Letter to the Editor

Schmorl's node of primarily developmental cause and Schmorl's node of primarily acquired cause: two related yet different entities

Yì Xiáng J. Wáng^

Department of Imaging and Interventional Radiology, Faculty of Medicine, The Chinese University of Hong Kong, Shatin, New Territories, Hong Kong SAR, China

Correspondence to: Dr. Yì Xiáng J. Wáng. Department of Imaging and Interventional Radiology, The Chinese University of Hong Kong, Shatin, New Territories, Hong Kong SAR, China. Email: yixiang_wang@cuhk.edu.hk.

Keywords: Spine; Schmorl's node; radiograph; developmental cause; acquired cause

Submitted Mar 01, 2023. Accepted for publication Apr 04, 2023. Published online Apr 12, 2023.
doi: 10.21037/qims-23-252
View this article at: https://dx.doi.org/10.21037/qims-23-252

According to the original definition, Schmorl's node (SN) corresponds histologically to nucleus pulposus herniation into the vertebral spongy bone with thickened trabeculae around the formed node (1,2). Very conflicting literature on the epidemiology of SN have been reported. Several earlier reports noted that, except for the early childhood period, SN prevalence among age groups remains stable (3-6). Dar *et al.* (4) studied skeleton vertebrae from a normal adult population and noted that SN was more common in men than in women, but SN was age independent. Hilton *et al.* (5) found no relationship between age and SN with their study of cadaveric spines and thus proposed a developmental pathogenesis for SN. Chen *et al.* (7) reported that aging was related to greater odds of endplate lesions; however, the prevalence of focal endplate defects remained stable. Sonne-Holm *et al.* (6) analysed lateral spine radiographs in an adult Caucasian population cohort and did not note any significant correlation between SN and gender, or age. On the other hand, some authors reported the association of SN with age. Wang *et al.* (8) reported that greater age was associated with the presence of SN among a male cadaver collection. A male predominance of SN had been frequently reported (5,9-11). Üstündağ (11) studied SN in a post-medieval skeletal sample and noted that males were more affected than females, and there was no relationship found between SN and aging. However, a few studies did not observe an association of SN with gender (6,12,13). The correlation between osteoporosis and SN also remains controversial (14-16). In a study on the cadavers of pre-Hispanic inhabitants González-Reimers *et al.* (14) did not find a relationship between osteoporosis and SN. On the other hand, other evidence suggests osteoporosis is a cause of SN. Mäkitie *et al.* (15) reported a high prevalence (61%) of SN in their case-control study of 18 patients diagnosed with WNT mutation-induced osteoporosis. Based on a CT study, Güngör *et al.* (16) suggested that low bone mineral density may be a predisposing factor for the development of SN in patients younger than 40 years. Recently we (17) described a study of thoracic spine MR imaging among community elderly subjects (mean age: 82 years) and noted a number of features of SN paralleled those of osteoporotic vertebral fracture (OVF). SN prevalence in women (55.5%) almost doubled that in men (25.9%). SN was statistically significantly correlated with lower bone mineral density, and subjects with SN were more likely to have OVF. In vertebrae with osteoporosis, the endplate becomes weakened due to the loss of support from trabecular bone and due to thinning of the endplate itself (18), thus this pathway may exist that: osteoporosis → weakened endplate → SN development → osteoporotic endplate fracture (17).

To make sense of these conflicting literature, we suggest that SNs should be classified into two categories: SN of

^ ORCID: 0000-0001-5697-0717.

