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Feyza Ünlü ÖZKAN[1]
Tuba NAZLIGÜL[1]
Fatma Nur SOYLU BOY[2]
İlknur AKTAŞ[1]

Case Report

A LATE DIAGNOSED CERVICAL MYELOPATHY CASE

Abstract

Cervical spondylotic myelopathy is the most important cause of myelopathy in the elderly. It is defined as spinal cord dysfunction secondary to the compression of the cord caused by degenerative disease of the cervical spine. Cervical spondylotic myelopathy is a slowly progressing disease, and disability caused by cervical spondylotic myelopathy can be life threatening. Despite its severity, it may remain undiagnosed for a long time. We report the case of an 80-year-old female with cervical spondylotic myelopathy leading to paraparesis. She progressively had difficulty in walking, which worsened in the last year. She had knee pain for several years, with a known diagnosis of bilateral gonarthrosis. She had been walking with the aid of a cane when her symptoms worsened and became unable to walk independently in the past months. Her walking disability was attributed to advanced bilateral gonarthrosis. Upon physical examination, she had bilateral knee contractures, extensor plantar responses, and tonus was slightly increased in the legs. Muscle strength was also decreased in the right upper and bilateral lower extremities. Magnetic resonance imaging of the cervical spine revealed spondylotic myelopathy from C3 to C6. Cervical decompression and a stabilization operation were performed. She responded well to the rehabilitation programme. Cervical spondylotic myelopathy should always be considered for elderly patients with walking disabilities, and physicians should be aware that this disease develops insidiously and coexisting diseases leading to disability may mask the symptoms and delay the diagnosis.

Key Words: Cervical Vertebrae/physiopathology; Spinal Cord Diseases; Spondylosis/therapy.

Olgu Sunumu

GEÇ TANI ALMIŞ BİR SERVİKAL MYELOPATİ OLGUSU

Öz

Servikal spondilotik myelopati; servikal omurganın dejeneratif patolojilerine bağlı olarak spinal kordun kompresyonuna sekonder gelişen spinal kord disfonksiyonudur. Servikal spondilotik myelopati yavaş ilerleyen bir hastalıktır. Neden olduğu disabilite hastaların hayatını ciddi derecede etkilemesine rağmen uzun zaman tanı almadan kalabilmektedir. Bu makalede servikal spondilotik myelopati sonucu paraparezi gelişen 80 yaşındaki bir olgu sunulmuştur. Polikliniğimize yürüme güçlüğü ile başvuran hastanın öyküsünde son yıllarda kötüleşen ilerleyici bir yürüme güçlüğü mevcuttu. Olgunun uzun yıllardır olan diz ağrısı şikayeti ve bilinen bilateral gonartroz tanısı vardı. Şikayetlerinin artışından önce tek kanedyenle yürüyebilen hastanın son birkaç aydır bağımsız yürüyemediği, artan yürüme güçlüğünün ileri evre gonartroza bağlandığı öğrenildi. Hastanın fizik muayenesinde bilateral diz kontraktürü mevcuttu. Kas kuvveti sağ üst ekstremitede ve bilateral alt ekstremitelerde azalmış bulundu. Plantar yanıt bilateral ekstansör ve alt ekstremite kas tonusu hafifçe artmıştı. Servikal manyetik rezonans görüntülemede C3 seviyesinden C6 seviyesine uzanan spondilotik myelopati saptandı. Servikal kompresyon ve stabilizasyon operasyonu yapılan hasta rehabilitasyon programından fayda gördü. Servikal spondilotik myelopati yürüme güçlüğü bulunan yaşlı hastalarda ayırıcı tanıda mutlaka düşünülmelidir. Servikal spondilotik myelopati yavaş ilerleyen ve progresif seyir gösteren bir hastalıktır ve dizabiliteye neden olan diğer kronik hastalıklar semptomları maskeleyerek tanıyı geciktirebilir bu konuda dikkatli olunmalıdır.

Anahtar Sözcükler: Servical Vertebra/Patofizyoloji; Spinal Kord Hastalıkları; Spondilozis/tedavi.

Correspondance

Tuba NAZLIGÜL
Fatih Sultan Mehmet Training and Research Hospital, Physical Medicine and Rehabilitation Clinic, İSTANBUL

Phone: 0216 578 30 00
e-mail: drtuba_@hotmail.com

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[1] Fatih Sultan Mehmet Training and Research Hospital, Physical Medicine and Rehabilitation Clinic, İSTANBUL
[2] Fatih Sultan Mehmet Training and Research Hospital, Radiology Clinic, İSTANBUL

Introduction

Cervical spondylotic myelopathy (CSM) is defined as spinal cord dysfunction secondary to chronic progressive compression of the cervical spinal cord caused by degenerative disc disease, spondylosis or other degenerative pathology of the cervical spine (1). CSM causes progressive disability and severely impairs the quality of life. It is the most common cause of acquired spinal cord dysfunction in adults over 55 years (2). The incidence of CSM was reported to be 26.6% in a study of 585 patients with tetraparesis and paraparesis (3). CSM is a slowly progressive disease, and disability caused by CSM can be seriously devastating. Despite its severe consequences, it might remain undiagnosed for a long time. We report the case of an 80-year-old patient with CSM in whom symptoms were masked by coexisting severe bilateral gonarthrosis, which caused a delay in the diagnosis of CSM.

