

## Case Report

**Cervical myelopathy due to single level disc herniation presenting as intramedullary mass lesion: What to do first?**Murat Şakir Ekşi, Emel Ece Özcan Ekşi, Baran Yılmaz<sup>1</sup>, Zafer Orkun Toktaş<sup>1</sup>, Deniz Konya<sup>1</sup>Department of Orthopedic Surgery-Spine Center, University of California, San Francisco Medical Center, California, USA, <sup>1</sup>Department of Neurosurgery, Bahçeşehir University Medical School, Istanbul, TurkeyCorresponding author: Dr. Murat Şakir Ekşi, University of California, San Francisco, Medical Center, 500 Parnassus Avenue MU320 West, San Francisco, California 94143-0728, USA. E-mail: [muratsakireksi@gmail.com](mailto:muratsakireksi@gmail.com), [EksiM@orthosurg.ucsf.edu](mailto:EksiM@orthosurg.ucsf.edu)

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## Abstract

Cervical myelopathy (CM) is mostly a degenerative process ending in myelopathic and/or radiculopathic syndromes. On T2-weighted magnetic resonance imaging (MRI), CM appears as a hyperintense area near the spondylotic spine. This high intensity signal depends on the impact of outer forces and their duration. It also determines the prognosis of the surgical candidate. A 40-year-old male patient admitted to our clinic with right upper extremity weakness and hypoesthesia that had started 2 months earlier. On neurological examination there was 2/5 motor weakness of right biceps brachii, and hypoesthesia over right C6 dermatome. Right upper extremity deep tendon reflexes were hypoactive, but lower ones were hyperactive. After clinical and radiological work-up, preliminary diagnosis was directed to a spinal intramedullary tumor. Total resection of the herniated cervical disc fragment and the mass lesion was managed. Pathology of the mass lesion was compatible with subacute infarct tissue and inflammatory response. Final diagnosis was CM under effect of cervical disc herniation. Contrast-enhanced spinal cord myelopathic lesions are very rare and resemble much more tumors and inflammatory processes. However, the principal treatment approach totally differs depending on pathology. When there are both a disc herniation and a high clinical suspicion; biopsy should be delayed. The most probable solution will be surgery for the disc disease with thorough preoperative scanning of vascular malformations; clinical and radiological close follow-up after surgery. Biopsy or surgical resection can be performed if patient deteriorates despite the primary surgery.

**Key words:** Cervical disc herniation, myelopathy, magnetic resonance imaging, spinal cord tumor

## INTRODUCTION

Cervical myelopathy (CM) is mostly a degenerative process ending in myelopathic and/or radiculopathic syndromes.<sup>[1]</sup> On T2-weighted magnetic resonance imaging (MRI), CM appears

as a hyperintense area near the spondylotic spine.<sup>[1-7]</sup> This high intensity signal depends on the impact of outer forces and their duration. It also determines the prognosis of the surgical candidate.<sup>[1,4]</sup> Intravenous contrast material use is routine in radiological workup of neoplastic, vascular, inflammatory, infective, and demyelinating diseases.<sup>[8]</sup> It has been used in spinal cord injury models in laboratory conditions or sparse clinical settings.<sup>[9-11]</sup> Contrast enhancement of *in vivo* myelopathic lesions has been reported infrequently [Table 1].<sup>[1-4,8]</sup>

Herein, we present a unique case of a cervical spinal cord mass lesion-mimicking tumor, which was proved to be a myelopathic process through histopathology.

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### CASE REPORT

A 40-year-old male patient admitted to our clinic with right upper extremity weakness and hypoesthesia that had started 2 months earlier. On neurological examination there was 2/5 motor weakness of right biceps brachii, and hypoesthesia over right C6 dermatome. Right upper extremity deep tendon reflexes were hypoactive, but lower ones were hyperactive.

Cervical plain films were nonspecific [Figures 1a and 2a]. Cervical MRI depicted a C5-C6 central disc herniation extending to the right foramen. Also the spinal cord below the herniated

disc level was very swollen with T2-hyperintense areas diffusely scattered intramedullary from C6 to C7 [Figure 3a and c]. After intravenous (IV) contrast administration, the swollen area was enhanced [Figure 3b and d]. There was no trauma or infection history. Laboratory test results were nonspecific. Cranial MRI was done to rule out any other concomitant lesions in the brain, brainstem, and cerebellum. After excluding an intracranial mass lesion, lumbar puncture was performed. Cerebrospinal fluid (CSF) glucose amount was 65 mg/dl and protein level was 45 mg/dl. No leukocyte or oligoclonal band was observed. Gram and acid-fast staining of CSF proved to

