Neurologic Abnormalities in Children Presenting With Diskitis

Moshe Nussinovitch, MD; Nir Sokolover, MD; Benjamin Volovitz, MD; Jacob Amir, MD

**Background:** Neurologic impairment is not considered a clinical manifestation of diskitis in children and has seldom been associated with it in the medical literature.

**Objective:** To describe neurologic findings and their implications in children with diskitis.

**Study Design:** Retrospective medical records review of children discharged with a diagnosis of diskitis between January 1992 and December 2000. The study included children in whom the diagnosis was based on the presence of clinical findings, laboratory evidence of an inflammatory process, and findings on imaging studies compatible with diskitis.

**Results:** Neurologic findings of decreased muscle strength or hyporeflexia in the lower extremities were found in 7 (41%) of 17 children who met the diagnostic criteria for diskitis. Five of the 7 underwent magnetic resonance imaging, 2 of whom demonstrated intraspinal inflammatory involvement.

**Conclusions:** Neurologic impairment does not exclude the diagnosis of diskitis and may be a common manifestation of the disease in children. Nevertheless, when neurologic findings are present, advanced imaging studies are needed to exclude intraspinal involvement.

Arch Pediatr Adolesc Med. 2002;156:1052-1054

Diskitis, or intervertebral disk inflammation, is an uncommon disease occurring mostly in children. Its exact incidence is unknown, with most investigations following a prolonged retrospective design. Although the disease is believed to be of infectious etiology, positive bacterial cultures are rarely obtained, and some authors oppose the use of antibiotic treatment.

The onset of diskitis is usually subtle, and clinical manifestations vary with age. Therefore, a high index of suspicion is required for diagnosis. Diagnosis is established by narrowing of the intervertebral disk space on radiography and irregularities of the adjacent vertebral bodies, or by increased uptake of technetium Tc 99m in the disks on bone scan early in the course of the disease.

In our review of the relevant literature, we found only anecdotal descriptions and little information about neurologic signs and symptoms associated with diskitis. To determine the incidence of neurologic impairment in children with diskitis and to discern the implications, we conducted a retrospective study of all children discharged from our department during 8 years with a diagnosis of diskitis.

**METHODS**

The medical records of all children discharged from the Schneider Children’s Medical Center of Israel between January 1992 (opening of the pediatrics department) and December 2000 with a diagnosis of diskitis were reviewed. We included only those children in whom the diagnosis was based on the presence of typical clinical findings (limp, refusal to walk, or pain on changing position), laboratory evidence of an inflammatory process (elevated erythrocyte sedimentation rate or C-reactive protein level), and imaging study results compatible with diskitis (narrowing of the intervertebral disk space on radiography or increased uptake in that area on bone scan). The neurologic examinations were performed by a neurologist. All children were initially treated with antibiotics for 3 to 4 weeks.