Case

An 80-year-old female with walking difficulty was referred from the neurosurgery clinic. Her medical history revealed that she had progressive difficulty in walking, which worsened in last years. She had knee pain for several years that was diagnosed as bilateral gonarthrosis and was walking with a cane. She did not want to have an operation and had been treated conservatively. Over time progressive worsening was thought to be caused by gonarthrosis and knee contractures. Once she had visited a neurologist for the stiffness in her legs. Cranial magnetic resonance imaging (MRI) revealed small vessel disease, and spinal stenosis and multiple intervertebral bulging discs were detected in the lumbar MRI. She was given symptomatic drugs. She became unable to walk independently in the last year. Six weeks ago, she was referred to a neurosurgeon for the pain in her arm, and MRI of the cervical spine revealed spondylotic myelopathy from C3 to C5 (Figure 1). Cervical decompression and a stabilization operation were performed and she was referred for rehabilitation. Upon physical examination, she had bilateral knee contractures (20 degrees extension limitation and 80 degrees flexion). Muscle strength was decreased in the right upper and bilateral lower extremities (manual muscle testing 4-/5). Deep tendon reflexes were found to be increased in the legs and normative in the arms. Plantar responses were extensor and tonus was slightly increased in the legs. After a 4-week inpatient rehabilitation programme, she was able to walk with the aid of a walker, and she is still following up with a home-based rehabilitation programme.

Discussion

The pathophysiology of CSM is thought to be multifactorial, with static factors causing stenosis and dynamic factors

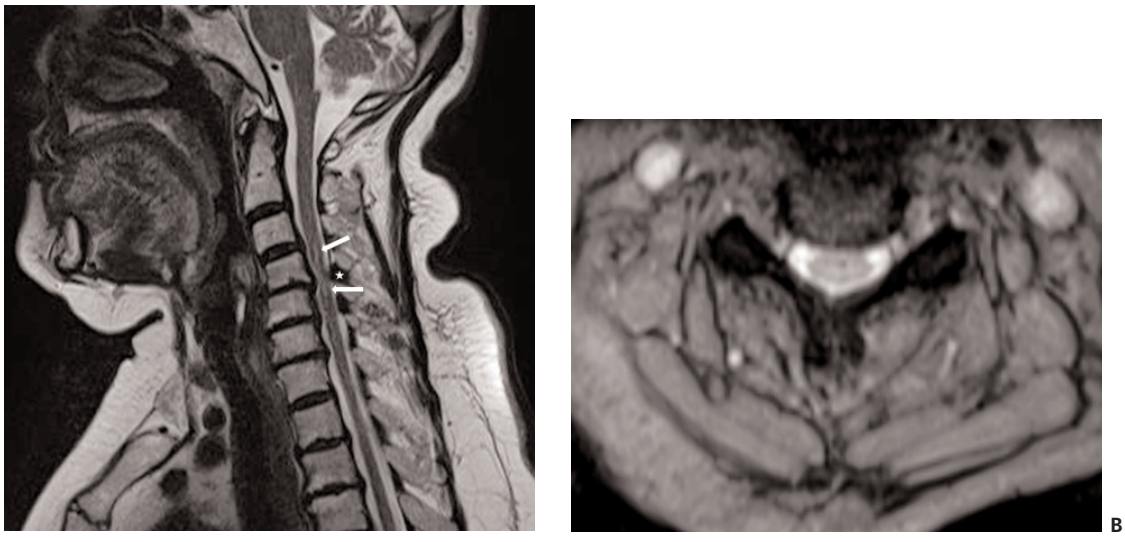

Figure 1— Cervical spondylotic myelopathy. T2-weighted **A.** sagittal and **B.** axial magnetic resonance images showing cervical spondylotic changes including spinal cord compression by disc osteophyte complexes and hypertrophic flaval ligaments (asterisk) accompanied by intramedullary high signal intensity reflecting moderate stage spondylotic myelopathy at C4-C5 (arrows).

resulting in repetitive injury to the spinal cord. The clinical findings related to the compression of the cervical spinal cord leads to a wide range of symptoms. CSM usually begins insidiously; therefore, patients often do not know the time of onset of symptoms and they may be unaware of subtle changes in balance or hand dexterity. Preliminary diagnosis of CSM could be considered with clues obtained from a detailed history and physical examination. The initial symptoms are loss of muscular strength and dysesthesia in the upper extremities, decline in fine hand skills, difficulty in writing, balance impairment and gait difficulty (4). Therefore, medical history often elicits difficulty in activities such as buttoning buttons, using keys, or changes in hand writing. Gait and balance disorders are more specific to cervical myelopathy; therefore, questioning patients in terms of these symptoms is important. Sadasivan conducted a retrospective study of 22 patients with cervical spondylotic myelopathy who were admitted to the hospital for surgical treatment of their neurological condition (5). The earliest consistent symptom in their patients was gait abnormality. Recent use of assistive devices, such as a cane or walker, may reflect progression of myelopathy. The patient in our case also had progressive walking difficulty, but her walking disability was attributed to advanced bilateral gonarthrosis, and this misinterpretation led to the progression of myelopathy causing paraparesis. Similarly, Sadasivan mentioned a considerable delay in the diagnosis of CSM and reported an average 6.3 year delay in their retrospective study. Sphincter dysfunction is also often a symptom seen later, and severe cases of CSM may present with symptoms of incontinence (4). Other common complaints are shoulder and arm pain, neck pain and stiffness and paresthesia and weakness in the upper extremities (6). Patients may have concomitant radicular signs and symptoms, which our patient also suffered; therefore, referred to a neurosurgeon. Radicular symptoms were reported in 25% of cases with the Spurling test (7).

