The Spectrum of Traumatic Schmorl's Nodes: Identification and Treatment Options in 3 Patients

John R. Dimar II, MD, Senthil T. Nathan, MD, and Steven D. Glassman, MD

Abstract

Schmorl's nodes may present as a simple endplate intravertebral herniation following trauma or as a large cystic lesion of the vertebral body.

In this article, we report on 3 patients with severe back pain following trauma resulting in traumatic Schmorl's nodes and pathologic fracture of the vertebral body. All 3 cases had antecedent trauma. Radiographs showed a cystic lesion in the vertebral body with continuity into the disc space. T2-weighted magnetic resonance imaging showed herniation of the intervertebral disc into the vertebral body through the endplate with surrounding bony edema, indicative of fracture. In 2 cases, the integrity of the vertebral body was severely compromised, requiring vertebrectomy and fusion.

The wide spectrum of presentation and treatment options of traumatic Schmorl's nodes are presented, ranging from symptoms that are responsive to treatment to pathologic fracture of the vertebral body leading to significant collapse and the need for major surgical stabilization.

Schmorl's nodes occur when disc material extrudes into the vertebral body through the endplate. They are usually incidental findings on plain radiographs or magnetic resonance imaging (MRI) of the thoracolumbar spine. The radiographic appearances of these nodes are typical and generally appear as irregularities of the vertebral endplates or small radiolucent pockets in the vertebral body with continuation into the disc through the vertebral endplates.[1-3] In addition, they often involve multiple disc levels, occur in conjunction with Scheuermann's kyphosis, and are associated with degenerative disc disease.[4-6]

However, certain Schmorl's nodes may have a dramatically different clinical appearance and symptomatology. These lesions may present following trauma as a large cystic lesion of the vertebral body, which are called giant cystic Schmorl's nodes.[2] When one of these giant nodes is present, it may lead to pathological fracture of the involved vertebral body, resulting in persistent, severe lower back pain, and neurologic compromise.

There have been rare single case reports of giant cystic Schmorl's nodes in the orthopedic spine literature without a review of the natural history and management of these lesions. We report on a series of 3 patients who demonstrate the wide spectrum of the clinical presentation of these lesions. While the etiology is less clear in 1 case, all 3 cases reported antecedent trauma. All patients were initially treated conservatively, with 2 patients subsequently requiring surgery after initial presentation. We present 3 interesting case histories demonstrating progressively more severe examples of the unusual presentation of pathologic, post-traumatic intravertebral body discal herniations (Schmorl's nodes), 2 of which required surgery for incapacitating pain. The patients provided written informed content for print and electronic publication of these case reports.

Case Reports

Case 1

A 46-year-old male was first seen in the office approximately 1 year after he sustained a lower back injury when a 250 kg whiskey barrel fell on top of him. He had immediate severe lower back pain and was treated with bed rest, nonsteroidal anti-inflammatory drugs (NSAID) and physical therapy for 6 weeks. His pain continued and MRI of the spine revealed an acute intervertebral disc herniation, reported as an acute Schmorl's node extruded through the superior endplate and into the L4 vertebral body (Figure 1A-C). He was treated with pain medications and physical therapy for 10 months with mild improvement. The patient continued to have chronic back pain and occasional severe pain during activities, but was able to return to work with lifting restrictions. He continued on NSAIDs with good relief of his symptoms. A repeat MRI showed no progression of the Schmorl's node and decreased T2 signal indicative of healing. He continues to have chronic lower back pain on a recent 3-year follow-up.

Dr. Dimar is Clinical Professor, Dr. Nathan is Fellow, and Dr. Glassman is Professor of Orthopedics, Department of Orthopaedic Surgery, University of Louisville School of Medicine, Louisville, Kentucky.

Address correspondence to: John R. Dimar II, MD, Norton Leatherman Spine Center, 210 East Gray Street, Suite 900, Louisville, KY 40202 (tel, 502-584-7525; fax, 502-584-6851; e-mail, sherry.whelan@nortonhealthcare.org).

