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BACKGROUND AND PURPOSE: **Literature regarding clinical pain syndromes associated with acute, traumatic Schmorl's nodes (SNs) is limited. Our purpose was to determine whether an SN could be related to a previous traumatic event producing either acute SN or a vertebral endplate fracture.**

METHODS: **Two neuroradiologists independently reviewed initial and follow-up MR examinations of 14 patients with a clinical diagnosis of acute, symptomatic thoracolumbar SNs or vertebral body endplate fractures that evolved into SNs to evaluate marrow edema, signal intensity, margin definition, presence of intravertebral extruded disk material, and pattern of contrast enhancement.**

RESULTS: **Edema of the affected vertebral body, adjacent to an endplate without wedging or collapse, was observed on the initial MR images in all cases. The initial MR images of six (43%) of 14 patients exhibited only edema of the marrow immediately adjacent to the endplate without wedging or collapse. The MR images obtained at the time of follow-up showed subsequent formation of a chronic and eventually asymptomatic SN for all six patients. The initial MR images of eight (57%) of the 14 patients showed the typical appearance of acute SNs with marrow edema of the affected vertebra. The contrast-enhanced images of three patients manifested enhancement of the invaginated disk material in three (100%) of three cases and enhancement of the surrounding vertebral body in one case (33%). Six (43%) of 14 patients had acute typical compression fracture of a vertebral body of at least one additional level.**

CONCLUSION: **Most (57%) of the SNs in this series could be traced to episodes of significant, sudden-onset, localized, nonradiating back pain and tenderness for which the MR images showed SNs surrounded by vertebral body marrow edema. The remaining SNs (43%) were not immediately apparent as SNs and manifested only as vertebral body edema representing endplate fracture but did evolve into classical chronic SNs that follow-up imaging revealed.**

Nonacute Schmorl's nodes (SNs) are common spinal abnormalities regarded as incidental observations on MR images or plain X-rays of the thoracolumbar spine. They may occur in 38% to 75% of the population (1, 2). The term *SN* represents a remote herniation or extrusion of intervertebral disk nuclear material through the vertebral body endplate, thought to be asymptomatic and of no clinical consequence (2). If it is assumed that SNs represent invagination of disk material into the vertebral body through a vertebral body endplate fracture, at one time, some of them may have been traumatically produced, symptomatic, and painful. Axial-loading trauma is known to result in vertebral endplate fractures with clinical features of localized, nonradiating low back pain, and local tenderness of sudden onset. The eventual observation of a classical chronic and asymptomatic SN may be assumed in select cases to have originated as an acute and painful event.

There have been isolated case reports of well-documented, clinically confirmed symptomatic acute SNs occurring in children and young adults (3, 4). Awareness that an acute SN may be a cause of acute back pain could facilitate an accurate early diagnosis, even though the therapeutic regimen may not change as long as no biomechanical instability is implied (5). This study seeks to determine whether an asymptomatic SN could be related to an acute SN or to a previous endplate fracture that eventually developed into a classical, asymptomatic SN.

Methods

Two senior members of the American Society of Neuroradiology retrospectively and independently reviewed original

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Summary of findings on initial MR examination in 14 patients with acute-onset low back pain after axial loading trauma

Age (yrs)/ Sex	Focal Endplate Defect	Diffuse Endplate Defect	Marrow Edema	Wedge at Adjacent Level	Narrow Disk Space
40/m (Fig 1)		+	diffuse	+	
38/m (Fig 2)	+		focal		+ (immediate)
37/m (Fig 3)	+		focal		
45/m (Fig 4)	+		focal		+
26/f		+	diffuse	+	
31/m	+		focal		+
28/m	+		focal	+	+ (immediate)
32/f		+	diffuse		
22/m	+		focal	+	+
25/m		+	focal	+	+
19/m	+		focal		+
30/m		+	diffuse	+	
26/m	+		focal	+	
29/m		+	focal		+

and follow-up MR images of the thoracolumbar spines of 14 patients. All 14 patients initially presented with acute onset of localized nonradiating back pain after axial-loading injuries. Diagnosis of acute SN or endplate fracture was made based on MR images that showed marrow edema in a nondeformed vertebral body or disk signal in an endplate SN defect with marrow edema, which imaging showed to develop into classical asymptomatic SN.

