Golfer's Myelopathy: Is It Like Surfer's Myelopathy?

Sir,

Myelopathy describes any neurologic deficit related to the spinal cord. It is seen in various conditions including, trauma, inflammation, infections, vascular disorders, metabolic abnormalities, degenerative spine conditions, rheumatoid disorders, and paraneoplastic syndrome.[1] Recently, the incidence of sports-induced myelopathy has been increasing, particularly in association with surfing, swimming, cheerleading, back handspring, and yoga.[2,3] Here, we present a case of thoracic myelopathy following a prolonged and strenuous golf session.

A 47-year-old female amateur golfer reported to the neurology department. She had recently taken up golf and played 36 holes for the first time 2 weeks prior to admission [Figure 1a]. Several hours after playing, she experienced mild thoracic back pain (2 on the Numerical Rating Scale for Pain). 10 days afterwards, she developed abnormal sensation on both soles of the feet and buttocks while walking and sitting. She had no sphincter-related complaints. Previous history included a thyroidectomy due to papillary cancer at the age of 32. For this, she took 0.175 mg of levothyroxine daily. Neurologic exam revealed bilateral hyperesthesia on the lateral aspect of the tibia and foot, posteromedial thigh, and perineal area. The sensations of position and vibration were normal for all joints; Romberg test was negative. Deep tendon reflexes were brisk for both knees. Clinical examination revealed no motor weakness in the lower limbs. Laboratory tests showed a decreased level of thyroid stimulating hormone (0.02 μIU/ml, normal range 0.3-4.0 μIU/ml) with normal T3 and free T4 levels, suggesting subclinical hyperthyroidism. Cerebrospinal fluid analysis revealed a white blood cell count of 14/μL with monocyte predominance (100%), red blood cell count of <3000/μL, and immunoglobulin G index of 0.86; oligoclonal bands were absent. Spinal magnetic resonance imaging (MRI) showed hyperintensities on T2-weighted images (T2WI) at the level of thoracic 5-6 involving the central cord [Figure 1b and 1e; with enhancement, Figure 1c and 1f]. Corresponding lesion identified in T2WI was not positive in

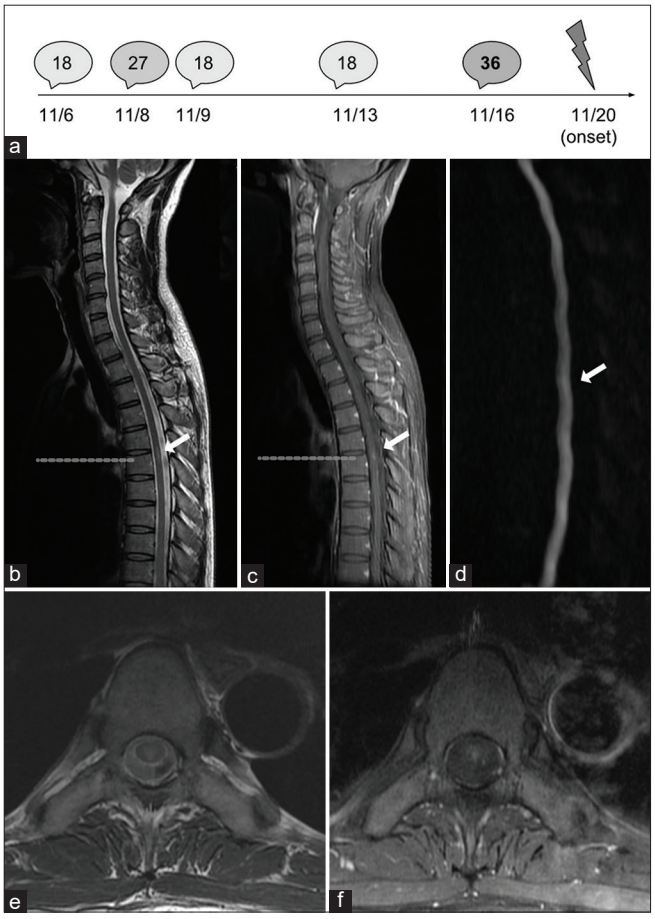

Figure 1: Patient's golf schedule (a) and cervicothoracic spine magnetic resonance image 10 days after symptom onset (b, c, d, e, and f). Patient played the golf strenuously before the presentation of symptoms (a). The holes of playing golf are depicted on the upper row and the date of playing golf are described on the lower row (a). The lesions are located at the thoracic 5-6 level seen in T2-weighted (b, arrow) and gadolinium-enhanced T1-weighted sagittal images (c, arrow). Corresponding lesion identified in T2WI was not positive in diffusion weighted images (d). The identical lesion is centrally located in T2-weighted (e) and gadolinium-enhanced T1-weighted axial images (f). The gray dotted line in b and c are scout lines of e and f, respectively.

diffusion weighted images (DWI, Figure 1d]. Brain MRI was normal. Autoimmune profiles, including anti-aquaporin 4 antibody and hypercoagulability were negative.

The patient was diagnosed with golf-induced myelopathy and treated with intravenous methylprednisolone (1 g/day) for 5 days. Risk factors for multiple sclerosis and thyroid-associated myelopathy were extremely low (negative for oligoclonal bands, normal brain MRI, normal thyroid hormone levels). Neurologic examination 20 days after discharge revealed no objective deficits.

The identification of spinal cord hyperextension is one of the most important findings in sports-related myelopathy. To date, spinal cord hyperextension has been suggested as the cause of neurologic deficits in surfing, swimming, cheerleading, and back handspring.[2-4] Modern golf swing technique favors limited pelvic rotation compared to shoulder rotation during backswing, and hyperextension of the spine during follow-through.[5] In addition, the compressive load to the low back can be 8 times body weight, further exacerbating spinal hyperextension.[5] This large and repetitive movement can cause ischemic insults to the spinal cord.

Our patient's lesion was located in the middle thoracic spine, an uncommon site for surfer's myelopathy. According to previous studies on surfer's myelopathy,[4] T2-signal abnormalities range from T5 to T10, which can reflect the anatomical variation of the Adamkiewicz artery. Furthermore, gadolinium enhancement was identified in 4 of the 11 patients.[4] The thoracic location of our patient's lesion corresponded to that found in a previous report,[4] although it is atypical for surfer's myelopathy. In addition to the location of lesion, positive DWI lesion has not been always revealed in surfer's myelopathy,[6] which conforms with our patients.

Surfer's myelopathy is associated with activities requiring repetitive spinal hyperextension. Physicians should be aware of sports-related spinal cord injuries as the popularity of sports, including golf grows.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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