Syndrome of the incidental herniated lumbar disc

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Lumbar myelographic defects consistent with herniated disc were found in 108 asymptomatic patients undergoing myelography for other reasons. Within 3 years, 64% of these patients developed symptoms of lumbosacral radiculopathy. The clinical features of these patients comprise a syndrome significantly different from that typically associated with classical lumbar disc herniation: the syndrome described here carries a much higher incidence of silent root compression with minimal pain. Incidental lumbar myelographic defects are not necessarily benign findings, and patients in whom they are encountered deserve close clinical follow-up review and appropriate treatment if the defects become symptomatic.

KEY WORDS • herniated disc • incidental finding • lumbar disc disease • myelography

Myelographic defects in the lumbar region consistent with disc herniations are not infrequent incidental findings in asymptomatic patients undergoing myelography for other reasons. While several authors have noted the presence of such defects in up to 56% of myelograms, there is little information relative to the clinical significance of incidental myelographic abnormalities in the asymptomatic patient. Specifically, it is not known whether these incidental defects do indeed represent herniated discs and, if so, whether such "minor" herniations have a tendency to become asymptomatic over time.

In order to answer these questions, we undertook a retrospective analysis of 108 patients with incidental lumbar myelographic defects treated at the University of Pittsburgh Health Center Hospitals over the past 5 years. We will attempt to define the natural history of this condition and to examine the possible pathophysiological mechanisms involved in the symptomatic onset of some of these lesions.

Clinical Material and Methods

From 1975 to 1980, 122 patients undergoing posterior fossa or cervical positive contrast myelography at the University of Pittsburgh Health Center Hospitals were discovered to have lumbar defects consistent with disc herniation. Myelography was performed via the lumbar intrathecal route with metrizamide in 82% and Pantopaque in 18% of the patients. The myelographic criteria of Taveras and Wood for lumbar disc herniation were used: 1) lateral indentation in the contrast column; 2) asymmetrical root sleeve filling; 3) ventral defect with double contour greater than 2 mm; and 4) partial or complete obstruction of the subarachnoid space (hour-glass deformity).

When a myelographic defect was discovered in the lumbar region, the patient's recorded medical history was carefully reviewed for symptoms of back pain or lumbosacral radiculopathy. If such symptoms were present or if physical findings consistent with root compression were found, the patient was excluded from the study. Follow-up data on those patients included in the study were obtained either by direct communication or detailed questionnaire.

A total of 108 patients were admitted to the study. They ranged in age from 37 to 64 years, with a mean of 43 years. Fifty-eight percent were male and 42% were female.

Results

Incidental lumbar myelographic defects consistent with herniated disc were found in 122 patients. Of these, 14 were excluded because of previous or current symptoms of back pain or lumbosacral radiculopathy, or because of the presence of physical findings compatible with root compression. The remaining 108 patients were considered to have incidental, asymptomatic herniated lumbar discs. Defects were present at the L4-5 level in 56.4% and the L5-S1 level in 41.6%. Lateral root-sleeve defects were seen in 76.8%, while ventral defects were seen in 13.8%; 9.4% showed partial ob-
To read this document naturally:

**TABLE 1**

Profile of 108 patients with incidental lumbar disc herniation

<table>
<thead>
<tr>
<th>Data</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>sex (M:F)</td>
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<tr>
<td>age (yrs)</td>
<td>37–64</td>
</tr>
<tr>
<td>myelographic defects</td>
<td></td>
</tr>
<tr>
<td>vertebral level (%)</td>
<td></td>
</tr>
<tr>
<td>L3–4</td>
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</tr>
<tr>
<td>L4–5</td>
<td>56.4</td>
</tr>
<tr>
<td>L5–S1</td>
<td>41.6</td>
</tr>
<tr>
<td>type (%)</td>
<td></td>
</tr>
<tr>
<td>lateral root sleeve</td>
<td>76.8</td>
</tr>
<tr>
<td>ventral</td>
<td>13.8</td>
</tr>
<tr>
<td>partially obstructed subarachnoid space</td>
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</tr>
</tbody>
</table>

**TABLE 2**

Presenting signs and symptoms in 70 of 108 patients in this series

<table>
<thead>
<tr>
<th>Signs &amp; Symptoms</th>
<th>Cases</th>
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</thead>
<tbody>
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</tr>
<tr>
<td>back pain</td>
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<tr>
<td>radicular pain</td>
<td>22</td>
</tr>
<tr>
<td>no pain</td>
<td>45</td>
</tr>
<tr>
<td>positive straight-leg raising test</td>
<td>22</td>
</tr>
<tr>
<td>motor/sensory deficits</td>
<td>48</td>
</tr>
<tr>
<td>foot-drop</td>
<td>5</td>
</tr>
<tr>
<td>bowel/bladder dysfunction</td>
<td>0</td>
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</tbody>
</table>

**Fig. 1.** Upper Left: Large extradural defect at L4–5 in a patient with no symptoms or signs of lumbar disc disease. Upper Right: Six months later the patient developed a painless foot-drop, and a repeat myelogram demonstrated an unchanged L4–5 defect. Lower: Computerized tomography scanning substantiates the presence of disc pathology.

