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REVIEW ARTICLE

Schmorl's nodes

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Abstract
Introduction First described in 1927, a Schmorl's node (SN) is the herniation of nucleus pulposus (NP) through the cartilaginous and bony end plate into the body of the adjacent vertebra. SNs are common findings on imaging, and although most SNs are asymptomatic, some have been shown to become painful lesions. In this manuscript, we review the literature regarding the epidemiology, clinical presentation, pathogenesis, imaging, and management of SNs.
Materials and methods Using databases from the US National Library of Medicine and the National Institutes of Health, relevant articles were identified.
Results While several theories regarding the pathogenesis of SNs have been proposed, an axial load model appears to have the greatest supporting evidence. Symptomatic SNs are thought to be due to the inflammatory response solicited by the herniation of NP into the well-vascularized vertebral body. Management options for symptomatic SNs vary, ranging from medical management to surgical fusion.
Conclusion SNs are common lesions that are often asymptomatic. In certain cases, SNs can cause back pain. No consensus on pathogenesis exists. There is no established treatment modality for symptomatic SNs.

Keywords Schmorl's node · Review · Spine

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Introduction

In 1927, pathologist Christian Georg Schmorl [1] described a specific type of vertebral lesion, seen primarily in the thoracolumbar spine, which is now known as Schmorl's node (SN). Unlike the better-known horizontal disc herniations into the spinal canal or neural foramina, SNs are herniation of nucleus pulposus (NP) through the cartilaginous and bony end plate into the body of an adjacent vertebra [2]. It has been reported that multiple SNs are highly associated with lumbar disc disease and lower back pain [3].

A number of theories have been proposed in an attempt to explain the pathogenesis of SNs; however, no consensus currently exists. Some researchers view SNs as a developmental disease [4], while others see SNs as a degenerative bone disease [5]. Still some researchers theorize that SNs are a result of pathologies that weaken the discs and vertebral bodies [6]. Direct trauma to the vertebra has also been implicated as a contributor to the development of SNs [7]. In addition, Zhang et al. [8] theorized a possible role of autoimmunity in symptomatic SNs.

A fair amount of research effort has been expended to characterize SNs, yet their pathogenesis, clinical significance, and management are still in debate. The aim of our study is to present a critical review of the SN literature with a focus on pathogenesis, clinical presentation, and management of symptomatic lesions.

Materials and methods

Databases of the US National Library of Medicine and the National Institutes of Health (i.e., www.pubmed.gov) were queried to identify studies pertaining to SNs. The term *Schmorl's node* was used as the search key word, and all 89

English-language articles were read for mention of the pathogenesis, epidemiology, clinical presentation, imaging, or management of SNs. From this initial list, 23 articles were selected to be included in this review as they substantially addressed one or more of the areas of interest. Pertinent references cited in these articles were then examined to identify additional studies of relevance.

Results

Epidemiology

The exact cause (or causes) of SNs is currently unknown. Symptomatic SNs usually resolve spontaneously or respond to conservative treatment. Some, however, can become a source of chronic lower back pain [9]. It has been shown that SNs are more frequent in males than females [5]. Some researchers have reported a positive association with increasing age [10], while others argue that age is not a significant factor [5]. Yet, a closer look at the study by Hilton et al. [5] revealed an age-dependent relationship between SNs and disc degeneration in the T10-L1 region, an observation acknowledged by the authors as "unexpected". In spite of this, they assume a developmental etiology, in conjunction with thoracolumbar susceptibility to stress as an explanation. The authors discount the possibility that age-dependent disc degeneration could have a role in the etiology of SNs, which would better explain the age discrepancy. Age-dependent degeneration may, therefore, play a larger role in the formation of SNs than the authors recognized.

