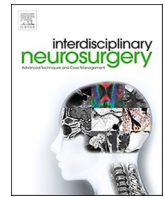




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Case Reports & Case Series

Massive obstruction of the spinal canal and progressive cervical myelopathy due to osseous hypertrophy of a facet joint: A case report

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ABSTRACT

Cervical spondylotic myelopathy is one of the most common causes of cervical spinal cord dysfunction. A 65-year-old patient was admitted with slowly-progressing myelopathic symptoms. Imaging studies indicated a massive lesion, which was thought to be a primary bone tumor. Laminectomy and microsurgical resection of the suspected lesion with a posterior approach at the C4 level was performed. Histopathological examination showed a hypertrophic facet joint. Age-related degenerative changes and adaptation mechanisms result in hypertrophy of the joint and synovial cyst formation. In our case, it was enormous and could be easily mistaken for a tumorous process. Such extensive hypertrophic changes are rarely addressed in the literature and should be considered as a differential diagnosis.

1. Introduction

One of the most common causes of cervical spinal cord dysfunction is cervical spondylotic myelopathy (CSM) [1]. The clinical course of CSM is quite variable, from an insidious and stepwise decline in neurological function to rapid deterioration. Different pathophysiological factors are involved at each step during the development of the disease. Anatomically the spinal cord can be compressed due to various reasons such as protruding vertebral discs, deformed vertebral bodies, facet joint hypertrophy, osteolytic lesions, hypertrophic ligamentum flavum, and ossified posterior longitudinal ligament [1]. Early recognition and treatment are essential for an optimal clinical outcome. Surgical intervention has been shown to be superior for moderate and severe cases of CSM [3]. During ten years from 1993 to 2002, the incidence of CSM and a subsequent number of cervical spinal surgery demonstrated a 7-fold increase in the United States [1]. Even though it is one of the most common causes of cervical spinal cord dysfunction, there has been only one other case report addressing this issue in the literature: unilateral facet joint pathology causing rapidly progressive CSM [6]. Our case had a chronic clinical course for two years. We report a patient with exceptional facet joint hypertrophy, compressing more than 50 % of the

spinal canal, thus causing myelopathy. It presents a prominent facet joint hypertrophy, which was initially suspected to be a primary bone tumor.

2. Case report

A 65-year-old male presented with progressive tingling and numbness in his both legs, unsteady gait and stool and urine incontinence. His neurological examination showed hypesthesia from T8 level downwards, hyperreflexia in the lower extremities, atactic gait and a positive Romberg test. The rest of the physical examination was within normal limits.

The preoperative cervical- CT images (Fig. 1) showed reduction of the spinal canal (more than 50 %, reducing the canal diameter from 1.5 × 2.4 cm to 0.7 × 1.2 cm) and a raised bone-like structure involving both the lamina and vertebral joint at the left side. The preoperative cervical MRI (Fig. 2) showed an unclear tumorous lesion at the C4 level with the absolute stenosis of the spinal canal and the displacement of the cervical spinal cord to the right side, starting from the bone to the left facet joint at this level, without intradural involvement. We first suspected a space-occupying process due to the given radiological findings. Laminectomy

Abbreviations: CSM, Cervical spondylotic myelopathy; CT, Computed tomography; MRT, Magnetic resonance tomography; T8, 8th thoracic vertebra; C4, 4th cervical vertebra.

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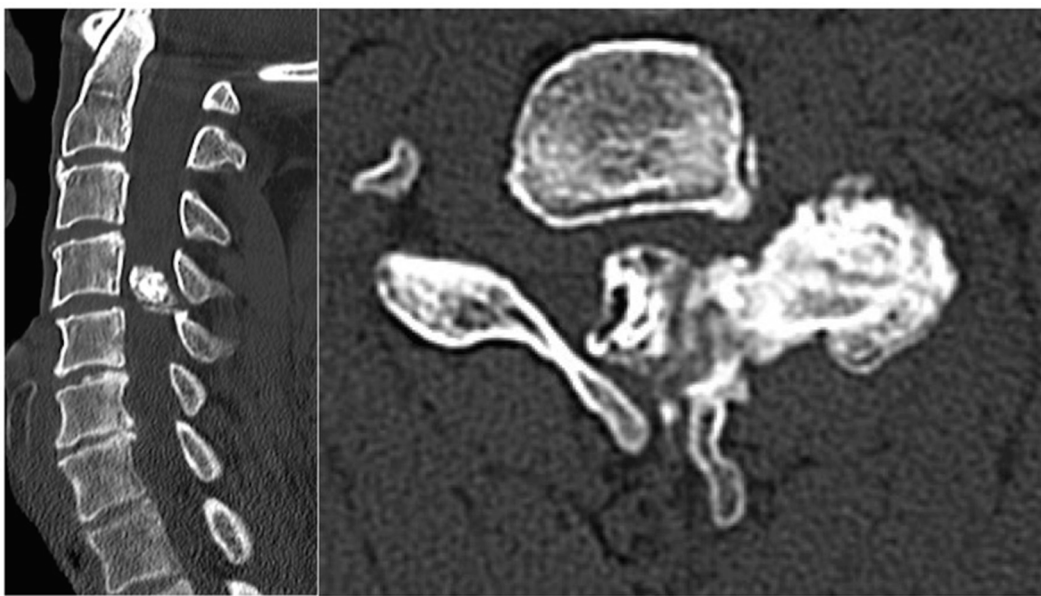


Fig. 1. Showing pre-operative CT scans, sagittal and horizontal views of the cervical spine, progressive reduction of the spinal canal (more than 50 %), and raised bony structure involving both lamina and vertebral joint on the left side.