Am J Orthop. 2012;41(9):427-431.

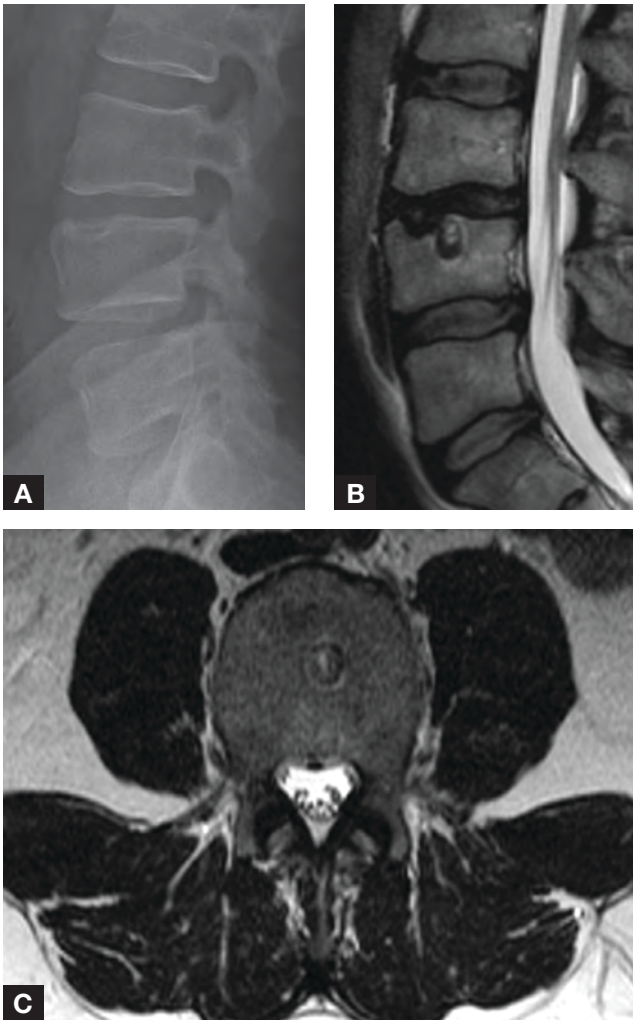

Figure 1. (A) Plain radiograph showing cystic radiolucency at anterosuperior endplate of L4. Sagittal (B) and axial (C) T2 MRI show disc material with L4 vertebral body and surrounding edematous changes.