**RESULTS**

Seventeen children were discharged with a diagnosis of diskitis during the study period. Neurologic findings of decreased muscle strength or hyporeflexia in the lower extremities were found in 7 (41%) of them. Neurologic findings and imaging results are found in the Table. Nine of the children were male. The median age at presentation was 16 months (range, 11-174 months). The median temperature on admission was 38°C (range, 36.8-39.0°C). The median erythrocyte sedimentation rate was 60 mm/h (range, 39-120 mm/h); the median white blood cell count was 11.7 × 10^9/µL (range, 6.5-18.5 × 10^9/µL). Symptoms were present for a median duration of 14 days (range, 7-35 days). Blood
**Clinical Symptoms and Neurologic Signs Among Children With Diskitis**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Symptoms</th>
<th>Neurologic Signs</th>
<th>Imaging Diagnosis and Findings</th>
<th>Duration of Symptoms, d</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/19</td>
<td>Intermittent limping, antalgic walk, refusal to bear weight</td>
<td>Decreased tendon reflexes in right leg and absent in left leg</td>
<td>BS: increased uptake in S1-L5 interspace</td>
<td>21</td>
</tr>
<tr>
<td>2/30</td>
<td>Refusal to bear weight, back pain, abdominal pain, constipation</td>
<td>Decreased muscle power (L4-5), absence of tendon reflexes was more pronounced in the left leg</td>
<td>XR: narrowing of disk space, interspace endplate irregularities; BS: increased uptake in L4-5; CT: disk space narrowing, lytic lesions in endplates, paravertebral mass; MRI: disk destruction with disk space narrowing and involvement of adjacent vertebrae, inflammatory tissue depressing the left psoas, no intraspinal protrusion</td>
<td>21</td>
</tr>
<tr>
<td>3/174</td>
<td>Low back pain, limping</td>
<td>None</td>
<td>XR: disk space narrowing; BS: increased uptake in L4-5 interspace</td>
<td>7</td>
</tr>
<tr>
<td>4/15</td>
<td>Anorexia, pain and crying during movements, limited flexion of back</td>
<td>None</td>
<td>XR: disk space narrowing</td>
<td>10</td>
</tr>
<tr>
<td>5/22</td>
<td>Limping, limited flexion of back, refusal to bear weight or spontaneously move</td>
<td>Decreased tonus and absent tendon reflexes in both legs</td>
<td>XR: disk space narrowing; BS: increased uptake in L2-3 interspace; MRI: inflammatory process involving the vertebral bodies and intervertebral disk</td>
<td>25</td>
</tr>
<tr>
<td>6/12</td>
<td>Refusal to bear weight, opisthotonus, irritability</td>
<td>Hypotonus, head lag, decreased muscle power (4/5)</td>
<td>XR: disk space narrowing; BS: increased uptake in T9-10 interspace; MRI: inflammation in the intervertebral disk involving the adjacent vertebral bodies and the paravertebral tissue</td>
<td>21</td>
</tr>
<tr>
<td>7/11</td>
<td>Irritability, refusal to stand, leg dragging in crawling</td>
<td>None</td>
<td>BS: increased uptake in L5-S1 interspace</td>
<td>10</td>
</tr>
<tr>
<td>8/15</td>
<td>Pain and crying in walking and standing, refusal to bear weight</td>
<td>Bilateral Babinski sign, decreased muscle power in both legs (4/5)</td>
<td>BS: increased uptake in L3-4 interspace; MRI: narrowing of the intervertebral space and enhancement on gadolinium image of mass pressing the dura with involvement to L3-4 disk</td>
<td>35</td>
</tr>
<tr>
<td>9/168</td>
<td>Low back pain, limitation in movements, hyperlordosis</td>
<td>None</td>
<td>XR: narrowing of disk space; BS: increased uptake in T12-L1 interspace</td>
<td>20</td>
</tr>
<tr>
<td>10/14</td>
<td>Refusal to sit, hyperlordosis, wide-base walking</td>
<td>None</td>
<td>XR: narrowing of disk space; BS: increased uptake in L3-4 interspace</td>
<td>16</td>
</tr>
<tr>
<td>11/13</td>
<td>Refusal to bear weight or walk, standing only with support</td>
<td>Hypotonus, decreased muscle power in both legs (4/5)</td>
<td>XR: narrowing of disk space L5-S1; BS: increased uptake in L5-S1 interspace</td>
<td>14</td>
</tr>
<tr>
<td>12/13</td>
<td>Refusal to stand or sit</td>
<td>None</td>
<td>XR: narrowing of disk space L2-3; BS: increased uptake in L5-S1 interspace</td>
<td>7</td>
</tr>
<tr>
<td>13/16</td>
<td>Limp in left leg</td>
<td>None</td>
<td>XR: narrowing of disk space T12-1; BS: increased uptake in L12-L1 interspace</td>
<td>7</td>
</tr>
<tr>
<td>14/25</td>
<td>Limping, refusal to stand</td>
<td>None</td>
<td>XR: narrowing of disk space L3-4; BS: increased uptake in L3-4 interspace</td>
<td>8</td>
</tr>
<tr>
<td>15/16</td>
<td>Limping, hyperlordosis</td>
<td>None</td>
<td>XR: narrowing of disk space L5-S1; BS: increased uptake in L5-S1 interspace</td>
<td>7</td>
</tr>
<tr>
<td>16/18</td>
<td>Back pain, refusal to stand, hyperlordosis</td>
<td>None</td>
<td>XR: narrowing of disk space L4-5; BS: increased uptake in L4-5 interspace</td>
<td>14</td>
</tr>
<tr>
<td>17/13</td>
<td>Restlessness, refusal to bear weight or spontaneously move</td>
<td>Hypotonus, decreased tendon reflexes in lower extremities</td>
<td>BS: increased uptake in L5 interspace; CT: subacute lytic lesion in L4-5 region, extradural involvement in L5-S1 region; MRI: inflammatory process involving L4-5 vertebrae and intervertebral disk, extending to epidural fat</td>
<td>35</td>
</tr>
</tbody>
</table>

*BS indicates bone scan; XR, radiography; CT, computed tomography; and MRI, magnetic resonance imaging.*

cultures were sterile in 15 (88%) of the 17 patients with diskitis. Among patients who had positive blood culture results, *Kingella kingae* was isolated from 2. Penicillin G was given to patients requiring intravenous therapy, and oxacillin sodium or a first-generation cephalosporin (cefazolin) was given to those requiring oral therapy.

