Compressive Thoracic Myelopathy

Asia Filatov[1]*; Thomas C Hammond[2]; Marc A Swerdloff[2]

[1]Florida Atlantic University, Charles E. Schmidt College of Medicine, Boca Raton, Florida, USA.

[2]Marcus Neuroscience Institute, Boca Raton Regional Hospital, Boca Raton, Florida, USA.

*Corresponding Author(s): Asia Filatov

Florida Atlantic University, Charles E. Schmidt College of Medicine, Boca Raton, Florida, USA.

Tel: 561-334-3437; Email: asia.filatov@gmail.com

Abstract

Thoracic myelopathy is quite rare and can present with a false localizing sensory level. Knowledge of its clinical features is essential to a timely diagnosis and treatment. Magnetic Resonance imaging of the thoracic spine is the test of choice to confirm the diagnosis. Lack of imaging of the neural elements by computed tomography masks spinal cord injury. We present four cases highlighting diagnostic missteps and clinical features to guide clinical management before and after surgical decompression.

Thoracic Myelopathy

The purpose of these case reports is to emphasize the role of careful physical examination and proper image selection to aid in the diagnosis of thoracic myelopathy. False localizing levels complicates clinical diagnosis of spinal cord compression [1]. Thoracic Myelopathy may present with back pain, gait difficulty, leg weakness, sensory loss, and bladder or bowel dysfunction. The thoracic spinal cord conveys the corticospinal tract. Its dysfunction will manifest as weakness with hyperreflexia and spasticity in the legs (Case 1). A subacute hemi-cord syndrome (Brown-Sequard syndrome) is reported as the most common presentation [2], though none of our patients presented this way. Like Cervical Spondylotic Myelopathy, a sensory level may falsely localize, i.e., appear to arise from a segmental level below the area of pathology. Cervical cord compression may present with a thoracic sensory level [3]. Similarly, a thoracic cord compression may present with a lumbar spinal level. These false localizing signs may shift focus away from the site of pathology, leading to additional diagnostic testing, ineffective remedies, and diagnostic confusion. The presence of several neurological illnesses (e.g., cervical myelopathy and peripheral neuropathy) may complicate diagnosis of a thoracic myelopathy (Case 2). Case 4 demonstrates a thoracic myelopathy arising from an intradural extramedullary tumor presenting with a lumbar spinal level that may be confused with a progressive distal peripheral neuropathy.

Cite this article: Filatov A, Hammond TC, Swerdloff MA. Compressive Thoracic Myelopathy. Neurol Neurol Sci Open Access. 2021; 4(2): 1023.

Case 1

A 66-year-old male presented to the clinic with complaints of bilateral leg numbness tingling, and difficulty with balance. He felt his legs were jelly-like particularly when he was sitting or lying for a long time. He required a moment of standing to test his legs before attempting to walk. He was able to play tennis, but his knees would sometimes buckle. He prided himself on his athletic prowess and had sought multiple therapeutic avenues to regain his lost athleticism. He recently underwent unsuccessful lumbar epidural injections from a pain management specialist. He was puzzled and sought neurological consultation.

He had undergone decompressive neurosurgery two years prior when a Magnetic Resonance Imaging (MRI) of the spine revealed severe degenerative canal stenosis at T11-T12 with spinal cord compression, gliosis and myelomalacia (Figure 1). He underwent T11-T12 laminectomies and T11-12 medial facetectomies. His preoperative complaints of numbness in his legs and incoordination persisted one year after thoracic decompression.

Figure 1: The yellow line and white arrow point to the area of maximum compression from synovial cyst and facet hypertrophy.

His athletic past produced severe deforming osteoarthritis of his hands. He had a left carpal tunnel syndrome that caused left hand numbness while griping a tennis racket. He had non-compressive cervical canal stenosis; neck flexion elicited no electric shocks (Lhermitte's sign).