At the time of original presentation, all patients experienced the sudden onset of localized, nonradiating low back pain and tenderness to palpation referable to the level of observed abnormality. Six (43%) of the 14 patients whose initial MR images revealed endplate edema and endplate fracture underwent clinical and MR follow-up examinations ranging from 8 months to 11 years and were observed to develop classical SNs in the location of the original endplate fracture. The patients' clinicians directly attributed the acute onset of localized low back pain and tenderness to deep palpation, which was found during the examinations of the 14 cases at the time of original presentation, to acute SN or endplate fracture that MR imaging showed to evolve into classical SNs. Resolution of the patients' pain (median time to resolution of pain, 3.5 months) correlated well with the disappearance of marrow edema and subsequent MR appearance of evolution of the vertebral body defect or endplate edema into a classical, chronic SN. The case of each patient was presented to an institution-wide, multispecialty spine conference, at which a diagnosis of acute, symptomatic SN or endplate fracture eventually evolving into SN was reached by consensus.

Initial MR imaging was performed in all cases within 7 days of the precipitating injury and at follow-up examinations using 1.5-T whole-body magnets and lumbar spine surface coils. In two of the initial images, T1-weighted (550/35/2 [TR/TE/number of excitations]) and T2-weighted spin-echo (2500/60/2) sequences were obtained. In the remainder, for both initial and follow-up studies, T2-weighted images using a gradient-echo sequence (415/20) were substituted for the T2-weighted spin-echo sequence. Because some of these studies were performed before the advent of fat-suppressed T2-weighted images, these sequences were not included in all cases. Sagittal and axial planes were acquired with both T1- and T2-weighted imaging. In three cases, contrast-enhanced T1-weighted sagittal and axial sequences were obtained after the IV administration of contrast material. A clinician requested contrast material administration because some additional information might be gained from contrast enhancement; it is not strictly necessary nor routinely administered in cases of spinal trauma imaging at our institution.

Results

The 14 patients consisted of 12 male and 2 female patients, ranging in age from 19 to 45 years; all except three were younger than 38 years (the Table). No patient had significant concurrent medical problems. All patients complained of sudden onset of localized low back pain, without radiculopathy or neuropathy, but with spinal localized tenderness to deep palpation. Nine patients had been involved in motor vehicle accidents in which they were thrown from the conveyance, and they sustained impact head injuries resulting in mixed axial loading and impact injuries. Five patients were injured in ski jumping incidents, which were more purely axial loading mechanisms of injury. None had biomechanical instability as a result of the trauma, and none required spinal fixation procedures as therapy. None of the vertebral bodies that eventually showed SNs were significantly compressed or wedged, but six (43%) of 14 patients had acute wedge compressions at additional levels. Vertebral body marrow edema, diagnosed by decreased signal on T1-weighted images and increased signal on T2-weighted images, was present in the region of the actual or subsequent SNs in all 14 cases, although the patterns differed. Eight of the 14 patients had initial marrow edema confined to the area immediately surrounding the acute SN, whereas in the remaining six, the edema was more diffuse and an actual vertebral endplate defect was not yet clearly visualized on MR images. In two of these latter patients, the edema pattern was associated with a more diffuse but very subtle deformation of the involved vertebral endplate, which was difficult to differentiate from simple vertebral body endplate fracture. The pattern and the extent of edema on the initial images did not correlate with the intensity of pain; clinical and imaging follow-up did not indicate that edema duration was related to duration of symptoms.

