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CASE REPORT OPEN ACCESS

MULTIPLE SCHMORL'S NODES ON YOUNG ADULT WITH MYOFASCIAL PAIN SYNDROME: A CASE REPORT

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ABSTRACT

Background: Schmorl's node (SN) is described as intraosseous disc herniations which penetrate into the spinal canal, which is caused by the weakening of the cartilaginous endplate and subchondral bone. SN is usually detected on imaging which appears small, focal, and rounded with varying degrees of adjacent reactive sclerosis.
Case Report: We present a rare case of symptomatic SN in 25-year-old male patient with complaints of discomfort and muscle strain in the back. These complaints were exacerbated by physical exercise. Severe tenderness was found at several levels of the thoracic vertebrae during palpation, where the trigger points were found in the lower trapezius muscle. Computerized tomography (CT) scan of the vertebra showed multiple bony lesions, noted as Schmorl's nodes, at the inferior endplates of the T6-T11, and superior endplate of the T11 vertebra, in accordance to the location of severe pain during palpation.
Conclusion: SN is associated with disc degeneration due to osteonecrosis process which leads to ischemia in the vertebral body. Microtrauma from excessive physical exercise could also contribute to the herniation through the weak points of the vertebral endplates. Knowledge and awareness of SN as one of the causes for acute or chronic back pain is essential for deciding early examination and diagnosis. Therefore, appropriate management could be delivered.

Keywords: Schmorl's node, symptomatic, myofascial pain, CT scan

Introduction

In 1927, a German pathologist Christian Georg Schmorl described a unique lesion most frequently seen in the thoracolumbar spine. This spinal lesion is referred to as Schmorl's node (SN). SN is described as intraosseous disc herniations or Geipel hernias. Disc herniations were commonly horizontal, which penetrate into the spinal canal. Meanwhile, SN herniated vertically through the cartilaginous and bony endplate into the body of the adjacent vertebra.[1] SN is usually detected on imaging which appears small, focal, and rounded with varying degrees of adjacent reactive sclerosis.[2]

SN is caused by the weakening of the cartilaginous endplate and subchondral bone due to ischemic necrosis.[3] This condition is commonly associated with pathologic conditions such as metabolic diseases, neoplasms, and trauma.[2] Most cases of SN are asymptomatic and detected on incidental findings. However, there are symptomatic cases in the manifestation of back pain.[1,4] Here, we report a case of a patient with a symptomatic SN which was discovered from a typical imaging finding.

Case Report

A 25-year-old man, Caucasian race, came to the neurology clinic of a private hospital with complaints of discomfort and strained muscle in the back area around the ribs parallel to the right and left nipples. The discomfort arose a week ago after falling from a height of 1.5 meters with his back hitting the edge of a table. These complaints are exacerbated by lifting weights during physical exercise or in a hanging position with both hands on the exercise pole. There is no pain and the patient can freely move his body to all positions. There were no complaints of fever, chronic medication, or a history of previous surgery. There are no complaints or history related to neurological disease. The patient was athletic, muscular, and active in sports to maintain his fitness.

The patient immediately went to the ER (Emergency Room) for a check-up, and an X-ray of the lumbosacral spine was performed. There were no severe clinical symptoms nor abnormality on the X-ray. There was no evidence of fracture, compression, or listhesis. The ER doctor gave the patient analgesic and anti-muscle spasm medications. The patient is scheduled for follow-up next week. At the time of control, the patient felt that there was no improvement and asked to be consulted by a neurologist.

Neurological examination showed no limitation of motion either passive or active hence normal range of motion. There is no pain that appears when the patient moves his body. However, when the vertebrae were systematically palpated from cervical to lumbar, severe tenderness (NPRS

8-9) was found in the spine at the level of the T9-T11 (thoracic) vertebrae without any radiation of pain. Pain did not appear at all when no pressure was given. Trigger points were found in the lower trapezius muscle. There were no motor, sensory, or autonomic disturbances in the patient. All reflexes were normal.

