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A Retrospective Study of Percutaneous Balloon Kyphoplasty for the Treatment of Symptomatic Schmorl's Nodes: 5-Year Results

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Manuscript Preparation E
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Background: Despite literature supporting the efficiency of percutaneous balloon kyphoplasty (PKP) in treating osteoporotic and malignant vertebral compression fractures, few reports exist that document its use for treatment of symptomatic Schmorl's nodes (SNs) refractory to conservative treatment. Patients with symptomatic SNs could have pain in the vertebrae similar to an acute vertebral compression fracture. MRI is very helpful in diagnosing symptomatic SNs when x-ray and CT scan are unremarkable. In painful cases, the vertebrae bone marrow around the SNs is hyperintense on T2-weighted subsequence. We evaluated the long-term safety and effectiveness of PKP for the treatment of symptomatic SNs not responding to conservative therapy.

Material/Methods: From January 2008 to December 2012, 32 patients suffering from symptomatic SNs underwent 43 PKP procedures. Outcome data, including mean height ratio of anterior and middle vertebral body, Visual Analog Scale (VAS score) for pain measurement, Oswestry Disability Indexes (ODI score) and SF-36 questionnaires for function measurement were recorded preoperatively, postoperatively, and at one month, six months, two years, and five years after treatment.

Result: Thirty-two patients were treated successfully with PKP. Clinically asymptomatic cement leakage was observed in three (6.98%) of the treated vertebral bodies. The mean height ratio of anterior and middle vertebral bodies changed from 98.2±1.6% preoperatively to 98.5±1.4% postoperatively ($p>0.05$) and 98.3±1.5% preoperatively to 98.8±1.9% postoperatively ($p>0.05$). The mean VAS scores, ODI score, and SF-36 scores for physical function (PF), bodily pain (BF), social functioning (SF), and vitality (VT) all showed significant improvements ($p<0.05$). During the 5-year follow-up, the stabilization of the height of the vertebral body and functional improvements were all maintained.

Conclusions: PKP is a safe and effective procedure for the treatment of symptomatic SNs refractory to conservative therapy.

MeSH Keywords: Fractures, Stress • Kyphoplasty • Vertebroplasty

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Background

Schmorl's nodes (SNs) were first described by Von Luschka in 1858 and rediscovered by Christian Georg Schmorl in 1927 [1,2]. This specific type of vertebral lesion usually represents herniation of the intervertebral nucleus pulposus into the adjacent vertebrae. The etiology of SNs is not properly understood and could be degenerative, metabolic, malignant, infectious, traumatic, or might be owing to weakening of endplates due to idiopathic diseases or some other etiology [3].

SNs are common in normal individuals and are often asymptomatic; incidental findings are usually via magnetic resonance images (MRI). The reported prevalence of SNs ranges from 3.8% to as high as 79% and SNs usually occur in the thoracolumbar junction; there is a male predominance [4,5]. Prior epidemiological studies have revealed that patients with SNs may have a higher incidence of back pain compared to the normal population. However, the relationship of SNs and their clinical significance causing back pain remain unknown. Vertebral bone marrow edema which surrounded the symptomatic SNs on MRI image was thought to contribute to pain generation and correlate with the recent occurrence of nucleus pulposus extrusion into the endplate [5].

There are no differences in findings between asymptomatic and symptomatic patients on plain x-ray films and diagnosis is difficult in symptomatic SNs presenting with acute back pain. MRI is very helpful in diagnosing symptomatic SNs when plain film radiographs and CT scan are unremarkable. In painful patients, the vertebrae bone marrow around the SNs is seen as hyperintense on T2-weighted subsequence [4,5]. The change is located in the area around the SN. In contrast, the vertebrae bone marrow circles the SNs in asymptomatic cases are hypointensity on T2-weighted subsequence, similar to normal bone marrow.

Patients with symptomatic SNs may have pain on manual compression and percussion of the vertebra similar to that of a traumatic vertebral compression fracture [6]. Symptomatic SNs are much less frequently identified on routine images than are their asymptomatic counterparts, and they have been described in the literature only in isolated case reports [7,8]. Some research has indicated that these symptomatic SNs might either respond to conservative therapies such as analgesics, bed rests, and external bracings, or heal spontaneously in most instances. However, some patients are refractory to these therapies in terms of pain relief; thus surgical treatment (such as eradication of the SNs and segmental fusion or other minimally invasive surgery such as vertebroplasty and ramus communicans nerve blockage), should be considered [9].

Percutaneous balloon kyphoplasty (PKP) and percutaneous vertebroplasty (PVP) are two minimally invasive vertebral augmentation procedures, and both rely on percutaneous injection of bone cement. PKP utilizes inflatable bone tamps to create a cavity prior to the insertion of cement and is now considered as effective as conventional PVP for rapid pain relief in patients with painful vertebral compression fractures resistant to conservative treatment [10]. So far, few reports exist documenting the outcomes of minimally invasive vertebral augmentation procedure as an alternative to traditional conservative therapy for symptomatic SNs, most of which are limited to case reports and small series reports [11–13]. To our knowledge, there have been no reports focusing on PKP in the treatment of symptomatic SNs.

Therefore, we assessed the safety and effectiveness of PKP for the treatment of symptomatic SNs. In this report, we present a series of 32 patients with symptomatic SNs not responding to conservative therapy treated with PKP; we assessed the radiological and clinical outcomes over a long-term follow-up period.

Material and Methods

This retrospective study was approved by local Ethical Committee of our institution. From January 2008 to December 2012, 32 patients were diagnosed with symptomatic SNs. General characteristics of the patients are summarized in Table 1 and the levels treated by PKP were shown in Figure 1.