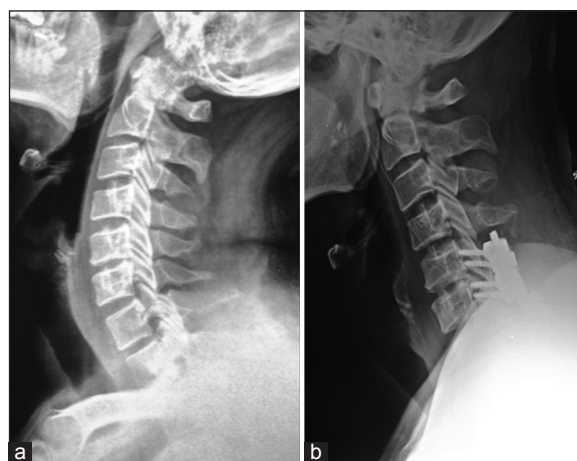
**Table 1: Spinal enhancing myelopathic lesions mimicking spinal cord tumor**

Authors/Year	Age/sex	Presentation	Level	Treatment	Follow-up
Takahashi et al., 1989*	74/M	Mild weakness and hypoesthesia	C3-C4	N/A	N/A
Matsuda et al., 1991	29/M	Gait disturbance	C5-C6	Discectomy with bone grafting at C5-C6 using Cloward technique	Clinical status got better 3 months after surgery, T2 hyperintensity decreased, and contrast enhancement disappeared
Morimoto et al., 1996	59/M	Progressive severe myelopathy	C5-C6	C4-C6 laminectomies, C3 and C7 laminotomies and clipping of concomitant AVF	Sensorial and gait difficulties regressed, spastic paraparesis and deep sensorial impairment persisted, contrast enhancement disappeared, and T2 hyperintensity continued
Boet et al., 2004	50/F	Paresthesia of hands more on the right side, difficulty in fine motor skills, and tightness of lower limbs	C6	Discectomy and anterior fusion with cage	T2 hyperintensity and contrast enhancement diminished 12 months after surgery
Cabraja et al., 2008	36/M	Neck and bilateral upper extremity pain, paresthesia, gait instability, and urinary difficulty	C4-C5	Biopsy and spinal fusion	Signs and symptoms improved 3 months after surgery. T2 hyperintensity and contrast enhancement persisted
Present case	40/M	Right upper extremity weakness and hypoesthesia	C5-C6	Surgical excision and posterior stabilization with lateral mass screws	Signs and symptoms improved 3 months after surgery

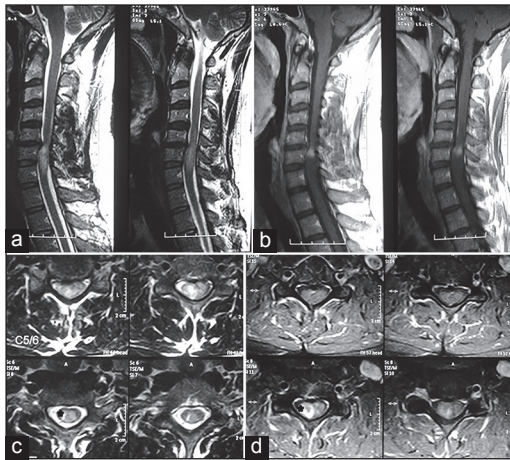
N/A = Not available; M = Male; F = Female, AVF = Arteriovenous fistula; \*Another probable enhancement in a T2-hyperintensity area from another patient was also reported



**Figure 1: Preoperative (a) and postoperative (b) cervical AP plain X-rays. AP = Anteroposterior**



**Figure 2: Preoperative (a) and postoperative (b) cervical lateral plain X-rays**



**Figure 3: Preoperative sagittal (a) and axial (c) T2-weighted MRI reveals a disc herniation at the level of C5-C6 level. Below the same level, the spinal cord had become swollen and hyperintense relative to normal cord tissue. It enhances on T1-weighted scans (b and d) after IV contrast material administration. MRI = Magnetic resonance imaging, IV = Intravenous**