A computerized tomography (CT) scan of the vertebrae was performed to confirm the patient's severe tenderness. Multiple bony lesions, noted as Schmorl's nodes, were found at the inferior endplates of the T6-T11, and superior endplate of the T11 vertebra. Lesions at the inferior endplate of the T10 vertebrae and superior endplate of the T11 vertebrae showed the largest lesions, which in accordance to the location of severe pain during palpation. Bony structures were intact, and there was no evidence of compression or collapse of the thoracal body. No evidence listhesis of the thoracal internal architectures were demonstrated either. Complete haematological laboratory examination showed normal results.

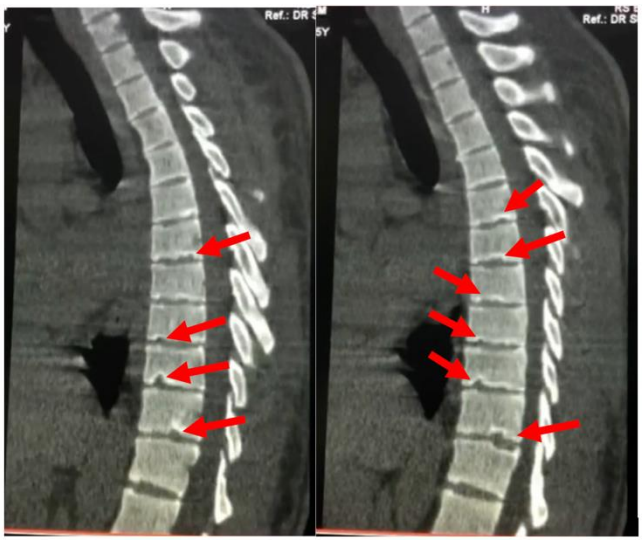

Figure 1. Sagittal view of vertebrae CT scan examination demonstrated multiple Schmorl's nodes at the inferior endplates of the T6-T11, and superior endplate of the T11 vertebra. The Schmorl's nodes were pointed by the arrow.

The initial diagnosis was of acute back pain due to post-traumatic mixed pain with myofascial syndrome. After the CT scan was carried out, the additional diagnosis was of Schmorl's nodes which appeared incidentally due to chance findings. Conservative treatment or watchful waiting was chosen for the management of this patient.

The patient was scheduled for magnetic resonance imaging (MRI) to check for marked bone marrow oedema to explore the etiology of the condition. However, he didn't show up for the next scheduled follow up.

Discussion

Spinal abnormality in the form of Schmorl's node (SN) was present in 38% to 75% of the population.[5,6] SN is mostly found in men and increases with age due to weakening of the cartilage endplate and decreased bone density, with a peak in the fourth decade of life.[3,4] Based on the location, a study in the Iraqi population found that SN was most common in the L1-L2 vertebral region, and the least in the L5-S1 vertebral region.[3] In a study in India, SN was found in the thoracic and lumbar regions, where 59.5% had SN at only one intervertebral disc and 40.4% had multiple SN at different vertebral levels.[4] Another epidemiological analysis revealed that SN occurs frequently in thoracolumbar transition (T7-L1) and commonly affects the inferior surface of the vertebral body.[7] The prevalence of SN varies in the literature depending on several factors such as the examination method, the criteria for the subject in the study, and the characteristics of the population that are influenced by genetics and ethnic distribution.[3,8]

In general, SN is asymptomatic and usually found incidentally on radiographic examination. However, the clinical presentation of SN can also be back pain. Research by Hamanishi et al. showed 19% of SN in the group of patients with low back pain and 9% of SN in the asymptomatic control group.[9,10] In another study, it was found that the prevalence of SN through MRI examination was 22% in the adult population with low back pain.[3] Symptomatic features of SN are usually very severe pain with a visual analogue scale (VAS) score close to 10 and affect the patient's quality of life. The location of SN is often found in the lower thoracic region between T8 and T12.[10]