Patients included in the research underwent plain film, multislice computed tomography (MSCT) and magnetic resonance imaging (MRI) to assess the main causes of pain and confirm the presence of symptomatic SNs in absence of other causes of low back pain such as body vertebral fracture, intervertebral disc herniation, or body malignant lesions. SNs considered to be symptomatic may present with an edematous rim around the nodes in the cancellous vertebral body with high signal intensity on T2-weighted and STIR sequence of MRI images [14]. As a result, MRI was the most confirmative screening examination used to determine the presence of symptomatic SNs. MSCT was used also to evaluate the structures of vertebral body before the PKP procedure. The 32 patients all presented with severe back pain of at least eight weeks duration that was refractory to conservative treatment, including bed rest, rigid bracing, analgesics, and physical therapy. In our study, symptomatic disc diseases are ruled out by using discography. Patients with local infections, non-correctable coagulation disorders, and other systemic diseases were also ruled out from the study treatment.

PKP procedure

All the PKP procedures were performed by senior spinal surgeons from our institution. Patients all received general

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Table 1. Patient characteristics.

Characteristic	Value
Patient	
Number	32
Total number of PKP procedures	43
Number of symptomatic SNs treated per patient	1.34 (1–2)
Age (years)	53.4±6.8
Gender (F/M)	13/19
BMI (Kg/m^2)	28.5 ± 4.2
BMD T score	–2.4±0.7
Pain duration (months)	3.25±1.15
Follow-up (months)	60
Symptomatic SNs located Region (Number/Percentage)	
T5 through T9 vertebrae	9/(20.9%)
T10 through L2 vertebrae	26/(60.5%)
L3 through L5 vertebrae	8/(18.6%)
Symptomatic SNs distributed Region (Number/Percentage)	
Superior endplate	27/(62.8%)
Inferior endplate	16/(37.2%)
PKP Procedure	
Operation time per vertebrae (minutes)	23.2±6.3
Fluoroscopy time per vertebrae (minutes)	8.3±0.8
Bipedicular access (vertebraes)	32
Unilateral access (vertebraes)	11
Injected cement volume (mL)	4.1±0.8
Cement leakage	
Number of vertebraes	3/(6.98%)
Location	
Adjacent disks area	3
Venous plexus	0
Paravertebral soft tissues	0
New fractures	
Recollapse of treated vertebrae	0
Adjacent vertebral fractures	2 (4.65%)

Data are mean ± standard deviation; median, with the range in parentheses; or number of findings, with the percentage in parentheses.

anesthesia and were placed prone on a radiolucent C-arm operation table. The skin was then disinfected and covered with sterile drapes. Small incision was made and a probe was pushed forward discreetly and penetrated close to the symptomatic SN within the vertebral bodies with the help of fluoroscopy. Guide wire was initially inserted in order to obtain bilateral trans-pedicular access. In cases in which pedicle was not visualized on C-arm fluoroscopy, the unipedicular approach was then performed. PKP was then performed using the procedures suggested by Yang et al. [15,16] and biopsies around the nodes were obtained for pathological examination. The openings were then gradually enlarged using successively larger trocars. The bone was drilled and a balloon (Kyphon Inc., Sunnyvale, CA, USA) was inserted into the vertebral bodies to create a cavity

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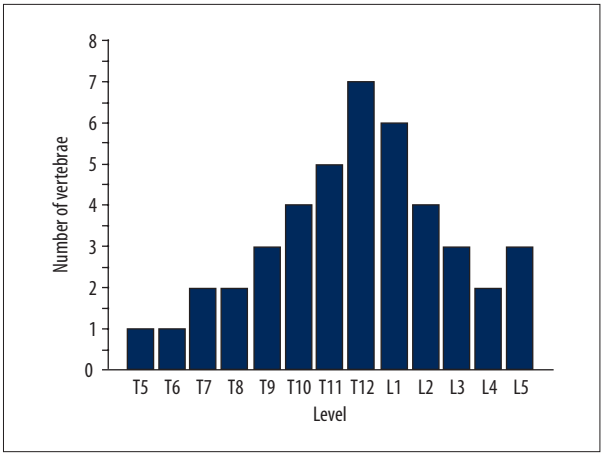

Figure 1. Distribution of vertebral bodies treated by PKP (n=43).

before injection of cement. The inflation process was stopped when the pressure reached 280 to 320 psi. Then, the balloon was deflated and removed. During the cement injection process, fluoroscopic monitoring was conducted in both planes. The PMMA cement introducers were then pulled out slightly until the bone cement hardened. After completion of the PKP procedures, patients were monitored for six to eight hours.

Radiographic assessment

Radiographic and clinical assessments were taken preoperatively, postoperatively, and at one-month, six-months, two-years, and five-years after treatment. The anterior vertebral height and middle vertebral height were obtained from lateral plain radiographs for symptomatic SNs compromised and adjacent control vertebral body. For each vertebral body, the normal height of the SNs compromised vertebral body was measured from the means of the data from the closest uncompromised vertebral body cephalad and caudad to the SNs compromised level. The anterior vertebral height and middle vertebral body height ratios were calculated using this method: (SNs compromised vertebral body heights/normal vertebral body heights)×100% [15].

Clinical evaluation

Back pain was evaluated using a visual analogue scales (VAS) with values in the range of 0 to 10, where 0 indicates no pain and 10 indicates the worst pain imaginable. Furthermore, Oswestry Disability Index (ODI) scores, which are used to assess functional capacity where a lower percentage indicates a better health status, were also documented. The SF-36 questionnaire is an in-depth quality of life evaluation consisting of 36 questions, where a higher score indicates a better health status. The results are categorized into eight domains and physical function (PF), the social function (SF), bodily pain (BP) and vitality (VT) have been shown to have higher reliability and responsiveness and were selected for assessment in our research [16].

