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Historical Schmorl's Node: Time to Differentiation and Renaming (A Mini-Review)

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Abstract

"Schmorl's nodes" (SN) are lytic lesions at endplates which are believed to be due to the herniation of the nucleus pulposus through the cartilaginous endplate into the body of a vertebral endplates. Our observations disclose the fact that all Schmorl's nodes are not single entities and a subset of SNs seems to be a discrete clinical condition with similar (but not identical) imaging manifestations. They share many features of seronegative spondyloarthritis. We called them "Owlia's nodes".

Keywords: Schmorl's nodes; Back pain; Inflammation; Herniation; Spondyloarthritis; Seronegative spondyloarthropathy; Owlia's node

Introduction

Schmorl's nodes" (SN) are destructive lesions at endplates which are believed to be due to the herniation of the nucleus pulposus through the cartilaginous endplate into the body of a vertebra [1]. Schmorl first described them in 1927. Recent observations indicate two different entities with traditionally single name: degenerative and inflammatory lesions. Most clinicians assume that SNs are accidental finding in spine imaging and of no clinical significance. In some reviews SNs are divided into asymptomatic and symptomatic SNs.

Based on our findings, Inflammatory SNs are clearly distinguishable from classic degenerative Schmorl's nodes frequently seen in spine MRIs as a co-incidental finding (Figure 1).

Epidemiology

Schmorl's nodes have been noted in 10-38% of all spine studies, with a slightly higher incidence in males. Some researchers proposed a positive association with increasing age, while others argue that age could not be a significant factor. Heredity may play an important role [1]. Hilton et al. found an incidence of 75%, with a higher frequency in the thoracolumbar region than in the mid and lower lumbar spines [2]. This regional difference has been noted by others. Studies have been restricted to the thoracic and lumbar spine and reports seldom indicates to Schmorl's nodes in the cervical spine. These nodes have been identified with increased frequency in athletic adolescents. With regard to the prevalence of SNs, cadaver studies vary in their estimates, ranging from 38% to as high as 79% [1].

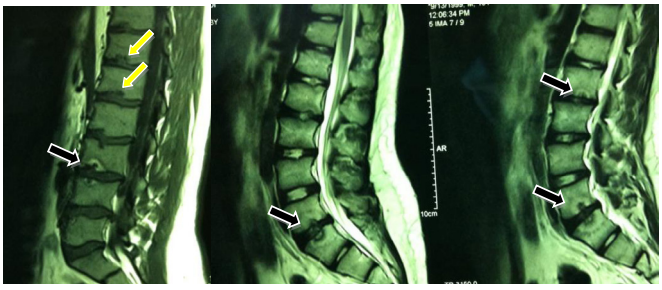

Figure 1: Regular classic Schmorl's node (yellow arrows) in comparison with newly described inflammatory Schmorl's node (black arrows) with typical active kissing lesions demonstrated on MRI.

Pathogenesis

Developmental factors, degenerative mechanisms, infection, neoplasia, metabolic derangement and trauma have been proposed [3]. But none of them have been critically evaluated.

Some researchers categorize SNs as a developmental disease, while others see SNs as a degenerative bone disease. Some researchers theorize that SNs are a result of pathologic processes that involve discovertebral complex [1]. Direct trauma to the vertebra has also been implicated as a risk factor in developing SNs. The role of inflammatory processes is less addressed in respect to clinical settings. Edema in the bone marrow was observed and terminology of edematous SN is sometimes used [4]. Inflammatory process or a possible autoimmunity may increase 18-fluorodeoxyglucose (*FDG*) activity in PET study in the Schmorl's node in a subset of symptomatic SNs [3].

Schmorl's nodes may be the consequence of ischemic necrosis beneath the cartilaginous endplate and that herniation into the body of the vertebra could be a secondary epiphenomenon. The topographic distribution of Schmorl's node varies case by case. In cases with no anatomic abnormality, or disc alteration, they are most frequent in the lower thoracic and upper lumbar areas and are less frequent in the middle and lower lumbar vertebra. Occurrence of nodes at the thoracolumbar junction is more prevalent when marked variation in orientation of the articular facets is observed. These types of SNs usually involve the lower end plates and are more common in men than women, especially in younger age groups. In cases in which Schmorl's nodes are a manifestation of other disease processes, their distribution depends on the site of spinal involvement by the primary process.