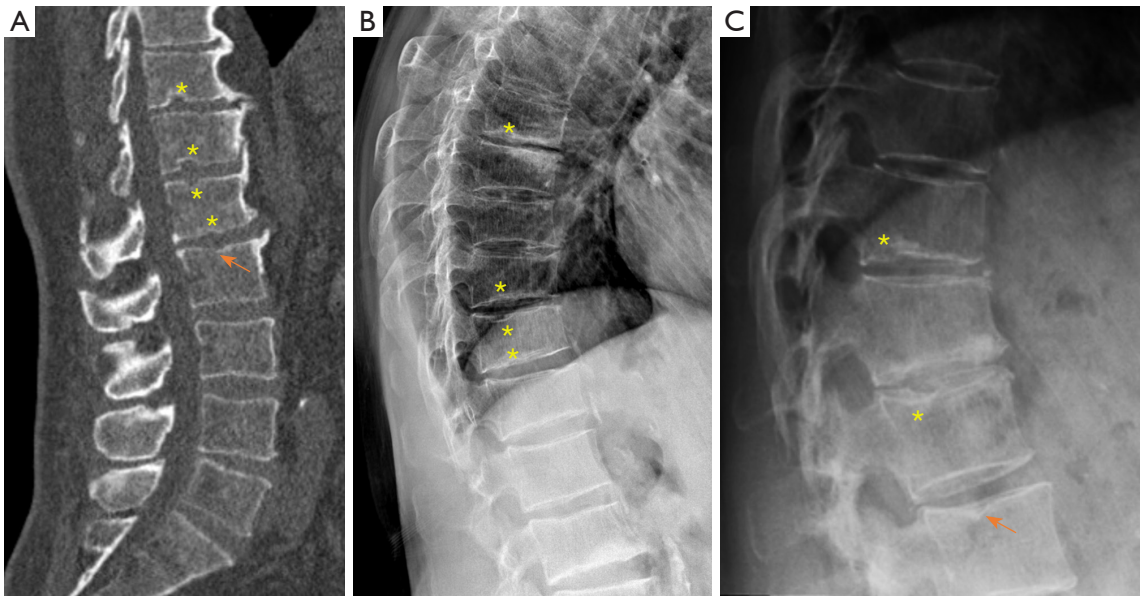

Figure 1 Image examples of SNd. (A) Reconstructed CT image. (B) Radiograph. Multiple acquired short vertebrae are noted. However, a few SNd might have been missed due to the projectional nature of radiograph. (C) Radiograph. * indicates SNd, arrow indicates a possible SNd. SNd, Schmorl's node of primary developmental cause; CT, computed tomography.

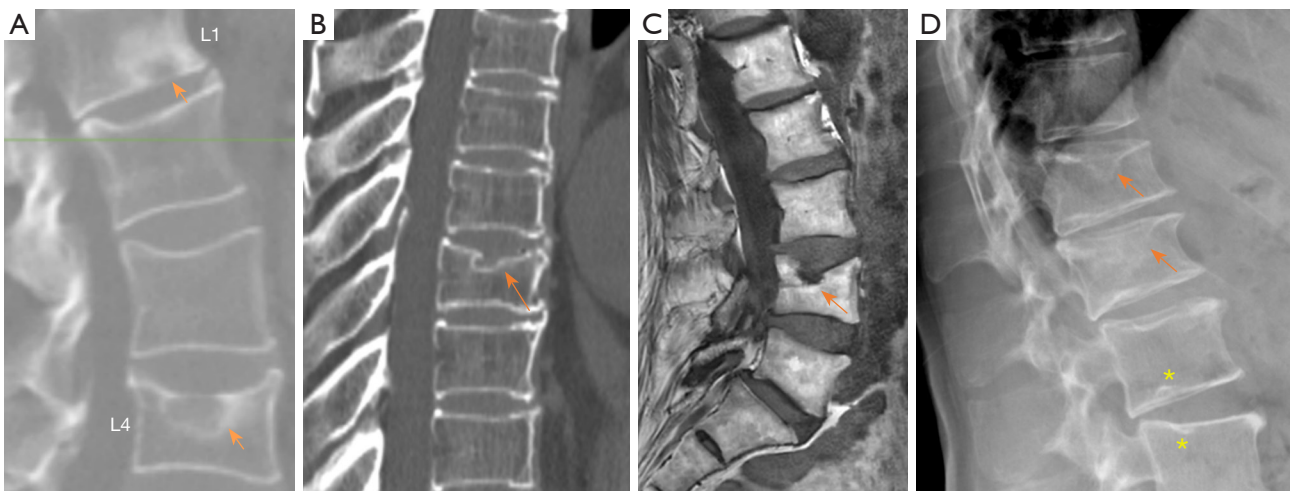

Figure 2 Image examples of SNa. (A) reconstructed CT image. L1 lower endplate assumed SNa (arrow) with apparent reactive bone changes, and L4 upper endplate SNa (arrow) and depression. (B) Reconstructed CT image. Arrow indicates upper endplate SNa (arrow) and depression. (C) MR image, L4 OVF with apparent upper endplate depression and a SNa (arrow). (D) Radiograph. L1 upper endplate SNa together with compressive vertebral deformity. L2 upper endplate SNa together with apparent endplate fracture. *, Schmorl's node of developmental cause. (A) reproduced with permission from (17). (B,C) modified with permission from (19). SNa, Schmorl's node of primarily acquired cause; CT, computed tomography; OVF, osteoporotic vertebral fracture.

primarily developmental cause (SNd, *Figure 1*) and SN of primarily acquired cause (SNa, *Figure 2*). Note that, SN can be considered as a 'general phenomenon' (rather than a specific disease entity) where a portion of disc materials herniated through endplate into the vertebral spongy bone, and the surrounding sclerosis in vertebra reflects

