall, and at all the 29 sites examined by MRI. Most cases showed mirror oedema (10/29) which could involve up to 100% of the two vertebrae contiguous to the spondylodiscitis.

Abscesses were found at 17 sites (52%); they were paravertebral or involved the psoas muscle at 13, and were epidural at 10 sites. Both types of involvement were present at six sites, while only epidural abscesses were present at four.

**Antibiotic treatment**

All 29 patients received antibiotic therapy. Twenty-six of them initially received intravenous antibiotic therapy, for a mean duration of 23 days (6–90 days). Three received only oral treatment. The mean overall treatment period was 98 ± 27 days. Of the 29 patients, 22 were treated for 90 days, seven were treated for longer, because of the pathogen (*Brucella*, one case) or an underlying health disorder (six cases).

**Clinical and biological outcome**

Median follow-up lasted 18 months overall (3–59 months) and 12 months (0–51 months) after the completion of treatment. The infection was cured in every case: initial pain disappeared or improvement, general symptoms and biological markers of inflammation disappeared and there were no relapses.

Analgesic consumption decreased over time. The proportion of patients who took major opiates was 18/29 at M0 (62%), 6/29 at M3 (21%) and 1/19 at M6 (5%), while the proportion of patients who did not take analgesics was 0/29 at M0, 10/29 at M3 (21%) and 1/19 at M6 (5%), while the proportion of patients who took major opiates was 18/29 at M0 (62%), 6/29 at M3 (21%) and 1/19 at M6 (5%).

Twenty-two patients made a full recovery. One patient died under treatment. The mean overall treatment period was 98 ± 27 days. All but two patients had an elevated CRP or polymorphonuclear neutrophil count. The spondylodiscitis had a documented bacterial cause in every case. Blood cultures were positive in 14 patients (48%). Fifteen patients with negative blood cultures underwent radioguided needle biopsy, and of them 11 were positive. The remaining four patients had surgical biopsy that was positive in all cases. Bacterial isolates are listed in Table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>N (%)</th>
</tr>
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<tbody>
<tr>
<td>Men</td>
<td>17 (59)</td>
</tr>
<tr>
<td>Age (yrs)*</td>
<td>58 ± 14</td>
</tr>
<tr>
<td>Immunosuppression</td>
<td>15 (52)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>6 (21)</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>3 (10)</td>
</tr>
<tr>
<td>Spinal pain</td>
<td>29 (100)</td>
</tr>
<tr>
<td>Neurological signs</td>
<td>29 (100)</td>
</tr>
<tr>
<td>Radicular compression</td>
<td>12 (41)</td>
</tr>
<tr>
<td>Spinal cord compression</td>
<td>3 (10)</td>
</tr>
<tr>
<td>Fever &gt;38°C</td>
<td>16 (55)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>7 (24)</td>
</tr>
<tr>
<td>Time to diagnosis (days)*</td>
<td>29 (4–730)</td>
</tr>
<tr>
<td>Site of involvement</td>
<td>29 (4–730)</td>
</tr>
<tr>
<td>Cervical</td>
<td>5 (15)</td>
</tr>
<tr>
<td>Thoracic</td>
<td>8 (24)</td>
</tr>
<tr>
<td>Lumbar</td>
<td>20 (61)</td>
</tr>
<tr>
<td>Multifocal</td>
<td>4 (14)</td>
</tr>
<tr>
<td>Biological data</td>
<td>29 (4–730)</td>
</tr>
<tr>
<td>Leucocytes (per mm³)*</td>
<td>9708 ± 3607</td>
</tr>
<tr>
<td>Polymorphonuclear neutrophils (per mm³)*</td>
<td>7107 ± 3469</td>
</tr>
<tr>
<td>C-reactive protein (mg/l)*</td>
<td>102 ± 490</td>
</tr>
<tr>
<td>Bacterial isolates</td>
<td>29 (4–730)</td>
</tr>
<tr>
<td>Staphylococci</td>
<td>15 (52)</td>
</tr>
<tr>
<td>Streptococci</td>
<td>7 (24)</td>
</tr>
<tr>
<td>Others</td>
<td>3 (10)</td>
</tr>
<tr>
<td>Polymicrobial</td>
<td>4 (14)</td>
</tr>
</tbody>
</table>

*aMean ± s.d.

*bRange.
**Imaging outcome (Fig. 1)**

Imaging studies were repeated in all the patients at M3, 22 at M6 (67%), three at M9 and 5 at M12. Decreased disc height was present at both M3 and M6 at all the sites examined. Discal abscesses were still detectable in 42% of sites (14/33) at M3 and in at least 18% (6/33) at M6.

