

Diagnosis and surgical treatment of back pain originating from endplate

Baogan Peng · Jindong Chen · Zengda Kuang ·
Duanming Li · Xiaodong Pang · Xinyu Zhang

Received: 9 January 2009 / Revised: 21 February 2009 / Accepted: 4 March 2009 / Published online: 18 March 2009
© Springer-Verlag 2009

Abstract Twenty-one patients with back pain originating from the endplate injuries were selected to explore the methods of diagnosis and surgical treatment. All patients underwent examinations using radiography, CT, and MR imaging. Pain level of disc was determined through discography in each patient. The principal outcome judgments were pain and disability, and the efficacy of surgical treatment was assessed through the use of the visual analog scale (VAS) for pain and the Oswestry disability index (ODI) for functional recovery. All 21 patients with a diagnosis of back pain originating from endplate injuries according to discography were treated with anterior or posterior fusion surgery. The mean follow-up period was 3 years and 5 months (range, 2–6 years). Of the 21 patients, 20 (20/21) reported a disappearance or marked alleviation of low back pain and experienced a definite improvement in physical function. Statistically significant and clinically meaningful improvements in the VAS and ODI scores were obtained after treatment in the patients with chronic low back pain originating from the endplate injuries ($P = 0.0001$). The study suggests that discography and fusion surgery may be very effective methods for the diagnosis and treatment, respectively, of chronic back pain originating from the endplate injuries.

Keywords Back pain · Endplate · Schmorl's node · Discography · Fusion surgery

Introduction

Low back pain is the most commonly diagnosed orthopaedic clinical disease. Approximately 70–90% of the population will suffer low back pain at some point in their lives and at any time, about 15–20% of the population suffers low back pain. Low back pain ranks as the second-most diagnosed condition among all patients visiting the hospital, second only to upper respiratory tract infection [2, 4, 6]. In spite of its pervasion, low back pain is one of mankind's most confusing common disorders, because in a majority of cases, low back pain cannot be accurately diagnosed via pathological anatomy.

In theory, any bony and soft tissue structures in the lumbar spine that contain nerve fibers may be the origin of low back pain. Intervertebral discs are such a structure in which, apart from the outer annulus fibrosus, the vertebral endplate also is innervated. Usually, one part of the nerves distributed in the vertebral endplate enter the vertebral body, accompanied by the blood vessels at the edge of the vertebral body; the other part of these nerves are branches of the sinus vertebral nerves entering the vertebral body through the intervertebral foramen. The density of the endplate nerves is similar to the density of the annulus fibrosus nerves, suggesting that the endplate is also an important source of discogenic pain [17].

The current basic and clinical research into discogenic pain mainly focuses on discogenic low back pain caused by annulus fibrosus injuries [21, 22], and rarely involves low back pain caused by endplate injuries. In fact, low back pain caused by endplate injuries is quite common in clinical research. Epidemiological investigations indicate that in the population without low back pain, the incidence rate of endplate injuries is about 30% [30]. This paper reports a group of cases of low back pain caused by endplate injuries

B. Peng (✉) · J. Chen · Z. Kuang · D. Li · X. Pang · X. Zhang
Department of Spinal Surgery, Institute of Spinal Surgery,
General Hospital of Armed Police Force, 69 Yongding Road,
100039 Beijing, People's Republic of China
e-mail: pengbaogan@163.com

and explores the diagnosis and surgical treatment of these cases.

Patients and methods

Patients

From January 2000 to December 2005, in total, we treated 21 patients who were diagnosed as having low back pain originating from the endplate. Fifteen of them were male and six of them female. Their ages ranged from 21 to 55 years, with the average being 33 years. The courses of treatment varied from 12 months to 6 years, with an average of 3 years and 5 months. In five cases, patients had a clear trauma history.

All patients appeared to have severe low back pain without radicular symptoms that failed to be cured through a variety of conservative treatments. Meanwhile, patients suffered accompanying dual hip and/or dual thigh pain in six cases, and unilateral hip and/or ipsilateral thigh pain in five cases, but the pains usually did not appear below the knee. The thigh pains included pains throughout the entire thigh, in the anterior thigh, and in the posterior thigh. There was unilateral ectocnemial pain and numbness in one case, a unilateral groin pain in one case, and bilateral groin pains in two cases. Apart from activity limitation or widespread tenderness in lumbar spine, no patient experienced obvious impairment of the dual lower limbs or of motor, sensory, or reflection.