Complications such as bleeding, stroke, infection, cardiac arrest, and pulmonary embolism were recorded. The rates of cement extravasation outside the vertebral bodies were also measured postoperatively using plain radiographs and CT scans.

Statistical analysis

Mean changes, including mean values and standard deviations, in anterior and middle vertebral body height ratios, SF-36 scores, ODI scores, and VAS scores were assessed and analyzed using SPSS software (SPSS 19.0, Inc., Chicago, IL, USA). Comparisons between different time points in the follow-up time were performed using a paired Student's *t*-test and tested by the repeated-measures analysis of variance (ANOVA). The level of significance was set at $p<0.05$.

Results

Safety and efficacy

We performed a five-year follow-up for the 32 patients. Significant improvements in SF-36 scores, ODI scores, and VAS scores were recorded postoperatively, and at one-month, six-months, two-years, and five-years after the treatment. Symptomatic SNs were confirmed by pathological examination, which demonstrated the presence of edema and inflammation in the vertebral body bone marrow around the nodes. There were no complications such as bleeding, infection, reactions with the bone cement, pulmonary embolism, vertebral new fractures, or recurrence of back pain in the follow-up time period.

The mean anterior and middle vertebral heights variation changed from 98.2±1.6% preoperatively to 98.5±1.4% postoperatively ($p>0.05$) and 98.3±1.5% preoperatively to 98.8±1.9% postoperatively ($p>0.05$), respectively. Mean VAS scores, ODI scores, and SF-36 scores for PF, BF, SF and VT all showed notable improvements ($p<0.05$) (Table 2). During the 5-year follow-up, the stabilization of the height of the vertebral body and functional improvements were all maintained.

In our study, the postoperative CT images of the PKP-treated vertebras indicated a clinically asymptomatic cement extravasation in three vertebral bodies, representing 6.98% of all treated levels. All cement extravasation was located in the adjacent disc areas and there were no major complications such as pulmonary embolism or epidural leakage.

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Table 2. Mean outcome measures among 32 patients with symptomatic SNs treated by PKP.

	Preoperative	Postoperative	1-Month follow up	6-Month follow up	2-Year follow up	5-Year follow up
Vertebral body height ratio (%)						
Anterior	98.2±1.6	98.5±1.4#	98.4±1.2#	97.8±2.1#	96.9±2.3#	95.2±3.6#
Middle	98.3±1.5	98.8±1.9#	98.1±2.0#	97.5±2.4#	96.3±2.8#	95.0±3.2#
VAS score	8.7±1.1	2.3±1.2**	2.5±1.4**	2.9±1.5**	3.2±0.8**	3.1±1.0**
ODI score	82.1±10.2	29.2±8.0**	17.8±8.1**	24.6±12.4**	29.1±6.5**	31.5±10.9**
SF-36 score						
BP	12.1±3.2	38.6±7.4*	43.0±8.2*	52.1±12.3**	54.0±19.7**	52.5±13.6**
PF	23.8±5.3	47.2±10.8*	48.5±14.0*	52.1±11.9*	49.8±11.6*	48.8±16.0*
VT	31.3±10.4	49.8±8.7*	52.5±9.0*	54.4±15.6*	58.9±13.2*	59.4±12.9*
SF	13.2±5.6	38.1±8.0**	44.7±9.3**	41.9±10.1**	45.7±11.7**	43.6±9.8**

VAS – visual analog scale; ODI – Oswestry disability index; BP – bodily pain; PF – physical function; VT – vitality; SF – social function; # P>0.05 compared to preoperative value; * P<0.05 compared to preoperative value; ** P<0.001 compared to preoperative value.

Case presentation

A 52-year-old man was admitted to our spine outpatient clinic with severe symptoms of acute localized back pain for six weeks. He sprained his back six months ago and was treated with an external bracing. He gradually recovered from the back pain and give up the external bracing. In the recent four months, the back pain recurred and could be aggravated by trivial activity. He was not responding to conservative therapy such as analgesics, bed rests, and external bracings. His usual lifestyle was limited and he was basically confined to bed. The patient reported back pain usually reoccurred and his condition progressively deteriorated.

Physical examination revealed conspicuous tenderness and percussion pain in the L1 vertebral body. The straight leg raising test was negative on both sides and both knee and ankle reflexes were present. He denied paresthesia, numbness, or weakness in the lower extremities. Routine laboratory results, including total blood count, electrolyte values, sedimentation rate, hormone levels, CRP, and alkaline phosphate levels, were found to be within normal ranges. Furthermore, plain radiograph of the lumbar spine showed no abnormalities (Figure 2A,2B). Due to patient's suffering and the severity of the symptoms, further imaging studies were performed. MSCT showed a Schmorl's node in the L1 vertebral body (Figure 2C, 2D) surrounded by a sclerotic margin. On sagittal MRI images, bone marrow of vertebral body surrounding the SN was characterized by hypointensity on T1-weighted sequence (Figure 3A), hyperintense on T2-weighted sequence (Figure 3B), and especially higher hyperintense on STIR sequence (Figure 3C).