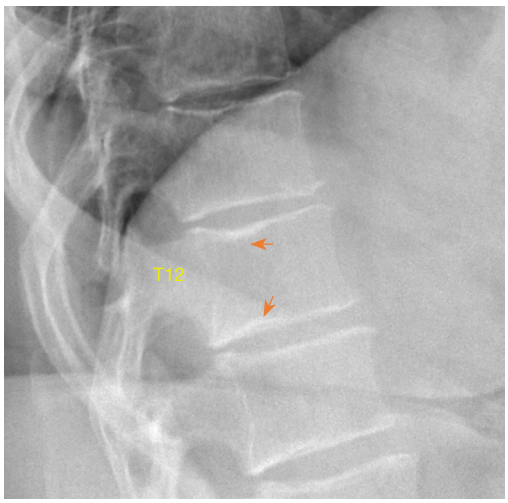

Figure 3 Radiograph shows depression of posterior parts of vertebra T12 upper and lower endplates (arrows) and associated sclerosis. These changes may be a small version of SNd or a small version of cupid bow change. SNd, Schmorl's node of developmental cause.

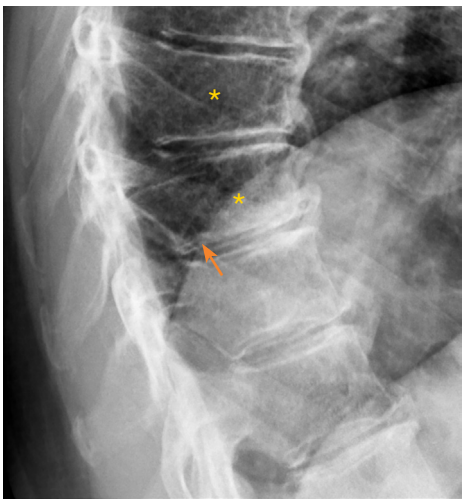

Figure 4 Radiograph shows a very small SNd (arrow). Note that this is a typical location of SNd. *, both vertebrae have minimal grade osteoporotic-like vertebral deformity. SNd, Schmorl's node of developmental cause.

reactive healing. An analogy can be made to congenital spondylolisthesis, traumatic spondylolisthesis, and degenerative spondylolisthesis (20).

SNd is characterized by that they more likely involve multiple adjacent vertebrae, more likely to be small or modest in size, they likely to have relatively consistent location and more likely involve a posterior portion of the lower endplate (though other locations are also common). Moreover, compared with SNa, SNd tend to have a more solid border on radiograph due to their longstanding and sometimes static nature. It is also possible some SNd and cupid bow may belong to the same spectrum of developmental changes (*Figure 3*). Note that Pfirrmann and Resnick described cases showing '*the transition of Schmorl nodes to a cupid's bow contour*' (21). SNd are known to be a common but not obligate manifestation of Scheuermann disease. SN associated with Scheuermann disease shall belong to SNd. It is also possible that some very 'tiny' SNd may not have clinical relevance (*Figure 4*), and some SNd may not have a true disc material herniation process. Heritability of SN has also been demonstrated (15,22,23). Though the upper endplate is less resistant to compressive pressure and more likely to fracture (24), in a female twin volunteer study (mean: 53 years), for their study subjects Williams *et al.* (22) noted that SNs (assumed largely SNd) were more prevalent in lower endplate than in upper endplate.

A vertebral endplate consists of perforated cortical bone with a layer of hyaline cartilage bonded to its disc surface. The cortical bone layer contains a network of small cavities which allow bone marrow to lie adjacent to calcified hyaline cartilage for approximately 10% of the central endplate area, which is an important route for metabolite transport into the discs. The nutritional demands of the discs result in that vertebral central endplates are thin and porous, and which can be subject to fracture under stress force even if bone strength is normal. For the pathogenesis of SNa, whatever the cause of the damage to the cartilaginous endplate, to the subchondral bone of the vertebral body, or to both structures, a weakened area is created that is unable to resist the expansive pressure of the adjacent nucleus pulposus. SNa are more likely to be modest or large in size, and their borders are not always clearly defined on radiograph. Trauma and endplate micro-fractures are triggers for SN (25-29). Dar *et al.* (25) proposed an axial load model which suggests that the human spine must accommodate increased axial forces in addition to balancing the need for spinal mobility and stability, and it may accumulate micro-traumas that can, over time, lead to the formation of SN. In a cohort of children who had suffered from stable compressive vertebral fractures, Möller *et al.* (26) reported the occurrence of SN at advanced ages (40 years) at adjacent