With regard to the prevalence of SNs, cadaver studies vary in their estimates, ranging from 38 % to as high as 79 % [2, 5, 11]. A magnetic resonance imaging (MRI) study of 150 monozygotic and 366 dizygotic living twins found SNs in 30 % of subjects at any vertebral level, and multiple SNs in 14 % of participants [3]. A strong hereditary association was noted, with lumbar and thoracic SNs having heritability values of 80 and 72 %, respectively. These heritability findings may suggest a genetic or embryologic etiology to the pathogenesis of SNs, which are discussed below [12, 13].

Clinical presentation

SNs are often incidental findings on MRI (Fig. 1). However, Hamanishi et al. [14] studied 400 patients with lower back pain and 106 controls without back pain and reported that 19 % of the patients with lower back pain had SNs, while only 9 % of the control group had SNs. This result suggests that SNs may account for a significant number of back pain cases. It does not, however, rule out the possibility that SNs could be histologic or radiologic findings in degenerated disc disease, and not necessarily the source of the pain as evidenced by the prevalence of asymptomatic SNs. A number of studies have found SNs to be mostly localized to the lower thoracic area, between T8 and T12. Symptomatic SNs are often very painful, with visual analog scale (VAS) scores prior to treatment close to 10 on a 10-point VAS, with significant reductions in quality of life [15–17].

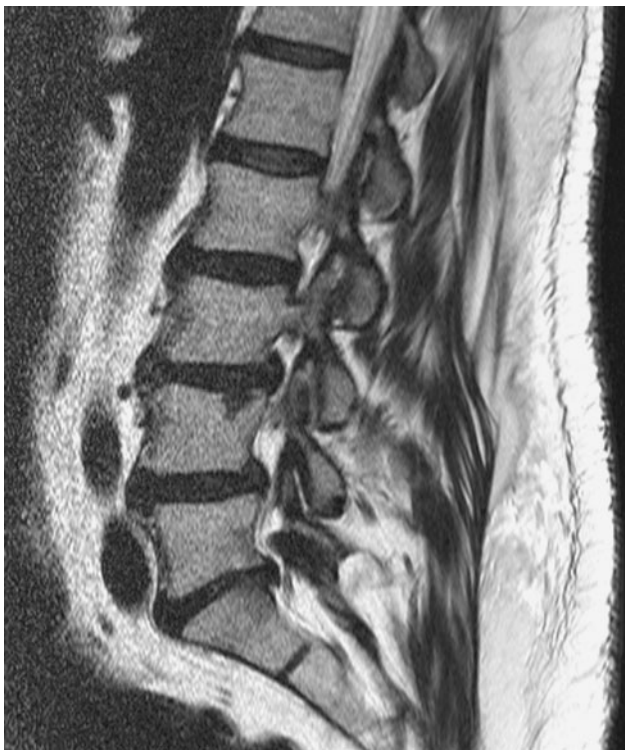

Fig. 1 Sagittal MRI showing an incidentally discovered Schmorl's node at level L4

Pathogenesis

The pathogenesis of SNs is a topic of debate. As mentioned above, there are a number of theories that attempt to explain their development.

Axial load (trauma)

In a study of 70 thoracolumbar spines from cadavers of individuals killed in motor vehicle collisions, Fahey et al. [7] reported a link between acute trauma and the occurrence of SNs. The authors found that 10 % of their samples manifested SNs. Of these, 40 % were from motorcyclists. The authors believed this to be a significant finding given the typically axial trajectory of a motorcyclist from his/her vehicle to the ground in an accident. It is known that cyclists often land head first in an inverted position leading

to axial loading on the vertebrae. It also known that falls often produce axial loading on the spine [18]. Interestingly, a study comparing vertebral abnormalities in elite gymnasts versus non-athletes [19] found SNs in 71 % (17 out of 24) of gymnasts and only 44 % (7 out of 17) of non-athletes. Gymnasts experience greater-than-average axial forces on their vertebrae. Hence, such a finding is evidence that axial loading may play a crucial role in the development of SNs.