The patient underwent ramus communicans nerve blockage but experienced little benefit and refused to receive a segmental fusion surgery. However, he had a strong desire for immediate pain relief. We successfully performed a PKP procedure with a bilateral access under fluoroscopy (Figure 4). The diagnosis of symptomatic SNs was confirmed by pathological examination obtained around the node that demonstrated the presence of edema and inflammation in the vertebral body bone marrows (Figure 5). Bone cement distribution was diffuse and homogeneous, and cement extravasation was not noticed (Figure 6). The patient experienced complete pain relief. A routine follow-up revealed no residual abnormalities, and the patient continues to enjoy good health. At the five-year follow-up evaluation, the patient was satisfied with the procedure results and was able to perform all normal daily activities.

Discussion

SNs are intraspongious herniations or extrusions of intervertebral disc materials of variable sizes (typically >2.0 mm) through the cartilaginous and bony endplate into the vertebral body [5,17]. They are usually observed as chronic entities in approximately one fourth of the population, although one cadaveric related study reported a 79% incidence [5,9]. A number of theories have been proposed in an attempt to explain the etiology of SNs; however, no consensus currently exists. It is accepted that weakening of the endplate facilitates the migration of disc materials and the cartilaginous endplate weakness of a vertebrae is related to several mechanisms including traumatic, degenerative, or vascular causes due to bone fragility, Scheuermann's disease, or immunologic factors [18]. Pathologically, SNs represent the nucleus

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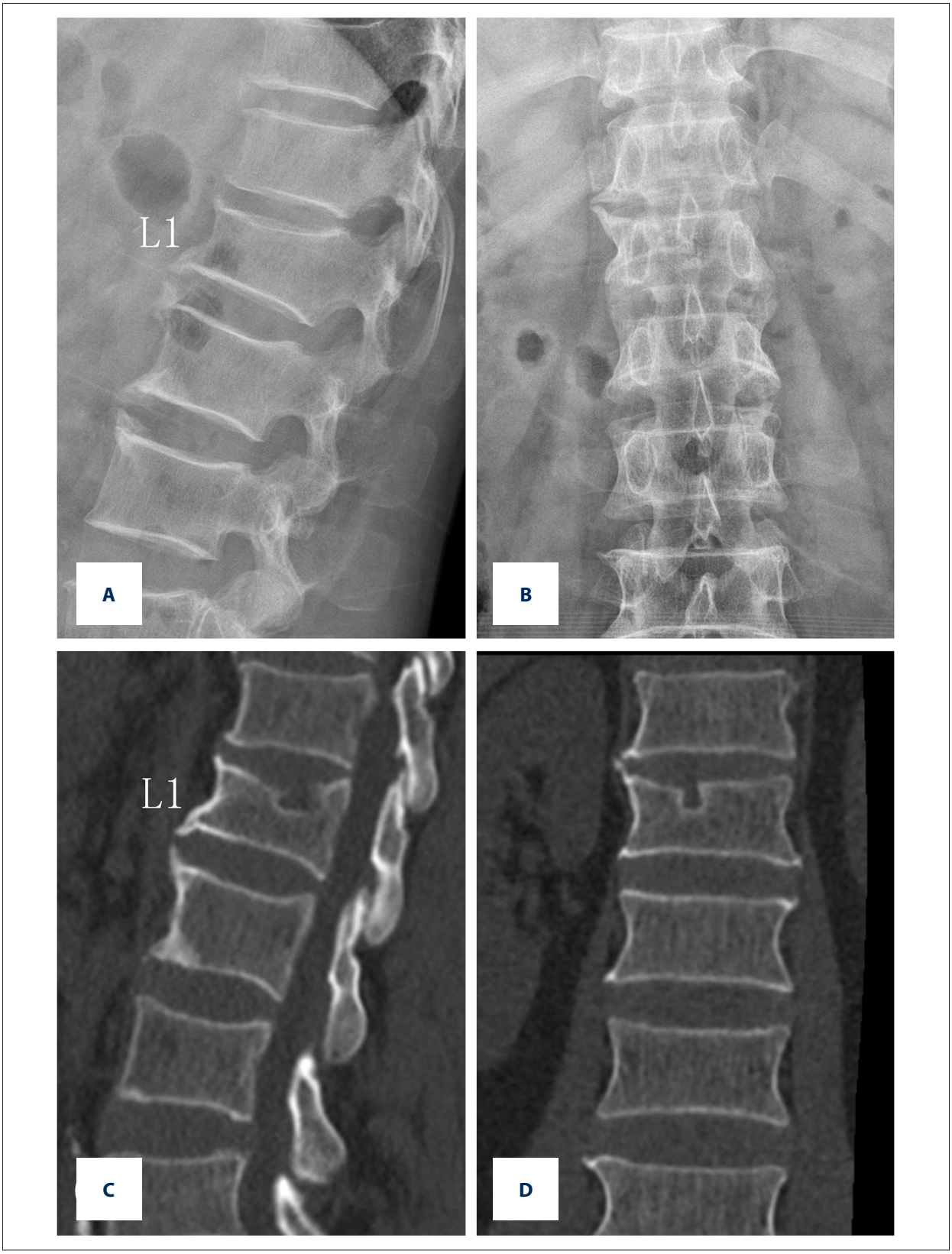

Figure 2. Lateral (**A**) and anteroposterior (**B**) plain radiograph of the lumbar spine showed no abnormalities. Sagittal (**C**) and coronal (**D**) CT images of the lumbar spine demonstrate a Schmorl's node in the L1 vertebral body surrounded by a sclerotic margin.

