Role of MRI in Cervical Spondylotic Myelopathy with Other Pathological Findings: Case Report and Literature Review

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ABSTRACT

Introduction: Cervical spondylotic myelopathy (CSM) is a neck condition that arises when the spinal cord becomes compressed due to the wear-and-tear changes that occur in the spine as we age. **Case report:** The patient was a 52-year-old male, who complains of neck pain, paranesthesia in upper extremities, lower back pain and bilateral calf pain, muscle weakness in the lower and upper extremities, fatigue and general body pain that started four weeks ago associated with other clinical manifestation. Magnetic Resonance Imaging (MRI) of the cervical spine revealed canal stenosis and increased T2 signal within the spinal cord. CSM is a rare condition there are very few or no cases described in literature when CSM in C3/C4 is associated with a lack of B12 and other pathologies such as and hypoplasia of the mastoid air-cell with system maxillary sinus aplasia. **Conclusion:** The case described in the present study suggested that the incidence of CSM may be correlated with VB12 deficiency, particularly for cases in which the clinical manifestations and the imaging do not fully match. **Keywords: Cervical myelopathy, clinical characteristics, hypoplasia of the mastoid air-cell system, MRI, prognostic value.**

1. INTRODUCTION

Myelopathy is an injury to the spinal cord (SC) due to severe compression that may result from trauma, congenital stenosis, degenerative disease or disc herniation. Spinal cord compression can cause neurologic symptoms such; pain, numbness, or difficulty walking and is the conduit that enables communication between your brain and body and begins at the base of the brain and ends at the first lumbar vertebra. Cervical spondylotic myelopathy (CSM) is a neck condition that arises when the spinal cord becomes compressed due to the wear-and-tear changes that occur in the spine as we age. CSM is considered the commonest cause of SC dysfunction in individuals above 55 years of age and if left untreated, permanent cord damage may occur. In asymptomatic individuals, imaging evidence of spondylosis is present in 25-50% Of 50-year-old and 75-90% of individuals above 65 years of age (1). Myelopathy develops when the degenerative process causes a reduction in both the spinal canal diameter and sagittal mobility of the spinal cord (2). This results in ischemic cord compression leading to cord atrophy, neuronal loss in the grey matter and white matter demyelination. The most common symptoms include neck stiffness, arm pain, numbness in the hands and weakness of the arms and legs, stiff legs, difficulty using your hands or walking steadily, and loss of bladder or bowel control (3). On physical examination upper motor neuron signs are present including hyperreflexia, clonus, spasticity, Lhermitte's phenomenon, up-going plantar response, and Hoffmann's sign) (4). In a prospective study that assessed the relative frequencies of cervical cord compressive myelopathy, 24% of patients with no traumatic tetra-paresis or para-paresis admitted to a regional neuroscience center had cervical cord compressive myelopathy (5). Magnetic Resonance Imaging (MRI) plays an essential role in the management of patients with CSM. There have been many advances in MRI technology over the past few years and the resolution and image quality have improved greatly. With these improvements, the application of MRI in CSM has progressed in parallel, on T2weighted is usually observed. The novel MR techniques not only offer a diagnostic modality, but also can be used to predict neurological outcome and response to intervention (6). The work has been reported in line with the SCARE criteria (7).

2. CASE REPORT

A 52-year-old man who complains of neck pain, lower back pain and bilateral calf pain, muscle weakness in the lower and upper extremities, fatigue and general body pain that started four weeks ago. He also demonstrated poor to fair upper or lower extremity strength with more pronounced weakness on the right. The symptoms are constant throughout the day and these symptoms are relieved by rest. The patient did not have any bowel or bladder incontinence. Motor and sensory disturbances were found to be more expressed on the left side. Severe deficit was diagnosed on the left at C3, C4, C5 and C6 and bilaterally at C7 and C8 (grade 3-4/5 muscle strength. The cervical compression test (Spurling's test) was positive for reproduction of arm pain bilaterally. Upper motor neuron signs elicited were and bilateral upper extremity hyperreflexia, bilateral Babinski reflex, the Hoffmann's

Grade O	Signs or symptoms of root involvement but without evidence of spinal cord disease
Grade 1	Signs of spinal cord disease but no difficulty in walking
Grade 2	Slight difficulty in walking that did not prevent full-time em- ployment
Grade 3	Difficulty in walking that prevented full-time employment or the ability to perform all housework but that was not severe enough to require someone else's help to walk
Grade 4	Able to walk with someone else's help or the aid of a frame
Grade 5	Chair bound or bedridden

Table 1. The Nurick score-the higher the grade, the more severe the deficit (1)environmental, and occupational influences may play a role. These spondylotic changes may result in direct compressive and ischemic dysfunction of the spinal cord known as cervical spondylotic myelopathy (CSM

Lower limb	
Grade 0	Intact
Grade 1	Independent walker, but gait is not normal
Grade 2	Walk with support
Grade 3	Can stand, unable to walk
Grade 4	Unable to stand and walk, slight movement present
Grade 5	Paralysis
Upper limb	
Grade 0	Intact
Grade 1	Only sensory symptoms
Grade 2	Mild motor deficit with mild functional impairment
Grade 3	Major functional impairment in at least one of the upper limbs, but can perform simple tasks
Grade 4	No movement or flicker of movement, but no useful func- tion
Grade 5	Paralysis

Table 2. The Cooper myelopathy scale (1)environmental, and occupational influences may play a role. These spondylotic changes may result in direct compressive and ischemic dysfunction of the spinal cord known as cervical spondylotic myelopathy (CSM