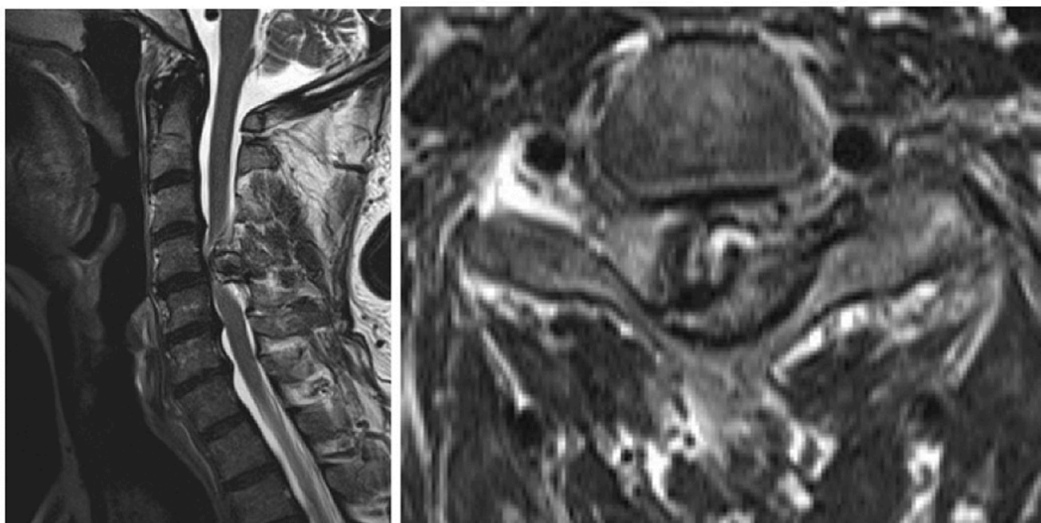


Fig. 2. Cervical MRI shows the cervical spinal cord compression due to an unclear tumorous lesion at the C4 level and cervical spinal cord displacement to the right side, starting from the bone to the left facet joint at this level.

and microsurgical resection of the suspected lesion with a posterior approach at the C4 level was performed. Intraoperative examination of the dural sac showed a hard, nodular structure that was difficult to remove with the punch tool because of its adherence to the dura. The lesion was firstly downsized with a diamond burr to detach it from the dura and then removed in total. After this procedure the dural sac unfolded again, thus signaling the release of the compression (Figs. 3 and 4).

Postoperative CT scans confirmed the laminectomy and total removal of the bony structure (Fig. 5). After the surgery, the atactic gait and incontinence symptoms of the patient showed moderate clinical improvement.

The histopathological examination of the resected obstructive tissue showed degenerative changes in the facet joint and synovial cyst formation without neoplastic changes or malignancy (Fig. 6).

3. Discussion

The facet joint is a diarthrodial joint with opposing articular cartilage surfaces that reduce friction and a capsule covering the joint space. [2] Along with the disc, the facet joints transfer stress, guide and constrain spinal motions due to their geometry and mechanical functions. They prevent two adjacent vertebrae from engaging in relative motions that could overload and damage the surrounding structures.

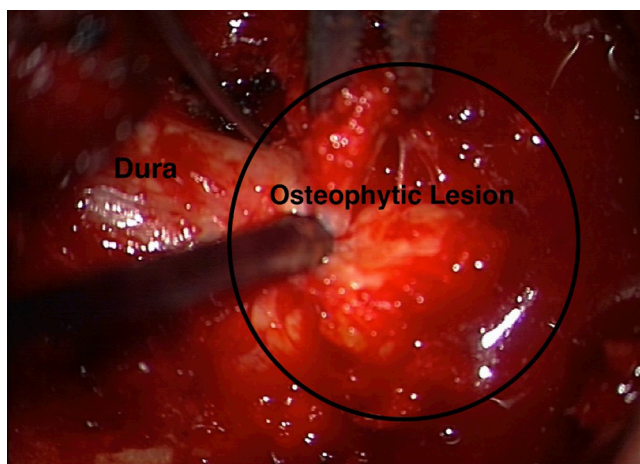


Fig. 3. Intraoperative demonstration under the microscope, in toto removal of the suspected lesion.