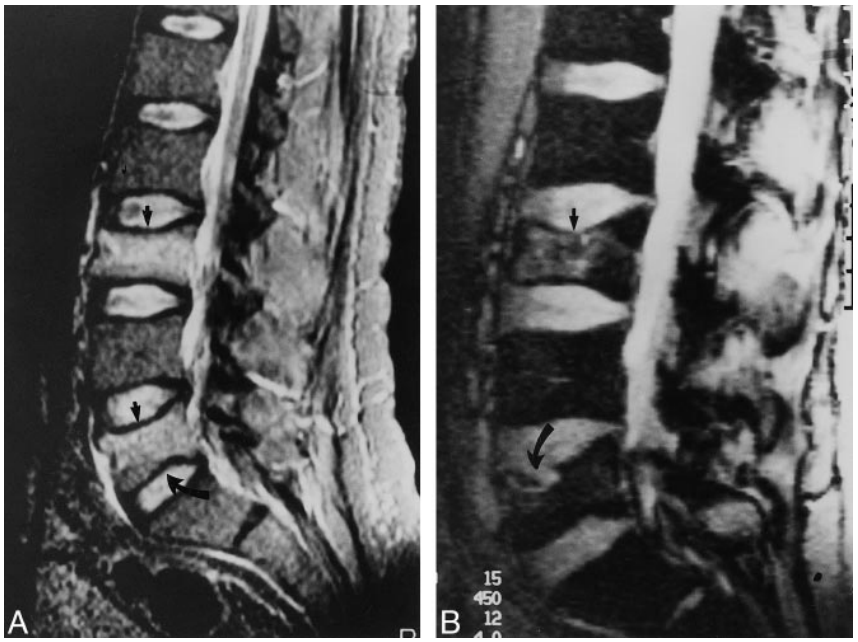

FIG 1. Images of a 40-year-old man who experienced axial loading injury when thrown from a car and who presented with nonradiating lumbar pain and tenderness directly over the L3 and L5 vertebral bodies.

A, Sagittal midline short inversion time inversion recovery (1400/15/2) image, obtained during the initial MR examination, shows vertebral body edema and mild compression of the superior surfaces of the L3 and L5 vertebral bodies, but no SN is present at either level. Both levels are consistent with simple endplate fractures. Note the smooth superior surface of vertebral bodies L3 and L5, with what appear to be intact cortical margins (*straight arrows*). The inferior endplate of L5 may also be slightly compressed (*curved arrow*).

B, Sagittal gradient-echo (450/12/1) MR image, obtained 2 months later when the patient experienced some but not complete resolution of pain and tenderness, shows that a chronic SN formed at the superior endplate of L5 (*curved arrow*). Note resolution of marrow edema at L5 with persistent but improved edema of L3 (*straight arrow*). Further follow-up did not show SN formation at L3. This case illustrates that some endplate fractures may evolve into SNs and some may not.

The initial MR examination of each patient showed either diffuse (n = 6) or focal (n = 8) concave abnormality or indentation of a vertebral endplate. On the MR images of the six patients with a diffuse endplate pattern, a cortical rim around the intruding disk material and endplate defect could not initially be easily seen, causing the abnormality to resemble a diffuse mild compression fracture or microfracture of the endplate more than an acute SN. In each of these cases, a classical SN was noted to have developed at that exact site at the time of the 7-month follow-up, leading to the supposition that one may be a precursor of the other (Fig 1). In the eight patients with a focal endplate deformity pattern on the initial MR images, disk signal was noted to be present, extending into the vertebral body through the endplate as a recognizable SN (Fig 2). In two of the cases with a diffuse pattern, there was heterogeneous signal on the T1-weighted images and a bright signal on the T2-weighted images, but invagination of disk material into the endplate could be appreciated only on the T2-weighted images (Fig 3). Seven patients had contemporaneous acute endplate fracture at a different vertebral body level (Fig 1). Another six patients manifested nonacute SN nodes (without marrow edema) at other levels in the lumbar spine (Fig 4).

The acute images showed narrowing of the disk space at the involved level for only two patients, whereas the remainder had intervertebral disks of normal height and signal. At follow-up, the initially normal-height disk spaces at the level of the acute SNs were noted to have narrowed over time, along with the development of classical SNs, for eight of the remaining 12 patients. The initially narrowed disk space also was noted to have become more narrowed or collapsed during the 2-year follow-up period.

Contrast-enhanced images were obtained at the time of the initial MR examination for one patient and at the time of follow-up examinations for two other patients. Enhancement within the SN as well as in the adjacent vertebral body was shown to have occurred in one patient. The contrast material helped for differentiating the margins of the acutely formed SN edema from the vertebral marrow edema surrounding it (Fig 3). Enhancement within the vertebral body consisted of a thick rim of enhancement surrounding the invaginated or extruded disk material with homogeneous enhancement in the disk material itself as well as in the parent intervertebral disk. The follow-up images of two additional patients, obtained 2 months and 4 years after the precipitating event (Fig 4), showed enhancement of the invaginated disk without vertebral body enhancement. These images exhibited contrast enhancement within the SN itself, probably representing scar within the extruded disk material, but showed no enhancement in the surrounding vertebral body.