Imaging examination

All patients underwent lumbar spine X-ray radiography films, CT scanning and MRI examinations. The CT and MRI examinations of all patients revealed that there was no lumbar disc herniation or nerve root compression. Larger endplate injury lesions in the lumbar spine X-ray films, especially in the lateral films, reflected the vertebral endplate depression and marginal sclerosis. The lumbar spine CT scanning revealed that, around the lesions below the lumbar vertebral endplate, there were one or more irregular, or nearly round, multi-cystic zones with irregular bone density or showing as diffuse spots, usually surrounded by hardened bands. MRI indicated that the nucleus pulposus was connected to the endplate lesions.

Discography

The lumbar discography was conducted on all patients in this group. Disc segments in discography relied on MRI. At least one adjacent normal disc was selected as a control disc. When the fluorescence imaging in discography

showed the contrast flowing into the endplate lesions, the patients would produce accurate pain provocation. CT after discography revealed that some contrast had infiltrated into the endplate injury lesions. Considering that pain can be caused by ruptured annulus fibrosus, cases where the annulus fibrosus was ruptured were excluded. In 14 cases within this group, there appeared single endplate injuries in one disc level; two cases were L2 superior endplate injuries, five cases were L3 superior endplate injuries, two cases were L4 inferior endplate injuries, four cases were L4 inferior endplate injuries, and one case was an L5 superior endplate injury. Superior and inferior endplate injuries in one disc level appeared in three cases—one of which was in the L2–L3 disc, and two of which were in the L3–L4 disc; two- (or more than two) disc level multi-endplate injuries happened in four cases. Additionally, two cases displayed four-disc multi-endplate injuries in MRI, but the discography showed pain provocation in one and two discs, respectively.