Neurologic findings on physical examination at admission revealed a refusal to bear weight or move spontaneously in 7 of the 17 patients. Four had a bilateral decrease in tendon reflexes (patients 1, 2, 5, and 17), 4 had a decrease in muscle tone (patients 5, 6, 11, and 17), and 4 had decreased muscle power (4/5) (patients 2, 6, 8, and 11). Patient 8 had bilateral Babinski sign, and patient 6 had a head lag that could not be explained by inflammatory lesions. Five of these 7 children underwent magnetic resonance imaging; 2 patients improved before the imaging was done. Magnetic resonance imaging demonstrated intraspinal inflammatory involvement in 2 of the 5. In patients 8 and 17, pressure on the spinal cord was demonstrated, and they were referred to the Department of Neurosurgery at another hospital for treatment with laminectomy and discectomy. In patients 1 and 2, hyporeflexia and hypotonia were lateralized and confined to the
lower extremities, suggesting a localized injury, and disappeared within 3 weeks. In all children, the neurologic findings and other clinical manifestations of the disease resolved with antibiotic treatment and bed rest. The mean time to diagnosis after onset of symptoms in patients with neurologic signs was 20 days, compared with 17 days in patients without neurologic findings.

**COMMENT**

Disk inflammation (pyogenic spondylitis) in children is believed to result from an infection. Staphylococcus aureus and *K* kingae are the organisms most commonly isolated from disk aspiration.1,6

The differential diagnosis of neurologic impairment in the lower extremities of children with low back pain generally includes spinal tumors, vertebral osteomyelitis,1 or epidural abscess formation,6 but not a clinical presentation of diskitis.6,5,2 Our review of the literature revealed only a few case reports and larger series3,8,13 describing neurologic involvement associated with diskitis. Kelfer and Haller2 described a patient with diskitis at the L4-5 intervertebral space who had Gowers sign as a notable physical finding. Rocco and Eyring described “the meninges symptoms complex,”4(p449)8 which consists of meningeal irritation as manifested by the presence of Kernig or Brudzinski signs. Hyperreflexia and clonus may be present.

Crawford et al, in a review of 36 children with diskitis, suggested that the protrusion of the disk and its contents into the spinal canal (as seen on magnetic resonance imaging scan in only 1 patient among that group) may explain the “occasional neurologic sign associated with diskitis.”9(p78) Ring et al10 noted neurologic involvement in the form of radicular pain with a mildly reduced patellar reflex in the foot of 1 of 47 children with diskitis; the child developed a paravertebral abscess.

In another review, Scoles and Quinn11 did not describe any neurologic impairment among 29 children with intervertebral diskitis. Likewise, Ventura and colleagues12 described 12 children with diskitis, 1 of whom had exaggerated patellar reflexes. Despite the presence of a paraspinal inflammation or a small collection of hemorrhage in 36 patients with diskitis, Fernandez et al2 observed only 1 patient in whom adverse neurologic symptoms or signs were noted.

In the present review of the medical records of 17 children treated in our hospital for diskitis, we found signs of neurologic impairment in 7. These signs could be explained by other causes, such as normal variation in reflexes, tone, or pain. These reflex changes were present at initial evaluation and evolved during the course of the illness. Although this small sample size does not permit evaluation of the frequency of neurologic impairment in children with diskitis, our findings suggest that it is not as rare as generally thought. Furthermore, resolution of the neurologic impairment and other clinical manifestations in 5 children after antibiotic treatment and in 2 children with neurosurgical intervention points to an inflammatory pathogenesis. Therefore, neurologic findings may serve as an indication for the follow-up and evaluation of the progression of the disease.

**What This Study Adds**

Neurologic impairment is not a clinical manifestation of diskitis in children and has rarely been referred to in the medical literature in association with this disorder. The aim of this study was to describe neurologic findings in children with diskitis and their implications. We conclude that neurologic impairment does not exclude a diagnosis of diskitis, as was suggested in the past, and may be a clinical manifestation of the disease. Nevertheless, when neurologic findings are present, imaging studies are needed to exclude intraspinal involvement.

The findings of decreased muscle tone, muscle weakness, and decreased tendon reflexes in our patients are compatible with lower motor neuron injury. This kind of injury is expected when pressure is applied on the spinal cord or on the root of the nerves in the paravertebral region.

In summary, the spectrum of neurologic involvement associated with diskitis in children ranges from no involvement to radicular pressure to intraspinal pressure, depending on the extent of the inflammatory process that evolves from the intervertebral disk. Because we define diskitis more heterogeneously in terms of etiology and severity, the incidence of neurologic complications in our series was higher than that among other surveys of diskitis in children.

We conclude that neurologic impairment does not exclude a diagnosis of diskitis, as suggested in the past,2,4,5,12 and may be a clinical manifestation of the disease. Nevertheless, when neurologic findings are present, imaging studies are needed to exclude intraspinal involvement.

Accepted for publication June 13, 2002.

We thank Gloria Ganzach and Phyllis Curchack Kornspan for their editorial assistance.

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**REFERENCES**


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