Discussion

Depending on terminology, SNs are herniations, extrusions, or invaginations of intervertebral disk material into the vertebral body endplates and were first described in 1927 by Schmorl (6). They are usually observed as chronic, asymptomatic entities in approximately one third of the population, although one study reported a 74% incidence (1). They are generally thought to be of no clinical con-

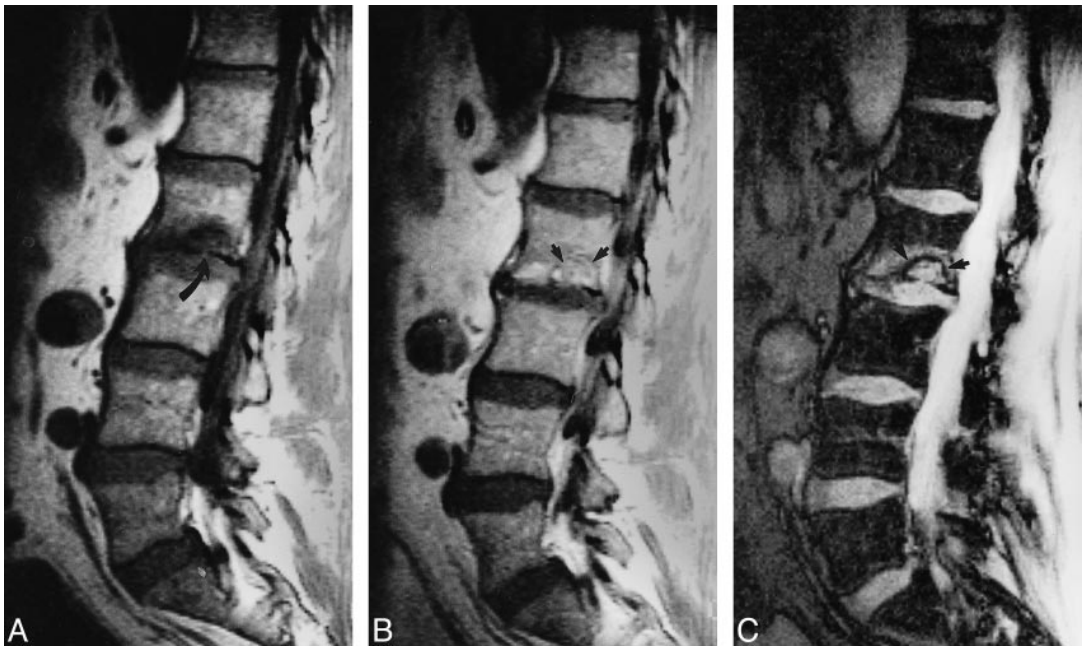

FIG 2. MR images obtained 6 days after injury of a 38-year-old man who experienced the sudden onset of pain and point tenderness referable to the L2 level immediately after experiencing axial-loading injury.

A, T1-weighted sagittal (600/15/2) image shows subtle concave compression of the caudal L2 endplate without obvious disk extension but with localized marrow edema. The margin of the SN is not well delineated because of the marrow edema, but a very small cortical defect representing an endplate fracture is present (*arrow*).

B, After the administration of contrast material, enhancement of the marrow edema peripheral to and within the SN defect can be seen. The margins of the SN are slightly more visible after the injection of the contrast material (*arrows*).

C, Gradient-echo (400/15/1) image best shows endplate invagination of disk material, establishing this abnormality as an acute SN. The margin of the SN can be most clearly appreciated as a linear area of decreased signal surrounding the extruded disk material (*arrows*) on the gradient-echo image. At 4-month follow-up, when the patient's pain had resolved, the edema had disappeared but the appearance of the SN on T2-weighted images was otherwise unchanged.

sequence, probably because their inception is assumed to be remote, and they are mentioned only as incidental findings. Reports in the literature of symptomatic acute SNs caused by trauma are sparse, with diagnosis being based on high clinical suspicion after exclusion of other causes (3, 7).

SNs are thought to be the long-term result of displacement of intervertebral disk material through cracks in an otherwise normal endplate after axial loading trauma (2, 8). Predisposing causes for endplate weakness might rarely include primary or secondary bone dysplasias, neoplasms such as multiple myeloma or metastases, disk space infection, diskitis, or any other processes that may weaken the endplate or the underlying bone. Weakening of the endplate is not a necessary condition for such extrusion and is thought to be present as an underlying cause in only a very small percentage of SN cases. Most SNs form after axial-loading trauma results in the preferential extrusion of nuclear material through the vertebral endplate rather than through an intact and normal annulus fibrosis. The annulus is actually biomechanically more resistant to mechanical failure than the vertebral endplate, especially in younger individuals (2, 8).