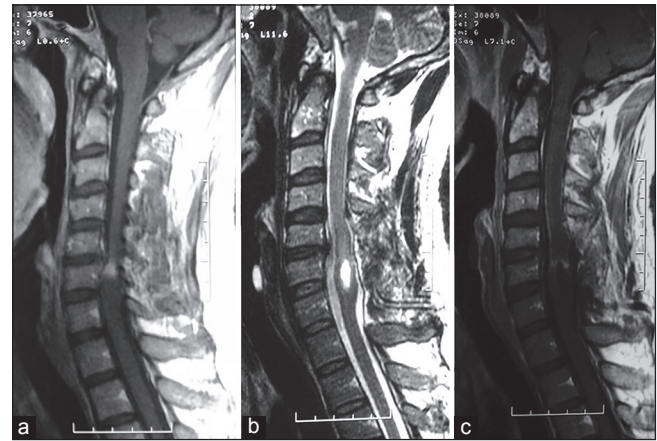
include no microorganism. CSF cultures turned to be negative. Corticosteroids were prescribed and they made no symptom relief during clinical work-up. After excluding inflammatory, demyelinating, and infectious entities; our diagnosis was spinal intramedullary tumor due to rapid onset of clinical presentation. Surgery was planned. We made C5 and C6 laminectomies and resected the right foraminal C5-C6 extruded disc portion. After duratomy, a central posterior myelotomy was performed. A yellowish hard piece of mass lesion was excised totally. After watertight closure of the dura, posterior spinal stabilization with lateral mass screws was accomplished from C5 to C7 [Figures 1b, 2b, and 4]. No change on neuromonitorization and no complication occurred throughout the surgery. The patient had no additional neurological deficit, postoperatively.

Diffuse axonal injury, piloid gliosis, perivascular lymphocytic inflammation, microgliosis, and subacute infarction zones were present on histological examination. Glial fibrillary acidic protein (GFAP) in astrocytic cells was positive. Neurofilament protein (NFP) was immunopositive in axonal structures. CD3 +, CD5 +, and CD20 + lymphocytes were present. Synaptophysin was detected in the neuropils. Ki-67 index was lower than 1%. Reticular structures were present around vessels. To rule out possible vascular pathology underneath the current evidences, a craniocervical digital subtraction angiography (DSA) was performed and no unusual vascular malformation or anomaly was observed.

Inpatient and outpatient physical therapy was conducted. After 3 months, his motor strength improved to normal, but hypoesthesia persisted in the same manner. Final diagnosis was CM caused by a herniated intervertebral disc.

## DISCUSSION

The most common spinal intramedullary tumors in adults are gliomas. Astrocytomas and ependymomas make up



**Figure 4: Enhancing lesion (a) has been totally excised with a posterior approach (b and c)**

90% of them.<sup>[12-15]</sup> The other less foreseen tumor types are hemangioblastomas, gangliogliomas, paragangliogliomas, lipomas, schwannomas, primitive neuroectodermal tumors (PNETs), metastases, teratomas, dermoids, epidermoids, and melanomas.<sup>[13,15]</sup> Mass lesion other than neoplasm is rarely observed in the intramedullary zone (4%).<sup>[12]</sup> But there are many diseases in differential diagnosis such as tuberculoma, amyloid angiopathy, plasma cell granuloma, sarcoidosis, vascular abnormalities, demyelinated, and inflammatory diseases.<sup>[12-14]</sup> Spinal cord myelopathy mimicking a mass lesion is even rare than all above mentioned diseases.

CM is usually observed in patients aged 60 years or older and having long-lasting symptoms. Patients present with pain that radiates in the dermatome of the lesion level with accompanying motor weakness and sensory deficits. Hyperreflexia, pathological reflexes, spasticity, and urinary problems (urgency and incontinence) are common signs.<sup>[12-14]</sup> CM presents with hyperintensity on T2-weighted MRI, which also determines the disease prognosis for surgical candidates.<sup>[1,4]</sup> Cabraja *et al.*,<sup>[1]</sup> presented a young male patient with kyphotic cervical spine including enhancing myelopathic lesion. Boet *et al.*,<sup>[8]</sup> followed-up a similar case of a middle-aged lady for 1 year. They observed the enhancement had regressed in time after cervical disc herniation surgery. In our case, it was very hard to make proper diagnosis because of short duration of symptoms, young patient age, and contrast enhancement on MRI.

