Cervical Radiculopathy Due to Disc Herniation with Adjacent Facet Hypertrophy: Case Report

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Abstract

We report a rare case of cervical radiculopathy associated with facet hypertrophy and disc herniation. The patient was a 38-year-old woman with sudden-onset left arm pain. As conservative therapy failed to alleviate her symptoms she was referred to us. On physical examination she manifested no neurological deficits except pain and dysesthesia in the left C7 territory. Computed tomography revealed hypertrophic ossified changes in the left T1 facet joint with encroachment on the spinal canal. Magnetic resonance imaging showed compression of the spinal cord at C6/7 by disc herniation at C6/7. Anterior cervical decompression and fusion by corpectomy (C7 corpectomy and C6/T1 fixation with a titanium cage) ameliorated her pain. Facet hypertrophy in a morphologically normal cervicothoracic spine is extremely rare and its etiology is unknown. We speculate the possibility that our patient harbored a congenital anomaly and that the morphologic changes were the consequence of an injury she sustained in a traffic accident.

Key words: cervical radiculopathy, cervical disc herniation, bone anomaly, facet hypertrophy

Introduction

Hypertrophy of the facet joints is common in the lumbar region of the spinal column.¹ It is usually associated with spondylotic changes and often involves multiple spinal segments. Although isolated hypertrophy of a single thoracic or lumbar facet has been documented,²,³ hypertrophy of cervicothoracic facet joints appears to be more rare. To our knowledge, no case of massive unilateral facet hypertrophy in this area has been reported. Here we describe a patient with radiculopathy due to cervical disc herniation with unilateral facet joint hypertrophy and discuss its clinical significance and pathophysiology.

Case Report

This 38-year-old woman presented with a one-month history of numbness and pain in the left arm. She had been in good health until 2 months earlier when she suddenly suffered severe pain in the left neck and shoulder. Two days later she consulted a local hospital. Based on magnetic resonance imaging (MRI), the primary diagnosis was cervical radiculopathy due to disc herniation. Conservative therapy with a neck collar failed to mitigate her symptoms and she was referred to our hospital. Her pain radiated to the index- and middle finger and was exacerbated by movement of the hand. There was no remarkable past medical history except for a whiplash injury she sustained in a traffic accident 15 years earlier.

On physical examination there were no neurological deficits including deep tendon reflexes except for pain and dysesthesia in the left C7 territory. Cervical X-ray study (anteroposterior and lateral views) revealed no spondylotic changes; dynamic study showed no instability. On the left oblique view there was stenosis of the intervertebral foramen at C7/T1. Computed tomography (CT) disclosed hypertrophic changes in the left C7/T1 facet joint and encroachment on the spinal canal (Fig. 1A). Sagittal reconstruction of CT images showed hypertrophic changes of the left Th1 superior facet (Fig. 1B). Axial T2-weighted MRI revealed cervical disc herniation with protrusion at the C6/7 level (Fig. 2A); the dural sac was compressed at the level of the C7 vertebral body with extension above the C6/7 level attributable to cervical disc herniation at C6/7 and left facet hypertrophy at the level of the C7 vertebral body with extension above the
Fig. 1  A: Axial computed tomography (CT) images demonstrating hypertrophy of the left facet at C7/T1 resulting in marked compression of the dural sac (arrow). B: Sagittal reconstruction of CT images revealing facet hypertrophy at the left C7/T1 level (arrow).

Fig. 2  A: Axial T₂-weighted magnetic resonance imaging (MRI) of the C6/7 intervertebral space level revealed disc herniation in the left postero-lateral direction (arrow). B: Axial T₂-weighted MRI of the C7 vertebral body level demonstrates stretching of the spinal cord by facet hypertrophy (arrow). C: Contrast-enhanced axial T₁-weighted MRI of the same level shown in B showed no enhancement. D: Sagittal T₂-weighted MRI disclosed compression by a low-signal mass due to cervical disc hernia at the C6/7 level (arrow). Note the bony protrusion behind the C7 vertebral body.
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herniation (Fig. 2D). The provisional diagnosis was nerve root compression by disc herniation at C6/7 and an ossified mass at the left C7/Th1 facet. We planned C7 corpectomy, resection of the mass, and discectomy at C6/7 followed by fixation with a titanium mesh cage.

A 6-cm horizontal skin incision was made on the left side of the anterior cervical surface. After cervical dissection, the C6/7 and C7/T1 discs were removed under a microscope. C7 corpectomy was completed with a William’s microsurgical oscillating saw and a high-speed drill. The posterior longitudinal ligament on the right side was identified. On the left in the spinal canal, we noted a bony structure that was not an ossified posterior longitudinal ligament but rather a protrusion from the left facet (Fig. 3A). The surface portion of the protrusion was removed with a high-speed drill and the middle-to-deep portion was drilled out with an ultrasonic bone curette, carefully protecting the dura mater and spinal cord. There was mild adhesion between this bony structure and the dura. After adequate decompression of the left C7 nerve root and dural sac, (Fig. 3B) a titanium mesh cage filled with autologous bone chips from the corpectomy was introduced. Her postoperative course was uneventful and she was discharged with milder symptoms 8 days after the operation. Postoperative CT confirmed decompression of the spinal cord (Fig. 3C).