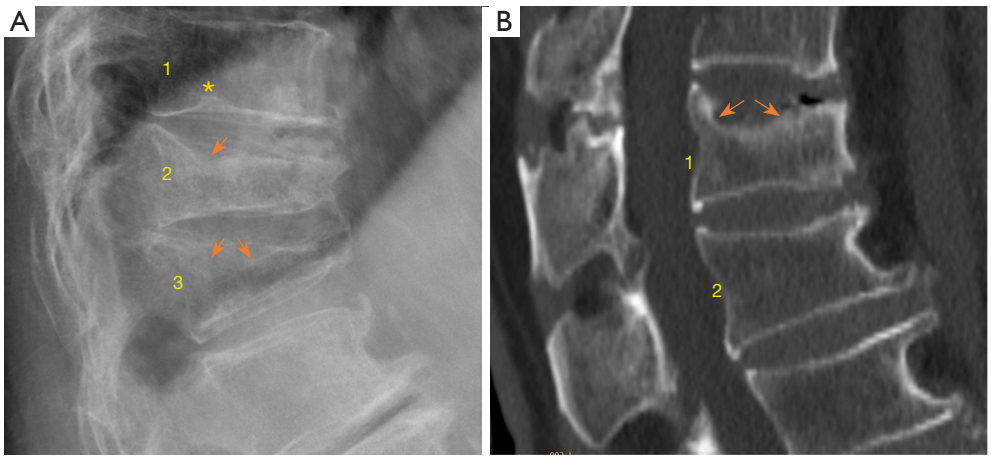

Figure 5 Schmorl's nodes associated with advanced osteoporotic vertebral fracture. (A) Vertebra-2 and vertebra-3 have collapse grade (>2/3 vertebral height loss) osteoporotic vertebral fracture. Arrows indicate Schmorl's nodes. The height of vertebra-1 is reduced likely also due to osteoporotic compression. *, a Schmorl's node of assumed acquired cause (its border does not appear to be as solid as those of developmental cause). (B) Vertebra-1 and vertebra-2 have osteoporotic vertebral fracture. Arrows in vertebra-1 indicate a Schmorl's node of assumed osteoporotic cause.

disc levels. In a study of 70 thoracolumbar spines from cadavers of individuals killed in motor vehicle collisions, Fahey *et al.* (27) reported a link between trauma and the occurrence of SN. Swärd *et al.* (28) compared vertebral abnormalities in elite gymnasts versus non-athletes, they found SNs in 17 out of 24 (71%) gymnasts with nodes in 57 endplates and in 7 out of 17 (44%) non-athletes with nodes in 23 endplates. Certain pre-existing conditions can facilitate herniation occurs due to axial forces. The theory of weak spot presence within vertebral endplate has been considered. The possible endplate weakness can be due notochord regression, ossification gaps, or vascular channels (2,30,31). The vertebral level distribution of SNa will be similar to traumatic vertebral fracture (high energy trauma) or OVF (low energy trauma) (19,32). SNa less likely involve posterior portion of an endplate which is not a weak point of biomechanics. Among elderly subjects, SNa more likely involve upper endplate which is the same as osteoporotic endplate fracture (24), and SNa are commonly associated with endplate depression. Osteopenic/osteoporotic SN may be a precursor of OVF, a specific type of endplate fracture, or a co-phenomenon for advanced OVF (*Figure 5*). Tumorous changes of a vertebra can also increase the fragility of an endplate, and lead to disc materials herniation through endplate into the vertebral spongy bone and form SNa (33,34). In these cases, the surrounding sclerosis may not necessarily develop when the SNa is detected.

Some earlier authors already discussed SN sub-classifications. Hansson and Roos (35) classified the SNs situated just above or below the nucleus pulposus, symmetrically on both sides of the nucleus with well-rounded smooth bone surroundings as type A nodes. The SNs which are situated asymmetrically in relation to the nucleus and/or surrounded by rough often sclerotic bone and were classified into type B. However, according to our reading of the literature, how our classification can correspond to the classification of Hansson and Roos remains unclear. On the other hand, Hansson and Roos did note that some SNs were associated with lower vertebral bone strength while others were not.

We advocate that, for future studies, SNd and SNa should be separately described as much as possible, as SNd and SNa may have different clinical significance. Small SNd may not have clinical relevance, and some SNd may be well covered with the endplate. SNa are usually associated with endplate fracture, and some SNa may be an indicator of compromised vertebral bone strength (17). On the other hand, the physiopathology of SN formation can be multifactorial; and for some SNs, a definite separation of SNd and SNa may not always be possible. More research is required to elucidate the classification of SNs and their relationship to developmental causes or degenerative causes.

Acknowledgments

Funding: None.

Footnote

Provenance and Peer Review: This article was a standard submission to the journal. The article has undergone external peer review.

Conflicts of Interest: The author has completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-23-252/coif). YXJW serves as the Editor-in-Chief of *Quantitative Imaging in Medicine and Surgery*. The author has no other conflicts of interest to declare.

Ethical Statement: The author is accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Cite this article as: Wáng YXJ. Schmorl's node of primarily developmental cause and Schmorl's node of primarily acquired cause: two related yet different entities. Quant Imaging Med Surg 2023;13(6):4044-4049. doi: 10.21037/qims-23-252

