Facet and Facet-Joint Infections: Case Report and Review

A 40-year-old male, active iv drug user who experienced chronic low back pain after a fall, presented with fever, worsening back pain, and sciatica of 1 month’s duration. The patient had been HIV positive, with a CD4 cell count of 519/mm³ 2 months before. Previous radiographs of the lumbar spine revealed spondylolisthesis at the L4–L5 level. There was no history of back surgery.

On examination, the patient was afebrile and comfortable. His heart, lungs, abdomen, skin, and neurological examination were unremarkable. Palpation of the lower back revealed focal tenderness over L4 and L5. The erythrocyte sedimentation rate (ESR) was 66 mm/h; other laboratory results were normal. Gadolinium-enhanced MRI of the lumbar spine revealed destruction of the right L4–L5 facets, with adjacent fluid collection and inflammation. Laminectomy was performed. Culture of purulent material obtained from the facet joint area yielded Streptococcus equisimilis and Staphylococcus aureus, both susceptible to oxacillin; acid-fast staining and fungal cultures were negative.

Two sets of blood cultures were positive for S. equisimilis but negative for S. aureus. It was presumed the patient had bacteremia due to both organisms but that the inoculum of S. equisimilis was larger than that of S. aureus. The presumed portal of entry was via iv drug use; there was no workup of the urinary or gastrointestinal tracts.

The patient received a 6-week course of iv nafcillin, 1.5 g every 4 hours. Two months later he had minor residual lower back pain, but no systemic symptoms. No follow-up MRI was performed; subsequently, he was lost to follow-up.

We found six cases of facet infection reported since 1966 in the English-language literature [1–6] (table 1). Predisposing factors included chronic back pain, steroid use, previous bacteremia, and parenteral drug use. Presentations ranged from subacute to acute. Six patients were febrile, and all had significant spinal pain; four had sciatica. All cases involved the lumbar spine except for one case that involved the cervical spine. The WBC count was elevated in three cases. The ESR was frequently elevated. Radionuclide studies, CT (especially with thin cuts), and MRI were generally helpful for diagnoses.

References


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**Figure 1.** Survival of patients for whom bronchoalveolar lavage (BAL) was positive after treatment for Pneumocystis carinii pneumonia. The second BAL (BAL 2) was performed at the end of effective treatment; P. carinii was readily identified by staining with toluidine-blue-O. There was no correlation between positivity of BAL 2 and survival.

suggest that without clinical improvement during treatment for PCP, persistence of P. carinii is not indicative of ineffective therapy, and another etiology must be sought.

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Clinical Infectious Diseases 1998;26:510–2
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Table 1. Data from cases of facet and facet-joint infections reported since 1966.

