Nontraumatic Myelopathy Associated With Surfing

Israel Avilés-Hernández, MD^{1,2}; Inigo García-Zozaya, MD²; Jorge M. DeVillasante, MD²

¹University of South Florida, College of Medicine, Tampa, Florida; ²James A. Haley VA Hospital, Tampa, Florida

Received June 12, 2006; accepted October 9, 2006

Abstract

Background/Objective: Ischemic nontraumatic spinal cord injury associated with surfing is a novel diagnosis believed to be related to prolonged spine hyperextension while lying prone on the surfboard. Only 9 cases have been documented. This report features possible risk factors, etiology, diagnostic imaging, and outcomes of surfer's myelopathy.

Design: Case report.

Results: A 37-year-old man developed T11 American Spinal Injury Association (ASIA) A paraplegia shortly after surfing. The clinical history and magnetic resonance imaging findings were compatible with an ischemic insult to the distal thoracic spinal cord. Our patient did not have any of the proposed risk factors associated with this condition, and, contrary to most reports, he sustained a complete spinal cord lesion without neurological recovery by 8 weeks post injury.

Conclusions: Surfer's myelopathy, because of its proposed mechanism of injury, is amenable to medical intervention. Increased awareness of this condition may lead to early recognition and treatment, which should contribute to improved neurological outcomes.

J Spinal Cord Med. 2007;30:288–293

Key Words: Spinal cord ischemia; Ischemic myelopathy; Paraplegia; Diffusion magnetic resonance imaging; Surfing

INTRODUCTION

Surfer's myelopathy is a relatively new entity first described by Thompson et al in 2004 (1). They presented a case series of nontraumatic spinal cord injury affecting inexperienced surfers. This case report of a 37-year-old man with surfer's myelopathy illustrates distinctive features from the original case series with regard to possible risk factors, etiology, and outcomes.

CASE REPORT

A 37-year-old man residing in Hawaii took surfing classes and completed the first 2-hour lesson without any traumatic events. After a 20-minute uneventful drive back home, he began to feel mild low back pain (rated 1/10 on a scale of 1 to 10). He took a bath, during which the pain progressed to 10/10 in a 15- to 20-minute period and paresthesias developed in both lower extremities.

Physical Examination

In the emergency room, the man's temperature was 98.1°F, pulse was 72 beats/min, regular respiratory rate was 16 breaths/min, and blood pressure was 125/85 mmHg. Mental status, speech, and cerebellar and cranial nerve examinations were normal. The neck was soft without rigidity. Respiratory, cardiovascular, and abdominal examinations were normal. The back showed no spinal deformity, and the skin was intact.

Muscle stretch reflexes were present (2+) at patellar tendon but not elicited at the ankles. Sensory examination showed decrease sensation in both lower extremities in a nonspecific dermatomal distribution. The patient had 3/5 strength in lower extremities and was unable to stand or walk. The rectal examination demonstrated normal tone. Within 2 hours, the symptoms progressed to complete sensory loss in his lower body, paralysis of both lower extremities, and loss of bowel and bladder control.

Past History

Past medical history was unremarkable. The patient denied any travel, history of trauma, or illness, including cold or flu-like symptoms during the preceding weeks

Please address correspondence to Inigo García-Zozaya, MD, San Diego VA Healthcare System, Spinal Cord Injury Service (128), San Diego, CA 92161; phone: 858.552.8585, ext. 3112; fax: 858.552.4315 (e-mail: Inigo.Garcia-Zozaya@va.gov).

Figure 1. Sagittal T1W and T2W images at 15 hours after the beginning of symptoms demonstrated mild fusiform expansion of the distal spinal cord and increased T2W signal (arrows).

prior to the event. At the time of the spinal injury, he was an army officer stationed in Hawaii for 2 years and performing regular physical training as required. He had practiced “bodysurfing” for the previous 2 years but not “regular” surfing on a surfboard.