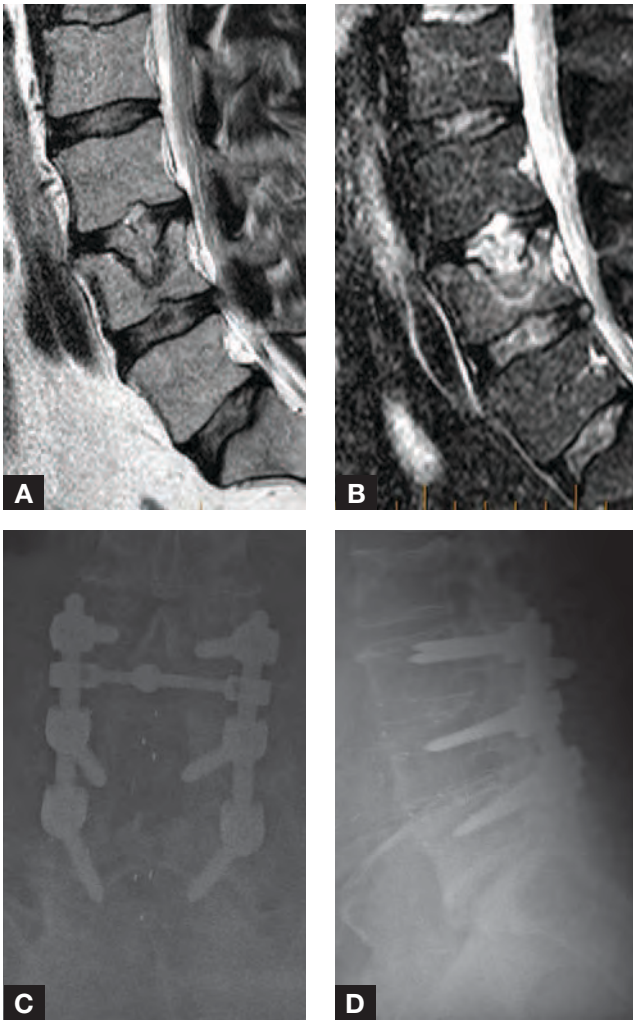

Figure 2. (A) Sagittal T2 and (B) STIR MRI shows disc material with L4 vertebral body and surrounding edematous changes. (C) Follow-up postoperative anteroposterior and (D) lateral radiograph.

Case 2

A 45-year-old male smoker picked-up a heavy boat trailer hitch and felt a "pop" in his lower back, followed by immediate severe lower back pain, and bilateral leg numbness. He was seen in the emergency department where plain radiographs revealed a L4 compression fracture. Upon examination, he had bilateral L5 sensory radiculopathy with no loss of motor, sensory or bowel, and bladder function. MRI revealed a massive protrusion of L3-4 disc into the vertebral body of L4, with an estimated 40% of the body involved. There were associated T2 bright signal changes within the bone, suggestive of an acute fracture (Figures 2A-D). A subsequent computed tomography (CT) scan revealed a penumbra of bone fracturing around the disc with an appearance that resembled an extremely large Schmorl's node. The patient was initially treated with a high Knight brace, pain medication, NSAIDs, activity and weight restriction, and kept off work. He was unable to return to work due to persistent lower back pain. One year later, the patient noted changes in his bowel and bladder function, sexual function, and worsening severe bilateral lower extremity radiculopathy. A CT-myelogram revealed a complete block at L3-4, severe concurrent spinal stenosis from L3-4 to L4-5, and collapse of the L4 disc space. The lesion in L4 remained with a "dumbbell" shaped appearance and sclerotic bony edges, involving 40-50% of the volume of the L4 vertebra. There was a large defect in the inferior L4 endplate which precluded the use of a traditional anteriorly placed interbody cage or femoral ring. An interbody cage could possibly subside through the defect and result in segmental kyphosis and a nonunion. Due to his severe symptoms, the patient underwent a partial anterior L4 corpectomy and interbody fusion, where the defect was filled with graft material to circumvent the large endplate defect, and thus allow for the spanning of the intervertebral body cavity defect from the Schmorl's node. This also allows some elevation of the disc space to more thoroughly decompress the spinal stenosis. The second staged procedure consisted of a wide facet sacrificing,