Vertebral destruction was present at 94% of sites (31/33) at M3, consisting of mirror erosions alone in 10 cases (30%); it had worsened at seven of the 18 sites (39%) at M3. At M6, evidence of vertebral destruction was still present at all 22 sites that were controlled at this time point. Vertebral oedema was assessed at 30 sites at M3 and persisted at 22 sites (76%). It had disappeared at five sites, and had become a fatty signal in two. Two patients had fused vertebrae, which hindered the interpretation of signal changes. Oedema was assessed by MRI at M6 in 20 cases: the oedema persisted in five cases (15%) and had become a fatty signal in one (12%). Of the five sites controlled by MRI at M12, the signal corresponded to oedema in two and to fat in three cases.

Two new vertebral compression fractures were found at M3 and one at M6. Overall, four of the 33 affected vertebrae collapsed (12%).

None of the 10 epidural abscesses persisted at M3. Three (18%) of the 17 paravertebral abscesses persisted at M3, representing 3/33 sites (9%). One of the three abscesses still present at M3 persisted at M6 but had shrunk by about 50%. Thus, one (8%) of the 13 paravertebral abscesses persisted at M6, corresponding to 3% of the 33 sites.

**Characteristics of patients with and without painful and/or neurological signs**

There was no significant difference in imaging abnormalities between the six patients with severe sequelae and the remaining patients, either initially or at M3 (data not shown).

**Discussion**

There is disagreement on imaging changes over time, their correlation with clinical outcome, and the clinical value of repeat imaging during follow-up of infectious spondylodiscitis. In the study by Modic et al. [1], 37 patients with spondylodiscitis had MRI at M0, and the examination was repeated at 6 weeks in 12 cases and at both 3 and 15 months in six cases. MRI abnormalities remained visible at 6 weeks and 3 months. In 1994, Korvin et al. [7] reported an MRI follow-up of 15 patients with spondylodiscitis, based on findings at M0, M3, M6 and M9. All the patients were cured, clinically and biologically, at three months, whereas some MRI abnormalities persisted. In particular, vertebral oedema persisted in 12 patients and worsened in three patients. Paravertebral and epidural abscesses also persisted but had improved. In a retrospective study, Flipo et al. [8] examined MRI findings in 10 patients, comparing nine who were clinically cured at months with one who was not cured. The nine cured patients had persistent vertebral and discal abnormalities, but these were less marked than at M0. In contrast, the MRI findings in the patient with an unfavourable clinical outcome were stable relative to M0. The authors, nonetheless, concluded that MRI was predictive of clinical outcome. In the series of 103 patients reported by Eugene and Carragee [4], 15 patients had a second MRI between 1 and 17 weeks after the beginning of treatment. MRI abnormalities were stable or worse in 12 patients who responded to the treatment. This led to unnecessary surgery in three cases (no residual abscesses were found and all samples were sterile). The abnormalities worsened in the three patients who had unfavourable clinical outcomes.

Overall, these studies tend to show that MRI abnormalities often persist after clinical recovery and some authors have found that a lack of improvement on MRI portends a poor clinical outcome.

Our study was prospective with a clinical and biological cure obtained in all the patients and confirmed by the absence of relapse during follow-up. Nevertheless, MRI abnormalities persisted at M3 in two-thirds of our patients. At M6, treatment was completed in all but one patient: discal abscesses persisted in 21%, vertebral oedema in 17% and paravertebral abscess in 3% of the patients. Decreased disc height remained stable even in patients who had later follow-up MRI examinations. Vertebral destruction was found at all sites, with compression fractures in 15% of the patients. However, vertebral destruction and fracture should not be considered as progressive lesions, but rather as sequelae of the pyogenic process. Furthermore, no significant difference was found in imaging findings at M0 or M3 between the six patients with neurological or painful sequelae and the remaining patients. We, therefore, conclude that imaging abnormalities present either at diagnosis or after treatment completion are not predictive of neurological or painful sequelae.

In conclusion, imaging abnormalities frequently persist despite clinical and biological cure of pyogenic spondylodiscitis, and are not associated with a greater risk of relapse. Consequently, such abnormalities do not require antimicrobial chemotherapy to be prolonged if the clinical and biological outcomes are favourable. In our opinion, follow-up imaging studies are not necessary when pyogenic spondylodiscitis responds favourably to treatment.

The authors have declared no conflicts of interest.

**References**