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Case report

Surfer's myelopathy: A rare presentation in a teenage gymnast and review of the literature

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ABSTRACT

This article describes a novel setting for a rare nontraumatic spinal cord injury referred to as Surfer's myelopathy. The patient is a 16 year-old female cheerleader who presented following a gymnastics practice where she was repeatedly performing back handsprings. She demonstrated progressively worsening midthoracic back pain and evolving paraplegia and hypesthesia of the lower extremities. Magnetic resonance imaging findings were consistent with T5-T7 spinal cord ischemia at 8 h and 16 h after symptom onset. The clinical and radiologic findings for this patient are consistent with previous case reports of Surfer's myelopathy. The authors also provide a summary of the current literature describing Surfer's myelopathy, which to date includes 64 reported cases. The diagnosis of nontraumatic spinal cord injury, referred to as Surfer's myelopathy, in a gymnast highlights the importance of greater physician and patient awareness of this rare condition.

1. Introduction

Surfer's myelopathy (SM) was first described in 2004 by a group of physicians within the Pacific Islands that reported a series of nontraumatic spinal cord injuries associated with people participating in first time surfing lessons [1]. The clinical syndrome begins with a nontraumatic back pain and evolves to include paraparesis and sensory disturbance [1–3]. The duration of symptoms is variable with some sources reporting resolution by discharge after 1 hospital day ranging to permanent neurologic dysfunction [1–3].

The etiology of SM has not been histopathologically verified but theories exist based on radiographic and clinical findings. The generally accepted theory of pathogenesis is related to a hyperextension induced ischemic spinal cord injury, supported by the acuity of presentation and uniform presence of restricted diffusion on magnetic resonance imaging (MRI) [2,3]. The ischemic injury is theorized to result from hyperextension of the back while paddling prone on a surf board, which induces dynamic vascular compression, vasospasm, or a thrombosis induced infarction of the artery of Adamkiewicz [2]. The variation in the level of spinal cord infarctions described within the literature is speculated to relate to the variation in collateralization of the radicular arteries [2,4] and due to the inconsistent level of origin for the artery of Adamkiewicz [3]. There is a notable predominance of lower thoracic spinal ischemia in the setting of SM within the literature [1,5–7], correlating with the most common location of origin for the artery of Adamkiewicz at T9-T12 in 75% versus having an origin at T5-T8 in 15% and L1-L2 in 10% of patients [8].

2. Methods

Literature review was performed on Surfer's myelopathy through a PubMed search for articles recalled by keywords Surfer's Myelopathy and an Ovid search for Surfer's Myelopathy. The literature search resulted in 14 articles, 3 retrospective reviews, 9 case reports, 1 case series, and 1 review article.

3. Case report

A 16-year-old female Caucasian female cheerleader with history of diabetes mellitus type 1, taking injectable insulin, presented

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to an outside hospital emergency department due to progressive severe midthoracic back pain that began during a gymnastics practice. During practice, the patient completed a series of backhand springs and reported acute-onset midline needle-like paresthesias within the midthoracic region of the spine. The paresthesias evolved to severe shooting pain on extension of the back. Despite this she continued to practice and after completing a round-off back tuck maneuver she reported coming down hard on her feet and noted her discomfort had become a constant severe sharp pain in the mid-thoracic region.

Upon arriving to the outside hospital she was able to ambulate to the emergency department waiting room. After waiting ten minutes in the waiting room she required assistance walking to the evaluation room due to right foot drop, evolving hypesthesia distal to the ankle, and loss of proprioception in the right foot. Over the next hour the hypesthesia ascended the right leg to the level of the T12 dermatome and subsequently began to involve the left foot and leg sequentially in a similar pattern. Finally the hypesthesia ascended bilaterally to the level of the T8 dermatome with paresthesias persisting within the T7 dermatome. She had 0/5 strength bilaterally within the lower extremities at time of evaluation at the outside hospital. She was subsequently transferred to the University of Kentucky Healthcare center for further management.