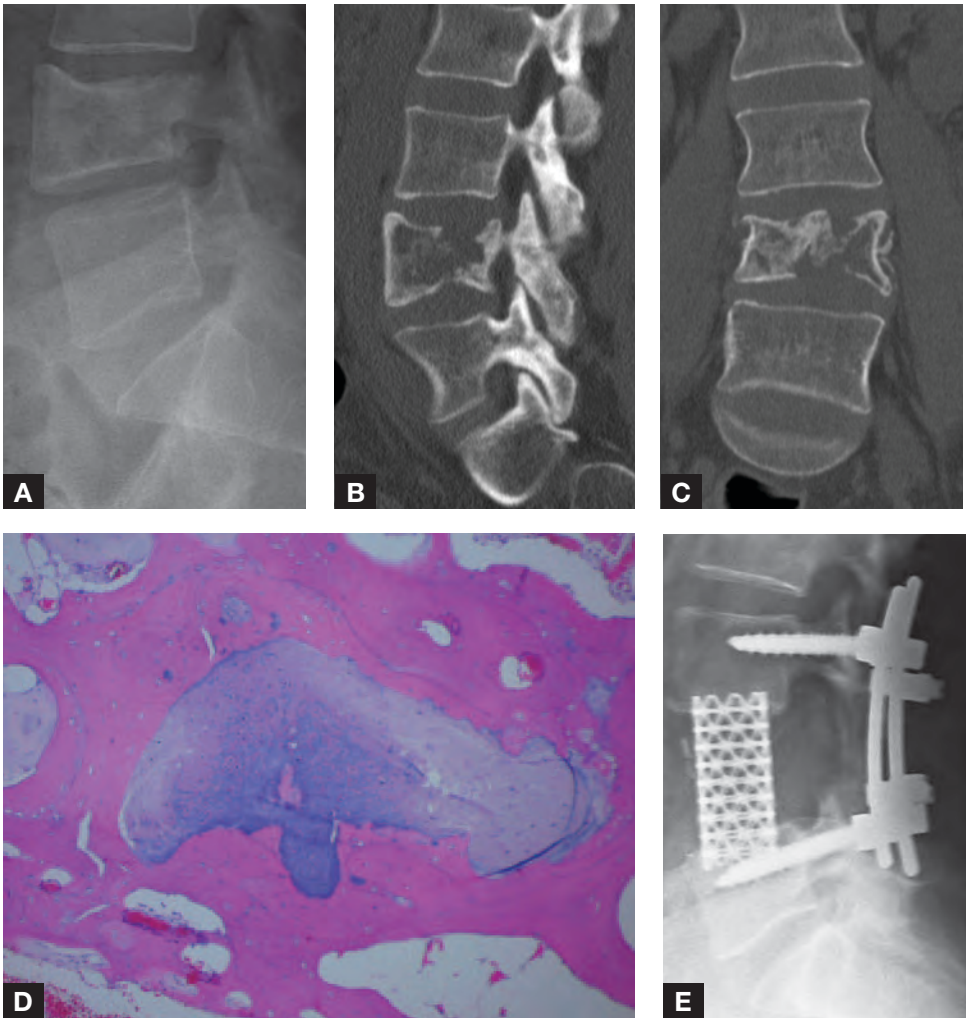

Figure 3. (A) Lateral radiograph showing pathologic fracture of L4. Sagittal (B) and coronal (C) CT reconstruction delineating extent of vertebral body destruction. (D) Biopsy showed intervertebral disc and attached hyaline cartilage endplate into the vertebral body with abundant callus formation. (E) Follow-up postoperative lateral radiograph.

posterior decompression, and instrumented spinal fusion from L3-5. At the time of surgery, the defect within the L4 body extended almost to the inferior endplate and was filled with fibrocartilagenous material. His neurologic symptoms resolved and at his 6-year follow-up, the patient was neurologically intact, but still reported chronic lower back pain with activity.

Case 3

A 32-year-old female was first seen for lower back pain following a fall which was treated nonoperatively. Imaging studies showed no evidence of spine fracture and no MRI was done following her injury. Three years later, the patient developed severe lower back pain following pregnancy and delivery. She had difficulty standing up from a sitting position. Radiographs of the spine showed an L4 fracture (Figures 3A-E). A CT scan of the spine showed a destructive lesion of the L4 vertebral body. MRI showed a superior endplate fracture of L4 and abnormal signal intensity in L4 vertebra. Over the following year, the patient was treated with observation, NSAID, physical therapy, bracing, and mild narcotics without avail. Her mechanical back pain and radicular pain became constant and severe, even with light activity. Repeat MRI and CT scanning revealed a large multi-cystic defect within the vertebral body with multiple fractures of the vertebral body cortex, fragmentation of the internal architecture of the body, and an estimated 60-80% loss of the bony volume of the body. Due to the patient's disabling back pain, an intralesional biopsy and marginal excision was elected, since the patient appeared to have suffered a pathological fracture that was unlikely to heal without anterior column support. The biopsy revealed a cartilaginous appearing lesion admixed with broken bone fragments, near complete replacement of the body with what was believed to be benign cartilaginous tumor. An anterior corpectomy, cage and posterior pedicular rod/screw instrumentation, and fusion was performed. Final pathology confirmed a rare giant cystic Schmorl's node with pathologic fracture.