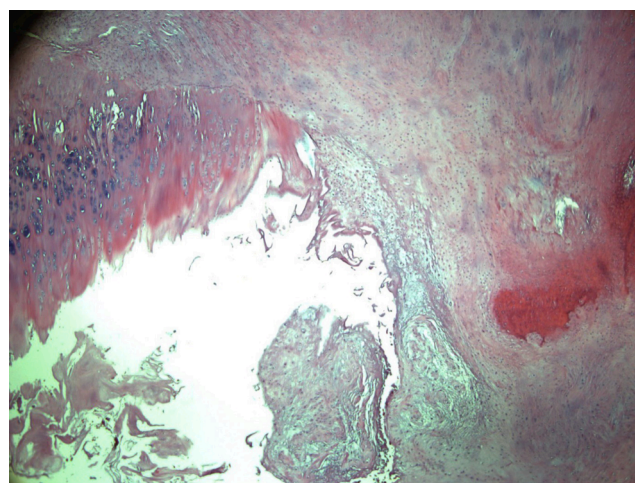


Fig. 6. Fibrotic periarticular tissue with pseudocyst formation representing a synovial cyst (Hematoxylin and Eosin (HE), $\times 40$).

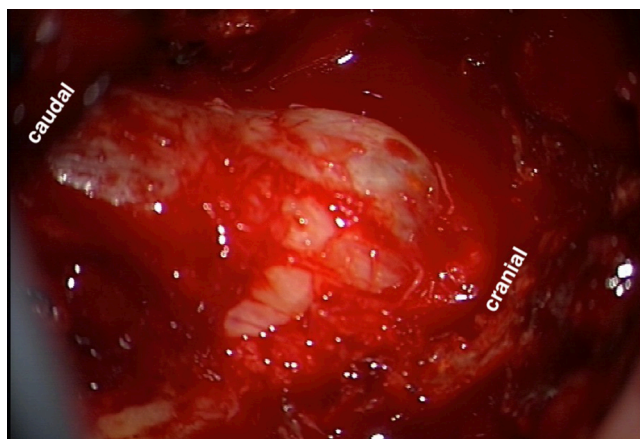


Fig. 4. After removing the structure, unfolded dural sac indicating compression release.

The pathophysiology of CSM can be explained with a “static and dynamic model.” Degenerative changes result in static compression and exacerbate the compression of the spinal cord during various movements. As a result, static and dynamic compression leads to axonal stretching, spinal cord ischemia from vascular constriction and venous congestion [5]. Degenerative changes of the fibrous tissues and the loss of the cushioning cartilage may result in ruptures of the capsule, resulting in leakage of the inflammatory cytokines into the intraspinal space, irritating nerve roots and triggering pain signaling and triggering synovial cyst formation. At the molecular level, neural cells undergo apoptosis instead of necrosis under CSM [4].

Only one case in the literature reported a unilateral facet joint hypertrophy resulting in rapidly progressing CSM [6]. In this case, the rapid progression and the demonstration of the “facet joint gap” sign on the cervical radiogram was acute. Our case illustrates a unilateral facet joint pathology that constricted the cervical spinal canal to a much larger degree, resulting in more pronounced clinical symptoms.

Our case report illustrates an unusual case with the far-reaching and extensive adaptations to the degenerative changes, that could have been

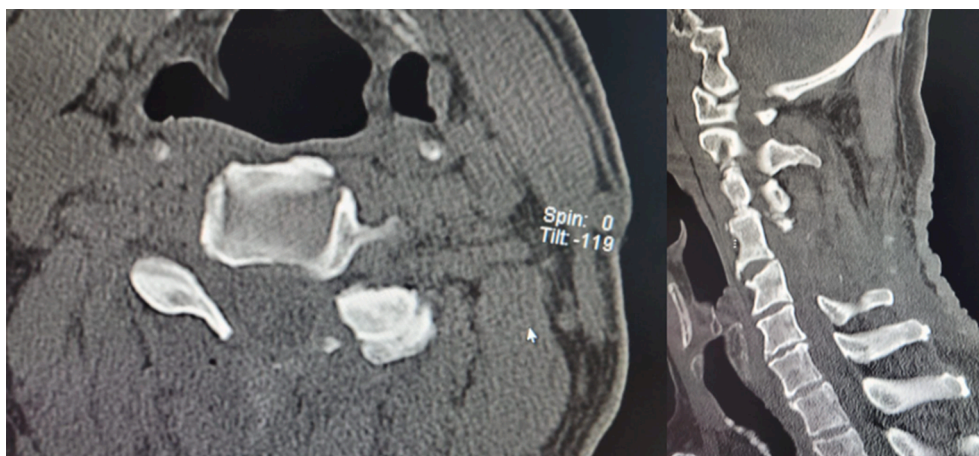


Fig. 5. Showing postoperative CT scans, sagittal and horizontal views of the cervical spine, confirming the laminectomy and total removal of the bony structure.

easily mistaken as a tumorous process. Such extensive hypertrophic adaptative tissue reactions are possible and should be considered in the differential diagnosis.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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