Neoplasms do not invade intervertebral disc, although osseous lesions with disruption of the cartilaginous endplate and subchondral bone plate may lead to cartilaginous node formation with disc space loss and thereby simulate SNs.

If endplate spondylitis be the culprit mechanism for so-called

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SN, then similar radiologic features are against the nature of classic SNs. So differentiation of real SNs from the other entity of local early spondylitis is crucial in clinical and imaging standpoint. "Kissing lesions" are frequently seen in case of SNs. This phenomenon implies to an inflammatory nidus hurting both sides of vertebral end plates rather than a focal pressure point causing herniation of disc material to the end plates [5]. This seems to be true for bilateral damage in fibrocartilaginous joints of sacroiliac joint and symphysis pubis [6]. This unique key event can potentiate the similar mechanism for SN, sacroiliitis and osteitis pubis as different neglected futures of seronegative spondyloarthritis (Figure 2).

Clinical Manifestation

Schmorl's nodes are frequently asymptomatic or at least most physicians think so. The most frequent reason for seeking an imaging study on spinal column is pain. Subtle manifestations of connective tissue diseases are frequently overlooked by physicians [7]. Our observations revealed most of these patients suffer from inflammatory back or low back pain which decreased with regular daily activities and accentuates during or after rest. Nocturnal pain was not rare in our series. Kyphosis may be the other manifestation of underlying pathology in cases of SNs. Pathologic fracture and deformity other than kyphosis is rare. Neurologic manifestations secondary to extrusion of intervertebral disc originated from atypical posterior SNs may be seen in Scheuermann's disease [8].

Due to subtle clinical manifestations of seronegative spondyloarthritis (SpA) in early stages, any correlations between inflammatory spinal pain and inflammatory Schmorl's node (as shown on MRI) could be taken into consideration.

Imaging Evaluation

The radiographic appearance of Schmorl's nodes is similar in respect to the specific cause. The radiological manifestation of Schmorl's nodes is an endplate indentation with an indistinct sclerotic marginal line [9]. In the lateral projection, the vertebral bodies are rather rectangular in shape.

Leakage of radiopaque contrast medium from the disc space during discography is observed limited to superficial layers of the cartilaginous endplate indentation. On CT scans SNs are appeared as a round or oval irregular area of bone density with a sclerotic circumferential margin beneath the cartilaginous endplate. These findings are similar to that of osteonecrosis.

Histological examination of the nodes revealed that the round or multi-cystic irregular areas beneath the endplate, which are zones of osteonecrosis, separate from the herniated nucleus pulposus as described by Schmorl.

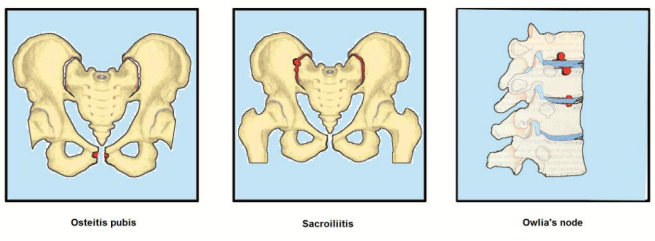

Figure 2: Kissing lesions, symphysis pubis (osteitis pubis), sacroiliac joint (sacroiliitis), and vertebrae (spondylodiscitis).

A radiolucent lesion surrounded by "helmet-shaped" sclerosis is observed. In the presence of intervertebral chondrosis, a specific feature of Schmorl's node would be degenerative clefts and collapse of the nucleus pulposus which cause vacuum phenomenon and considerable disc space narrowing; the displaced disc material may not extend deeply into the vertebral body.

MRI is a useful tool in discriminating symptomatic from asymptomatic SNs [10]. Takahashi et al. [11] analysed MRI findings in both groups. In symptomatic patients, the vertebral body marrow surrounding the Schmorl's node showed high signal intensity on T2-weighted images. This indicated the presence of inflammation and edema in the vertebral bone marrow which is less emphasized clinically so far [11]. These MRI findings were not present in asymptomatic individuals which suggested that Schmorl's nodes became asymptomatic when the inflammation subsided [9].