Histological examination of the biopsied material revealed the presence of herniated intervertebral disc and attached hyaline cartilage endplate into the vertebral body with abundant fracture callus formation. The hyaline type cartilage noted was of reactive infiltrative pattern and consistent with callus formation rather than any cartilage neoplasm.

Discussion

Schmorl's nodes were first described by Von Luschka[4,5] in 1858 and rediscovered by Schmorl in 1927. The underlying pathologic lesion is usually a breach in the endplate allowing herniation of disc material into the spongiosa. Theories concerning the pathologic origin of Schmorl's node include:

- Developmental, embryonic defects such as ossification gaps, vascular channels, and notochord extrusion defects.[5,6]
- Degenerative, in which aging produces sites of

weakness in the cartilaginous end plates.[4,7]
- Pathologic, diseases that weaken the intervertebral disc/vertebral body.[5,6]
- Traumatic, where acute or chronic trauma injures the cartilaginous endplates resulting in intervertebral disc herniation.[6]

Although it is generally accepted that Schmorl's nodes may occur due to trauma, to our knowledge, there have been no reports in the literature which clearly delineates the cause to be traumatic.

The first 2 cases described in this series clearly sustained antecedent traumatic injuries that presented with significant pain. The third case had a documented history of a fall prior to the development of chronic low back pain. However, the origin of the Schmorl's node in this patient is less clear, as it is unknown what prompted the injury to develop into a giant cystic Schmorl's node.

There is a strong resemblance between the giant cystic Schmorl's nodes and subchondral cysts found in other joints, particularly as a sequelae to bone injury.[2,8] Various hypotheses have been put forward to explain the development of cystic changes in the intraosseous component of the herniated disc. One possibility is that the trauma produces trabecular fracture with secondary hemorrhage, which prevents chondrification leading to cystic degeneration. The other possibility is the occurrence of altered biomechanical stress may lead to intramedullary vascular disturbance resulting in a foci of bone necrosis, which may heal by fibroblastic proliferation and mucoid degeneration of connective tissue.[9,10] As similarities exist between the synovial joints and discovertebral endplates, which were noted by Resnick and Niwayama,[8] these hypotheses may explain cystic changes in the giant cystic Schmorl's node.

The natural history of giant cystic Schmorl's node appears to be diverse. All case reports[2,3,9-14] describe patients who developed incapacitating lower back pain that persist for at least 24 months, followed by spontaneous pain reduction without any intervention. This was attributed to the maturation of the cystic degeneration of the intraosseous component. Trauma has also been described as a causative factor in the development of giant cystic Schmorl's node. Most of the cases described[11,14] report no significant traumatic episode that could have led to the the node. However, in our study, 2 of the patients had a history of significant trauma and 1 case reported a history of a fall preceding the giant cystic Schmorl's node.

In the current case series, 2 cases required surgical intervention: Case 2 developed progressive neurologic claudication and incapacitating lower back pain, while Case 3 continued to have persistent, disabling lower back pain for more than 24 months, culminating in severe radicular symptoms. The documented traumatic injury in Case 2 almost certainly had some degree of pre-existing congenital spinal stenosis with superimposed acquired degenerative changes. Any alteration of the biomechanical integrity of the bone leads to presence of residual back pain and a potential risk of a complete fracture especially in younger patients with increased physiological loading. This concept may be the etiology of the development of the neurologic symptoms. Specifically in the third case, where alteration of the cystic contents of the intraosseous Schmorl's node within the vertebral body resulted in a subsequent pathological fracturing, and the development of canal compromise.

