Spontaneous Regression of Herniated Nucleus Pulposus

J. George Teplick and Marvin E. Haskin

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Spontaneous Regression of Herniated Nucleus Pulposus

Spontaneous regression of herniated nucleus pulposus has not been previously documented. Reported here are 11 patients in whom there was unequivocal regression or disappearance of a herniated lumbar disk on follow-up CT study. Two patients with herniated disks were without symptoms. In the nine patients with symptoms, those attributed to the original herniation disappeared or were diminished in all cases. The mechanism of regression of a disk herniation is unknown. Whether or not regression of herniated disk is a frequent occurrence in patients who recover with conservative therapy should be investigated by more frequent use of follow-up CT scans.

Spontaneous regression of a herniated nucleus pulposus (HNP) in the lumbar or cervical spine might have been suspected and perhaps even postulated because of the disappearance of symptoms with nonsurgical treatments. However, no reports on this subject could be found, even after a computer literature search. Until the advent of CT, demonstration of regression would have required repeat myelography, which would not have been indicated if clinical improvement had occurred. We present 11 cases in which serial CT studies disclosed an unequivocal spontaneous regression or complete disappearance of an HNP.

Materials and Methods

Although the diagnosis of HNP has been made in over 1000 patients by CT in the past 3 years in the course of well over 10,000 CT spine examinations at Hahnemann Hospital, 55 patients had follow-up CT scans without either intervening surgery or a chymopapain injection. In these 55, reexamination was prompted by persisting symptoms or symptoms of a new type, sometimes on the opposite side. Such reexaminations provided opportunity to reassess HNP that may or may not have been originally symptomatic.

In nine patients the CT findings of a regression of an HNP were serendipitous. In two others, however, the follow-up CT was deliberately arranged in a search for regression of the HNP. None of these 11 had any interventional therapy; bed rest, physiotherapy, and, in some cases, exercises were the only treatments. In the remaining 39, the CT findings indicated similar or additional abnormalities.

All these patients had their CT studies performed on the same unit (a GE 8800) and by the same group of technologists. The techniques used for the original and the later scans were identical. In each case, virtually identical sections were available for comparing changes in the size and appearance of the HNP. Such uniformity of technique is essential in a study of this kind; different slice angulations or nonidentical positions during scanning can produce projection differences that would make accurate comparison of changes of the herniation difficult. The author who made all the interpretations (J. G. T.) is confident that the CT changes were real in all the cases, and not due to technical factors. Any cases in which he was uncertain of real change were excluded.
In 11 patients, an unequivocal decrease or total regression of the disk herniation was seen on the follow-up scans (figs. 1–12). The time intervals between the original and follow-up scans ranged from 5 months to 3 years.

Clinical improvement accompanying the regression of herniation occurred in each of the nine patients who were originally symptomatic. Radicular symptoms disappeared in every one. In two others, regressed HNP was apparently asymptomatic from the start; the clinical problems were from herniations at other interspace levels. The most gratifying findings were in the two patients whose clinical symptoms had disappeared and who consented to have a scan at our request. In both of these, the herniation had disappeared (figs. 1 and 2). The most unexpected finding was regression and disappearance of calcified herniations (figs. 9 and 10 [see Addendum]). They were also asymptomatic.

**Results**

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SPONTANEOUS REGRESSION OF HERNIATED DISK

Fig. 5.—Regression of HNP at L3–L4: 28-year-old man suffered low back pain and severe left radiculopathy. A, HNP at L3–L4 level. Herniated disks were also identified at L4–L5 and L5–S1. All three were left-sided. About 2 years later, radiation to buttocks had decreased, but radiation to left leg and foot had increased. On CT, HNP at L5–S1 had increased, while L3–L4 HNP (B) had disappeared. L4–L5 herniation was unchanged.

Fig. 6.—Regression of HNP at L4–L5. A, Successive sections of L4–L5 3 months after auto accident, which resulted in right radiculopathy. Large right-sided HNP is clearly visible (arrows). One year later, right radiculopathy was greatly improved. B, Corresponding sections show virtually complete disappearance of HNP. Questionable residual small herniation was seen (arrows).

Fig. 7.—Virtual disappearance of HNP in 5 months. A, Section at L4–L5. Clear-cut right-sided HNP (arrow), which was accompanied by typical clinical symptoms. By 5 months later, right-sided radiculopathy had abated, although low back pain persisted. B, No definitive HNP. C, Enhanced study done at same time displays no posterior rim enhancement and shows perfectly normal-appearing anulus. These three images are almost completely identical sections, but A is lighter (lower window level) exposure.

Discussion

The clinical course of a symptomatic lumbar disk herniation is variable and generally unpredictable. The back pain and radiculopathy may persist, worsen, diminish, or even totally disappear. Recurrent episodes after long or short symptom-free intervals are quite common [1]. An acute episode of low back pain and radiculopathy is generally treated conservatively; if improvement or remission occurs, CT is usually not done. Many such cases are probably relatively acute disk herniations, but undocumented.