<table>
<thead>
<tr>
<th>Reference(s)</th>
<th>Age (y) Sex</th>
<th>Predisposing factors</th>
<th>Site of lesion(s)</th>
<th>ESR (mm/h)</th>
<th>Tests diagnostic of infection</th>
<th>Organism</th>
<th>Treatment (duration)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[1]</td>
<td>66/M</td>
<td>Prior staphylococcal bacteremia</td>
<td>L4–L5 facet joint</td>
<td>113</td>
<td>CT scan, $^{67}$Ga, $^{99}$Tc, needle aspiration</td>
<td><em>Staphylococcus aureus</em></td>
<td>Surgical debridement, iv nafcillin (6 w), oral dicloxacillin (3 mo)</td>
<td>Recovered</td>
</tr>
<tr>
<td>[2]</td>
<td>82/F</td>
<td>Sacroiliitis, prior buttock abscess</td>
<td>L4–L5 facet joint</td>
<td>140</td>
<td>$^{67}$Ga, CT scan, needle aspiration</td>
<td><em>Pseudomonas pyocyanea</em></td>
<td>Unspecified antibiotics (8 mo)</td>
<td>Residual pain</td>
</tr>
<tr>
<td>[3]</td>
<td>66/M</td>
<td>Back &quot;strain&quot;</td>
<td>L3–L4 facet joint</td>
<td>99</td>
<td>$^{99}$Tc, blood culture</td>
<td><em>S. aureus</em></td>
<td>iv cloxacillin (6 d), po fl oxacin (6 w)</td>
<td>Residual pain</td>
</tr>
<tr>
<td>[4]</td>
<td>68/M</td>
<td>Chronic low back pain</td>
<td>L3–L4 facet joint</td>
<td>80</td>
<td>$^{99}$Tc, CT scan, needle aspiration</td>
<td><em>Klebsiella pneumoniae</em></td>
<td>iv pefloxacin and amikacin (4 w), po pefloxacin (8 w)</td>
<td>Recovered</td>
</tr>
<tr>
<td>[5]</td>
<td>76/M</td>
<td>Retropharyngeal abscess</td>
<td>C1–C2 facet joint, odontoid process</td>
<td>“High”</td>
<td>Surgical exploration with aspiration of retropharyngeal abscess</td>
<td>Presumed <em>S. aureus</em></td>
<td>Surgical debridement, iv nafcillin and po cloxacillin (6 mo)</td>
<td>Recovered</td>
</tr>
<tr>
<td>[6]</td>
<td>65/M</td>
<td>Steroids, chronic back pain</td>
<td>L2–L3 facet joint</td>
<td>46</td>
<td>CT scan, $^{99}$Tc, SPECT</td>
<td><em>S. aureus</em></td>
<td>Surgical debridement, unspecified duration of antibiotics</td>
<td>Recovered</td>
</tr>
<tr>
<td>[PR]</td>
<td>40/M</td>
<td>Spondylolisthesis, iv drug use, HIV infection</td>
<td>L4–L5 facet joint</td>
<td>66</td>
<td>MRI, surgical aspiration</td>
<td><em>S. equisimilis</em></td>
<td>Surgical debridement, iv nafcillin (6 w)</td>
<td>Residual pain</td>
</tr>
</tbody>
</table>

NOTE. ESR = erythrocyte sedimentation rate; $^{67}$Ga = gallium-67 citrate scan; PR = present report; SPECT = single photon emission computed tomography; $^{99}$Tc = technetium pyrophosphate isotope scan.

Diagnoses were based on open surgical aspiration in three cases, by needle aspiration in three cases, and by blood culture in one case. Two patients had associated epidural abscesses [1, 3]. *S. aureus* was the most common etiologic organism, and there was one polymicrobial infection.

Four patients underwent surgical decompression, and three were treated only with antibiotics. The iv antibiotic treatment courses ranged in duration from 6 days to 6 weeks; the duration of treatment with subsequent oral antibiotics ranged from zero to 6 months. Four patients recovered fully, and three had residual back pain. The mean follow-up time was 8.5 months (range, 2–24 months).

Infection of the vertebral body usually involves the vertebral body rather than the vertebral arch. Several early reports describe bacterial osteomyelitis involving the posterior elements of the spine; most cases were diagnosed by use of spinal radiographs [7]. Since the 1960s, reports of such cases are rare, although posterior spinal tuberculosis is well described [8]. Infections of the facets and facet joints have been particularly uncommon.

For a patient with chronic back pain who presents with fever, worsening back pain, and an elevated ESR, the diagnosis of facet-joint infection is occasionally made. MRI and CT are the most useful studies for diagnosis. Definitive diagnosis requires needle or open aspiration.

Another unusual aspect of our case is the isolation of group C streptococci. Of group C streptococci, *S. equisimilis* is the most common human pathogen; however, reports of arthritis and osteomyelitis due to this organism are infrequent [9, 10]. Most clinical experience in treating this condition has been with penicillin.

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References
Successful Treatment of an Acyclovir- and Foscarnet-Resistant Herpes Simplex Virus Type 1 Lesion with Intravenous Cidofovir

Systemic administration of acyclovir is the treatment of choice for prophylaxis and treatment of herpes simplex virus (HSV) infections [1, 2]. However, clinical resistance to acyclovir is a growing concern, especially for immunocompromised individuals. Most commonly, acyclovir-resistant HSV emerges as a result of mutations within the viral thymidine kinase. This kinase performs the initial phosphorylation step in the intracellular activation of acyclovir and other antiviral nucleoside analogs such as ganciclovir and penciclovir [3]. Although less common, mutations in the viral DNA polymerase can also confer resistance to acyclovir. Foscarnet, which does not require the viral thymidine kinase for its activity, is the only agent currently approved for the treatment of acyclovir-resistant HSV infection [1, 4].