There are several theories related to the etiology and pathophysiology of SN, such as intervertebral disc weakness, spinal degeneration due to overload, autoimmune reactions, and vertebral morphology.[3,10–12] Sadiq et al. found that SN is associated with disc degeneration, especially changes in the vertebral endplate in the form of inflammation and edema, known as modic changes (MC). SN is caused by an osteonecrosis process where bone cell death occurs due to ischemia in the vertebral body under the endplate which causes disc herniation into the vertebral body.[3] Microtrauma is also thought to be the etiology of SN that causes herniation of the nucleus pulposus through weak points on the endplate.[13]

Axial loads can also be caused by acute or chronic trauma. A correlation was found between SN and the incidence of acute trauma in motorcycle accidents that caused falls, where falling resulted in an increase in axial load on the spine. In addition, a study was conducted on professional gymnastics athletes and found that the prevalence of SN was higher in athletes than non-athletes (71% and 44%). Gymnastics causes high axial pressure on the spine, especially to maintain body mobility and stability. This causes microtrauma, especially in the thoracolumbar region which is more prone to SN.[10]

In this case, a 25-year-old male patient was found with multiple symptomatic NS in the thoracic region, with complaints of discomfort during activities and severe pain on palpation. Epidemiologically, SN is more often found in men because they tend to have a larger body size with higher vertebrae and discs, causing a heavier mechanical load on the vertebral endplate. The incidence of SN was significantly increased as age advanced due to weakened aged cartilaginous endplates and reduced bone density in older age groups.[3] However the patient in this study was young at 25 year old. Therefore, the microtrauma due to high axial pressure was suspected to be the cause of SN, especially since this patient was very athletic and actively exercising related to bodybuilding.

Moreover, the patient started complaining of discomfort and strained after a traumatic trigger. Traumatic triggering was suggested as a risk factor which differentiate symptomatic and asymptomatic SN.[7]

Imaging modality is essential for detecting SN. MRI is the gold standard due to its ability to detect acute SN lesions, which lead to earlier diagnosis and management of symptomatic lesions.[10] MRI is accurate to evaluate SN irrespective of the size, where it shows SN surrounded by vertebral body marrow edema with increased free water.[7,8,14] MRI also has the ability to differentiate symptomatic and asymptomatic SN from the accentuation of edema in T2-weighted images in all symptomatic cases.[10] Modic change (MC), which was associated with SN, could be seen on MRI as inflammatory changes and edema.[3,7]

Plain film radiographs can be useful at the chronic stage of the lesion when calcification around the SN has occurred, however it has limited value in diagnosing acute SN. CT scan is able to see sclerosis in an early stage and more detailed view. Several case reports presented CT scan of SN with well-defined contours and thin marginal sclerosis. This finding might be useful to identify small SN.[2] Bone edema can also be seen on dual energy CT.[8,15] In this study, the SNs were clearly identified through CT scan due to its size and multiple lesions. Sclerosis around the nodes' margin was also demonstrated in our CT scan findings.

A symptomatic SN is usually treated by conservative management with analgesic medication, bed rest, and bracing. Pain control and symptom improvement were the aim of the therapy.[1] Surgical management is proposed when the patient still suffers from persistent and disabling back pain, which does not respond to conservative management.[7,8] Vertebroplasty is suggested in SN which represents an early stage of vertebral body fracture, and fusion is suggested in SN which is associated with intervertebral disc degeneration and discogenic pain.[7]

Conclusion

This case report presented a rare case of a young adult with multiple Schmorl's nodes in his thoracic vertebra, which manifested as discomfort and severe tenderness during palpation. Awareness of SN as one of the causes for acute or chronic back pain is essential for deciding early examination and diagnosis. Therefore, appropriate treatment could be delivered.

Acknowledgement

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Conflict of Interest

None.

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