Symptoms consistent with lumbar disc herniation developed in 70 of the 108 patients (64%) during a follow-up period of 1 to 5 years after the initial myelogram. The interval between myelography and the onset of symptoms ranged from 6 months to 3 years, with a mean of 9 months. Presenting symptoms were primarily those of numbness, paresthesias, and/or extremity weakness (68.5%). Back pain rarely occurred (14.2%), and radicular pain was infrequent (31.4%). Back pain and radicular pain were entirely absent in 64.2% of patients. The straight-leg raising test was positive in only 31%. Sensory abnormalities, reflex changes, and/or motor weakness were present in various combinations in 68.5% of the cases. Significant weakness to the point of overt foot-drop was present in 7% (Table 2).

A second myelogram and subsequent lumbar disc surgery were performed in 65 patients whose symptoms and signs did not improve with conventional conservative therapy. In all cases, the myelographic defect previously visualized was essentially unchanged on repeat examination. Laterally placed root-sleeve defects were present in 85% and partial obstruction of the subarachnoid space was found in 15% of patients. Sixty-nine percent of the patients subsequently underwent electromyography and nerve conduction velocity testing, which corroborated the presence of root dysfunction consistent with the myelographic and clinical findings. Lumbar computerized tomography was carried out in 31%, in all cases confirming the myelographic findings of disc pathology.

At operation, root compression by herniated disc material was found in 61 patients; two other patients had facet hypertrophy, and two had localized canal stenosis. Surgical intervention resulted in complete pain relief in 84%, with significant amelioration in another 8%. Paresthesias were relieved or reduced in 75%. The reversal of foot-drop to the point of full functional use without bracing was achieved in two of the five patients so affected. The remaining patients had no further progression of their weakness. Sensory and reflex changes rarely improved with surgery; however, no
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FIG. 2. Clinical presentation of incidental herniated disc syndrome versus classical lumbar disc disease. SLR = straight-leg raising test.

patient has developed progressive deficits in up to 5 years of follow-up review.

Discussion

Since the introduction of positive contrast myelography, incidental abnormalities have been noted in the contrast column that could not be correlated with the patients’ signs and symptoms. Generally, such abnormalities were considered false-positive myelographic signs caused by minor bone changes, needle artifacts, thickening of the ligamentum flavum, or arachnoid adhesions. Leader and Rassell and Gurdjian, et al., reported an incidence of 2% to 15% of such defects in their myelographic studies. The majority of these incidental defects were noted in the lumbar region in patients with no symptoms of lumbar disc disease. As these defects were indistinguishable from those seen in symptomatic patients, the argument sometimes arose as to whether they should be considered as benign incidental findings.

McRae was the first to address this problem comprehensively. Before him, several autopsy studies had already shown that anular tear and bulging and frank herniation of the lumbar discs were present in up to 68% of cadaver spines. McRae also reported a 61% incidence of anterolateral disc protrusion and a 39% incidence of posterior disc protrusion in his cadaver studies. In an attempt to provide clinical correlations, McRae also analyzed the myelograms of 46 patients with no symptoms of lumbar disc disease and included only “medium-sized or large posterior or posterolateral” defects in the contrast column as being indicative of disc herniation. He found 23 cases of such defects in 40 patients (58%) aged 30 to 50 years and similar defects in all six patients over 60 years of age. McRae concluded that “disc protrusions are common but they seldom produce symptoms” and further asserted that everyone over 40 years old had at least one asymptomatic disc protrusion.

Trowbridge and French studied 25 patients undergoing cervical myelography, and found 14 (56%) with lumbar defects without a history of back pain, sciatica, or other lumbar root symptomatology. They thought that such defects were not artifacts but were due to structural abnormalities causing incomplete filling, and postulated that “it is entirely conceivable that any of the patients with abnormal lumbar myelograms might develop lumbar sciatica at a later date.” Unfortunately, their patients were not followed.

Only one other series addressed the problem of incidental asymptomatic lumbar myelographic defects. Hitesberger and Witten found a 24% incidence of such defects in 300 myelograms done for posterior fossa lesions, and warned of the danger of overemphasizing their clinical significance.

From the limited data available, incidental asymptomatic lumbar disc herniation subsequently developed symptoms within 3 years after the abnormality was first identified. In all cases, the myelographic findings at the time of symptom onset were identical to those noted at the time when the lesions were asymptomatic (Fig. 1). Moreover, all patients initially found to have partial obstruction of the subarachnoid space subsequently became symptomatic. Of those with lateral root-sleeve defects identified initially, 72% went on to develop symptoms. No patient with an incidental ventral defect ever became symptomatic.