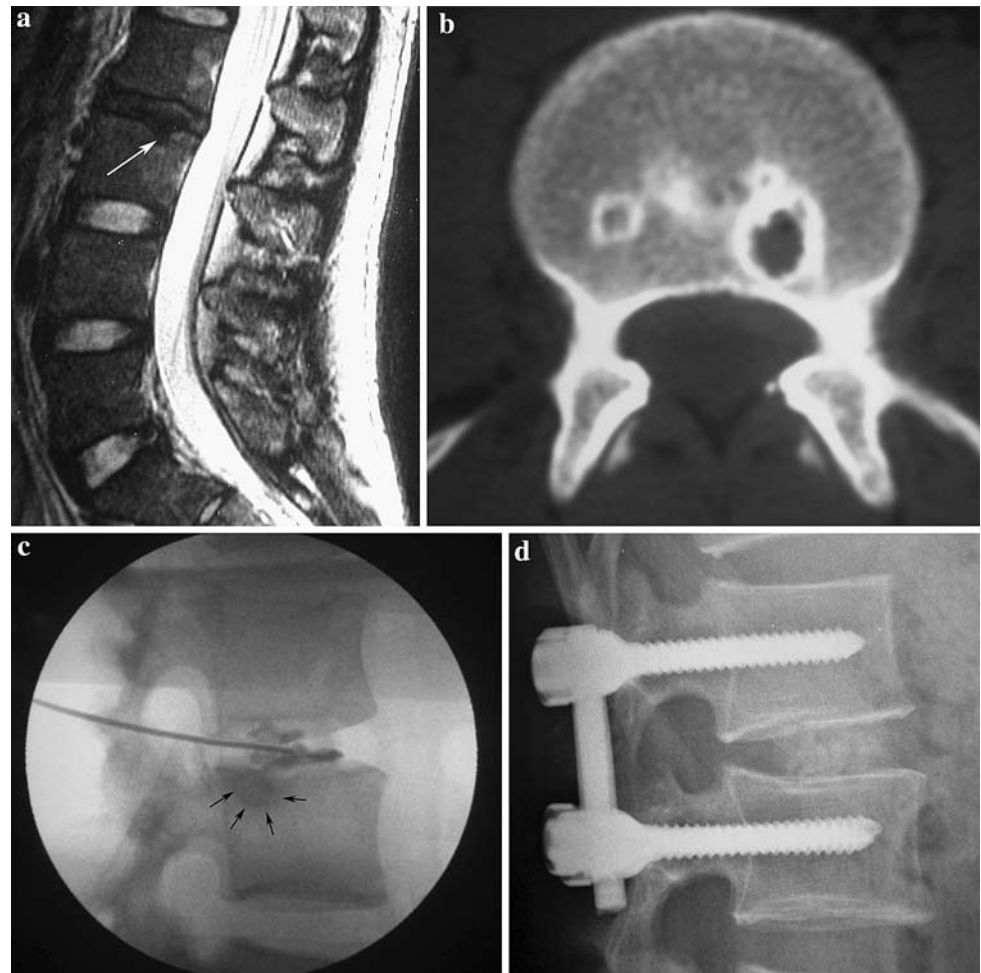
Treatment

The decision to fusion segment in lumbar spine depended on the level of pain provocation in lumbar discography. In order to further investigate the pathogenesis of Schmorl's node afterward, we removed the lesions of Schmorl's nodes in en bloc during the fusion procedure for histological examination. The anterior intervertebral body fusion was undertaken for those with painful Schmorl's nodes located in anterior or central endplate. Posterior intervertebral body fusion was performed for these with painful Schmorl's nodes located in posterior margins of vertebral body. Eleven cases underwent the posterior lumbar disc excision, pedicle screw system internal fixation, and the posterior lumbar interbody fusion (PLIF) operation. The bone-grafting materials were prepared through the laminectomy and/or spinous process removal bones (Fig. 1). In three cases, the posterolateral fusion operations were run after lumbar disc excision and pedicle screw system internal fixation. Seven cases underwent the extraperitoneal lumbar anterior disc excision and anterior lumbar interbody fusion (ALIF); of these, two cases accepted the pure ALIF, while three cases additionally adopted the single- or double-cortical bone screw fixation, and two cases the VestroFix single-rod system internal fixation.

Clinical efficacy evaluation

Preoperative and postoperative low back pain symptoms were evaluated through the use of the visual analog scale (VAS). Through the Oswestry dysfunction questionnaires, we scored the preoperative and postoperative lumbar disc

Fig. 1 **a** In a 26-year-old male who suffered low back pain for 4 years, MRI showed L2/3 disc degeneration and L3 superior endplate Schmorl's node injuries (*white arrow*). **b** CT scanning showed similar round injury lesions in the L3 vertebral superior endplate. **c** Discography revealed that the contrast flowed into L3 superior endplate injury lesion (*arrows*), and at the same time produced a sharp pain provocation. **d** Take the L2–L3 posterior lumbar interbody fusion and pedicle screw system internal fixation. Low back pain symptoms disappeared following the operation



dysfunction index (Oswestry disability index, ODI) of each patient [9]. The postoperative fusion assessment was performed as follows: If the consecutive bone trabecula can pass through the transplanted bone and the vertebral interface, in which there are no gaps, and/or lumbar flexion and extension X-rays show no movements in the fused segments, then it can be regarded as fused.

Statistical analyses

The preoperative and postoperative VAS scores and the ODI lumbar dysfunction indices were compared through a paired comparative *t* test. The significance level was 0.05.

Results

The postoperative follow-up time ranged from 2 to 6 years, with an average of 3 years and 5 months. In 14 cases, the PLIF or posterolateral fusion and the pedicle screw system internal fixation were conducted; in 12 of those, low back pain symptoms nearly completely disappeared following

the operation, and mild low back pain still existed in two other cases. Among the seven cases that took the lumbar anterior disc excision and ALIF, one case was still suffering low back pain; in the rest of the cases, low back pain symptoms basically disappeared. From the preoperative and postoperative low back pain VAS scores, we found that the preoperative VAS changed from 5.3 to 9.1, with an average of 7.15; the postoperative VAS ranged from 0 to 5.0, with an average of 1.64. After the preoperative scores were compared with the postoperative scores through a paired comparative *t* test, it was found that these two sets of scores held significant differences ($P < 0.01$). Among 21 postoperative cases, 20 experienced significantly improved lumbar function. Based on comparisons between the preoperative and postoperative ODI scores, the postoperative ODI scores were markedly decreased and therefore significantly improved (Table 1).

Among 11 cases taking the posterior PLIF, ten obtained complete pain relief and bone fusion in 4–6 months following the operation, and one case formed pseudoarthrosis (no consecutive bone trabecula between the interfaces), and still experienced mild low back pain. Hence, the fusion rate

Table 1 Preoperative and postoperative ODI scores and the VAS (mean \pm SD, $n = 21$)

	Before operation	After operation	<i>t</i> value	<i>P</i> value
VAS (0–10)	7.15 \pm 1.02	1.64 \pm 1.13	16.26	0.0001
ODI (0–100)	57.52 \pm 8.35	10.09 \pm 6.53	23.15	0.0001

was 91%. Three cases with posterolateral fusion obtained complete relief of back pain and bone fusion. Among the seven cases taking anterior fusion, one case accepted the two-level fusion and had an intervertebral space that was not fused; at present, that one case still suffers from low back pain. Therefore, this fusion rate was 86%.