Dar et al. [20] proposed an axial load model in which they argued that because of their erect posture and bipedal locomotion, humans must accommodate increased axial forces in addition to balancing the need for spinal mobility and stability. Given that the thoracolumbar spine bears great axial stress and is relatively mobile, it may accumulate micro-traumas that can, over time, lead to the formation of SNs in the general population [20]. They concluded that the combination of increased range of rotational movement, anteriorly located instantaneous axis of rotation, and low-disc thickness relative to vertebral body height in the thoracic spine makes this region more vulnerable to develop SNs. This predominance of SNs in the lower thoracic region has been verified by other studies [3, 7].

There is also evidence suggesting that in 46 % of people, a sharp coronal-to-sagittal transition in zygapophyseal joint orientation occurs in the lower thoracic region as it begins to interface with the lumbar vertebrae, and that asymmetry of paired zygapophyseal joints—articular tropism—is marked at the T11–T12 levels [21]. Moreover, Cyron and Hutton [22] observed that torsional stress within a vertebral segment is greatest where the zygapophyseal joints are oriented close to the sagittal plane. It is possible, then, that the greater incidence of SNs reported in the thoracic-lumbar transitional region is significantly influenced by the unique mechanical and anatomical characteristics of this region, which demonstrates increased susceptibility to axial and torsional forces.

Disc degeneration

Studies have shown that intervertebral disc (IVD) degeneration is very common, occurring in about 50 % of people over the age of 40 years and in up to 85 % over the age of 60 [23, 24]. Lumbar disc degeneration has also been shown to be a major cause of lower back pain [25]. Hence, disc degeneration leading to SN formation is a possibility. Williams et al. [3] found that SNs were highly correlated to lumbar disc degeneration and back pain, although they were not themselves independent predictors of back pain. It is important to note, though, that the formation of a SN does not automatically imply the presence of pain. It is possible for a SN to form without associated pain [8, 9]. Hence, a degenerated disc can lead to the formation of asymptomatic SNs that may or may not become symptomatic and cause pain. Also, a degenerated (and therefore weakened) vertebral endplate is likely less resistant to axial forces, allowing disc herniation more so than an intact one.

Embryogenesis

During embryogenesis, the development of an individual vertebra begins with a Sonic hedgehog (Shh)-mediated induction by the notochord on the early somite to form the sclerotome. Under the influence of Shh, the ventromedial portion of the somite ultimately forms the centrum (body) of the vertebra. Interestingly, the notochord disappears from the bodies of the vertebrae before embryogenesis is complete, expanding into the IVDs, and persisting as the NP [26]. These events are highly regulated genetic processes, and disruption can lead to malformation of the vertebrae, IVDs, and associated structures. Proponents of the embryogenic theory assert that SNs form due to a developmental insult which results in a gap in the developing vertebrae, leaving an indentation in the bone into which disc material can herniate [5]. Such indentations can conceivably be caused by abnormal regression of the notochord, incidental ossification gaps in the centrum of the vertebrae, vascular channels, or even Scheuermann's disease [15].

Pathologic processes

In this view, various pathologies involving the spine may weaken the IVDs and vertebral bodies, allowing SNs to form [12]. Identified candidates include osteomalacia, hyperparathyroidism, Paget's disease, infections, neoplasm, and osteoporosis [15].

Autoimmune involvement

Zhang et al. [8] postulate that the immune system may play a key role in the development of symptomatic SNs. Although less likely to directly cause SNs independently, an immune reaction to herniated NP via one of the aforementioned pathogenic pathways might exacerbate symptoms. They contend that a disc that herniates into the vertebral endplate and eventually the bone marrow could be considered as "non-self" tissue once in contact with blood. This could then incite an immune reaction to the herniated material. The authors point out that IVDs are the largest avascular structure in the body and, hence, could be recognized as foreign matter when met by a well-vascularized source, such as a vertebral body. Such an event could lead to an immune reaction, edema, an influx of cytokines, and pain.