Physical examination findings usually reveal lower motor neuron symptoms at the lesion level and upper motor neuron symptoms below the level of the lesion. Upper extremity symptoms may be unilateral but lower extremity symptoms are mostly bilateral. Sensory deficits, usually related to dorsal column function (reduced joint position and vibratory sense) and loss of pain sensation, can be elicited in the lower extremities and may contribute to the gait impairment. Physical examination should include a balance assessment with clinical tests such as heel-to-toe tandem walking and the Romberg test where disturbance shows posterior column involvement.

Imaging modalities including plain radiographs, computed tomography (CT) and MRI are all important in the diagnosis and each may provide helpful information in patients with CSM. Initial evaluation includes anteroposterior and lateral radiographs of the cervical spine where narrowing of disc space, osteophytes, facet arthrosis and ossification of the posterior longitudinal ligament may be visualized together with the evaluation of cervical lordosis. CT improves the depiction of bone spurs on the spinal canal and sometimes shows cord compression by disc osteophyte complexes. However, because of superior soft tissue resolution and multiplanar capability, MRI has replaced CT and became the mainstay in noninvasive evaluation of patients with painful myelopathy. MRI provides valuable information about disc herniation, morphology and internal signal of the spinal cord. Many authors have considered that intramedullary high signal intensity in MRI might represent a variety of histologic changes, including edema, ischemia, demyelination, gliosis, microcavities and cavities (4,5,8). In our patient, MRI showed apparent cervical spondylotic changes including loss of cervical lordosis and disc osteophyte complexes between C3-C5. The cervical spinal canal was narrowed and the spinal cord was compressed by disc osteophyte complexes anteriorly and hypertrophic flaval ligaments posteriorly which were apparent at the C4-C5 level. T2-weighted images also showed the intramedullary high signal of the spinal cord at the C4-C5 level reflecting moderate stage spondylotic myelopathy (Fig. 1). There was no pathologic finding on the T1-weighted images.

Differential diagnosis is important in CSM. Amyotrophic lateral sclerosis is one of the most frequently involved diseases to rule out (8). Neoplasia, multiple sclerosis, spinal cord infarction, syringomyelia and vitamin B12 deficiency should also be considered in the differential diagnosis (8). Parasagittal cerebral lesions, multiple strokes and brainstem strokes can mimic CSM, although onset tends to be acute (9).

The natural history of CSM has not been clearly defined. The debate about the course of CSM and the efficacy of conservative treatment is still ongoing. A longer duration of symptoms has been reported to present a poor prognosis (5,10). In a recent investigation Oshima (11) suggested that 62% of patients with mild CSM will not deteriorate or undergo surgery 10 years after diagnosis and that malalignment and instability are adverse prognostic factors. Matz, PG (10) in a systematic review of the literature regarding the natural history of CSM suggested a mixed course with many patients having quiescent disease for long periods of time and others experiencing a stepwise decline. Given the lack of definitive data regarding the natural history of CSM it is crucial to inform patients prior to conservative treatment about the potential for progression and possibility of spinal cord injury.

Conservatively treated patients should be advised to avoid hyperextension injury or even minor trauma, which may lead to progression of CSM. Physical therapy agents, exercise programmes and cervical collars may be used in the conservative treatment but there is limited evidence for them (8) and prolonged collar immobilization may result in muscle deconditioning.

The duration of the patient's complaints should also be considered while planning conservative treatment; chances to benefit from conservative treatment decrease as the duration of symptoms increases. Surgery should be considered in patients with refractory symptoms despite conservative measures, progressive symptoms, bowel or bladder dysfunction, or overt weakness. The number of surgical procedures in elderly patients has been increasing as the population ages. Kanchiku et al. investigated 43 cases with an average age of 79 years that underwent surgical treatment for CSM. They reported favourable postoperative outcomes and stated that when the surgical methods were compared selective laminoplasty is less invasive and well-suited for the elderly compared to conventional C3-C7 laminoplasty and anterior decompression and fusion (12).

In conclusion, CSM is the most common cause of spinal cord dysfunction in the elderly and underdiagnosis is frequent due to the vagueness of symptoms. Gait disturbance, which is one of the most common symptoms of CSM, might be mistakenly attributed to a coexisting disease such gonarthrosis or coxarthrosis or simply to ageing. Therefore, clinicians should be cautious and gait disturbance should be investigated with high suspicion of CSM.

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