Before lumbar CT, in cases of HNP documented by myelography, a follow-up myelogram was virtually never obtained; consequently there is little or no information about regressive changes in the size of the myelographic defect. In one case (personal communication), a clear-cut myelographic defect of a herniated disk associated with typical clinical symptoms was no longer seen on a myelogram done 1 year later for other reasons. This is the only case that has come to our attention in which a definite herniation defect has apparently disappeared spontaneously as shown by a later myelogram.

The decrease in the size of an HNP on the follow-up scans
Fig. 8.—Regression of HNP at L4–L5. A, Section at L4–L5. Central and left-sided HNP (arrow). Small central HNP was also seen at L5–S1. Clinically, radiculopathy was left-sided. B, Corresponding section 10 months later. No evidence of HNP. Clinically, left radiculopathy had disappeared, but new right radiculopathy had developed. Right-sided HNP had developed at L5–S1.

Fig. 10.—Disappearance of calcified HNP at L4–L5: 40-year-old technologist had developed severe back pain with radiation down right leg. A, Section at L4–L5. Densely calcified herniated disk (arrows) right of midline. By 3½ months later, radiculopathy had entirely subsided, although occasional backaches were experienced. B, Scan shows complete disappearance of calcified disk; anulus remains prominent. There was no sign of calcification, even after search using 1.5-mm-thick sections through anulus.

Fig. 9.—Disappearance of calcified HNP at L4–L5: 34-year-old man had recurrent episodes of right radiculopathy due to severe foraminal stenosis at L5–S1. Calcified herniation at L4–L5 was essentially asymptomatic. A, Calcified herniation at L4–L5 is clearly seen (arrow). B, About 3 years later, both soft-tissue herniation and calcification have totally disappeared. Anterior border of thecal sac can now be clearly delineated (arrows). These two sections are virtually identical and comparable. Images at bone window settings are shown to illustrate calcification more convincingly. This disappearance of calcified herniated disk is totally unexpected and contrary to our current understanding. A similar case has been seen (see fig. 10).

came as a complete surprise. We had conjectured that a herniated disk would not be able to retract into the anulus, even with an intact posterior longitudinal ligament. The clinical improvement in a patient with a documented disk herniation has been generally attributed to decreased swelling of the affected nerve root rather than retraction of the extended disk. However, a factor favoring improvement is related to the posterior retraction of the anulus (and its HNP) after extreme flexion of the spine, which can be seen on myelography. The decrease of anulus pressure on the thecal sac is dramatically demonstrated on a cross-table lateral myelogram taken in the regular and in the markedly flexed position (fig. 13). The usual flexed position that patients assume in conservative bed-rest treatment of a symptomatic HNP endorses the significance of this mechanism. Theoretically, the retraction of the anulus and its associated herniation during the flexion of bed rest may contribute to decreased nerve root irritation. Our findings suggest that there may also be a real decrease in the size of the HNP. Perhaps this regression is an important factor in many cases of clinical improvement or recovery.
Fig. 12.—Spontaneous disappearance of HNP at L5–S1. A. Original CT scans clearly reveal large right-sided herniation (arrows) at L5–S1. B. Corresponding sections 14 months later. No evidence of prior herniation. Normal anulus is seen (arrow). Right radiculopathy had disappeared within 3 months after first scan. Follow-up scan was done because of recent episode of low back pain.

Exactly how an HNP decreases in size remains a subject for speculation. Theoretical considerations would include (1) dehydration and shrinkage of the HNP, (2) regression of the HNP into the anulus via the tear in the anulus, and (3) fragmentation and subsequent sequestration at a distance from the anulus.

Of these three possibilities, fragmentation and a subsequent sequestration of the fragment seems quite unlikely. In all our cases, the posterior margin of the anulus remained quite sharp and regular, suggesting an intact posterior longitudinal ligament, which is strong evidence against fragmentation. Of the remaining two possibilities, we consider retraction of the nuclear material back into the anulus as a more likely possibility, since we cannot postulate conditions that would lead to a physiologic dehydration.

Regression of an HNP has been seen frequently after successful chymopapain therapy. In these cases, the anulus often regains a normal CT appearance, suggesting retraction of the herniation into the anulus.

We suspect that spontaneous regression of a herniated disk may not be a rare occurrence. In two cases, regression was suspected, searched for, and found. It probably would be rewarding if a series of follow-up CT scans could be made on many more patients with CT documented disk herniation whose symptoms have ameliorated or disappeared on conservative therapy. We hope other investigators will corroborate our findings.

REFERENCES

Addendum
Since submission and acceptance of this manuscript, we have encountered four additional cases of spontaneous regression of a lumbar herniated disk and one case of regression of a cervical disk. Since one of the lumbar cases was another instance of regression of a calcified HNP, the illustrations have been added as figure 10.