

## Surfer's Myelopathy

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**Study Design.** The authors reviewed a series of non-traumatic spinal cord injuries associated with surfing lessons.

**Objectives.** To characterize a unique syndrome of paraplegia/paraparesis to improve clinical recognition, treatment, and prevention.

**Summary of Background Data.** Surfer's myelopathy is a previously unreported nontraumatic spinal cord injury that affects inexperienced surfers. Nine patients with paraparesis/paraplegia were evaluated and treated after nontraumatic surfing events.

**Methods.** An office-based registry tracked patients with surfer's myelopathy between July 2001 and December 2002. A retrospective review of hospital records searched for additional patients. Nine cases of surfer's myelopathy are retrospectively analyzed to characterize the incidence, risk factors, and outcome. The literature related to surfing injuries is reviewed.

**Results.** Nine patients were detected with surfer's myelopathy between June 1998 and January 2003. The average age was 25 years. Most patients presented with back pain, paraparesis, and urinary retention. Other presenting symptoms included paraplegia, hypesthesia/hypalgesia, and hyperesthesia. At the time of discharge, three patients had a complete recovery and four patients had mild weakness without sensory deficits. Three in this group had residual urinary retention. One patient remained paraplegic. All patients had abnormal signal change in the lower thoracic spinal cord by magnetic resonance imaging.

**Conclusion.** Surfer's myelopathy is a nontraumatic paraparesis/paraplegia that affects first-time surfers. Although most patients have a complete or near-complete recovery, complete paraplegia has occurred.

**Key words:** myelopathy, paraparesis, surfing. **Spine** 2004;29:E353–E356

Practicing medicine in the Pacific Islands provides a number of unique experiences. Over the past several years, we have detected a recurring pattern of nontraumatic spinal cord injury associated with surfing. The average patient is a young, thin male learning to surf. During the course of a first surfing lesson, the patient develops nontraumatic back pain, progressing to paraparesis with sensory and urinary symptoms. With a few exceptions, most patients have obtained a complete or near-complete

recovery. One patient remained paraplegic. Our series is the first known report of nontraumatic paraparesis/paraplegia resulting from surfing. We review our series of patients with transient and permanent spinal cord injuries associated with surfing, the associated neuroimaging, and the possible etiologies.

### Materials and Methods

Nine patients were identified between 1998 and 2003. Seven patients were identified through an office registry initiated in July 2001 when the authors recognized a recurring, novel syndrome. A retrospective review of hospital records from April 1993 through January 2003 uncovered one additional case. A manual search of the magnetic resonance imaging (MRI) logbook examined all thoracolumbar MRI ordered by the neurologist, neurosurgeon, or emergency department staff. Patients' charts and radiologic studies were then reviewed in detail (Table 1).

### Case Report

**Case No. 1.** A 23-year-old Japanese tourist was taking surfing lessons. During the demonstration on the beach, he hyperextended his back and developed mild low back pain. The hyperextended posture simulated the prone posture that is common on a surfboard. The patient proceeded with his surfing lesson. He was able to stand on the board for the first three or four attempts at surfing, but then developed weakness in his legs and fell off of the surfboard. There was no trauma associated with the fall into the water. He attempted to continue surfing for another six rides, but felt weak and returned to the shore. He was ambulatory but noticed weakness and a loss of sensation in the legs. He returned to his hotel room, developed "shaking" legs, pain in both buttocks, hyperesthesia of the feet, and a progressive sensory loss in the lower extremities.

At the time of arrival in the emergency department, the patient noted mild back pain. Initial evaluation revealed weakness in both lower extremities with a lower thoracic sensory level. Strength was 5/5 in the bilateral psoas, hamstrings, quadriceps, and plantar flexors, but 4/5 in the right dorsiflexor and 4-/5 in the extensor hallucis longus (EHL). The left dorsiflexor and EHL were 4+/5. Sensation of sharp/dull, light touch, proprioception, and vibration was intact. Deep tendon reflexes were normal and symmetric. The plantar response was flexor bilaterally. Rectal tone was normal. The back was nontender to palpation. Urinary retention was present.