There are many disease entities with lesion enhancement on MRI. Spinal intramedullary tumors are usually observed as expanded masses with hyperintensity on T2-weighted scans.<sup>[12,14]</sup> Rarely, they may include hemorrhagic components that appear as inhomogeneous zones.<sup>[14,16]</sup> They usually enhance after IV contrast material injection.<sup>[14,16]</sup> Depending on the pathology type, they may have a well-defined border (ependymomas) or not (astrocytomas).<sup>[16]</sup> Inflammatory processes appear as expanded or not.<sup>[12-14,16]</sup> Hyperintensity on T2-weighted MRI is also obvious in many lesions. Patchy enhancement with peripheral involvement, although not pathognomic, is seen.<sup>[12,16]</sup> Accompanying central canal syrinx, which is usually not seen

in astrocytomas, has also been described by Solmaz *et al.*, in inflammatory conditions.<sup>[13]</sup> MR spectroscopy can be a useful additional tool; but it may lead to misdiagnosis.<sup>[14]</sup> Multiple sclerosis (MS) plaques enhance at the initial stage, later they turn into high-intensity signal areas only on T2-weighted imaging.<sup>[1,15]</sup> Brain MRI should be checked up for other additional plaques. Rarely, sole spinal cord involvement is seen as a first manifestation of the disease. Inflammatory pseudotumor or plasma cell granuloma shows homogenous enhancement with mild cord enlargement. It involves B- and T-lymphocytes, plasma cells, and histiocytes. Systemic presentation is usual. Granulomatous lesions, mostly sarcoidosis, enhance homogeneously or heterogeneously with minimal cord swelling.<sup>[15,16]</sup> They present with leptomeningeal and peripheral cord enhancement. Other than the spinal cord, lesions present in brain, peripheral nerves, and other organ systems; so it is easy to differentiate them from neoplastic processes. Spinal infections (bacterial, viral, fungal, and parasitic) enhance with cord swelling. But; they present with fever, malaise, and constitutional signs and symptoms. Multisystemic involvement is present in many cases.<sup>[15,16]</sup> Lesions related with vascular malformations and/or vascular insufficiencies enhance diffusely with a disseminated manner.<sup>[15]</sup> They sometimes arise with hematomas or solely as congestive myelopathies.<sup>[16,17]</sup> Also, angiography helps for exact diagnosis. Chronic progressive radiation myelopathies present as hyperintense masses on T2-weighted MRI. A ring-like enhancement with cord distension can be a distinct feature.<sup>[15]</sup> Contrast enhancement has also been described in acute and subacute injured spinal cords.<sup>[1]</sup> It is even rarer in chronic degenerative processes.<sup>[1-3,8]</sup> On MRI there are some nuances between these different pathologies. Neoplasms enhance more centrally; spinal cord injuries, demyelinating diseases, and sarcoidosis enhance more peripherally.<sup>[1,11,16,18-20]</sup> CM can mask underlying pathologies. Morimoto *et al.*,<sup>[3]</sup> described a 59-year-old male patient with contrast enhancement at the level of myelopathy. In fact what they found, during further examination with angiogram, was an arteriovenous fistula (AVF). After pathology result, we ordered cerebrospinal DSA of the patient and it was inconclusive for any vascular anomaly.

Although there are some differences, it is still hard to differentiate a spinal neoplastic lesion from a nonneoplastic lesion depending on solely clinical and radiological findings. Sometimes, CSF laboratory studies help clinicians differentiate neoplasms from inflammatory processes, but this is not true in a degenerative situation.<sup>[1,16]</sup> The patient in this case report was a young adult with a rapid worsening history. Conjoining with contrast enhancement on MRI, the preliminary diagnoses were spinal intramedullary tumor, demyelinating or inflammatory disease, and infection. Brain MRI was normal with nonspecific CSF parameters. On microbiological assessments, no organism was detected and there was no other systemic involvement. So, tumor option became more logical than others and surgery was planned. After surgical resection, the final diagnosis was

subacute infarction and gliosis. It should have occurred due to circulatory insufficiency caused by intervertebral disc herniation. Circulatory insufficiency would have resulted in blood-nerve barrier breakdown and contrast leakage into spinal cord tissue.<sup>[1,3,4,6,8]</sup> After the decompression and discectomy surgery, his signs and symptoms regressed. It is interesting that all reported cases of enhancing myelopathic lesions, including ours, are from cervical spinal cord [Table 1].<sup>[1-4,8]</sup>

There are both anterior and posterior approaches available for cervical spinal surgeries.<sup>[1]</sup> We preferred posterior approach because we had planned gross lesion resection. We performed laminectomies; after resection, we applied posterior stabilization with lateral mass screws from C5 to C7.

## CONCLUSION

Contrast-enhanced spinal cord myelopathic lesions are very rare and resemble much more tumors and inflammatory processes. But, the principal treatment approach totally differs depending on pathology. When there are both a disc herniation and a high clinical suspicion; biopsy should be delayed. The most probable solution will be surgery for disc disease with thorough preoperative scanning of vascular malformations; clinical and radiological close follow-up after surgery. Biopsy or surgical resection of the suspected mass can be performed if patient still deteriorates despite the primary surgery.

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