This suggested role of the immune system in symptomatic SN formation is supported by an MRI study by Takahashi et al. [9]. The authors compared the MRI

findings of 5 symptomatic SN cases to 11 asymptomatic cases and reported that all 5 symptomatic SNs were seen as low-intensity lesions on T1-weighted MRI, but high-intensity on T2-weighted images, most likely due to the presence of inflammation. These findings of hyperintense signal on T2 images were absent in all 11 asymptomatic SNs. Histologic examination of bone marrow from two symptomatic SNs cases showed the evidence of inflammatory cell infiltration and bone marrow edema in the vicinity of the SNs. The authors theorized that the pain generated by symptomatic SNs originated from nociceptors located in these edematous rings observed in the symptomatic SNs, but not in asymptomatic lesions [9]. Conceivably then, resolution of the inflammation would lead to conversion of a symptomatic lesion to an asymptomatic one [9, 27]. This transformation has indeed been observed [28]. Conversely, it is possible that a previously asymptomatic SN can induce pain symptoms if the NP continues to herniate deeper into the vertebral marrow over time. If contact is made with the bloodstream, an immune response could be initiated, causing pain and discomfort as well as further damage to the disc and vertebral body.

Further evidence for the possibility of key interactions between the immune system and bone dynamics is noted in their shared reliance on the activity of cytokines [8]. Hence, cross-talk between a dysregulated immune system and the processes of bone formation and resorption is a conceivable mechanism for sequelae after initial SN formation [29]. In other words, an improperly activated immune response, due to NP herniation into the vertebrae, can potentially result in an imbalance in bone resorption and deposition leading to bone loss. Bone loss may predispose affected vertebrae to herniation of more disc material, exacerbating the condition. Furthermore, it has been shown that aged SNs can become encased in calcified material [28]. This may contribute to the development of symptomatic SNs, perhaps due to compression of nearby nociceptive nerve endings.

Imaging

The best imaging modality for detecting SNs is MRI [9]. Although plain film radiographs can detect these lesions, they are mostly useful in the later stages of the lesion when some calcification around the SNs has occurred [28]. MRI is the gold standard, however, in part because it can detect acute SN lesions. This could translate to earlier diagnosis and prompt management of symptomatic lesions. In addition, MRI has been shown to be capable of distinguishing between symptomatic and asymptomatic SNs by accentuation of concomitant edema in T2-weighted images and low signal intensity in T1-weighted images in virtually all symptomatic cases, a distinction plain radiographs cannot make [9].

Management of symptomatic lesions

Although frequently incidental and asymptomatic, certain SNs have been purported to cause lower back pain and thereby considerably impact quality of life [3, 9, 17]. A number of strategies to alleviate the pain associated with symptomatic SNs have been reported in the literature.

Fusion surgery

It has been reported by Peng et al. [30] that segmental fusion surgery is efficacious in alleviating severe lower back pain due to SNs. The authors recruited 21 patients with painful SNs with concordance confirmed by discography. The authors performed an anterior intervertebral body fusion for painful SNs located in the anterior or central endplate. Posterior intervertebral body fusion was performed for those with painful SNs located in posterior margins of the affected vertebral body. Of the 21 cases, 11 underwent posterior lumbar disc excision, pedicle screw system internal fixation, and posterior lumbar interbody fusion (PLIF) operations. In three cases, posterolateral fusion operations were performed after lumbar disc excision and pedicle screw system internal fixation. The remaining seven cases underwent anterior disc excision and anterior lumbar interbody fusion (ALIF). Regarding fusion rates, 10 of the 11 cases that received PLIFs achieved complete fusion, while all three cases that underwent posterolateral fusion operations achieved complete fusion for an overall fusion rate of 91 %. Among the seven cases that received the anterior approach, one failed to fuse; hence, the fusion rate was 86 %. Of the 14 cases that underwent PLIF or posterolateral fusion and pedicle screw system internal fixation, low back pain resolved in all but 2 cases (including the case of pseudarthrosis) following the operation. Among those receiving anterior disc excision and ALIF, the authors reported that back pain disappeared in all but one case (the non-fused case). The authors reported that preoperative VAS scores ranged from 5.3 to 9.1, with an average of 7.15. In contrast, postoperative VAS scores ranged from 0 to 5.0, with an average of 1.64. The difference between the pre- and postoperative VAS scores was significant, with a p value <0.01. Hasegawa et al. [31] reported successful pain relief in a woman with an 8-year history of unexplained back pain due to SNs who underwent fusion surgery.

