Symptomatic intraosseous Schmörl herniation

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ABSTRACT
Intraosseous disc herniation—or Schmörl nodes (SN)—are a herniation or prolapse of the nucleus pulposus of the intervertebral disc through the vertebral plate and into the adjacent vertebral body. They are usually associated with vertebral deformities, such as Scheuerman's disease, or rheumatic diseases, such as ankylopoietic spondylitis. In general, they are spontaneous and asymptomatic findings, and there are only a few reported cases of symptomatic nodes. The etiology is supposedly related to a weakened spinal plate due to trauma or repeated stress. When the node is acute or recent, it can be difficult to differentiate a benign degeneration from a malignant infiltration or infection.
In this paper, we discuss the unusual case of a painful Schmörl node in a man with no relevant history and a masked metastatic lumbar spinal tumor originated from pancreatic cancer. We performed a literature review.
Key words: Symptomatic; intraosseous; hernia; Schmörl.
Level of Evidence: IV

Hernia intraesponjosa de Schmörl sintomática

RESUMEN
Las hernias discales intraesponjosas vertebrales o nódulos de Schmörl consisten en una herniación o una extrusión del núcleo pulposo del disco intervertebral a través del platillo vertebral hacia el cuerpo adyacente. Suelen asociarse a deformidades vertebrales, como la enfermedad de Scheuermann, o a enfermedades reumáticas, como la espondilitis anquilopoyética. En general, son hallazgos casuales y cuadros asintomáticos; son pocos los casos publicados de nódulos sintomáticos. Su aparición estaría relacionada con un platillo vertebral debilitado por traumatismo o estrés repetido. Cuando el nódulo es agudo o reciente, puede ser difícil diferenciar la degeneración benigna de una infiltración maligna o una infección. Presentamos un caso inusual de un nódulo de Schmörl doloroso en un hombre sin antecedentes de relevancia, que enmascaró una lesión metastásica de carcinoma pancreático a nivel lumbar. Se realiza una revisión bibliográfica.
Palabras clave: Hernia; intraesponjosa; Schmörl; sintomática.
Nivel de Evidencia: IV

INTRODUCCIÓN

Intraosseous disc herniation is commonly referred to as Schmörl nodes (SN), which were first described in 1927 by pathologist Christian Georg Schmörl.[1]

SNs are a herniation or an extrusion of the intervertebral disc nucleus pulposus through the spinal plate into an adjacent vertebral body.[2] The herniated tissue may form a defect in the upper or lower surface of the involved vertebra. SNs tend to occur at the central or posterior region of the vertebral plate, and more commonly in the thoracolumbar junction.

A weakened vertebral plate due to trauma or repeated stress may probably be SN etiology.[4] This situation could be the result of intrinsic factors in the endplate (indentations, ossification zones, vascular channels, Scheuermann's disease) or acquired factors (infection, cancer, osteoporosis or osteomalacia, hyperparathyroidism, rheumatic diseases or Paget's disease).[4,5]

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However, such endplate weakening is not considered to be a necessary condition for prolapsing and is thought to be present as an underlying cause only in a small percentage of SN cases.

Most SN develop following an axial-loading trauma which causes the preferential prolapse of nuclear material through the weakened vertebral endplate, rather than through an intact annulus fibrosus.[6]

SNs are usually asymptomatic and incidental findings. There are only a few reported cases of symptomatic SN with acute onset of back pain associated with this injury.[5,7,8]

In this paper, we discuss the unusual case of a painful SN and perform a literature review on this subject.

CLINICAL CASE REPORT

A 66-year-old male college professor, with a personal history of high-blood pressure, hypercholesterolemia, and hypothyroidism, for which he is undergoing treatment. He arrives at our Department with a two-year low back pain complaint. He has previous X-rays and MRIs showing osteoarthritic degeneration, hypertrophy of facets, disc space narrowing at L5-S1 level, with no spondylolisthesis or disc deformity (Figure 1).

Figure 1. MRI sagittal section. Image exhibiting osteoarthritic degeneration, hypertrophy of facets, disc space narrowing at L5-S1 level, with no spondylolisthesis or disc deformity.