Discussion

There are two types of endplate injury: those involving Schmorl's nodes and endplate degenerative changes. 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 [5, 29]. The hypotheses of their origin and pathogenesis include developmental factors, degenerative conditions, pathological processes such as infection, neoplasia and trauma, and bone necrosis beneath the endplate [1, 5, 8, 14, 23, 26, 28, 30, 31]. The endplate degenerative changes, also known as Modic changes, were proposed independently by de Roos et al. [7] and Modic et al. [18, 19] according to signal intensity changes of the endplate beneath the cartilage endplate and the adjacent marrow on MRI. Modic changes are divided into three types. Type I changes mean that the T1-weighted signal intensity decreases and the T2-weighted signal intensity increases, with the histological characteristic of bone marrow edema. Type II changes indicate that the T1-weighted signal increases and the T2-weighted signal slightly increases or is kept at the normal level, with the histological characteristic of bone marrow steatosis. Type III changes indicate that T1- and T2-weighted signals are reduced, corresponding to the bone sclerosis in the X-ray films, with the histological change that the compact bone replaces the fatty marrow.

The causal relationship between Modic changes and disc degeneration has not been explained, but Modic changes usually accompany severe disc degeneration. Theoretically, due to the histological characteristics of Modic changes, they may cause low back pain. However, it is difficult to distinguish this low back pain from the low back pain caused by disc degeneration in clinical research. Some researchers have tried to prove the relationship between Modic changes and low back pain via lumbar discography;

as a result, they found that lumbar discography mainly reflected low back pain caused by ruptured annulus fibrosus, but not the low back pain derived from endplate degeneration [15]. In order to exhibit the clinical characteristics accurately of the diagnosis and treatment methods for low back pain caused by endplate injuries, this study included only those patients with low back pain caused by Schmorl's node injuries; cases of low back pain caused by endplate degeneration were excluded.

Theoretically speaking, each lumbar spine and its adjacent soft tissue structures that contain rich innervations may be origins of low back pain. The endplate may be an important source of low back pain [3]. In our previous studies [24], we found that the histological performance of Schmorl's node lesions included focal bone necrosis; in addition, the bone marrow tissues beneath the endplate were replaced by the vascular granulation tissues. Based on these study results, we analyse that the possible mechanisms of endplate injury in generating pain are as follows: trauma, inflammation, or osteonecrosis in endplate injury lesion induce the production of inflammatory mediators and cytokines, such as bradykinin, prostaglandin E2 and IL-1, which can sensitize the silent nociceptors which usually do not respond to mechanical stimulation. During movement, pressure changes within the disc can activate nociceptors in endplate injury lesions and thus contribute to low back pain. There are different types of low back pain, including continuous dull pain and sharp pain, indicating that pain may be derived from the involvement of different nerve fibers, such as non-myelinated C fibers and fine myelinated A δ fibers [17]. The relationship between endplate injuries caused by Schmorl's nodes and low back pain has been proved by a number of clinical studies, but most injuries caused by Schmorl's nodes are painless [14, 24, 28, 29]. Clinical observations have found that nearly one-third of the population has Schmorl's nodes [29]. In current study, we observed that some patients had multiple Schmorl's nodes, but not all induced pain in discography. Edema or inflammation dissipation in the injury site may be the causes of painlessness [27]. To identify whether Schmorl's nodes are painful, we mainly rely on discography. According to our diagnosis and treatment results, the lumbar discography is a very dependable approach. In the process of discography, the injection of contrast would generate not only the expansion pressures on the annulus fibrosus, but also pressure on the endplates. Meanwhile, the contrast always flows to the injured endplates shown in the MRI. However, when patients with discogenic low back pain took the lumbar discography, the contrast always flowed to the posterolateral disc via the ruptured annulus fibrosus fissures. This difference is also the main identifier of low back pain originating from the endplate and discogenic low back pain caused by ruptured annulus fibrosus.

In 1993, Heggeness et al. [12] published the results of studies of cadaver discography. They found that the discography exerted obvious expansion and deflection effects on the endplates. The deflection of the central endplates in response to the nucleus pulposus was 0.12–0.69 mm, while the deflection of the endplates in regions responding to the annulus fibrosus was 0.06–0.35 mm. Their analysis revealed that the discography led to swelling in the annulus fibrosus and deflection in the endplate, and thus induced low back pain. Thus, the endplate could be one origin of low back pain.