Thoracolumbar MRI revealed signal change within the spinal cord between the T-9 and T12 vertebral levels on long TR sequences. High-dose methylprednisolone was initiated in the emergency department. Within 24 hours, the patient was neurologically normal. There was no tenderness in his back to palpation. The patient voided spontaneously.

**Case No. 2.** A 28-year old vacationing Japanese neurologist was participating in a surfing lesson. In attempting to stand up on his surfboard, he hyperextended his lower back and felt a "snap" and pain in the lumbar spine. There was no external

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**Table 1. Patient's with Surfer's Myelopathy**

Patient	Age (yrs)	Sex	Length of Stay	Examination				Magnetic Resonance Image	Flexion Extension	Angio
				Motor	Sensation	Urinary Retention	Babinski			
1	Admission	21	Female	3	4/5 Ps	L1 level mild	Present	Present	Signal change T8 Ascended to T6	
	Discharge				Normal	Normal	Absent	Absent		
2	Admission	25	Male	4	4/5 ps	L1 mild	Present	Absent	Signal change T8 to conus Improving at 72 hours	
	Discharge				Normal	Normal	Normal	Absent		
3	Admission	28	Male	2	Paraplegia	L1 complete	Present	Absent	Signal change T11 to conus, prominent vessels	Normal
	Discharge				Paraplegia		Present	Absent		
4	Admission	23	Male	3	4+/5 Ps, HS, Q, 4/5 DF, PF, EHL	Hyperesthesia T10	Present	Absent	Signal change T9 to conus	Negative
	Discharge				Normal	Normal	Normal	Absent		
5	Admission	30	Male	6	4/5 Ps, HS, Q, DF, PF, EHL Deteriorated at admission	Hyperesthesia Abnormal SSEP (decreased N-22 amplitude)	Normal	Present	Signal change T8-T11	
	Discharge				4+/5 Ps, HS, Q, DF, PF, EHL	Normal	Normal	Absent		
6	Admission	24	Male	5	4/5 Ps, HS, Q, DF, PF, EHL	Mild variable hypesthesia	Present	Present	Signal change T8-conus	
	Discharge				4+/5 Ps, HS, Q, DF, PF, EHL		Present	Present		
7	Admission	26	Male	4	4/5 Ps, HS, Q, DF, PF, EHL	Normal	Present	Absent	Signal change	Normal
	Discharge				4+/5 Ps, HS, Q, DF, PF, EHL	Normal		Absent		
8	Admission	24	Male		4/5 5 Ps, HS, Q, 0/5 DF, PF, EHL	T10	Present	Absent	Signal change T9 to conus Increased signal change T8-conus	
	Discharge				4+/5 5 Ps, HS, Q, 3/5 DF, PF, EHL	Normal Normal SSEP on day 3	Present	Present		
9	Admission	22	Male	1	4+/5 Ps, 5/5 HS, Q, DF, PF, EHL	Normal (paresthesias L1)	Present	Absent	Signal change T9-T11	Negative
	Discharge				Normal	Normal (paresthesias resolved)	Absent	Absent		

Ps = psoas; HS = hamstrings; Q = quadriceps; DF = dorsiflexors; PF = plantar flexors; EHL = extensor hallucis longus.

trauma associated with the onset of pain. He paddled to shore and walked onto the beach. Sitting on the beach to await his friends' return, he noticed only back pain. Within approximately 30 minutes, he noticed progressive bilateral lower-extremity weakness and could not ambulate.

On presentation, the patient had a normal sensorium and normal upper extremity examination. The back was non-

tender. Strength was 2/5 in the right psoas, 1/5 in the left psoas, and 0/5 in the bilateral hamstrings, quadriceps, dorsiflexors, plantar flexors, and extensor hallucis longus. The patient's reflexes were diminished at the ankles and absent at the knees. The plantar response was absent bilaterally. Rectal tone was diminished. Urinary retention was present.