Percutaneous fluoroscopy-assisted vertebroplasty

Wenger and Markwalder [27] reported on the use of percutaneous vertebroplasty to alleviate the pain caused by symptomatic SNs. In their report of a 31-year-old man

with a 10-year history of back pain unresponsive to conservative treatments (NSAIDs, corticosteroids), the authors made the diagnosis of symptomatic SN by exclusion and by the presence of an edematous SN at the L4 vertebra on MRI. The patient was deemed a candidate for vertebroplasty after responding to restrictive treatment with a rigid brace. This improvement was attributed to a lack of mechanical stress on nociceptors located within the edematous ring around the SN. Under fluoroscopy, vertebroplasty was performed by injecting polymethyl methacrylate cement into the edematous ring, taking care to avoid the node itself. At 18-month follow-up, this intervention resulted in the reduction of the patient's pain, as assessed by VAS, from a 7 before the surgery to a 4–5.

Tumor necrosis factor-alpha (TNF-α) blockade

As discussed, there is evidence that the immune system, specifically the inflammatory response, plays a key role in symptomatic SNs [8, 9]. The role of TNF-α in animal models of sciatica has been documented [32]. Furthermore, Olmarker and Rydevik [33] reported the efficacy of TNF-α in preventing NP-induced functional and structural nerve root injury in animal models. Subsequently, it has been demonstrated that the TNF-α inhibitor, infliximab, is efficacious in alleviating leg and back pain due to sciatica [34]. Sakellariou et al. [17] specifically tested infliximab on two patients with severely painful SNs that were symptomatic for at least 18 months. The treatment regimen consisted of infusion of infliximab at 3 mg/kg at weeks 0, 2, 6, and 14. The first patient had a VAS score of 9 out of 10 prior to infliximab infusion and showed immediate response, with the VAS score dropping to 7 within 24 h. Her pain completely resolved after the second infusion, but treatment was discontinued due to an allergic reaction. It is reported that the patient remained asymptomatic for 30 months. The second patient, who had an initial VAS score of 8, received all 4 infusions and also responded promptly to the first infusion. This patient's VAS scores ranged between 1.5 and 2 at weeks 2, 6, and 14. Remarkably, Seymour et al. [28] showed a correlation between improvement in back pain due to SNs and reduction in bone marrow edema. These findings underscore the likely role of the inflammatory response in symptomatic SNs and the potential therapeutic benefits of inflammation control in patients with symptomatic SNs.

To our knowledge, no systematic studies of the efficacy of common anti-inflammatory agents (such as NSAIDs) in the treatment of SNs have been reported in the literature. However, they are often administered as conservative treatment for SNs before other more invasive methods are considered [9, 35].

Rami communicans nerve block

Jang et al. [15] reported the use of rami communicans nerve block to alleviate the symptoms of SNs. The rationale for this treatment stemmed from the known distribution of nerve endings around the IVDs and vertebral bodies. These structures have been shown to be innervated by two extensive microscopic nervous plexuses that run along the anterior and posterior longitudinal ligaments. It is thought that the anterior and posterior plexuses are connected via a lateral plexus formed by branches of the gray rami communicans. The entire circumference of the vertebral bodies and IVDs is thought to be innervated by branches from these nervous plexuses [36]. Jang and colleagues [15] performed a nerve block on the gray ramus communicans at the L4 level where the SN was located. The nerve block was performed by injecting 2 mL of 1 % mepivacaine and 10 mg of triamcinolone on each side at the L4 level. The treatment was administered once a week for 2 consecutive weeks. It is reported that the patient's pain improved immediately after the nerve block from 9 to 2 on the 10-point VAS. One month later, the patient still had a score of 2.

