

The pathogenesis of Schmorl's nodes

B. Peng, W. Wu, S. Hou, W. Shang, X. Wang, Y. Yang

From the 304th Hospital, Beijing, China

We examined the pathogenesis of Schmorl's nodes, correlating the histological findings from 12 lumbar vertebrae with the corresponding conventional radiographs, tomographs, MR images and CT scans. The last revealed round, often multiple cystic lesions with indistinct sclerotic margins beneath the cartilaginous endplate. The appearances are similar to the typical CT changes of osteonecrosis. Histological examination of *en-bloc* slices through Schmorl's nodes gave clear evidence of subchondral osteonecrosis. Beneath the cartilage endplate, we found fibrosis within the marrow cavities with the disappearance of fat cells. Osteocytes within bone trabeculae were either dead or had disappeared. We suggest that Schmorl's nodes are the end result of ischaemic necrosis beneath the cartilaginous endplate and that herniation into the body of the vertebra is secondary.

J Bone Joint Surg [Br] 2003;85-B:879-82.
Received 22 May 2002; Accepted after revision 21 November 2002

Schmorl's nodes have been widely assumed to be the herniation of the nucleus pulposus through the cartilaginous endplate into the body of a vertebra, ever since Schmorl first described them in 1927.¹⁻³ The hypotheses of their origin and pathogenesis include developmental factors, degenerative conditions, pathological processes such as infection, neoplasia and trauma.⁴⁻¹¹ These hypotheses have not been critically examined and therefore we have analysed both the histology of specimens of 12 lumbar vertebrae and the cor-

Table I. Distribution of Schmorl's nodes

Vertebra	Number
L3 superior endplate	3
L4 superior endplate	2
L4 inferior endplate	2
L5 superior endplate	1
L5 inferior endplate	1
S1 superior endplate	3

responding images from conventional radiographs, MRI and CT scans.

Patients and Methods

We examined 12 surgical specimens of Schmorl's nodes taken from ten patients who had severe low back pain, with or without leg pain. Their mean age was 34 years (14 to 61) and there were eight men and two women. The mean duration of pain from onset to first medical attendance was 3.5 years (4 months to 12 years). All the patients denied a history of lumbar injury. The distribution of Schmorl's nodes is shown in Table I. All patients underwent conventional radiography and CT of the lumbar spine, and five had MRI. Immediately before radiological examination and CT, three patients underwent discography of five intervertebral spaces with Schmorl's nodes, and in all three the pain was reproduced during the injection of contrast material.

For anterior intervertebral body fusion, we used a retroperitoneal approach for those patients with pain associated with lesions located in the anterior or central endplate. For posterior intervertebral body fusion with pedicle screw fixation, we used a standard posterior approach for painful Schmorl's nodes located in the posterior margins of vertebral bodies. The youngest patient, a 14-year-old boy, had the lesion removed without internal fixation. The lesions were excised *en bloc* during the fusion procedure, for histological examination. The specimens were fixed in 10% neutral formalin, decalcified in formic acid, slit mid-sagittally, embedded in paraffin, sliced into sections 5 to 6 μm in thickness and stained with haematoxylin and eosin.

Results

The radiological manifestation of Schmorl's nodes in our series was an endplate indentation with an indistinct scler-

B. Peng, MD, PhD, Associate Professor of Orthopaedic Surgery
W. Wu, MD, Professor of Orthopaedic Surgery
S. Hou, MD, Professor of Orthopaedic Surgery
W. Shang, MD, Associate Professor of Orthopaedic Surgery
Department of Orthopaedics
X. Wang, MD, Associate Professor of Pathology
Y. Yang, MD, Lecturer of Pathology
Department of Pathology
304th Hospital, 51 Fuchenglu, Beijing, 100037, People's Republic of China.

Correspondence should be sent to Dr. B. Peng.