Se le diagnosticó síndrome facetario, y fue tratado inicialmente con fisioterapia, ejercicios y analgesia, sin mejoría, por lo que fue sometido a un bloqueo de facetas lumbares y el dolor mejoró.

A los seis meses del bloqueo facetario, acude a una consulta por dolor lumbar de dos meses de evolución, esta vez, irradiado hacia la cara posterior del muslo izquierdo, sin sobrepasar la rodilla, que se ha ido incrementando progresivamente con un puntaje de 6/10 según escala analógica visual (EAV).

He was diagnosed with Lumbar Facet Syndrome and showed no improvement with the initial physical therapy, exercise and painkillers; consequently, he underwent a lumbar facet block and the pain wore off.

Six months after the lumbar facet block, he refers a two-month low-back pain, however, this time, it spreads towards his posterior thigh surface, not reaching the knee, and, after progressively escalating, he has a visual analog scale (VAS) score of 6/10. On physical examination, the patient was slightly overweight (although he mentions having lost some weight during the last month), presented with spinous tenderness on lower lumbar segments, a good range of motion of the trunk, no sensory or motor deficit in the lower extremities. He requests a new lumbar facet block, given the results he experienced following the first block. Owing to the lapse of time since the previous block and the increase of pain intensity, a new lumbar MRI is ordered.

The following week, the patient brings the MRI results and indicates that his low-back pain has significantly increased. He has a VAS score of 9/10. In addition to the osteoarthritic degeneration present in the previous MRI, the new MRI demonstrates a fracture in L4 vertebral body, on the central region of its vertebral inferior plate as a result of a SN exhibiting a "mushroom cloud" shape and associated edema (Figure 2).

Figure 2. MRI sagittal section. MRI demonstrating a fracture in L4 vertebral body, on the central region of its vertebral inferior plate as a result of a SN exhibiting a "mushroom cloud" shape and an associated edema.

In order to reach a complete diagnosis, a lumbar computed tomography (CT) and a 99mTc bone scintigraphy were requested. The CT confirmed the L4 inferior plate fracture and suggested that it was secondary to SN (Figures 3 y 4). The scintigraphy showed increased uptake in L4, with all other bones exhibiting normal structures. (Figure 5).

Figure 3. CT sagittal section. Image exhibiting a fracture on L4 inferior plate, secondary to SN.

Figure 4. CT transverse section. Image exhibiting a fracture on L4 inferior plate.

Figure 5. 99mTc bone scintigraphy. An increased uptake was noticed on L4, with all other bones exhibiting normal structures.

On account of the lack of positive outcomes produced by conservative treatment, bilateral transpedicular kyphoplasty at L4 level and surgical biopsy (Figure 6) are performed with no complications. No alterations were detected through the preoperative workup.