After two spinal surgeries and multiple epidural blocks, he expressed a desire to remain competitive. He denied bladder or bowel abnormalities. His exam showed marked deforming arthritis of his hands and an inoperable right sided Dupuytren's contracture. He had an enlarged arthritic right knee. There was increased tone in his legs with bilateral Babinski signs. Sensory examination showed a level to pinprick above the knees corresponding to the L2 sensory dermatome. There were brisk knee and ankle reflexes. He had a suprapatellar reflex bilaterally and a crossed adductor reflex indicating upper motor neuron dysfunction. His gait had a subtle tendency to scissor. His upper extremity strength and sensation were normal. There were absent bilateral, triceps and biceps reflexes from cervical radiculopathy. Vitamin B12 and methylmalonic acid levels were normal. A delayed post-operative MRI of the lumbar spine demonstrated decompression of the canal from previous stenosis with a stable area of myelomalacia (Figure 2). He felt improved after decompressive surgery but continued to have troubling sensory symptoms and a lack of leg dexterity required for competitive sports.
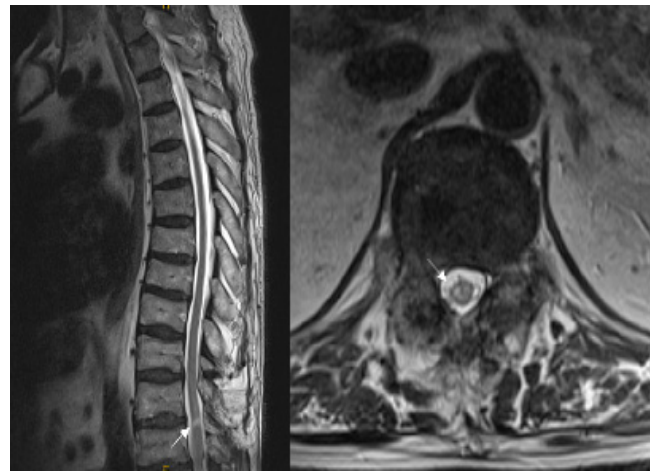
**Figure 2:** The white arrows point to myelomalacia (T2 hyperintensity).

Case 2

A 78-year-old female presented to the neurology clinic with worsening gait and balance. She had previous surgery of the lumbar and cervical spine for osteomyelitis. She had features of cervical myelopathy with hyperreflexia and spastic weakness. Distally she had a diabetic peripheral neuropathy with a level to the mid-foreleg. After cervical and lumbar surgical intervention, there was a period of gait stability. After a two-year interval, she had an abrupt decline in her ambulation. Her sensory level to pinprick had ascended symmetrically to above the knee. Diagnostic imaging of the cervical spine, brain and electromyography was performed. They pointed to small vessel cerebral vascular disease, cervical myelopathy and diabetic polyneuropathy as the cause of her decline. She had several falls and developed pain in her lower back. Her legs became progressively weaker. She had difficulty lifting or crossing her legs and no longer had the strength to stand or walk. She had Babinski signs bilaterally with unsustain clonus at the ankles. She became wheelchair bound. MRI of the thoracic spine showed severe central stenosis with abnormal T2 signal in the spinal cord at T10-T11 (Figure 3).
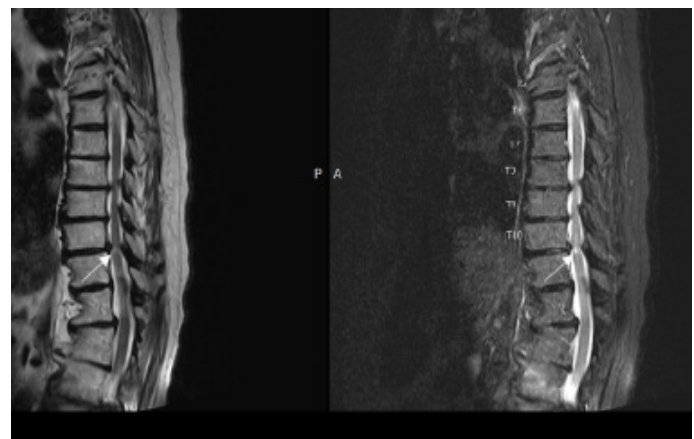
**Figure 3:** The white arrows point to myelomalacia (T2 hyperintensity).