Nonsymptomatic, chronic SNs are most commonly found in the thoracolumbar region (1). They are typically well corticated but radiolucent lesions seen adjacent to the vertebral endplate, with surrounding intact cortical margins or even sclerosis on both radiographs and computed topographic scans. With MR imaging, SNs are most often noted on sagittal images as intervertebral disk signal extending through a defect in the vertebral endplate with intact cortical margins that have a thin, decreased area of signal intensity on all image sequences, similar to the cortex of the vertebral body itself. In nonacute SNs, adjacent vertebral body marrow edema is not present, there is no associated pain or tenderness, and enhancement has been described (9). There is usually, but not necessarily, a loss of normal water signal and height of the disk in cases of chronic SNs. Loss of height of the disk space has not been described in any of the acute SN reports in the literature and was seen in only one of the presumed acute SN cases in this series. Technetium-99m bone scans can show focal increased uptake of radiopharmaceutical associated with acute/subacute SN absent in nonacute cases (10).

Acute, traumatic SNs are much less frequently identified on routine images than are their asymptomatic, chronic counterparts, and they have been described in the literature only in isolated case reports (4, 5, 7). Perhaps the relative infrequency of this diagnosis as a cause of nonradiating low back pain is attributable to the low degree of awareness that acute SN may be symptomatic.

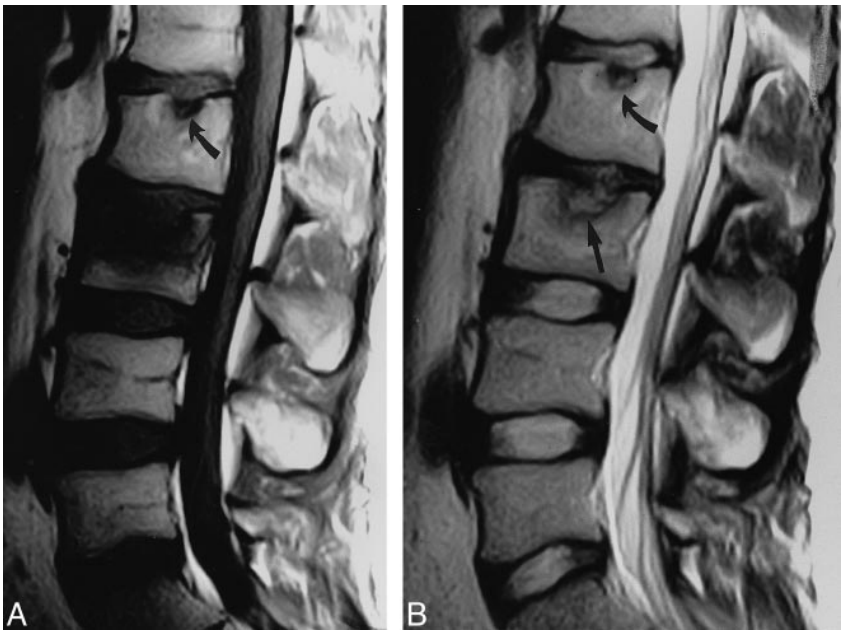

FIG 3. Images of a 27-year-old man who presented with sudden onset of pain and tenderness centered at L3 after a snow ski jump injury involving pure axial loading.

A, Sagittal T1-weighted (600/15/2) image shows diffuse marrow edema of the cephalad two thirds of vertebral body L3, centered along the superior endplate. On the T1-weighted image, this looks like a simple endplate fracture. Note the nonacute SN and the narrowed disk space at L2 (*arrow*).

B, Sagittal fast spin-echo T2-weighted (4000/90/2) image, obtained as part of the same examination, shows the margins of the acute SN at L3 to better advantage (*straight arrow*). The L2 SN is unchanged (*curved arrow*).