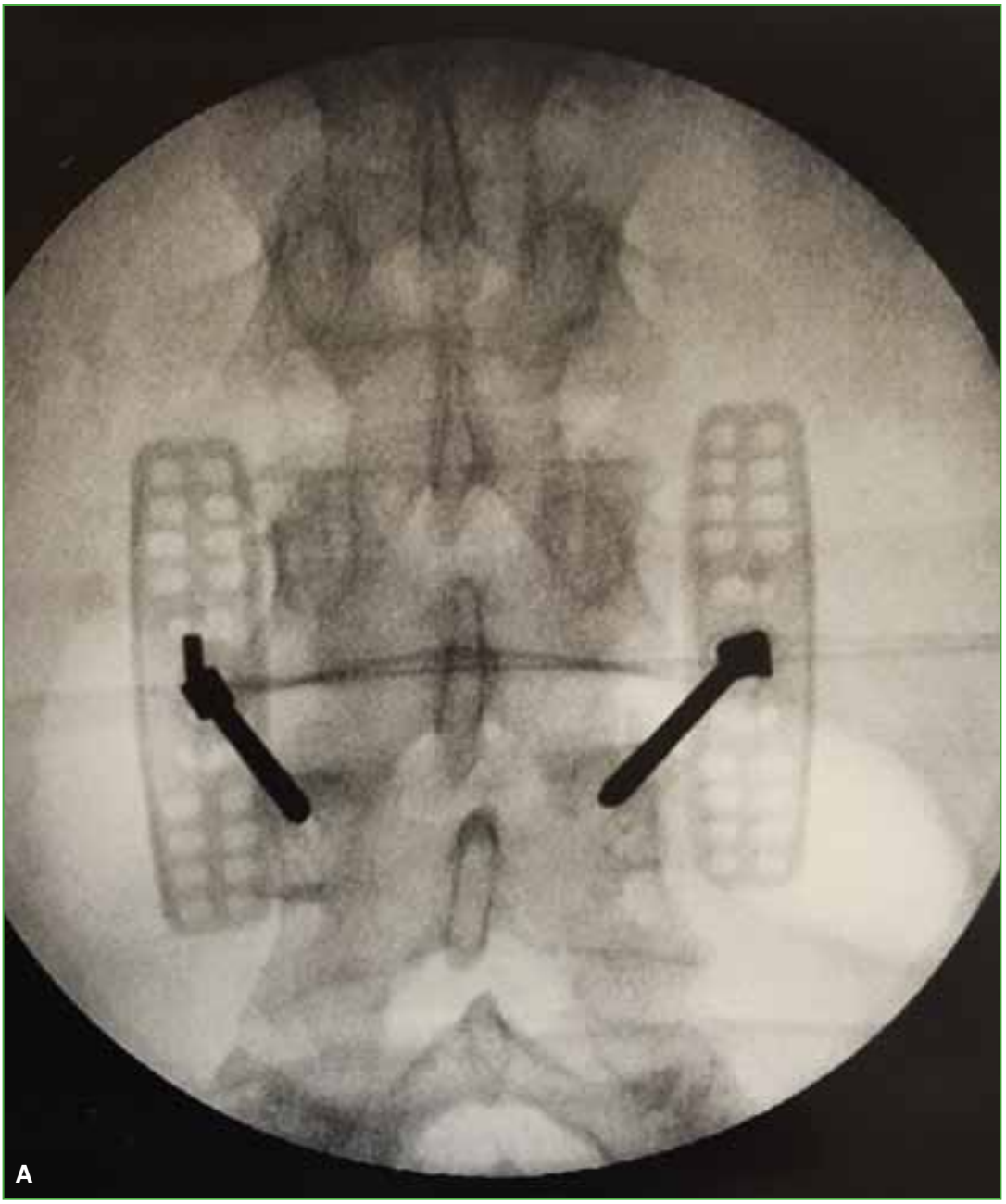

Figure 6. A and B. Intraoperative fluoroscopy during kyphoplasty. Lumbar spine anterior and lateral views**.** Image exhibiting bone cement in L4.

Contrary to what was expected, the patient experienced no postoperative relief. Pain keeps escalating and prevents him from standing. He has lost weight, which he claims to be the result not eating due to the pain.

Two days after the surgery, the first results from the biopsy show the presence of cancer cells; however, the results do not constitute an established diagnosis, pending the final results.

Tumor extension studies were conducted by a chest-abdomen-pelvis CT scan, more comprehensive tests were performed, and consultations were held with the Department of Internal Medicine.

The abdominal CT shows a pancreatic mass consistent with carcinoma and multiple metastatic spreading at the liver (Figure 7). The final results of the biopsy confirmed the pancreatic adenocarcinoma diagnosis. The patient died a week after being diagnosed.

Figure 7. A and B. Abdominal CT. Image exhibiting a pancreatic mass consistent with carcinoma and multiple metastatic spreading at the liver.

DISCUSSION

Reported asymptomatic SN prevalence varies widely, from 38% to 75% of the population, and has a male predominance.[9,10] This variation in prevalence could be attributed to several factors: differences between assessment methodologies, inclusion criteria, and socioeconomic status and other demographic characteristics considered.[1,11]

SN may be diagnosed through diagnostic imaging, such as plain X-ray, CT or bone scintigraphy.[8] However, MRI is the technique that provides the most valuable information.

Diagnosing SN through conventional radiography depends on the size of the nodes as well as the reactive process, the fibrosis and the sclerosis, in the adjacent trabecular bone.