Diagnostic Workup

Laboratory tests were unremarkable, including complete blood count, chemistry panel, liver function tests, and coagulation parameters. Cerebrospinal fluid (CSF) analysis showed the protein to be 52 mg/dL, the white blood cell count to be $29 \times 10^9/L$, the red blood cell count to be $68 \times 10^{12}/L$, and the glucose to be 70 mg/dL. CSF culture yielded no growth after 4 days. CSF serology was negative for HTLV I-II and nonreactive for presence of T-cell virus antibody in serum. CSF protein electrophoresis showed 1 gamma band, but no gamma band was present in serum.

Lumbosacral spine radiographs (antero-posterior, lateral, and oblique views) revealed normal curvature, normal height and alignment of vertebral bodies, well-preserved disk spaces, and normal pedicles and posterior elements, including apophyseal processes and joints. Radiographs also revealed normal sacroiliac joints and no evidence of abnormal paraspinal soft-tissue densities. Computed tomographic scan of the lumbosacral spine showed mild degenerative disk disease at L4-L5 and L5-S1, without canal stenosis or nerve root compromise, and there was no evidence of spondylolysis, spondylolisthesis, fracture, or soft-tissue abnormalities. Initial magnetic

resonance imaging (MRI) of the lower thoracic and lumbar spine performed approximately 15 hours after the beginning of symptoms demonstrated a mild degree of fusiform enlargement of the distal spinal cord and subtle increased T2-weighted signal (T2W) at the distal conus medullaris region (Figure 1).

Repeat MRI examinations in the next 2 days revealed progression of increased T2W signal within the distal spinal cord up to level T10 (Figure 2). Follow-up MRI examination at approximately 4 weeks performed with and without IV gadolinium demonstrated a new finding of patchy increased T1-weighted (T1W) signal at the distal spinal cord prior to administration of intravenous gadolinium contrast consistent with hemorrhagic products (Figure 3) and abnormal enhancement within the distal cord (Figure 4). Also noted was progression and cephalad extension of increased T2W cord signal up to level T6 (Figure 5). Diffusion-weighted images (DWI) (Figure 6) demonstrated no evidence of restricted water diffusion in the abnormal segment of the spinal cord.

All the above findings noted at 4 weeks were compatible with evolution of an ischemic lesion to chronic stage. Brain MRI with and without gadolinium contrast was normal. A course of steroids (dexamethasone) was part of initial management treatment. After the patient was admitted for spinal cord injury rehabilitation, the neurological examination revealed normal strength for bilateral upper extremity myotomes from C5 to T1 and flaccid paralysis in lower extremity myotomes from L2 to S1 bilaterally. Sensation was intact to pin prick, light

Figure 2. Sagittal T2W MRI on day 2 demonstrated cephalad progression of increased T2W signal extending from the tip of the conus to level T10 (arrows).

touch, and vibration from C2 to T11 and absent from T12 to S5 bilaterally. Muscle stretch reflexes were normal and symmetric for biceps, triceps, and brachioradialis, but the patellar and Achilles reflexes were absent bilaterally. The rectal examination showed absent sensation and absent voluntary contraction with decreased sphincter tone. Bulbocavernosus reflex was absent. Babinski response was absent bilaterally. The examination was compatible with T11 American Spinal Injury Association (ASIA) A paraplegia. Urodynamics study showed a neurogenic bladder with hypocontractile detrusor and no flow. Eight weeks post injury, the neurological examination remained unchanged. The patient successfully completed an acute spinal cord injury inpatient rehabilitation program and is functioning independently from the wheelchair level.

DISCUSSION

Various types of surfing injuries have been described in the medical literature (2–5), mostly lacerations and contusions. Traumatic spinal cord injuries associated with surfing have been reported by Nathanson et al (2), who described 7 cases of cervical spine fractures due to the surfer's head striking the ocean floor, 3 of which resulted in permanent neurological deficits and 4 of which resulted in thoracic spine fractures.

To our knowledge, this patient and those in the series described by Thompson et al (1) represent the only reported cases of nontraumatic spinal cord injury associated with surfing. However, considering that

Figure 3. T1W sagittal image without IV contrast medium at 4 weeks demonstrated increased T1W signal at the distal spinal cord consistent with hemorrhagic products (arrow).

surfing is popular worldwide and has been practiced in Hawaii for generations, it is likely that many other cases may not have been recognized, especially before the advent of MRI or in locations in which this imaging modality is not readily available.