A neurosurgery consultation agreed her deterioration might be from a thoracic myelopathy on top of pre-existing cerebrovascular disease, cervical myelopathy, and peripheral neuropathy. A T10-T11 laminectomy was performed in 2018 in the hope of optimizing her rehabilitation potential. Her gait significantly improved post operatively where she was able to ambulate with a walker. A T10 / 11 sensory level to pinprick was first noted post operatively (Figure 4).
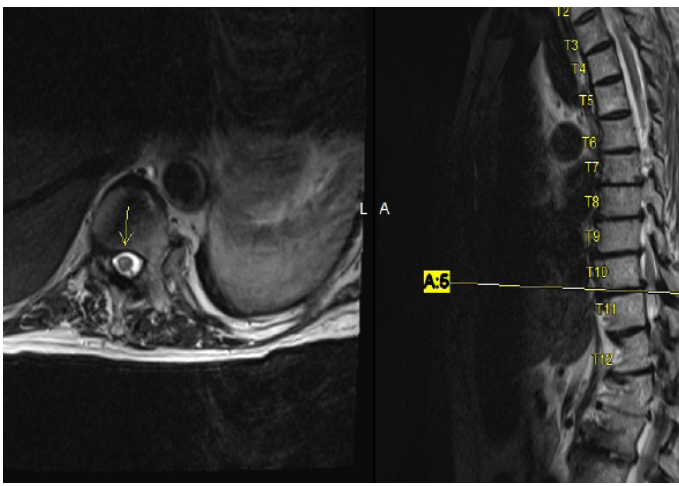
Figure 4: Postoperative MRI Thoracic spine. The yellow arrow and line indicate level of myelomalacia axial and sagittal T2 imaging.

Case 3

A 78-year-old male presented to the neurology clinic five weeks after being discharged from another hospital. He had presented there with lower back pain. CT scans of the thoracic and lumbar spine were performed with a diagnosis of thoracic spondylosis, but no further imaging was done. Subsequently, he developed increasing lower extremity paraparesis and became wheelchair-bound one week prior to presentation to our outpatient clinic. He denied saddle anesthesia. At the clinic visit, back pain was not his primary complaint. Physical examination revealed a spastic paraparesis with involuntary shaking of both legs each time he tried to move them. His hip flexion strength was 3/5 bilaterally, with a bilateral foot drop. He could not dorsiflexion his feet above gravity. His Patellar and Achilles' reflexes were brisk at 3+ without clonus. He was unable to perform heel-to-shin maneuvers. He had bilateral Babinski signs. Sensory examination showed a sensory level to pin above the knees bilaterally corresponding to the L2 level. He had no numbness or weakness of the upper extremities. There was no sensory level on the trunk. He was admitted to the hospital for further workup. MRI of the thoracic spine revealed T10-T12 severe canal stenosis with cord compression and severe bilateral neuroforaminal narrowing. There was an hyperintense T2 signal in the spinal cord at the area of compression (Figure 5).
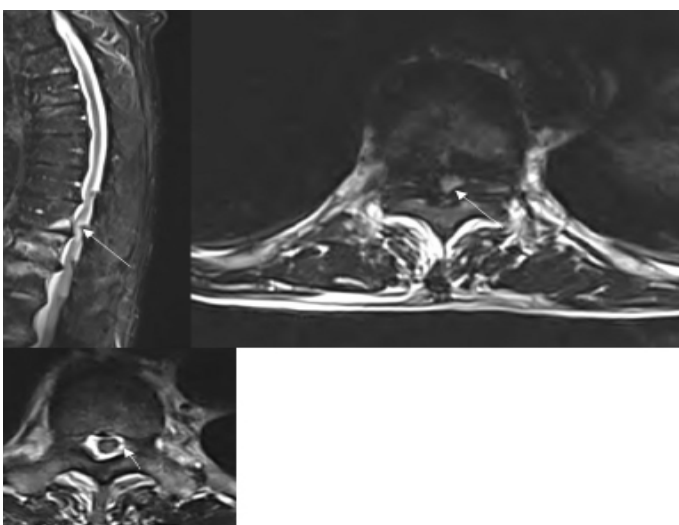

Figure 5: Note the lack of CSF around the cord (white arrow) and the reappearance of encircling CSF signal around the cord after decompression (white arrowhead).