Doubtlessly, conservative treatments including medication, physical therapy, bed rest, and massage may be effective for most patients. For some in whom conditions are gradually developing, or chronically disabled patients with low back pain, if a variety of non-surgical treatments are invalid, surgeries still deserve consideration. We elected to perform fusion surgery based on the following criteria. First, a case of consecutively or repeatedly recurrent serious low back pain should have failed to respond to the use of formal conservative treatments, and hence seriously affected the life or work of the afflicted individual for at least 12 months. Second, the CT or MRI examination would show no lumbar disc herniation and no nerve root compression, save for the endplate Schmorl's node injury lesions. Third, the lumbar discography would reveal that the contrast flowed from the nucleus pulposus to the endplate injuries, and when the contrast was injected, patients experienced accurate low back pain provocation.

Lumbar spinal fusion surgery has been proved in long-term clinical practices to be an effective treatment method for low back pain. A large number of prospective and retrospective clinical studies have shown that various fusion technologies can obviously improve low back pain [10, 11, 13, 16, 20, 24]. The clinical outcomes of our treatment showed a reduction in pain as measured by VAS of a mean of 5.51, a mean reduction in Oswestry disability scores of 47.43, and satisfaction rate of 95% after the treatment. The treatment results achieved in the current study were similar or exceeded those obtained by fusion surgery for the treatment of low back pain caused by internal disc disruption or Modic changes without endplate injury [10, 11, 13, 16, 20, 24, 25]. Our clinical results indicate that both anterior and posterior fusions achieve satisfactory efficacies in eliminating low back pain symptoms and improving both motor function and fusion rate, suggesting that as long as the choice of indication is appropriate, lumbar spine fusion is a very effective method of treating otherwise disabling low back pain caused by endplate injuries.

Acknowledgments Supported by the Foundation of capital medical development, Beijing, China. The funding sources had no involvement or influence in the preparation of the manuscript.

References

1. Aggrawal ND, Kaur R, Kumar S, Mathur DN (1979) A study of changes in the spine in weight lifters and other athletes. *Br J Sports Med* 13:58–61. doi:10.1136/bjism.13.2.58
2. Andersson GB (1999) Epidemiologic features of chronic low-back pain. *Lancet* 354:581–585. doi:10.1016/S0140-6736(99)01312-4
3. Brown MF, Hukkanen MVJ, McCarthy ID et al (1997) Sensory and sympathetic innervation of the vertebral endplate in patients with degenerative disc disease. *J Bone Joint Surg Br* 79:147–153. doi:10.1302/0301-620X.79B1.6814
4. Carragee EJ (2005) Persistent low back pain. *N Engl J Med* 352:1891–1898. doi:10.1056/NEJMc042054
5. Chandraraj S, Briggs CA, Opeskin K (1998) Disc herniations in the young and endplate vascularity. *Clin Anat* 11:171–176. doi:10.1002/(SICI)1098-2353(1998)11:3<171::AID-CA4>3.0.CO;2-W
6. Deyo RA, Weinstein JN (2001) Low back pain. *N Engl J Med* 344:363–370. doi:10.1056/NEJM200102013440508
7. de Roos A, Kresse H, Spritzer C et al (1987) MR imaging of marrow changes adjacent to endplates in degenerative lumbar disc disease. *AJR Am J Roentgenol* 149:531–534
8. Fahey V, Opeskin K, Silberstein M, Anderson R, Briggs C (1998) The pathogenesis of Schmorl's nodes in relation to acute trauma: an autopsy study. *Spine* 23:2272–2275. doi:10.1097/00007632-199811010-00004
9. Fairbank JC, Couper J, Davies JB et al (1980) The Oswestry low back pain disability questionnaire. *Physiotherapy* 66:271–273
10. Fritzell P, Hagg O, Wessberg P, Nordwall A (2006) The Swedish Lumbar Spine Study Group. 2001 Volvo award winner in clinical studies: lumbar fusion versus nonsurgical treatment for chronic low back pain. A multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. *Spine* 23:2521–2534
11. Fritzell P, Hagg O, Wessberg P et al (2002) Chronic low back pain and fusion: a comparison of three surgical techniques: a prospective multicenter randomized study from the Swedish lumbar spine study group. *Spine* 27:1131–1141. doi:10.1097/00007632-200206010-00002
12. Heggeness MH, Doherty BJ (1993) Discography causes end plate deflection. *Spine* 18:1050–1053
13. Hanley EN, David SM (1999) Lumbar arthrodesis for the treatment of back pain. *J Bone Joint Surg Am* 81:716–730
14. Hauger O, Cotton A, Chateil JF et al (2001) Giant cystic Schmorl's nodes: imaging findings in six patients. *AJR Am J Roentgenol* 176:969–972
15. Kokkonen S, Kurunlahti M, Tervonen O et al (2002) Endplate degeneration observed on magnetic resonance imaging of the lumbar spine: correlation with pain provocation and disc changes observed on computed tomography diskography. *Spine* 27:2274–2278. doi:10.1097/00007632-200210150-00017
16. Lee CK, Vessa P, Lee JY (1995) Chronic disabling low back pain syndrome caused by internal disc derangements. The results of disc excision and posterior lumbar interbody fusion. *Spine* 20:356–361
17. Lotz JC, Ulrich JA (2006) Innervation, inflammation, and hypermobility may characterize pathologic disc degeneration: review of animal model data. *J Bone Joint Surg Am* 88(suppl 2):76–82. doi:10.2106/JBJS.E.01448
18. Modic MT, Masaryk TJ, Ross JS et al (1988) Imaging of degenerative disk disease. *Radiology* 168:177–186
19. Modic MT, Steinberg PM, Ross JS et al (1988) Degenerative disc disease: assessment of changes in vertebral body marrow with MRI imaging. *Radiology* 166:194–199
20. Moore KR, Pinto MR, Butler LM (2002) Degenerative disc disease treated with combined anterior and posterior arthrodesis and