The case series reported by Thompson et al (1) describes 9 patients diagnosed with surfer's myelopathy from June 1998 through January 2003. All individuals were young (21–30 years); 8 were male, and 1 was female. None had underlying pathology, such as spondylosis or spondylolisthesis, and none had overt trauma associated with the onset of their symptoms. The common factor was their inexperience, that is, all of them were first-time surfers. All except one were Japanese, possibly a reflection of Hawaii's tourism demographics. The patients developed the symptoms while surfing or shortly thereafter, presenting with low back pain, lower extremity weakness, sensory changes, and urinary retention. On discharge, 4 patients had complete recovery and 4 patients had persistent lower extremity weakness with normal sensory function (3 of whom had persistent urinary retention). One patient remained paraplegic. In all patients, the MRI demonstrated abnormal signal change in the lower thoracic region of the spinal cord without evidence of acute or chronic canal stenosis. The proposed mechanism of injury is an ischemic event to the watershed zones of the spinal cord, brought about by the prone hyperextended posture associated with surfing.

Figure 4. Post IV gadolinium sagittal T1W MRI at 4 weeks demonstrated new abnormal enhancement of the distal thoracic cord consistent with infarct in chronic stage (arrows).

Our patient was also a beginner surfer who performed all the commonly required activities for the sport, including paddling with his upper extremities while lying prone on the surfboard with his neck and back hyperextended. He also developed symptoms shortly after surfing. Although a first-time surfer, he did have experience in “bodysurfing,” which may suggest better body conditioning for the practice of surfing. In contrast to most cases reported, our patient did not have any return of neurological function by 2 months post injury.

Potential risk factors mentioned by Thompson et al (1) that may predispose surfers to ischemic cord injury include body habitus (most of the patients in the case series were thin with underdeveloped musculature), dehydration (related to travel and prolonged hours at the beach), and long-distance travel (associated with hypercoagulable state and deep venous thrombosis). Interestingly, none of these risk factors applied to our patient. He was physically fit with a well-proportioned muscular body habitus. He drove just 20 minutes from his home to the beach to take the surfing lesson and had a low risk for dehydration. There was no history of prolonged air travel for 2 years prior to the injury. He is a lifelong nonsmoker and maintained an active lifestyle, minimizing his risk for coagulation problems.

Thompson et al (1) proposed arterial insufficiency related to spine hyperextension as the most likely etiology of surfer’s myelopathy. The anesthesiology

literature has reported spinal cord ischemia with supine hyperlordosis positioning while in the operating room (6,7). Roberts et al (6) described a patient who presented with dysesthesias and sensory changes at the thoracic and lumbosacral levels after being positioned in hyperlordosis for 6 hours for abdominal (nonaortic-related) surgery. An MRI revealed a thoracic-level spinal cord infarction. Although hypotension may have been related to these anesthesia-related cases, they share with surfer’s myelopathy the positioning of the spine in hyperextension. More studies are needed to confirm a relationship between spine hyperextension and spinal cord ischemia.

The blood supply to the spinal cord includes the anterior spinal artery, which extends from the ventral medullary area (anastomosis of 2 branches from the intracranial vertebral arteries) down through the conus medullaris, and supplies the anterior two thirds of the cord. From the lower cervical level downwards, this arterial system consists of anastomosing branches from the anterior radicular arteries (eg, Adamkiewicz artery). The 2 posterior spinal arteries supply the posterior one third of the cord. They commonly originate from the vertebral arteries and create an anastomotic loop with the anterior spinal artery at the level of the conus medullaris. In addition, the posterior spinal arteries receive contributions from numerous posterior radicular arteries. Already intramedullary, the anterior and posterior spinal arteries form a vascular plexus (peripheral vasocorona) that encircles the cord. Also, sulcal (central) arteries penetrate

Figure 5. Sagittal T2W MRI at 4 weeks demonstrated progression and further cephalad extension of increased of T2W signal up to level T6 (arrow).