Considering similar MRI finding in early inflammatory spinal disorders, these types of SNs merit further attention and consideration as a separate entity. Our unpublished data showed that considerable number of patients with high signal lesions on MRI actually had inflammatory low back pain typical of seronegative spondyloarthropathies. Frequent cases of our series had tenderness on sacroiliac joints and entheses along with similar positive findings in pubic area (osteitis pubis) or frank radiographic sacroiliitis.

Pathology

The classic descriptions of Schmorl's nodes are of two basic components, disruption of the cartilaginous endplate and herniation of the gelatinous material of nucleus pulposus into the vertebral body. It may suggest osteonecrosis beneath the cartilaginous endplate as the basic event. Secondary herniation of the nucleus pulposus may or may not ensue.

The intervertebral discs may appear abnormal or degenerative. The corresponding cartilaginous endplates may appear thinner than normal, which deforms the endplate.

The cartilaginous endplate may then degenerate, with some fissuring and ulcerations. Microscopic necrosis may occur.

Intraosseous nuclear displacements occur through the weakened endplates, and the displaced disc material becomes surrounded at first by a cartilaginous and osseous material then after.

The cartilaginous nodes similar to a normal disc eventually evolve to consist of both nucleus pulposus and annulus fibrosus. Fibrovascular proliferation (similar to pannus pathology seen in classic rheumatologic conditions) and narrowing of the intervertebral disc can ensue.

This narrowing is maximal on the anterior aspect of the disc. The pressure on the vertebral bodies in this area may impede normal growth and produce kyphosis.

Healing of the disc alterations can result in ossification, with synostosis of vertebral bodies (similar to syndesmophytes seen in seronegative spondyloarthropathies).

The pathologic alterations in Scheuermann's disease involve the intervertebral discs and the vertebral bodies including the cartilaginous endplate the detailed structure of the vertebrae differs in the cervical, thoracic and lumbar regions.

Etiology

The exact cause(s) of SNs is currently unknown. Cartilaginous

nodes can accompany any disease process that weakens the cartilaginous endplate or subchondral bone of the vertebral body and allows intraosseous disc displacement. A partial list of such processes includes trauma, neoplasm, metabolic disorders (hyperparathyroidism, osteoporosis, and Paget's disease), infection, intervertebral osteochondrosis, and articular disorders (rheumatoid arthritis). Interestingly while seronegative spondyloarthropathies and/or frank ankylosing spondylitis are the major differential diagnosis in inflammatory spinal disorders, this condition is "the overlooked" item in literature. Accurate radiographic diagnosis of these conditions is based on additional spinal and extra spinal manifestations. The combination of kyphosis, cartilaginous nodes, and irregular vertebral endplates is actually pathognomonic for Scheuermann's disease [8].

Treatment

Most cases of SNs are asymptomatic which are found as accidental finding on imaging techniques need no specific treatment. Management options for symptomatic SNs vary, ranging from reassurance, medical management to surgical intervention. Most of symptomatic SN are in setting of seronegative spondyloarthritis and are managed as per other SpA. Physical therapy may be of some help in cases of Scheuermann's disease. In complicated SNs, percutaneous fluoroscopy-assisted vertebroplasty or segmental fusion is proposed to control severe pain due to SNs [1].

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 mostly of inflammatory type. 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 specific treatment for SNs and any therapeutic approach aims to the underlying clinical diagnosis. These signal abnormalities are corresponding to imaging finding seen in classic AS such as "shiny corner" or "Romanos sign". We propose a distinctive terminology for the different clinical, pathophysiological and imaging entity of "inflammatory Schmorl's nodes" implying to the first person who critically described it and correlated this distinct entity from clinical and imaging standpoint (Owlia's node).

Conclusion

All Schmorl's nodes are not single entities in clinical settings. A subset of SNs seems to be discrete clinical pathologies with similar (but not identical) imaging manifestations.

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