©2003 British Editorial Society of Bone and Joint Surgery
doi:10.1302/0301-620X.85B6.13555 \$2.00



Fig. 1a



Fig. 1b



Fig. 1c



Fig. 1d

A 22-year-old man with a four-year history of severe lower back pain, without specific trauma. Figure 1a – A lateral radiograph showing severe degenerative changes and a Schmorl's node at L2/3 (arrow indicates the lesion). Figure 1b – A lateral radiograph after discography at L3/4, L4/5 and L5-S1. There is absence of contrast material in the Schmorl's node at L3. Figure 1c – A lateral radiograph after anterior intervertebral body fusion in L2/3. There are normal intervertebral spaces and physiological curvature in the lumbar spine. Figure 1d – An axial CT scan showing subchondral osteonecrosis in the Schmorl's node.

rotic marginal line. There was leakage of radiopaque contrast medium into the endplate indentation from the disc space during discography. The node appeared on CT scans as a round or multicystic irregular area of bone density with a sclerotic circumferential margin lying beneath the cartilaginous endplate. This is similar to the CT changes of osteonecrosis. After discography, CT showed the diffusion of radiopaque contrast to be in the superficial layer of the cartilaginous endplate indentation, not in the deep layer (Fig. 1).

Histological examination of the nodes revealed that the round or multicystic irregular area beneath the endplate, shown on CT scans or MR images, was a zone of osteonecrosis, separate from the herniated nucleus pulposus as described by Schmorl (Fig. 2). The cartilaginous endplates overlying the infarcted bone were intact, which con-

trasted with the appearance of disruption of the endplates with herniation of the nucleus pulposus into vertebral bodies demonstrated on radiographs. Areas of fibrosis, discovered within the marrow cavities beneath the cartilaginous endplate, together with the disappearance of fat cells and the disappearance or death of osteocytes within bone trabeculae (Fig. 2) were evidence of the end stage of an infarct. The fibrocartilage lying beneath the endplate appeared as a lobulated area on the CT scan because it was radiolucent (Fig. 3). The sclerotic margin surrounding the zones of osteonecrosis on CT with or without discography, was assumed to represent the interface between the osteonecrotic and normal bone.

The so-called microscopic Schmorl's node appeared to be an ingrowth of fibrous tissue or fibrocartilage from the necrotic area into the cartilaginous endplate since there was

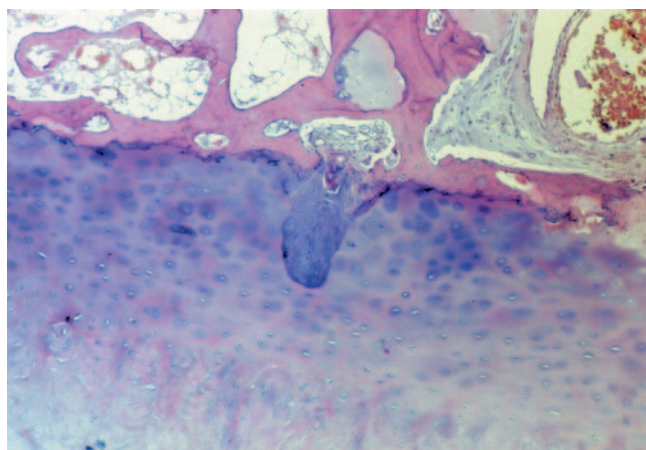


Fig. 2

Photomicrograph of a mid-sagittal slice of the lesion arrowed in Figure 1a. The osteonecrotic zone underlying the cartilaginous endplate shows fibrosis in marrow cavities, the absence of fat and the disappearance or death of osteocytes within bone trabeculae. The cartilaginous endplate is intact, without herniation of the nucleus pulposus into the vertebral body (haematoxylin and eosin x 27).

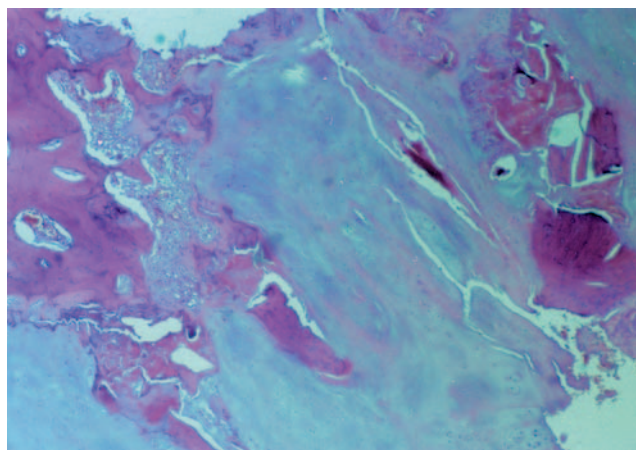


Fig. 3

Photomicrograph of large areas of avascular fibrocartilage formed in the zone of osteonecrosis underlying the cartilaginous endplate (haematoxylin and eosin x 27).