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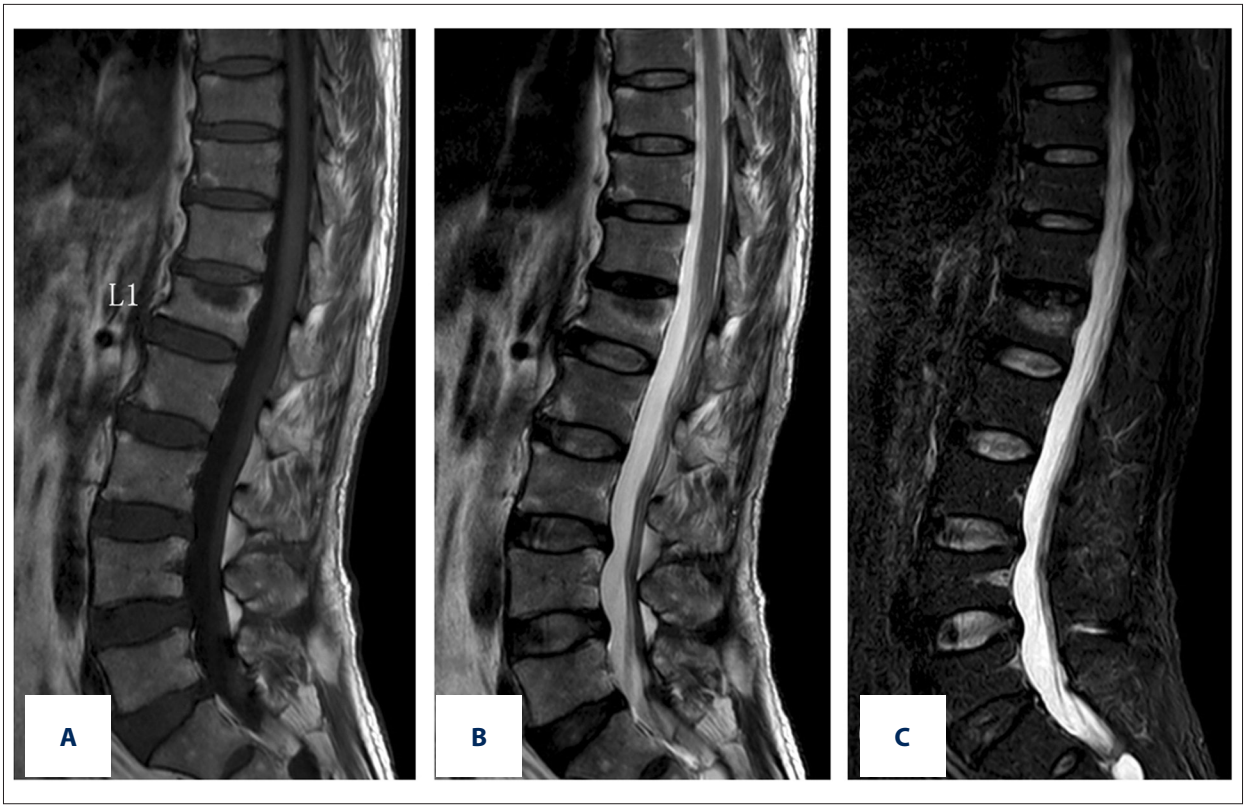

Figure 3. On sagittal MRI images, vertebral body bone marrow surrounding the node was characterized by low-intensity signal on T1-weighted sequences (**A**), high-intensity signal on T2-weighted sequences (**B**), and especially high intensity on STIR sequences (**C**). The appearance of the node was typical and appears as a small pocket in the vertebral body with continuation into the disc through the vertebral endplate (**C**).

pulposus with degenerative or inflammatory changes and a confined sclerotic response in the adjacent vertebral spongiosa [5]. Clinically, they are usually believed to be asymptomatic incidental findings on plain radiographs, computed tomography (CT), and MRI and do not require surgical treatment. However, in some cases, they may denote a severe symptomatic process and cause acute low back pain [19].

Several studies have demonstrated that SNs are of themselves related to back pain. However, the relationship of SNs and clinically significant back pain remains unknown in the general population. As we know, any spinal structure having a nerve supply may be a source of back pain when subjected to pain-producing tissue damage. The histological origins of pain are not yet fully established. It has been postulated that in affected vertebrae there are higher proportions of disc marrow contacts and that the herniating materials irritate a special intravertebral nociceptive system, ultimately generating pain [19,20]. Fields et al. [20] reported a higher nerve density was found in endplate defects than in normal endplates. Symptomatic SNs represent fresh intraosseous fractures in the vertebral body. We presume that inflammatory reactions in the bone marrow of the vertebral body induced by intraosseous fractures and biological reactions to nucleus pulposus might produce the pain. Like other authors [2,13], we believe that the relevant nociceptors are located in the edematous rim around the node, similar to edematous zones in and around osteoporotic vertebral compression fractures (OVCFs). Micromovements, inflammation, and pressure on the nociceptors within the edematous area probably induce back pain. We postulate that after fracture healing and subsidence of inflammation, the SNs become asymptomatic, in analogy with old OVCFs.

In the acute stage, symptomatic SNs are difficult to diagnose and even to detect on plain radiographs, because the sclerotic margin around the node has not had time to develop [9]. Therefore, plain radiographs have limited value in assessing SNs, and especially symptomatic SNs. MRI is the gold standard imaging modality to detect symptomatic SNs. Takahashi et al. [21] analyzed MRI findings in patients with symptomatic and asymptomatic SNs. In symptomatic patients, the bone marrow of vertebral bodies surrounding the SNs demonstrated a hypointensity on T1-weighted images and a hyperintense on T2-weighted images. This demonstrated the presence of edema and inflammation in the marrow of vertebrae. These signal changes in MRI were not present in asymptomatic