Discussion

It is important for spine surgeons to be aware of the relative prevalence of SNs. Although most lesions appear to be asymptomatic, certain SNs can be symptomatic, causing back pain. It is known that next to upper respiratory tract infections, lower back pain is the most frequently diagnosed condition among all patients visiting the hospital, and that a remarkable 85 % of them are due to unexplained causes [37, 38]. As reported by Williams et al. [3], multiple SNs are significantly associated with lumbar disc disease. Hence, it is possible that SNs may account for some of these unexplained cases of back pain, given that they are often considered to be incidental findings. It is, therefore, important for spine surgeons to appreciate the relationship between lower back pain and SNs, as they can be the source of significant lower back pain requiring surgical intervention when conservative methods fail [9].

The pathogenesis of SNs is still a subject of debate. Given the evidence discussed in this manuscript, an axial load model for the development of SNs is more likely to be independently capable of causing the development of SNs in the majority of cases. It has been reported that axial forces may be capable of soliciting sufficient counteracting turgor pressure within the NP that can act on the cartilaginous endplate, causing deformity and herniation [39].

The evidence in support of an axial trauma model is quite robust. However, certain preexisting conditions could also facilitate the ease with which herniation occurs due to

axial forces. Hence, a combination of the models can be likely explanations for a particular presentation of SN. For example, a developmental insult can result in an indentation in the vertebral endplate into which NP herniation can readily occur if sufficient axial force is experienced or accumulated over time. Similar arguments can be made in favor of the pathological and degenerative models.

Hilton et al. [5] proposed a strictly developmental pathogenesis for SNs. If this was true, one may expect to find SNs equally distributed in all regions of the vertebrae and not so highly localized to the thoracolumbar spine. In their study of postmortem thoracolumbar spines, they found no relationship between age and SNs. They, therefore, rejected a degenerative disease model and proposed a developmental/embryogenic model, arguing that SNs are already present during skeletal maturation; hence, the lack of a difference between specimens from their subject pool over and under the age of 50 years. To explain why SNs are not equally distributed in the spine, Hilton and colleagues [5] proposed that the thoracolumbar spine is under greater stress than other regions of the vertebral column, and, therefore, is predisposed to SN formation caused by these developmental insults.

To the contrary, Vernon-Roberts et al. [10] found SNs to be more prevalent in older spines (over 30 years) than younger ones, supporting the degenerative model. It is possible that the 20-year gap between the cut-off ages used in these studies affected the outcomes. Further studies are necessary to determine the point at which this discrepancy in SNs between old and young spines occurs, if at all.

A number of strategies to alleviate the symptoms of SNs have been reported in the literature. The quality of evidence for each of these treatment options is low, with no randomized studies. Of the four treatment modalities presented in this paper, the intervention with the strongest evidence of efficacy and utility to the patient was fusion surgery. Of the 21 cases of painful SNs reported by Peng et al. [30], fusion surgery was reported to completely attenuate the pain due to SNs in all but three of their patients, who also had a reduction but incomplete relief of pain. Hasegawa et al. [31] reported similar results in a case report. Fusion surgery was the only treatment modality found in the literature in which complete disappearance of pain was reported in a substantial majority of cases. Nevertheless, these results should be viewed in the context that the studies involved a small number of patients without a comparison group.

Conclusions

In summary, SNs are common lesions seen primarily in the thoracolumbar spine that are often asymptomatic, but in certain cases can be a source of back pain. A number of theories addressing their pathogenesis have been proposed, but no consensus currently exists. Painful or symptomatic SNs can lead to a significant decrease in quality of life. Currently there is no established treatment modality. Future investigations should address interventions for treating symptomatic SNs.

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