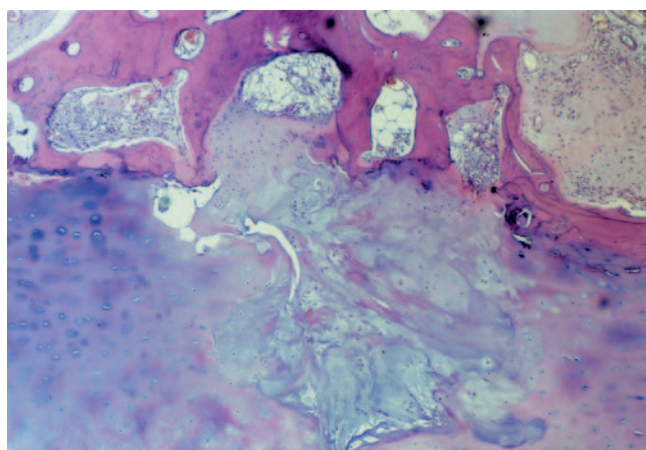


Fig. 4a

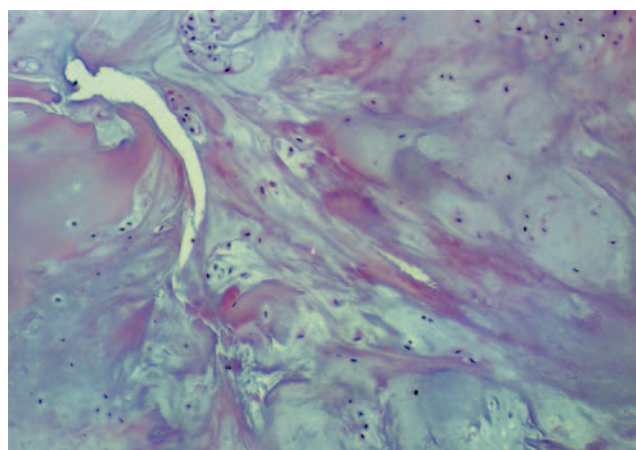


Fig. 4b

Photomicrograph of a Schmorl's node with a) the defect area of the cartilaginous endplate showing the ingrowth of fibrous tissue and fibrocartilage from the necrotic zone into the cartilaginous endplate with no evidence of the herniation of the nucleus pulposus through the disrupted cartilaginous endplate into the vertebral body (haematoxylin and eosin x 27), and b) enlargement of the central area in Figure 4a (haematoxylin and eosin x 70).

no evidence of the herniation of the nucleus pulposus through the disrupted cartilaginous endplate into the vertebral body (Fig. 4). The local defect of the endplate may have been the consequence of degeneration and necrosis of the cartilaginous endplate secondary to the underlying osteonecrosis or alternatively, a stress fracture of the endplate. Of the 12 specimens of Schmorl's nodes, four defects of cartilaginous endplates, measuring 0.5 to 4 mm in diameter, were found in three.

The cartilaginous endplates overlying necrotic areas showed acidophilic degeneration, focal proliferation, calcification and cell death, but were structurally intact.

Discussion

The classic Schmorl's node has at least two basic components, namely disruption of the cartilaginous endplate and herniation of the nucleus pulposus into the vertebral body.^{12,13} Our study suggests that the basic pathological lesion may be osteonecrosis beneath the cartilaginous endplate. Whether a herniation of the nucleus pulposus is present or not, seems to be a secondary phenomenon.

There is a close histological resemblance between the Schmorl's nodes which we have studied and lesions of avascular necrosis of the femoral head. Loosely textured fibrous

tissue with multiple small blood vessels completely replaced the marrow of the vertebral body underlying the cartilaginous endplate. There was an increase in reactive woven bone with thickened trabeculae and prominent osteoclasts and osteoblasts.