Neurosurgery performed a T10-T12 laminectomy and Dural repair at the area of maximum compression. The dura had torn from adhesive calcified ligament and boney overgrowth. He had improvement of leg spasticity and dorsiflexion of the ankles. On discharge, he was able to ambulate 200 ft with a rolling walker with minimal assist.

Case 4

A 66-year-old male presented to the neurology clinic with bilateral leg weakness for the last year. His legs would give out and buckle at the knees. He felt unsteady but did not fall. His legs jumped up on him at night, which he attributed to his restless leg syndrome. Prior imaging of the cervical spine demonstrated a sigmoid curvature, but no compression of the cord was seen. A nerve conduction study revealed sensorimotor polyneuropathy of the axonal type. He had a remote history of right temporal infarct with a secondary seizure disorder. Routine labs showed IgM monoclonal spike and hypogammaglobulinemia consisted with history of monoclonal gammopathy of undetermined significance, normal thyroid panel, Vitamin B12 and methylmalonic acid levels. Physical examination revealed spastic legs worse on the right than the left but no weakness proximally or distally. He had decreased toe tap on the right. He had absent patella and Achilles reflexes. He had stocking hypoalgesia to above the knees level bilaterally. Scoliosis was noted on thoracic spine examination. His gait was wide based and spastic. Babinski signs were absent bilaterally. The lack of explanation for his lower extremity spasticity prompted further investigation of the thoracic spine. It revealed an extramedullary thoracic tumor at the level of T6-T7 with significant cord compression (Figure 6). He was admitted urgently to the hospital for neurosurgical consultation. He underwent T6-T7 laminectomy and tumor removal. He was discharged from the hospital for continuing rehabilitation.
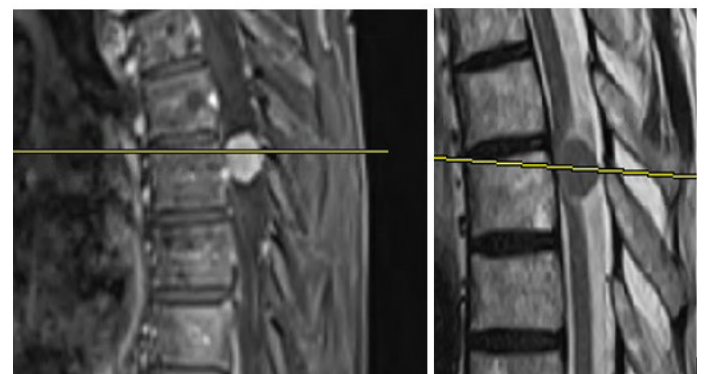
Figure 6: Thoracic extramedullary tumor with severe stenosis and cord compression at T6-T7 level. (Red arrow shows the tumor and White arrow shows the compressed cord)

Discussion

Myelopathy refers to pathology of the spinal cord [4]. Thoracic Spondylotic Myelopathy results from degenerative disease of the thoracic vertebrae [5]. Most Spondylotic and compressive changes in the thoracic spinal canal are asymptomatic.

Spondylosis can involve uncovertebral or intervertebral discs, vertebral bodies, or facets leading to spinal stenosis in the lumbosacral or cervical spine [2]. Spinal cord injury from stenosis may result from either ischemia from disruption of venous and arterial supply of the spinal cord and/or direct compression of neural elements [2]. Thoracic myelopathy is most frequently caused by thoracic ossification of the ligamentum flavum [5]. Degenerative changes may compress the cord at multiple levels (Case 2). Symptomatic thoracic disc herniation is rare [6], resulting in thoracic myelopathy at an annual incidence of one case per million [7,8]. Symptoms may develop gradually without preceding trauma or injury (Cases1-4). It is reported that the most common presentation is a hemi-cord syndrome [7,8]. Cases 1-4 did not present in that manner.