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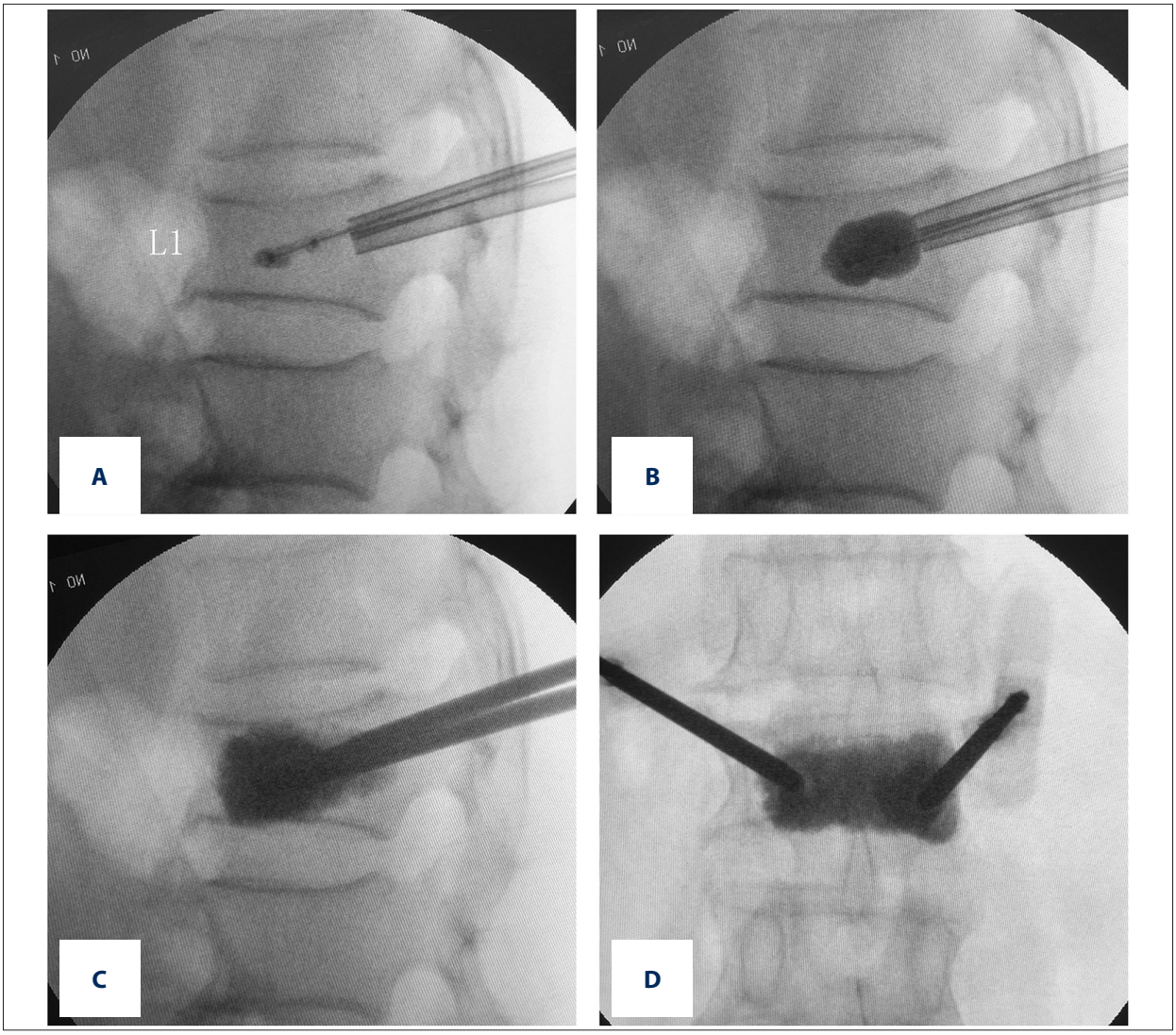

Figure 4. PKP was performed using a bilateral access under fluoroscopy. Inflatable balloons were inserted into the vertebrae (**A**); the balloons were then inflated with contrast medium and expanded to create a cavity in the vertebral body (**B**). The spaces created by the balloons were then filled with PMMA bone cement. Lateral (**C**) and anteroposterior (**D**) fluoroscopic images demonstrate PMMA filling the L1 vertebral body surrounding the Schmorl's node.

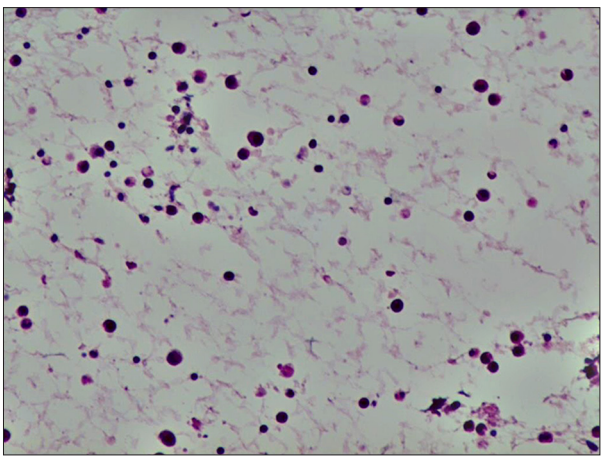

Figure 5. Biopsies around the Schmorl's node were performed during PKP procedure and histologic findings demonstrated the presence of severe inflammatory cell infiltration and bone marrow edema in the vertebrae (hematoxylin and eosin, 240×).