the cord while traveling through the anterior median fissure. An anatomical watershed zone susceptible to infarction is described at the midthoracic portion of the spinal cord from levels T3 to T8 due to poor vascularization by intercostal arteries (8). However, more recent reports from autopsy after cardiac arrest and hypotension suggest that the lumbosacral cord (conus medullaris) is more susceptible to ischemic events (9). Venous drainage of the spinal cord follows a similar pattern to that of its arterial supply (10). Intramedullary veins drain into the internal (epidural) vertebral plexus, which drains into 3 longitudinal anterior and posterior spinal veins that are freely interconnected. These veins drain cephalad into the posterior fossa dural sinuses and segmentally into medullary veins, which drain into the intervertebral veins and then into the external vertebral venous plexuses and the inferior vena cava and azygos system.

Proposed vascular mechanisms of injury by Thompson et al (1) include avulsion of perforating vessels, vasospasm of the artery of Adamkewicz, and transient ischemia in areas of borderline perfusion as a result of tension on the spinal cord with hyperextension. Other possible mechanisms include inferior vena cava obstruction and fibrocartilaginous embolism. The former may occur secondary to compression by the liver while lying prone over the surfboard aggravated by concomitant prolonged Valsalva maneuver during paddling, resulting in increased retrograde venous pressure in the epidural

Figure 6. DW sequence trace diffusion image obtained at 4 weeks demonstrated normal trace diffusion signal within the distal spinal cord (arrow) consistent with evolution of the spinal cord infarction to a chronic stage.

venous plexuses leading to infarction. Fibrocartilaginous embolism is an important consideration in spinal cord infarction, especially in the presence of back pain, delayed onset and gradual progression of symptoms as seen in our patient, and the variable outcomes reported by Thompson et al (1). This rare clinical entity, described in detail by Han et al (11), is the result of retrograde embolization of the central (sulcal) artery by nucleus pulposus material from the intervertebral disk. Such embolism has been occasionally related to physical effort, and, as in our patient, the clinical course is characterized by the sudden onset of back pain followed by a relatively rapid progression of neurological deficits. The diagnosis of fibrocartilaginous embolism myelopathy requires histologic confirmation, but this is a high-risk procedure. At the present time, there is no treatment available for this type of embolism. Finally, the relation between the 1 gamma band found in CSF electrophoresis with the development of surfer's myelopathy or the relation between this myelopathy as a clinically isolated syndrome suggestive of multiple sclerosis cannot be established at this time but appears highly unlikely (12), especially in the absence of any other findings to suggest an acute demyelinating process. Thompson et al (1) did not report CFS electrophoresis in their cases. Long-term follow up of surfer's myelopathy patients will be necessary for such purposes.

Concluding that surfer's myelopathy is the result of an ischemic event, Thompson et al (1) developed an intensive care protocol with rapid MRI, empiric steroids,

spinal angiogram, and hemodynamic management similar to that used for vasospasm after subarachnoid hemorrhage (hemodilution, hydration, and induced hypertension) and urologic evaluation with urodynamics study. However, taking into account the inherent risk and limited availability of spinal angiography and the probable ischemic etiology of surfer's myelopathy, the use of several MRI modalities for early diagnosis and treatment seems to be more practical. MRI with DWI is the single most important imaging study in the initial evaluation of suspected spinal cord infarction (13–17). MRI with DWI is extremely sensitive in detecting restricted water diffusion at the intracellular level. In the setting of acute ischemia, the cell membrane ion pump fails and excess sodium enters the cell, which is followed by a net movement of water from the extracellular to the intracellular compartment, resulting in cytotoxic edema. This process of restricted water diffusion occurs very shortly, within minutes after the ischemic insult. DWI will be positive within the first few hours, whereas the initial detectable change in the T2W signal is subtle and takes longer, approximately 8 to 24 hours (14,17). Once it is established that there is restricted water diffusion indicating evidence of acute infarction, aggressive medical management can be undertaken, possibly to include thrombolytic therapy (18). MRI with DWI is a very well-established imaging technique that is widely available in most community hospitals in the United States.