Coventry *et al*. report that only 3.6% out of 55 pathologically confirmed SN were visible on conventional X-rays. Similarly, Yasuma *et al*. reported that 5.6% out of 54 histologically identified SN were visible with conventional radiography.[12] Furthermore, Hamanishi *et al*. reported that only 33% of the SN detected through MRIs were noticed through X-rays.

Therefore, plain X-rays are of limited value in assessing for SN, in particular for acute SN, such as our case.

In contrast, only MRI enables detecting vascularization and bone marrow reaction related to bone edema which indicates an acute condition, especially when using frequency-selective fat suppression imaging.[14] Furthermore, it has been proven that the signal changes on MRI mirror bone marrow inflammation and edema detected through pathology.

Most authors considered SN to be asymptomatic since their detection is usually on people with no back pain.[15] However, Hamanishi *et al*. compared the SN findings through lumbar MRIs of 400 patients with low back pain

with those of a 106-patient control group and found that the SN incidence in the symptomatic group (19%) was significantly greater than in the control group (9%).

Takahashi *et al*.,[16] Walters *et al*.,[17] and Stabler *et al*.[14] showed that the bone marrow at the SN level had low signal intensity on T1-weighted sequences and high signal intensity on T2-weighted and short tau inversion recovery sequences in symptomatic patients.

Although its detection and the data provided by MRI pattern of contrast may be helpful, acute SNs or SN recent formations hinders the differentiation between benign degeneration, malignant infiltration, and infection. Furthermore, neoplastic and infectious processes may weaken the trabecular bone-supporting structure, which facilitates SN formation.

SN must be considered when bone edema extends from the endplate of one vertebra or two adjacent vertebrae without collapse or paraspinal mass.[18]

There is no unify agreement concerning the location of SNs. Mok *et al*.[1] conducted a cross-sectional MRI study with 2449 subjects and found most SN at upper lumbar levels, and the highest incidence was at L2-L3. In contrast, Dar *et al*.[11] skeletal study show that SN are more common at T7-L1 levels. Their findings are similar to a previous report by Pfirrmann y Resnick.[19] This SN distribution cannot be accounted for only by load magnitude differences along the spine. If that were the case, SN prevalence would be apparent at T1 and L5 (maximum load). Therefore, the thoracolumbar region being the most common SN location, other factors may be involved.

Dar *et al*.[11] also showed that SNs are more common on the lower surfaces of thoracic vertebrae (T4-T11) and on the upper surfaces of lumbar vertebrae (L1-L5), which is consistent with previous studies. To date, no convincing explanation has been found for this phenomenon.

An acute and painful SN is usually treated with conservative treatment, including analgesics, bed rest and wearing a girdle or a corset. In the event that medical treatment proves ineffective and the patient still sustains disabling and persistent back pain, some authors suggest surgical treatment.

Hasegawa *et al*.[20] published a clinical case of painful SN treated by extracting the intervertebral disc, including the SN and the segmental fusion. Masala *et al*.[21] suggested vertebroplasty for the cases of symptomatic SN where patients are patients are resistant to medical o physical therapy. Jang *et al*.[5] reported pain relief following the blocking of a communicating branch in a patient suffering from symptomatic SN.

In our case, we decided for kyphoplasty after the MRI showed classic T1 and T2 inflammation signs and the patient showed no improvement with the medical treatment.[21,22] At this point, we suggested taking a surgical biopsy before injecting the bone cement, on account of the recent history weight loss and the existing MRI resemblance between the imaging patterns of SNs and tumors.[23]

Vertebral metastasis very rarely is the first manifestation of pancreatic cancer. The most common sites of metastases in pancreatic cancer are the liver and the peritoneum. Other less common sites are the lungs, the brain, and the kidneys.[24,25] Skeletal metastases are less common but bear higher morbidity associated with pain and limited performance status. Its prevalence is estimated to be between 5-20%.[25]

CONCLUSIONS

SNs are usually incidental findings and asymptomatic. In the presence of symptoms, edema or imaging changes around the node, examination must be extended through analyses and bone scintigraphy directed to rule out infections, rheumatic diseases, and tumors.

MRI is the technique of choice for diagnosing SN.

If no medical treatments prove effective and it is decided to perform a vertebroplasty or a kyphoplasty, we advise taking a vertebral surgical biopsy.

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