Figure 1. A- Sagittal T2-weighted MRI reveals the spinal cord injury (curved arrow) predominantly at C3–C4 (intramedullary area of high intensity signal). B–Sagittal T1-weighted MRI demonstrates a hypointense strip on the posterior surfaces of the vertebral bodies and discs through C3-C4.C- transversal section T2-weighted at C3-CV4 demonstrate central canal stenosis and obliteration of the subarachnoid space. D- Coronar section T2-weighted at C3-CV4 demonstrate central canal stenosis and high intensity signal in spinal cord



Figure 2. A- Axial FLAIR sequence and Axial T2-weighted sequence showed T2 hyperintense signal in periatrial white matter. C- Coronar T2 sequenc showed hipoplasia of maxillar sinus and deviation nasal septum. D- Coronar T2 sequenc showed hipoplasia of of the mastoid air-cell system in left

test were positive, and a multi dermatomal decrease in upper extremity sensation during pinprick testing. Also, he had decreased grip strength and 4/5 strength in L2-S1, a positive Romberg sign with compromised coordination as evidenced by difficulty walking and placing one foot in front of the other (tandem walking). The patient demonstrated very poor standing balance, poor standing posture, bilateral lower extremity hyperreflexia with positive patellar and ankle clonus on right. He was graded as a 3 on the Nurick scale (Table 1) and 3 on the Cooper myelopathy scale [(Table 2).

A magnetic resonance imaging (MRI) of the head and the cervical spine were ordered to assess for a cerebrovascular accident and cervical myelopathy, respectively. Also, the cervical magnetic resonance imaging confirmed the diagnosis of Cervical spondylotic myelopathy (CSM). MRI imaging of the patient's cervical spine showed C3-C4 left central disc protrusion (disc herniation), which was abutting and flattening the ventral cord surface, intramedullary high signal intensity on T2-weighted imaging, mainly in the spinal gray matter causing cord deformity with associated cord edema and/or gliosis (Figure 1). The MRI also reported a mild diffuse disk osteophyte complex at C4-C5, asymmetric to the right, which led to mild-to-moderate foraminal narrowing without changes in the spinal cord (Figure 1). Laboratory examinations revealed that the level of serum Vitamin B12 was 153,5 pg/l, which was below the normal range (211-946 pg/l). The Vitamin B12 deficiency of the patient may have been the factor leading to the spinal cord damage. VB12 deficiency leads to a wide range of neuropathies, of which subacute combined degeneration of the spinal cord is particularly common. Neurological changes caused by Vit B12 deficiency may be correlated with subcortical dysfunction and neuronal metabolism changes.

Radiographic cervical spinal cord compression and hyper intense T2 in spinal cord signal abnormalities (MRI) correlate well with the presence of myelopathic findings on physical examination.

Except the changes described in the cervical spine, the patient has other associated pathologies such as: gliosis in white matter of parietal lobe. Also, maxillary sinus hypoplasia associated inflammation of the maxillary sinuses, hypoplasia of the mastoid air-cell system and deviation nasal septum that narrows the nasal passages (Figure 2).

3. DISCUSSION

Cervical spondylotic myelopathy (CSM) occurs as a late sequel to Cervical spondylosis as a result of static and dynamic factors (7). The static factors cause mechanical narrowing of the spinal canal and it includes congenital canal stenosis (AP diameter <13 mm), osteophytes, disc herniation and ligament hypertrophy. The dynamic factors include abnormal forces placed on the cord and spinal canal during flexion and extension of the cervical spine under normal physiologic loads. During flexion, the cord is susceptible to more compression in the presence of herniated discs or osteophytes, causing stretching of the cord; while during extension, cord compression could be increased by enfolding of ligamentum flavum or facet joint capsules leading to shortening and thickening of the cord. Also, cervical spondylosis could present with three syndromes: axial neck pain, cervical radiculopathy and CSM. Several authors have reported that CSM predominated in the lower cervical levels (C5-7). But in our study is at C3/4 level and moderate at the level C4/C5. Magnetic resonance imaging is choice for evaluation and diagnosis of this case study. Some researchers advocate the use of a low focal T1 and/or high T2 signal as a negative prognostic factor regarding post-treatment outcome, while others defy it. Rapid progressive myelopathy with advanced neurological impairment and "intramedullary T2-weighted hyper intensity" are common in patients with CSM and prior cerebral infarction (11). The initial diameter of the cervical spinal canal is the determining factor in the development of cervical myelopathy. Cord compression may occur if the initial diameter is 10 mm or less, but it is unlikely if the diameter of the spinal canal is 13 mm or more. Patients with spinal stenosis are unusual, and can develop an acute myelopathy following trauma without fracture or dislocation as early as the second decade of life. The older individuals described in this report developed signs of myelopathy in the presence of stenosis as a developmental phenomenon. The absence of available reserve space in the spinal canal renders the spinal cord vulnerable to the anatomical changes caused by aging and the effects of minor (12). Patients with cervical spondylotic myelopathy sometimes complain of cognitive dysfunction, which may be coincidence. However, cognitive dysfunction may be related to disorders of the cervical spine and/or spinal cord (13).

4. CONCLUSION

And in our work expect changes in spinal cord and clinical manifestation, the patient is also associated with headaches as a result of maxillary sinus aplasia and nasal septum. Also hypoplasia of the mastoid air-cell system and deviation nasal septum that narrows the nasal passages. Cervical myelopathy C3/C4 and hypoplasia of the mastoid air-cell with system maxillary sinus aplasia is an extremely rare condition. As well as the treatment of accompanying pathologies to enable a more stable life of patient. In conclusion, the case described in the present study suggested that the incidence of CSM may be correlated with VB12 deficiency, particularly for cases in which the clinical manifestations and the imaging do not fully match. In cases such as these, it may be benefitial to check serum VB12 levels. Further studies to investigate the incidence of VB12 deficiency in patients with cervical myelopathy may be of value.

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