The differential diagnosis of giant cystic Schmorl's node includes benign and low grade tumors of the spine, infectious spondylitis, and rheumatoid discitis. In our series, the patients were middle-aged, which was consistent with existing literature. Although Schmorl's nodes were reported in patients with pre-existing pathological bone disease like Scheurman's disease, the development of a Giant cystic Schmorl's node has been previously reported to occur in patients with no pre-existing bone disease.[2] The L3 and L4 level appears to be the most common level of occurrence of the lesion. This is in contrast to Schmorl's nodes, which occur in the thoracic or thoracolumbar region. Most often these nodes tend to be continuous with disc space through a fracture in the superior end plate.

Hauger and colleagues[2] differentiate the radiological features of a giant cystic Schmorl's node from a typical Schmorl's node by describing the giant cystic node as a large well-delineated cystic lesion of the vertebral body connected to a degenerative superior intervertebral disc, as evidenced by narrowing and low signal intensity on T2 weighted images. Most of the case reports depict a lesion to be more common in the lower lumbar spine. All our patients had the typical features of the MRI and Case 2 and 3 showed significant changes in their myelogram combined with CT scanning; these radiographic changes were quite dramatic in Case 3, with the giant cystic Schmorl's node.

Conservative treatment methods, including pain management and physical therapy, have been the mainstay of treatment for symptomatic giant cystic Schmorl's nodes, as none of the cases previously reported in literature had any neurologic deficits. Most patients stabilize with residual back pain at the end of approximately 9 to 24 months, indicating the maturation of the intraosseous component of the herniation. Nonoperative management may be continued as the primary modality of management in patients who have low functional demands. In young patients with greater physical demands and a history of significant trauma, there is a propensity to develop progressive neurological deficit and persistent lower back pain after maturation of the disc. This implies that the biomechanical characteristics of the spine have changed, which may require surgical management of a giant cystic Schmorl's node. Although most of these lesions can be treated nonoperatively, McLain and Weinstein[14] reported a case of giant cystic Schmorl's

node in a patient who underwent hemivertebrectomy with a preoperative diagnosis of spinal tumor. In our series, 2 patients developed severe neurologic symptoms following more than 1 year of disabling lower back pain. Consequently, Cases 2 and 3 underwent corrective surgery, with the latter undergoing biopsy of the lesion prior to their final surgery, since a spinal tumor was considered in the differential diagnosis.

Giant cystic Schmorl's nodes are a separate entity and differ significantly from the more benign Schmorl's nodes. They are rare, and according to the literature, do not often require surgical intervention. Because they differ in the pathogenesis, natural history, and pose a diagnostic challenge, controversy exists in the management of these lesions due to its confusion with the more benign Schmorl's node or other spinal tumors.

Two of our cases required surgical management due to their severe neurologic symptoms and persistent incapacitating low back pain. Patient 2 required surgery because of incapacitating lower back pain and concurrent spinal stenosis that led to progressive neurologic dysfunction. Serial radiographs showed L3-4 disc space collapse, which lead to worsening of the patient's spinal stenosis. We also hypothesized that the imploded disc material within the vertebral body and the concurrent damage to the disc, which led to degenerative disc disease, was the cause of the patient's severe lower back pain. The serial CT scanning of Case 3 demonstrated expansion of the lesion within the body and areas of endplate fracturing as the integrity of the vertebral body was compromised. Histological evaluation of the incisional biopsy revealed an extremely rare giant cystic Schmorl's node. Both of these patients in this series treated with surgery had complete relief of their neurologic symptoms, but not unexpectently, still have some mild lower back pain and decreased range of low back motion.