When they do become symptomatic, these incidental lesions produce clinical features distinct from those associated with classical lumbar disc disease (Fig. 2). Back and radicular pain, a hallmark of classical lumbar disc herniation, occurred infrequently in patients with incidental lesions, such patients being much more likely to present with paresthesias and painless motor loss. A positive straight-leg raising test was likewise uncommon in these patients, while sensorimotor deficits were much more prominent than in patients with the classical syndrome. Bowel and bladder disturbances were not encountered in patients with incidental disc herniation.

The results of surgical intervention in patients with incidentally diagnosed herniated discs are comparable to those achieved in classical disc disease. Pain was
relieved in the majority of patients so affected, and significant motor deficits were reversed in less than 50%.

In some respects, the symptomatology of the incidental herniated lumbar disc resembles that encountered in patients with extreme lateral herniation, first described by Abdullah, et al. Their 24 patients experienced no back pain; however, all had characteristic anterior thigh or leg pain. A positive straight-leg raising test was absent in all but one instance, and nerve-root compressive phenomena, such as sensorimotor deficits, were prominent.

It is not known why such incidental disc herniations become symptomatic after having remained silent for variable periods. In classical lumbar disc disease, back pain results from stretching of the pain-sensitive anular fibers (supplied by the sinuvertebral nerve of Luschka) due to outward bulging of the nucleus pulposus. Once the anulus is breached in cases of frank nuclear extrusion, the anular fibers are no longer stretched, and back pain then gives way to radicular pain as the nerve root itself becomes compressed by the dislocated fragment. However, painless sensorimotor paralysis due to lumbar disc herniation has been reported.

Garrido and Rosenwasser described painless foot-drop in two patients with myelographically and surgically proven L4–5 disc rupture. Bladder dysfunction caused by otherwise "asymptomatic" lumbar disc rupture has also been reported. These patients had chronic urinary retention secondary to asymptomatic central disc prolapse, and bladder function tended to improve following discectomy.

Three mechanisms may explain the lack of significant pain while other signs of nerve-root compression are prominent in patients with incidental disc herniations: 1) the episodes of back and/or leg pain are so fleeting that they are quickly forgotten or are attributed to other causes, such as renal colic or phlebitis; 2) the bulging and subsequent rupture of the anulus are of such a gradual and insidious nature as to allow for slow adjustment of the nociceptive nerve endings within the anulus; and 3) recurrent, subtle bulging causes repetitive neural injuries to occur in such minute installments that the ablative phenomenon of sensorimotor deficits occur without the irritative phenomenon of pain.

With regard to the first explanation, Taveras and Wood stated emphatically that "if the past history is reviewed carefully enough, the patient will indicate that there were symptoms on the side of the asymptomatic disc which at some time later disappeared." However, we were unable to elicit any history of previous back or leg pain in any of our patients, despite careful questioning. Nevertheless, it might be expected that, at least in some patients, previous episodes of pain have been forgotten or ascribed to other causes.

Scham and Taylor postulated that asymptomatic disc herniation results from the herniating process having developed gradually over a long period. Falconer, et al., agreed with this concept, and added that symptoms will eventually appear as a result of some abrupt and temporary increase in the size of the herniation.

To support this contention, Falconer, et al., studied 10 patients with sciatica and myelographic signs of disc herniation who experienced complete relief of symptoms with nonsurgical treatment alone. Myelographic defects identical to those seen initially were found subsequent to the abatement of symptoms, a situation analogous to that in patients with incidental disc herniation, only in reverse. In incidental disc herniation, myelographic findings at the onset of symptoms were indistinguishable from those present during the asymptomatic phase.

The third mechanism for painless root compression in incidental disc herniation involves recurrent minor disc bulging and repeated minimal nerve-root damage. Support for this mechanism can be found in the studies of Lindblom and Rexed. Eleven of their 17 patients who were found at autopsy to have marked lumbar disc protrusion did not have a clinical history of back pain or sciatica. In all 11 cases, however, there was evidence of repetitive neural damage; a mixed picture of degeneration and regeneration in the nerve roots suggested that multiple instances of repeated trauma had occurred over an extended period of time. In such a situation, sensorimotor deficits may ultimately become manifest without pain.

Conclusions

Incidental lumbar myelographic defects are not necessarily benign findings; in many cases they represent true disc herniations that are only temporarily asymptomatic. Many of these lesions will produce symptoms within the first few years following the initial myelogram. Patients with incidentally discovered partial obstruction of the lumbar subarachnoid space have the greatest likelihood of becoming symptomatic (100%); those with laterally placed root-sleeve defects have a greater than 70% chance of developing symptoms, while those with ventral defects do not become symptomatic. The clinical features of incidental herniated discs comprise a distinct syndrome different from that seen in classic lumbar disc disease: a much higher incidence of painless root compression occurs in the former group. However, surgical intervention produces comparable results in both syndromes. For these reasons, patients found to have incidental lumbar myelographic defects deserve to be followed closely, and appropriate treatment should be instituted promptly if symptoms or signs of radiculopathy arise.

References


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