Following transfer, her neurological exam revealed an American Spinal Injury Association Impairment Scale (ASIA) B from T8 and below, with reported hypesthesia of perianal sensation. Paresthesias were reported within the T7 dermatome and anesthesia to sharp/dull, light touch, proprioception, and vibration within all caudal dermatomes with preservation of normal perianal sensation. She had 0/5 strength bilaterally on attempted hip flexion, knee extension, knee flexion, dorsiflexion, and plantar flexion. Hoffman and Babinski signs were absent and she had 0 bilateral patellar and ankle reflexes. She exhibited tenderness to palpation of the spinous processes over the mid-thoracic region. Her rectal tone was determined to be mildly diminished. She had a normal cranial nerves exam and upper extremity motor exam was within normal limits for sensation, motor, and coordination tests. Thoracolumbar MRI revealed subtle T5-T7 spinal cord T2 signal hyperintensity at 8 h after symptom onset (Fig. 1). At 16 h after symptom onset a repeat MR thoracic spine was performed and included diffusion weighted imaging (DWI) sequences and T1 sequences with contrast. This scan demonstrated no abnormal enhancement within the T5-T7 ventral spinal cord and the DWI sequence was consistent with ischemia of those levels (Fig. 2A–C). The patient was subsequently started on a 4 day high-dose intravenous methylprednisolone dosage regimen. The patient had not urinated or experienced incontinence at 14 h after the onset of symptoms and urinary retention was evident upon bladder scan therefore a Foley catheter was placed. She had no fecal incontinence throughout her stay despite a diminished rectal tone. Lumbar puncture was completed to evaluate for any signs of transverse myelitis. Cerebral spinal fluid (CSF) was found to be normal except for a decreased ACE of 3 IU. CSF had 0 RBC and 1 WBC. Glucose was elevated in accordance to patient's blood glucose. Gram stain showed no microorganisms and CSF viral cultures were PCR negative. Serum neuromyelitis optica antibody was also negative. A repeat thoracic MRI, including contrasted and diffusion weighted imaging was obtained 5 days after initial injury, showing evolution of the patient's spinal cord injury (Fig. 3).

She was started on gabapentin and experienced good pain control. She remained inpatient for 6 days without any improvement in her motor or sensory exam. She regained some ability to void, but still required in and out catheterization. She was evaluated by PM&R and prescribed multipodus splints for prevention of plantar flexion contracture as well as heel protectors. She was discharged to an inpatient rehab facility.

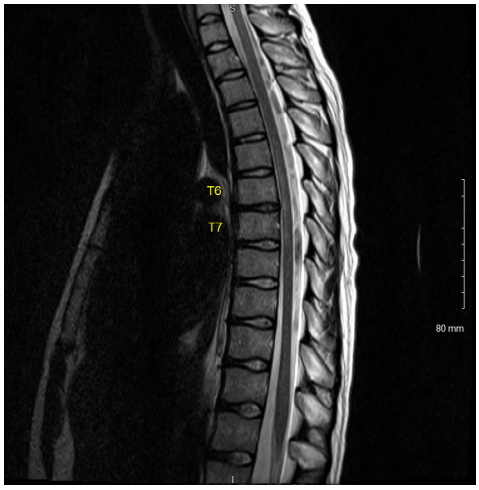

Fig. 1. T2 weighted sagittal MRI obtained 8 h after injury.

4. Results

The results of this brief literature search reveal that there are currently 64 reported cases of Surfer's myelopathy (Table 1). None of these case reports or retrospective reviews make mention of other activities outside of surfing as predisposing to this proposed non-traumatic hyperextension related mechanism of injury.

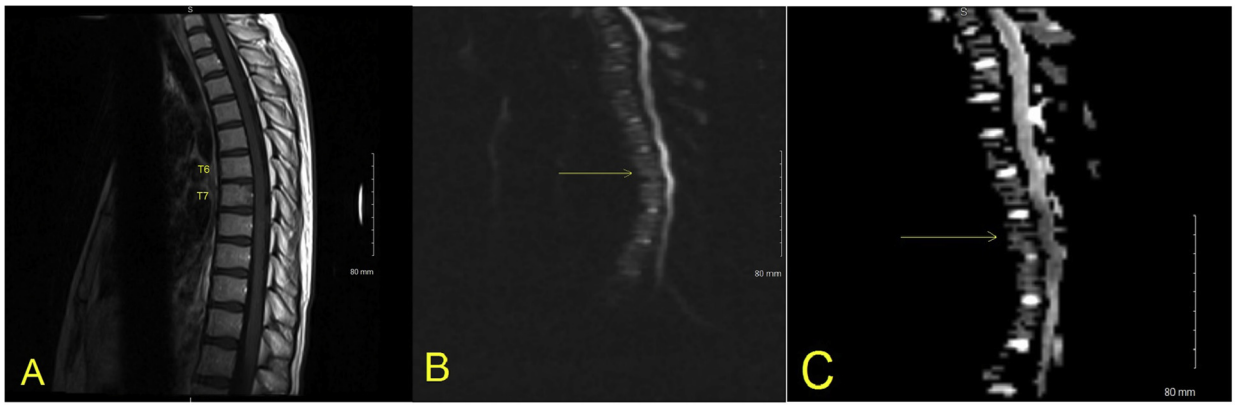

Fig. 2. T1 sagittal MRI with contrast obtained 10 h after injury (A). Sagittal diffusion weighted imaging. B400 (B), ADC (C). Arrow at site of restricted diffusion.

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