CONCLUSION

Nontraumatic acute myelopathy associated with surfing is a novel entity, with only 9 cases reported in the literature since 2004. It is proposed to be the result of an ischemic event in the distal thoracic and conus medullaris regions of the spinal cord, affecting inexperienced surfers and possibly related to spine hyperextension. Increased awareness of this rare condition may facilitate early recognition and treatment, which should contribute to improved neurological outcomes.

References

1. Thompson TD, Pearce J, Chang G, Madamba J. Surfer's Myelopathy. *Spine*. 2004;29(16):E353–E356.
2. Nathanson A, Haynes P, Galanis D. Surfing injuries. *Am J Emerg Med*. 2002;20:155–160.
3. Lowdon BJ, Pateman NA, Pitman AJ. Surfboard-riding injuries [abstract]. *Med J Aust*. 1983;2(12):613–616.
4. Sunshine S. Surfing injuries [abstract]. *Curr Sports Med Rep*. 2003;2(3):136–141.
5. Allen RH, Eiserman B, Straehley CJ, Orloff BG. Surfing injuries at Waikiki [abstract]. *JAMA*. 1977;237(7):668–670.
6. Roberts DR, Roe J, Baudouin C. Hyperlordosis as a possible factor in the development of spinal cord infarction. *Br J Anaesth*. 2003;90(6):797–800.
7. Beloeil H, Albaladejo P, Hoen S, Eschwege P, Benhamou D. Bilateral lower limb hypoesthesia after radical prostatectomy in the hyperlordotic position under general anesthesia. *Can J Anesth*. 2003;50(7):653–656.
8. Love B, Biller J. Neurovascular system. In: Goetz C, ed. *Textbook of Clinical Neurology*. 2nd ed. Philadelphia, PA: Saunders; 2003:395–424.
9. Duggal N, Lach B. Selective vulnerability of the lumbosacral spinal cord after cardiac arrest and hypotension. *Stroke*. 2002;33(1):116–121.
10. Williams A, Newell RLM, Collins P. Macroscopic anatomy of the spinal cord and spinal nerves: In: Standring S, ed. *Gray's Anatomy: The Anatomical Basis of Clinical Practice*. 39th ed. London, Elsevier Churchill Livingstone; 2005: 775–787.
11. Han JJ, Massagli TL, Jaffe KM. Fibrocartilaginous embolism, an uncommon cause of spinal cord infarction: a case report and review of the literature. *Arch Phys Med Rehabil*. 2004; 85(1):153–157.
12. Davies G, Keir G, Thompson EJ, Giovannoni G. The clinical significance of an intrathecal monoclonal immunoglobulin band A follow-up study. *Neurology*. 2003;60:1163–1166.
13. Holder CA, Muthupillai R, Mukundan S Jr, Eastwood JD, Hudgins PA. Diffusion-weighted MR imaging of the normal human spinal cord in vivo. *Am J Neuroradiol*. 2000;21(10): 1799–1806.
14. Kuker W, Weller M, Klose U, Krapf H, Dichgans J, Nagele T. Diffusion-weighted MRI of spinal cord infarction: high-resolution imaging and time course of diffusion abnormality. [abstract]. *J Neurol*. 2004;251(7):818–824.
15. Dinca-Avarvarei L, Valdes-Cruces JC, Quesada-Garcia M, Izquierdo G. The value of diffusion magnetic resonance imaging in diagnosing infarction of the anterior spinal artery. [Spanish]. *Rev Neurol*. 2005;40(5):282–285.
16. Zhang J, Huan Y, Qian Y, Sun L, Ge Y. Multishot diffusion-weighted imaging features in spinal cord infarction [abstract]. *J Spinal Disord Tech*. 2005;18(3):277–282.
17. Fujikawa A, Tsuchiya K, Takeuchi S, Hachiya J. Diffusion-weighted MR imaging in acute spinal cord ischemia [abstract]. *Eur Radiol*. 2004;14(11):2076–2078.
18. Restrepo L, Guttin JF. Acute spinal cord ischemia during aortography treated with intravenous thrombolytic therapy. *Tex Heart Inst J*. 2006;33(1):74–77.