Common compressive lesions of the spinal cord may present acutely, sub-acutely or chronically. These include slow growing tumors like meningiomas and schwannomas, subacute or chronic subdural hematomas, or acute or chronic intervertebral disc protrusions [9]. It is not unusual for a sensory level on physical exam to appear distant from the level of cord pathology [9]. Confusion may divert focus away from the site of pathology [10].

Red flags prompting spinal imaging include age over 65, sphincter incontinence, new-onset gait instability, posterior ecchymosis appearing over the spinal processes, midline back pain, and previous corticosteroid use. A meticulous exam with a metal pin looking for a sensory level, gait assessment, and reflexes are necessary to formulate a diagnostic workup. A Babinski sign revealing corticospinal tract dysfunction may not be present in up to 20% of patients with severe myelopathy [11]. A sensory level may falsely implicate the lumbar spine or be dismissed as secondary to peripheral neuropathy (Cases 1 and 4). Imaging to include the thoracic spine may reveal significant findings. Even after identifying thoracic pathology, it remains a difficult decision to proceed with surgery in the setting of co-morbidities (Case 1). Imaging modalities like CT and MRI are useful for quantifying spinal canal narrowing [2]. MRI is superior to CT scanning to demonstrate cord compression (Case 3). Only 65% of patients with compressive cervical myelopathy demonstrated T2 cord hyperintense lesions [12]. Phrased differently, a myelopathy may be present without intrinsic cord signal hyperintensity 35% of the time.

The diagnosis of myelopathy requires a detailed history and physical examination to define the clinical syndrome [4]. Neuroimaging is required in cases of new-onset myelopathy and when progressive myelopathic symptoms cannot be explained [3]. The diagnosis of thoracic myelopathy is challenging. Patients with progressive myelopathy require timely surgical intervention without diagnostic delay [4]. Surgical treatment often results in considerable degree of neurological recovery (cases 1-3) [5]. Even after surgical treatment, it is reported that continued deterioration may occur in 30% of cases [13].

References

1. Rousseff RT, Tzvetanov P. False localising levels in spinal cord compression. Neuro Rehabilitation. 2006; 21: 219-222.

2. Singleton JM, Hefner M. Spinal cord compression. Treasure Island (FL): Stat Pearls Publishing. 2021).

3. Rousseff RT, Tzvetanov P. False localising levels in spinal cord compression. Neuro Rehabilitation. 2006; 21: 219-222.

4. Marquis BO, Capone PM. Myelopathy. Handb Clin Neurol. 2016; 136: 1015-1026.

5. Yan C, Tan H-Y, Ji C-L, Yu X-W, Jia H-C, et al. The clinical value of three-dimensional measurement in the diagnosis of thoracic myelopathy caused by ossification of the ligamentum flavum. Quant Imaging Med Surg. 2021; 11: 2040-2051.

6. Nakajima H, Watanabe S, Honjoh K, Kubota A, Matsumine A. Differences in clinical and radiological features of thoracic disc herniation presenting with acute progressive myelopathy. Eur Spine J. 2021; 30: 829-836.

7. Sasani M, Ozer AF, Vural M, Sarioglu AC. Idiopathic spinal cord herniation: case report and review of the literature. The journal of spinal cord medicine. 2009; 32: 86-94.

8. Daroff RB, Bradley WG. Bradley's neurology in clinical practice. Philadelphia, PA: Elsevier/Saunders. 2012.

9. Larner AJ. False localising signs Journal of Neurology, Neurosurgery & Psychiatry. 2003; 74: 415-418.

10. Jamieson DR, Teasdale E, Willison HJ. False localising signs in the spinal cord. BMJ. 1996; 312: 243-244.

11. Houten JK, Noce LA. Clinical correlations of cervical myelopathy and the Hoffmann sign. J Neurosurg Spine. 2008; 9: 237-242.

12. Matsumoto M, Toyama Y, Ishikawa M, Chiba K, Suzuki N, et al. Increased signal intensity of the spinal cord on magnetic resonance images in cervical compressive myelopathy. Does it predict the outcome of conservative treatment?. Spine (Phila Pa 1976). 2000; 25: 677-682.

13. Cheung JP, Luk KD. Complications of Anterior and Posterior Cervical Spine Surgery. Asian Spine J. 2016; 10: 385-400.