Conclusion

When the functional demands of the patient result in incapacitating lower back pain and there is a significant, progressive change in their neurological status, surgical management may be necessary. The extent of the surgery may range from percutaneous cement injection to anterior corpectomy and fusion. Consideration of a giant cystic Schmorl's node should be considered in the differential diagnosis when a large expansive lesion is noted within the vertebral body on serial radiographs.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

References

1. Akahashi K, Miyazaki T, Ohnari H, Takino T, Tomita K. Schmorl's nodes and low back pain. Analysis of magnetic resonance imaging findings in symptomatic and asymptomatic individuals. *Eur Spine J.* 1995;4(1):56-59.
2. Hauger O, Cotten A, Chateil JF, Borg O, Moinard M, Diard F. Giant cystic Schmorl's nodes: imaging findings in six patients. *AJR Am J Roentgenol.* 2001;176(4):969-972.
3. Seymour R, Williams LA, Rees JI, Lyons K, Lloyd DC. Magnetic resonance imaging of acute intraosseous disc herniation. *Clin Radiol.* 1998;53(5):363-368.
4. Coventry MB, Ghormley RK, Kernohan JW. The intervertebral disc: Its microscopic anatomy and pathology Part II. Changes in intervertebral disc concomitant with age. *J Bone Joint Surg Am.* 1945;27(2):233-247.
5. Coventry MB, Ghormley RK, Kernohan JW. The intervertebral disc: Its microscopic anatomy and pathology Part III. Pathological changes in the intervertebral disc. *J Bone Joint Surg Am.* 1945;27(3):460-474.
6. Fahey V, Opeskin K, Silberstein M, Anderson R, Briggs C. The pathogenesis of Schmorl's nodes in relation to acute trauma. An autopsy study. *Spine (Phila Pa 1976).* 1998;23(21):2272-2275.
7. Hilton RC, Ball J, Benn RT. Vertebral end-plate lesions (Schmorl's nodes) in the dorsolumbar spine. *Ann Rheum Dis.* 1976;35(2):127-132.
8. Resnick D, Niwayama G. Intravertebral disk herniations: cartilaginous (Schmorl's) nodes. *Radiology.* 1978;126(1):57-65.
9. Peng B, Wu W, Hou S, Shang W, Wang X, Yang Y. The pathogenesis of Schmorl's nodes. *J Bone Joint Surg Br.* 2003;85(6):879-882.
10. Schajowicz F, Clavel Sainz M, Slullitel JA. Juxta-articular bone cysts (intraosseous ganglia): a clinicopathological study of eighty-eight cases. *J Bone Joint Surg Br.* 1979;61(1):107-116.
11. Takahashi K, Miyazaki T, Ohnari H, Takino T, Tomita K. Schmorl's nodes and low-back pain. Analysis of magnetic resonance imaging findings in symptomatic and asymptomatic individuals. *Eur Spine J.* 1995;4(1):56-59.
12. Wagner AL, Murtagh FR, Arrington JA, Stallworth D. Relationship of Schmorl's nodes to vertebral body endplate fractures and acute endplate disk extrusions. *AJNR Am J Neuroradiol.* 2000;21(2):276-281.
13. Walters G, Coumas JM, Akins CM, Ragland RL. Magnetic resonance imaging of acute symptomatic Schmorl's node formation. *Pediatr Emerg Care.* 1991;7(5):294-296.
14. Mclain R, Weinstein JN. An unusual presentation of a Schmorl's node. Report of a case. *Spine (Phila Pa 1976).* 1990;15(3):247-250.

Erratum

In the article, "Can Hylan G-F 20 With Corticosteroid Meet the Expectations of Osteoarthritis Patients?" (*Am J Orthop.* 2012;41(7):311-315), the funding source in the first sentence of the authors' disclosure was reported in error. The study was funded by Genzyme. This was corrected online. *The American Journal of Orthopedics®* makes every possible effort to ensure the accuracy in its articles and apologizes for the mistake.