Recently, Hauger et al⁶ reported a special type of Schmorl's node, which had a characteristic radiological appearance unlike that of the other, more aggressive cyst-like lesions of the vertebral bodies. They thought that it had resulted from altered mechanical stress, leading to vascular disturbance with consequent foci of osteonecrosis. Resnick and Niwayama¹⁴ noted the similarity between the discovertebral junction and a synovial joint in that both bone-ends were covered with hyaline cartilage. The annulus fibrosus is similar to a joint capsule and the gelatinous nucleus pulposus equivalent to synovial fluid. An animal experiment revealed that the repetitive mechanical stresses on the vertebral body may induce the formation of a Schmorl's node with damage to the vascularity of the endplate.¹⁵ Roberts, Menage and Urban,¹⁶ in a biochemical study, demonstrated that the composition of the cartilaginous endplate surrounding Schmorl's nodes was different from that of the normal endplate with a reduced content of proteoglycan, suggesting that there was degeneration of the cartilaginous endplate, before the formation of a Schmorl's node. On the basis of our study, we believe that the avascular necrosis of both the vertebral body beneath the endplate and the femoral head, share the same pathogenesis.

The relationship between a Schmorl's node and pain in the back is not clear. Takahashi et al³ analysed MRI findings in patients with symptomatic and asymptomatic Schmorl's nodes. In symptomatic patients, the vertebral body marrow surrounding the Schmorl's node gave a low signal intensity on T1-weighted and a high signal intensity on T2-weighted images. This indicated the presence of inflammation and oedema in the vertebral bone marrow. These MRI findings were not present in asymptomatic individuals which suggested that Schmorl's nodes became asymptomatic when the inflammation subsided. All patients in our study had severe and progressive back pain. The complete relief of the symptoms after lumbar interbody fusion implies that move-

ment at the level of the Schmorl's node was indeed the cause of the pain.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References

1. Ghelman B, Freiburger RH. An anterior disc herniation demonstrated by discography. *Am J Roentgenol* 1976;127:854-5.
2. Seymour R, Williams LA, Rees JI, Lyons K, Lloyd DC. Magnetic resonance imaging of acute intraosseous disc herniation. *Clin Radiol* 1998;53:363-8.
3. 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;4:56-9.
4. Chandraraj S, Briggs CA, Opeskin K. Disc herniations in the young and endplate vascularity. *Clin Anat* 1998;11:171-6.
5. Saluja G, Fitzpatrick K, Bruce M, Cross J. Schmorl's nodes (intravertebral herniations of intervertebral disc tissue) in two historic British populations. *J Anat* 1986;145:87-96.
6. Hauger O, Cotton A, Chateil JF, et al. Giant cystic Schmorl's nodes: imaging findings in six patients. *AJR Am J Roentgenol* 2001;176:969-72.
7. 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* 1998;23:2272-5.
8. Yasuma T, Saito S, Kihara K. Schmorl's nodes: correlation of x-ray and histological findings in postmortem specimens. *Acta Pathol Jpn* 1988;38:723-33.
9. Takahashi K, Takiata K. A large painful Schmorl's node: a case report. *J Spinal Disord* 1994;7:77-81.
10. Walters G, Coumas JM, Akins CM, Ragland RL. Magnetic resonance imaging of acute symptomatic Schmorl's node formation. *Pediatr Emerg Care* 1991;7:294-6.
11. Aggrawal ND, Kaur R, Kumar S, Mathur DN. A study of changes in the spine in weight lifters and other athletes. *Br J Sports Med* 1979;13:58-61.
12. Coventry MB, Ghomley RK, Kernohan JW. The intervertebral disc: its microscopic anatomy and pathology. Part II. Changes in the intervertebral disc concomitant with age. *J Bone Joint Surg* 1945;27:233-47.
13. Coventry MB, Ghomley RK, Kernohan JW. The intervertebral disc: its microscopic anatomy and pathology. Part III. Pathological changes in the intervertebral disc. *J Bone Joint Surg* 1945;27:460-74.
14. Resnick D, Niwayama G. Intravertebral disc herniations: cartilaginous (Schmorl's) nodes. *Radiology* 1978;126:57-65.
15. Revel M, Andre-Deshays C, Roudier R, et al. Effects of repetitive strains on vertebral end plates in young rats. *Clin Orthop* 1992;279:303-9.
16. Roberts S, Menage J, Urban JPB. Biochemical and structural properties of the cartilage endplate and its relation to the intervertebral disc. *Spine* 1989;14:166-74.