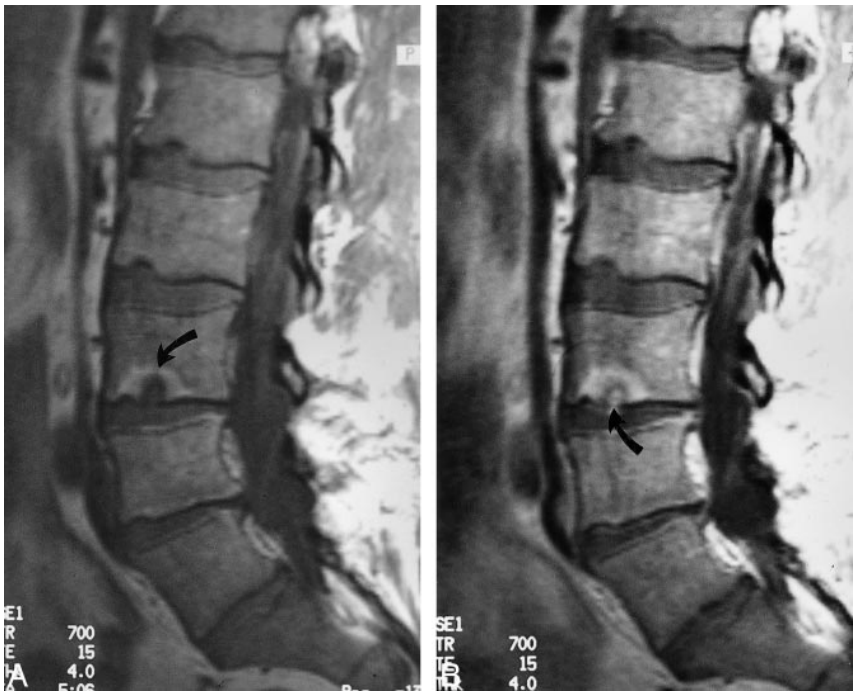

FIG 4. Images of a 45-year-old man who had experienced axial-loading injury 4 years before these images were obtained and who experienced the sudden onset of acute nonradiating low back pain with tenderness over L3.

A, Sagittal midline T1-weighted (700/15/2) image shows a small, nonacute SN at the inferior endplate of L3 with surrounding degenerative changes (*arrow*) and additional SNs without degenerative marrow changes at T12 to L2. The patient no longer experienced the pain and tenderness noted at the time of the original injury, and the original MR images are no longer in existence.

B, After the IV administration of contrast material, central enhancement within the asymptomatic SN was noted, probably in the granulation tissue that formed within the extruded fragment of disk (*arrow*). This illustrates one of the enhancement patterns that might be encountered with SNs. The other SNs from T12 to L2 do not enhance.

Vertebral body marrow edema was seen surrounding the acute SN on every initial MR image in this series and is thought to correlate with the recent occurrence of the disk extrusion into the endplate. Depending on the extent and the pattern, this may be indistinguishable from simple endplate fracture, and some endplate fractures may develop into classical SNs over time even if not shown to be present on contrast-enhanced images (Fig 3). The pattern of marrow edema in our series did not correlate clinically with the severity of trauma, symptoms, or the subsequent time to resolution of symptoms, and the edema was shown to be fully resolved on follow-up images obtained after 6 months.

The presence and pattern of contrast enhancement may be helpful in diagnosing acute SN, although most patients undergoing MR imaging for acute spinal trauma do not usually receive IV administered contrast material. A classic SN has been described as having peripheral contrast enhancement, aiding in the differentiation between a large SN and a metastasis, the latter of which will enhance diffusely, whereas the former may enhance in such a manner as to highlight the invaginated cortical margin (9). Some authors have found an

increased association of enhanced SN, thought to represent vascularized disk intrusions into the vertebral endplate. Symptomatic patients show greater extent of bone marrow edema than do asymptomatic patients (11). A follow-up MR image of one of the three patients in our series, obtained 4 years after the precipitating event, revealed contrast enhancement within the SN, even though symptoms were no longer present and surrounding edema had resolved (Fig 4). This enhancement could be the result of continued prominent vascularity of granulation tissue within the disk fragment.

Conclusion

On the basis of these observations, we conclude that an asymptomatic SN may be traceable to a specific occurrence of acute nonradiating low back pain in the patient's history in which an acute SN or simple endplate fracture occurred. The concept of the potential development of SN in the setting of an endplate fracture helps further the understanding of the origin and progression of these interesting and most often incidental spinal abnormalities.

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