CASE REPORT

Symptomatic giant Schmorl’s node treated by a decompression procedure

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Summary Schmorl’s nodes (SN) are common lesions that represent disc material prolapse through the vertebral endplate, into the vertebral body with generally limited clinical significance. Rarely they have been related to the onset of back pain, usually self-limited. Recently giant variants of SN have been described, with very discrete clinical and imaging expression. Knowledge of the typical imaging appearance in both CT-scan and MRI may preclude the use of unnecessary diagnostic and treatment procedures. The authors present a case of symptomatic giant SN resembling these rare variants by its size and persistent pain, but also featuring distinct classic SN characteristics. Although not usually needed when the image is typical, a percutaneous biopsy was performed which allowed both the definitive diagnosis and, surprisingly, the almost immediate and complete resolution of the symptoms. Five years later the patient is pain free and a new MRI showed a significant decrease of the lesion size and no edema of the vertebral body.

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Introduction

Schmorl’s nodes (SN) represent disc material prolapse through the vertebral endplate, into the vertebral body. These are common lesions, usually incidental findings on imaging studies or autopsies, with limited clinical significance [1].

Recently, SN have been implicated in the context of symptomatic discovertebral junction disorders such as acute intrasosseous herniations [2,3], lumbar posterior extramarginal disc hernia [4,5], and transvertebral intraspinal tunneling causing radiculopathy [6].

Giant cystic or fatty variants have also been described as uncommon diagnostic challenges that differ significantly from the classic description of SN [7,8].

KEYWORDS
Spine; Disc disease; Schmorl’s Node; Surgical treatment

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The authors report a case of symptomatic giant SN where percutaneous biopsy has allowed both the definitive diagnosis and the resolution of the symptoms.

**Case Report**

A 40-year-old man is referenced to our consultation by his general practitioner due to mechanical back pain resistant to 4 months of pain medicine and physiotherapy. His visual analogue scale (VAS) was 7. There was no relevant personal or family traumatic, surgical, rheumatologic or neurological history. On physical examination, only paravertebral contracture was found.

Radiographic study revealed a radiolucent zone in the body of the twelfth thoracic vertebra (Fig. 1), which on CT-scan presented as a well-delimited lesion, with sclerotic borders occupying nearly three quarters of this vertebral body (Fig. 2). MRI showed a low intensity lesion, both on T1 and T2 weighted images, with peripheral high intensity on T2, in continuity with the inferior disc, and slight peripheral enhancement (Fig. 3).

In the absence of a definite diagnosis and in face of the persistent complaints, a percutaneous biopsy was performed, and revealed fibrocartilaginous fragments, similar to intervertebral disc, with no signs of a malignant or infectious process.

The patient improved significantly after this procedure, with pain diminishing to 2 on VAS, and 1 month after new physiotherapy sessions, he became asymptomatic.

We reviewed this patient 5 years later and he still has no back pain. A new MRI was performed showing significant decrease of the lesion size and no edema of the vertebral body (Fig. 4).

**Discussion**

SN, described in the early 1930s, represent a nucleus herniation through the endplate of the superior or inferior vertebra [9]. They are more frequent in men, their incidence increases with age and occur mainly in the low thoracic or high lumbar spine [9–11].

The etiology is not properly understood. It is accepted that weakening of the endplate facilitates the migration of disc material. This defect can occur during development (through the vascular channels, through the region of the

![Figure 1](image1.png) Lateral X-ray revealing a hiperlucent area (white arrow) with discrete borders on the body of T12.

![Figure 2](image2.png) Axial CT-scan: the lesion occupied nearly three quarters of the vertebral body and was well delimited with sclerotic borders. A cortical interruption was noted on the posterior wall.

![Figure 3](image3.png) MRI (T1, T2 and T1-gadolinium) showed a low intensity lesion, both on T1 and T2 weighted images, with peripheral high intensity on T2, in continuity with the inferior disc, and slight peripheral enhancement.

![Figure 4](image4.png) Five years later MRI (T2 and STIR) shows significant decrease of the lesion size and no edema of the vertebral body.
regressed chorda dorsalis, or through ossification gaps in the first and second decades of life), or may occur because of weakening of the cartilaginous endplate or the subchondral bone by Scheuermann’s disease, chondro dysplasias, different forms of osseous metabolic disorders, neoplasms, degenerative disc disease, or traumatic lesions caused by compressive loads [9, 12–15].

Besides development disorders, mechanical factors seem determinant in the process of SN formation. Expansive pressure leads to nucleus herniation through the weakened area of the endplate [16, 17]. This pressure diminishes with age, which accounts for the quicker formation of central nodes in younger people as opposed to the older where herniation occurs in the periphery and gradually, sometimes associated with transverse prolapse, because normal stresses are transferred primarily by the annulus in this population [1, 13, 18].

Also, the incidence of SN is higher in athletes [10, 19].

Although they are usually considered asymptomatic lesions, some authors have related them to the onset of back pain [9, 10, 13, 20–22]. The histologic origin of pain is not yet established. It has been suggested that in affected vertebral bone there is a higher proportion of disc marrow contacts and that herniation irritates an intravertebral nociceptive system, generating pain during spinal motion [23, 24]. In symptomatic patients the vertebral bone marrow surrounding the node is generally seen as low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, as is the case in the present report, and on histologic examination edema and acute and inflammatory cells can be observed [1, 22]. Currently MRI fat saturation sequence is a good way to identify inflammatory liquid, although at the time it was not performed. Most of the times this is a regressive or self-limited process [21, 22, 25].

Recently giant variants of SN have been described, with very discrete features [7, 8]. They can present as large cystic lesions, with central location, involving the superior endplate, usually in women and exclusively found in the lower lumbar spine. In other cases, their content is fatty, also with central location and involving the superior endplate, more commonly in men and in the lower lumbar spine. Discography may help reveal the communication between the lesion and the superior disc. However, if all the criteria are not present, other diagnoses have to be considered and a histopathological confirmation by biopsy is needed [8, 21]. Differential diagnosis can be challenging, especially for cystic lesions, and includes infection, cystic hemangioma, fibrous dysplasia, giant cell tumor, and chordoma.

Since pain disappears as the lesion stabilizes, only symptomatic treatment is usually required. Nevertheless, sometimes disabling pain may persist, and some authors have suggested a surgical intervention for its resolution [13, 20].

The present report resembles these giant variants by the dimensions of the lesion and persistence of symptoms. However, thoracolumbar localization, involvement of the inferior endplate, and disc material are common features of classic SN.

A closer reading of the image examination, especially the communication with the adjacent disc, present both in the CT-scan and MRI, would have precluded the need for a biopsy, but with no definite diagnosis and with persistent pain we decided for this procedure. Surprisingly, this exam promoted an almost immediate and complete relief of pain.

It is well established that mechanical factors play a major role in SN formation and in the pain they generate, as mentioned above. Although a placebo effect should not be put aside, we think that biopsy may have promoted a decompression of the lesion and was the probable cause for this outcome. Further research on the potential success of this minimally invasive procedure as an option in persistently symptomatic and disabling SN is warranted.

Hauger et al. have reported the stabilization of a giant SN 2 years after the diagnosis [8]. Five years later, our patient is pain free, the lesion decreased in size and no signs of bone marrow edema were found on MRI.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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