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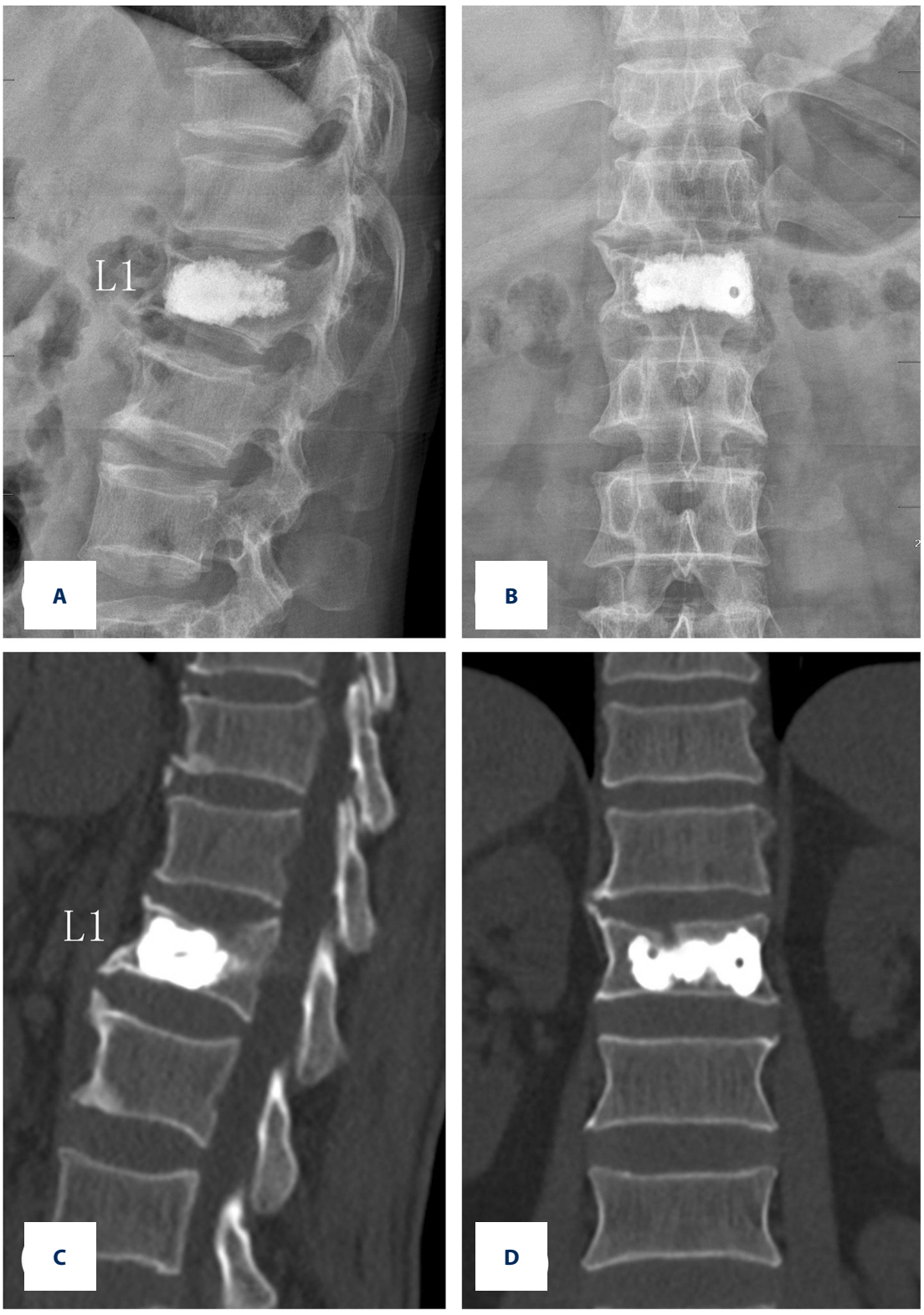

Figure 6. Lateral (**A**), anteroposterior (**B**) plain radiograph and sagittal (**C**), coronal (**D**) CT images showed cement distribution was diffuse and homogeneous in L1 vertebral body surrounding the Schmorl's node and cement leakage was not noticed.

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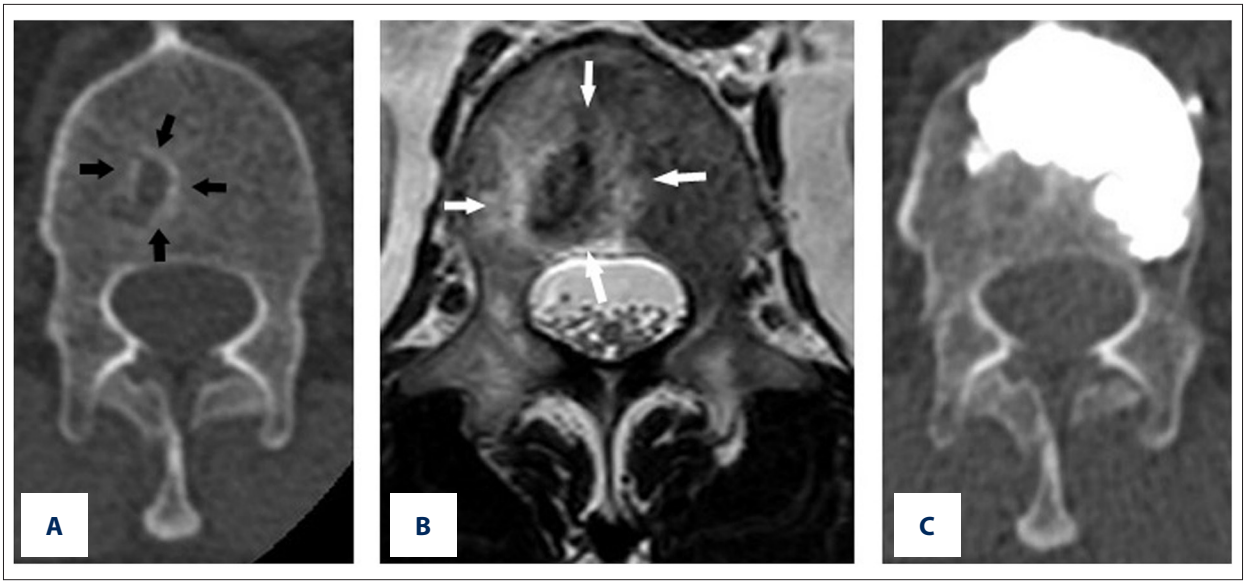