Cidofovir has been approved recently for the treatment of cytomegalovirus (CMV) retinitis in patients with AIDS and is the first of a new class of nucleotide antiviral agents with activity against a wide spectrum of herpesviruses, including HSV [5]. The phosphorylation of cidofovir to its active form does not depend on the HSV thymidine kinase [5], and thus cidofovir is active against acyclovir-resistant HSV both in vitro and in the clinical setting [4–8]. We describe the successful use of iv cidofovir to effect the rapid and complete resolution of an acyclovir- and foscarnet-resistant HSV-1 lesion in a patient who underwent umbilical cord stem-cell transplantation (UCSCT) and who had severe unremitting mucositis of the oropharynx.

A 36-year-old emaciated, ill-appearing male with acute myelogenous leukemia (AML), M4 subtype, presented with neutropenic fever and mild respiratory distress before undergoing UCSCT. Laboratory data indicated a WBC count of 300/mm³ with anemia and severe thrombocytopenia. Broad-spectrum antibiotic therapy was instituted. Three days before the UCSCT, the patient developed a grade II mucositis from which HSV-1 (isolate 1) was cultured. (table 1). Intravenous acyclovir (6 mg/kg iv q8h) was administered, and after 17 days of treatment, reculture of the oral lesion again yielded HSV-1 (isolate 2). Therapy was changed to foscarnet (40 mg/kg iv q8h) for 9 days, with minimal resolution of the mucositis; cultures continued to be positive for HSV-1 (isolate 3). The patient was then treated with iv ganciclovir (5 mg/kg q.d.) for 28 days, again without either clinical or virological improvement (isolate 4). Intravenous cidofovir (5 mg/kg once weekly) with concomitant probenecid therapy (2 mg orally 3 hours before and 1 g orally 2 and 8 hours after cidofovir administration), in addition to hydration to reduce the risk of nephrotoxicity (the dose-limiting toxicity associated with cidofovir therapy [5]) was started 97 days after UCSCT. After three consecutive once-weekly doses of cidofovir, the mucositis cleared, and the patient was able to tolerate oral nutrition.

The drug susceptibilities of the four HSV isolates obtained from this patient were tested in vitro (table 1), and the results were consistent with the clinical response. Compared with the isolate recovered prior to the initiation of therapy (isolate 1), the isolate recovered at the end of acyclovir therapy (isolate 2) exhibited decreased susceptibility (increased IC₅₀ value) to acyclovir and ganciclovir but not to foscarnet or cidofovir. The pattern of cross-resistance for this isolate suggests the presence of an acyclovir-resistant HSV variant containing a thymidine kinase mutation. The third isolate, obtained after foscarnet therapy, also exhibited decreased susceptibility to foscarnet. Resistance to foscarnet occurs as a result of mutations within the viral DNA polymerase, either in combination with the thymidine kinase mutation or in a separate virus population within the isolate. The final isolate, recovered at the end of the ganciclovir therapy, continued to exhibit decreased susceptibility to acyclovir, ganciclovir, and foscarnet. Notably, the cidofovir susceptibility of the isolates obtained during therapy (isolates 2–4) did not differ from that of the pretherapy isolate.

Table 1. In vitro drug susceptibilities of four consecutive herpes simplex virus type 1 isolates.

<table>
<thead>
<tr>
<th>Isolate no.</th>
<th>No. of days before and after UCSCT</th>
<th>Acyclovir</th>
<th>Foscarnet</th>
<th>Ganciclovir</th>
<th>Cidofovir</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>−3</td>
<td>0.42</td>
<td>26</td>
<td>0.18</td>
<td>1.2</td>
</tr>
<tr>
<td>2</td>
<td>+17</td>
<td>3.0</td>
<td>22</td>
<td>4.1</td>
<td>1.0</td>
</tr>
<tr>
<td>3</td>
<td>+59</td>
<td>220</td>
<td>195</td>
<td>32</td>
<td>1.6</td>
</tr>
<tr>
<td>4</td>
<td>+81</td>
<td>68</td>
<td>115</td>
<td>8.5</td>
<td>1.0</td>
</tr>
</tbody>
</table>

NOTE. UCSCT = umbilical cord stem-cell transplantation.

* In vitro drug susceptibilities (IC₅₀) were determined in plaque reduction assays with use of confluent monolayers of Vero cells (µm).