- posterior instrumentation. *Spine* 27:1680–1686. doi:[10.1097/00007632-200208010-00018](https://doi.org/10.1097/00007632-200208010-00018)
21. Peng B, Hao J, Hou S et al (2006) Possible pathogenesis of painful intervertebral disc degeneration. *Spine* 31:560–566. doi:[10.1097/01.brs.0000201324.45537.46](https://doi.org/10.1097/01.brs.0000201324.45537.46)
 22. Peng B, Wu W, Hou S, Li P, Zhang C, Yang Y (2005) The pathogenesis of discogenic low back pain. *J Bone Joint Surg Br* 87:62–67
 23. Peng B, Wu W, Hou S, Shang W, Wang X, Yang Y (2003) The pathogenesis of Schmorl's nodes. *J Bone Joint Surg Br* 85:879–882
 24. Penta M, Fraser RD (1997) Anterior lumbar interbody fusion. A minimum 10-year follow-up. *Spine* 22:2429–2434. doi:[10.1097/00007632-199710150-00021](https://doi.org/10.1097/00007632-199710150-00021)
 25. Resnick DK (2003) Spinal Fusion for Discogenic back pain: patient selection, operative techniques, and outcomes. *Tech Neurosurg* 8:176–190. doi:[10.1097/00127927-200308030-00006](https://doi.org/10.1097/00127927-200308030-00006)
 26. Saluja G, Fitzpatrick K, Bruce M, Cross J (1986) Schmorl's nodes (intravertebral herniations of intervertebral disc tissue) in two historic British populations. *J Anat* 145:87–96
 27. Takahashi K, Miyazaki T, Ohnari H et al (1995) Schmorl's nodes and low-back pain: analysis of magnetic resonance imaging findings in symptomatic and asymptomatic individuals. *Eur Spine J* 4:56–59. doi:[10.1007/BF00298420](https://doi.org/10.1007/BF00298420)
 28. Takahashi K, Takiata K (1994) A large painful Schmorl's node: a case report. *J Spinal Disord* 7:77–81. doi:[10.1097/00002517-199407010-00011](https://doi.org/10.1097/00002517-199407010-00011)
 29. Wagner AL, Murtagh R, Arrington JA et al (2000) Relationship of Schmorl's nodes to vertebral body endplate fractures and acute endplate disk extrusions. *AJNR Am J Neuroradiol* 21:276–281
 30. Walters G, Coumas JM, Akins CM, Ragland RL (1991) Magnetic resonance imaging of acute symptomatic Schmorl's node formation. *Pediatr Emerg Care* 7:294–296. doi:[10.1097/00006565-199110000-00009](https://doi.org/10.1097/00006565-199110000-00009)
 31. Yasuma T, Saito S, Kihara K (1988) Schmorl's nodes: correlation of X-ray and histological findings in postmortem specimens. *Acta Pathol Jpn* 38:723–733