Figure 7. (**A**) Pretreatment axial CT demonstrates a Schmorl's node (black arrows) in L1 vertebra with surrounding sclerosis. (**B**) Pretreatment axial MRI (STIR sequence) in symptomatic L1 vertebrae shows hyperintense in the vertebrae bone marrow around the SN (white arrows point at the edematous rim around the node). (**C**) Postprocedural CT demonstrates PMMA cement filling the L1 vertebrae around the Schmorl's node without extravasation into the node.

individuals, which also suggested that SNs became asymptomatic when the edema and inflammation subsided [21]. Although MRI findings are specific, differentiation from malignant disease or other inflammatory lesions can be indistinguishable [15]. Our experience has shown that identification of the endplate defects by MSCT may be helpful for the correct diagnosis of symptomatic SNs. In our study, each patient was diagnosed with symptomatic SNs using MRI and MSCT.

A number of strategies to alleviate the pain associated with symptomatic SNs have been reported in the literature. The first-line therapeutic method is conservative treatment with bed rest, rigid bracings, and analgesic drugs. However, this is ineffective in some patients and some authors have proposed several invasive surgical treatments. Peng et al. [18] conducted a segmental fusion surgery and found it efficacious in alleviating severe lower back pain due to SNs. Fukuta et al. [22] presented a case of a young man who suffered from severe back pain due to symptomatic SNs and the patient was treated successfully by anterior interbody fusion (AIF). Hasegawa et al. [23] presented a typical case of the eradications of intervertebral nucleus pulposus containing a SN and posterior lumbar interbody fusions to solve the symptomatic SN. Several other treatment methods, such as ramus communicans nerve blockade, discography, and discoblock have been proposed for the management of back pain secondary to suspected symptomatic SNs [8,24]. These techniques can provide rapid and lasting symptom relief. Masala et al. [25] and He et al. [26] reported that vertebroplasty, a minimally invasive transcutaneous injection of PMMA bone cement within the vertebrae, can be a possible alternative to treat symptomatic SNs refractory to conservative therapy. However, as these reports were case reports or small sample trials with short-term follow-up periods, their reliability is somewhat controversial.

PVP and PKP are minimally invasive, radiologically guided interventional procedures, involving injection of polymethylmethacrylate (PMMA) into the vertebral body. PKP is a modified edition of PVP and has gained wider clinical acceptance as a more effective treatment option in OVCFs [7,15]. Symptomatic SNs represent a fresh intraosseous fracture in the vertebral body and relevant nociceptors are located in the edematous rim. This suggests that symptomatic SNs could be treated with vertebral augmentation procedures and the cement injection should specifically target the edematous rims around the node (as seen on MRI images) which has been implicated as the main source of pain. PMMA cement should be injected at an early stage of polymerization for increased cement diffusion into the edematous area without extravasation into the node (Figure 7A–7C). The lack of cement leakage to the disc area may be due to increased pressure from the herniated disc or due to the presence of marginal peridiscal bone sclerosis caused by adjacent boney trabecular impaction occurring with the intravertebral body disc herniation [16]. It is likely that pain relief is obtained through stabilization of the intraosseous fracture by the PMMA cement. Another mechanism is that the injected PMMA cement causes thermal necrosis and chemotoxicity to the intraosseous nociceptors [16,27]. We propose the use of PKP over PVP as a therapeutic strategy in patients with symptomatic SNs because PKP has the

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advantage of reduced cement extravasation. To the best of our knowledge, there have been no reports focusing on PKP in the treatment of symptomatic SNs in the literature [5,9,15]. Here, we present a retrospective study, from the periods of January 2008 to December 2012, assessing 32 patients affected by symptomatic SNs refractory to conservative treatment, who underwent PKP procedures in our orthopedic center to solve their low back pain. Outcome data including anterior and middle vertebral body height ratios, VAS scores for pain, ODI scores and SF-36 questionnaire scores for function were collected preoperatively, postoperatively, and at one month, six months, two years, and five years after treatment. Notable improvements in all measurements were observed postoperatively and during the follow-up period. During the five-year follow-up, the stabilization of the height of the vertebral body and functional improvements were all maintained. There were no major complications or worsening of symptoms. We found PKP to be a safe and effective procedure in the treatment of symptomatic SNs refractory to conservative therapy.

The limitation of this research was its retrospective design, which has less creditability than a prospective study. Also, only patients who underwent PKP were included in our study; there was no control or alternative treatment, such as PVP, group. Further additional prospective studies are needed to assess the effectiveness and safety of PKP in treatment of symptomatic SNs.

Conclusions

Our study demonstrated that symptomatic SNs refractory to conservative therapy could be treated similarly to OVCFs. To achieve earlier detection and diagnosis, suspected patients with SNs who have symptomatic pain should undergo MRI and MSCT. These patients with incapacitating back pain refractory to conservative treatment can be treated with, and benefit from PKP. PKP is efficacious and safe for symptomatic SNs.

Conflict of interest

None of the authors has a financial interest or other intention in any of the products, devices, or methods mentioned in this article.

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