Discussion

We encountered a rare case of cervical radiculopathy associated with facet hypertrophy. This entity is usually associated with spondylotic changes. While hypertrophied lumbar facet joints due to degenerative changes have been documented, there are few publications on the cervicothoracic facet joint. Ours is the first report of massive unilateral facet hypertrophy associated with radiculopathy in a morphologically normal spine.

Degenerative changes of the cervical facet are thought to lead to hypertrophic changes. In their series of 215 patients, Morishita et al. identified 32 (14.9%) who manifested hypertrophic changes of the facet joint at the mid-level of the cervical spine. These changes tended to be unilateral and were seen more often in males. The mean age of their patients was 69.2 years. Citow and Macdonald encountered vertebral artery compression due to a unilateral osteophyte arising from the left C6 superior facet in a 69-year-old man. Spondylotic changes are usually bilateral and involve multiple spinal segments. Although laterality is a characteristic of cervical facet hypertrophy, the etiology of the hypertrophic facet in our patient was different from that in earlier reports because of her young age and the absence of other spondylotic changes in the spinal column.

Anomalies such as bone anomaly including facet hypertrophy have been reported to be associated with the cervical region. Shimizu et al. documented a rare case of vertebral artery occlusion due to an abnormal bone in the superior facet at C6 in a 39-year-old man; an excessive bone formation at the unilateral C6 superior facet compressed his vertebral artery. CT images yielded findings similar to ours. They speculated that the etiology of the bone formation was an anomaly rather than a spondylotic change because the patient was relatively young without evidence of degenerative changes. Goel et al. reported several patients with unilateral facet hypertrophy at the atlantal bone and suspected that a congenital abnormality was causative. The similarity of CT findings in our and the earlier patients suggests that the underlying pathology of the facet hypertrophy in our patient was a congenital anomaly.

Fig. 3 A: Intraoperative photograph obtained during anterior decompression and fusion surgery showing a bony structure after C7 corpectomy. The surgeon stood on the left side of the patient. The asterisk marks the facet hypertrophy as seen from the left side. B: Intraoperative photograph demonstrating dural sac decompression after removal of the facet hypertrophy. The arrow marks the level of the left C7 nerve root. C: Postoperative axial CT images confirming enlargement of the spinal canal. Note the residual part of the bony protrusion (arrow).

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A different etiology of facet hypertrophy has been described by Kirby and Maimaris. Their patient suffered a cervical vertebral injury in a car accident and in the course of 3 years he gradually developed unilateral facet hypertrophy (left C4/5) with radiculopathy at the same level. They suggested that unilateral facet joint hypertrophy can be elicited by post-traumatic myositis ossificans and that whiplash injuries of the cervical spine resulting in a musculo-ligamentous sprain of facet joints with periosteal tearing can lead to ectopic bone formation. The history of our patient and clinical findings reported by others suggest that her cervical radiculopathy was attributable to a congenital anomaly or to morphologic changes as sequelae of trivial trauma.

Some bone tumors elicit hypertrophic changes in the spinal column. Osteochondromas are the most common benign bone tumors; only 2–7% of all osteochondromas arise in the spine. Although rare, facet joint osteochondromas that produce hypertrophic facet changes have been reported. Sakai et al. presented three patients with spinal osteochondroma arising from an articular facet; they resulted in symptomatic spinal canal stenosis. Srikantha et al. reported a 40-year-old woman with radiating arm pain whose CT images showed a bony mass arising from the superior facet of C6. Documented radiological characteristics of osteochondroma are cortical and medullary continuity between the tumor and bone and cartilaginous caps. The growing part of the osteochondroma is characteristically covered by a cartilaginous cap that is hypointense on T1- and hyperintense on T2-weighted images.

We did not perform a histological evaluation in our case because preoperative enhanced MRI revealed no enhanced lesion suggestive of tumor pathology around the hypertrophic lesion. However, considering the reported radiographic characteristics of unusual bony hypertrophic changes, we suggest that osteochondroma be included in the differential diagnosis and that histopathological examination be performed to obtain a correct diagnosis even in patients with radiological findings of a non-tumorous nature.

Treatment options to address cervical disc herniation range from a conservative approach to surgery. As our patient's symptoms did not respond to conservative therapy, surgery was indicated. The primary contributor to her radiculopathy appeared to be cervical disc hernia at C6/7. Although we were unable to determine whether her facet hypertrophy that encroached on the spinal canal was due to an anomaly or to an asymptomatic bony structure, we were forced to decide whether surgical intervention was appropriate. The rarity of this clinical entity rendered the selection of the optimal treatment strategy difficult. We are following our patient closely on an outpatient basis and we continue to monitor her condition to detect early the possible development of any adjacent cervical disorders.

In conclusion, unilateral hypertrophy of the facet in the cervicothoracic region is a rare clinical entity. Although the mechanism(s) underlying the formation of facet hypertrophy remains to be elucidated, clinical findings reported by others suggest the possibility of a congenital anomaly or morphologic changes as a sequela of trivial trauma.

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Conflicts of Interest Disclosure

The authors did not receive any funding for this work. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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