Radiographs of the lumbar spine were normal. Long TR

sequences on the thoracolumbar MRI revealed increased signal in the inferior spinal cord. Contrast-enhanced images revealed a small area of enhancement on the dorsal dura. Long TR sequences did not demonstrate any paravertebral soft tissue signal change.

The patient was treated with 24 hours of high-dose methylprednisolone followed by oral dexamethasone. Within 24 hours, the small amount of movement in the psoas disappeared. The sensory level remained L-1. The patient's back pain resolved.

The patient was transferred to his hometown hospital 48 hours after admission. At the time of discharge, he remained paraplegic with a L1 sensory level. In Japan, the patient had a spinal angiogram. No abnormality was detected. No sensory or motor function had returned after 2 months.

## ■ Results

Nine patients (8 males, 1 female) were detected with surfer's myelopathy between June 1998 and January 2003. The average age was 25 years (range, 21–30 years). Patients presented with back pain (9), paraparesis (8), paraplegia (1), hypesthesia/hypalgesia (3), hyperesthesia (2), and urinary retention (8). At the time of discharge, three patients had a complete recovery and four patients had mild weakness without sensory deficits. Three in this group had residual urinary retention. One patient remained paraplegic.

All patients had abnormal MRI of the thoracolumbar spine. MRI showed increased long TR signal change extending from low thoracic vertebral levels to the conus. Three patients had sequential MRI. At 24 hours after injury, one patient exhibited increased signal change, ascending from T8 to T6. This patient continued to have urinary retention and mild right leg weakness 4 days after the onset of symptoms. A patient with severe paresis and abnormal signal change from T9 through the conus had a follow-up MRI 72 hours after the onset of symptoms, revealing extension of the signal abnormality to T8. The intensity of the signal change was diminished. Over a 4-month interval, this patient slowly improved with a mild residual spastic paraparesis. One patient had an MRI 3 hours after the onset of symptoms, revealing increased T2 signal from T8 to T11. Approximately 24 hours later, follow-up imaging showed no change. On the fourth day, MRI showed resolution of the T2 signal abnormality. This patient was neurologically intact at discharge. No patient had a fracture, spondylolysis, spondylolisthesis, spinal stenosis, or acute disc herniation.

Two patients had prominent vasculature surrounding the thoracolumbar spinal cord on MRI. Both patients had spinal angiography to rule out a vascular malformation. Both studies revealed normal vasculature.

## ■ Discussion

Despite the graceful appearance, surfing occasionally inflicts tremendous forces on the surfer. Successful surfing requires tremendous strength and training. Experienced surfers develop strong back and neck musculature for

lying prone on the surfboard, shoulder and arm musculature for paddling the surfboard, and leg strength for maneuvering the board and swimming. Like with all athletics, improper conditioning and inexperience can contribute to injuries.

The incidence of surfing injuries and water-sport injuries in Hawaii has been evaluated previously. Hartung *et al* found that most ocean sport-related injuries were related to swimming and surfing.<sup>1</sup> The majority of the reported 276 injuries over a 10-month period in Hawaii occurred in men, with only 10% requiring hospitalization. Unlike our population, most injuries occurred in local residents. The incidence of surfer's myelopathy is unknown, but believed to be a significant risk among first-time surfers. Colleagues at other Maui and Honolulu hospitals have confirmed the presence of similar cases at their institutions.

The most frequent surfing injuries are lacerations (41%) and soft tissue injuries (35%). Over a 56-month survey published in 1977, Allen *et al* found 36 hospitalized patients resulting from surfing injuries. Thirty-four percent of the injuries involved the head and spine.<sup>2</sup> Allen *et al* estimated the risk of injury in surfboarding as one per 17,500 surfing days. This differs dramatically from a direct survey of surfers in 1983 that estimated the rate of moderate and severe injury as 3.5 per 1000 surfing days.<sup>3</sup> Nathanson's Internet survey recorded traumatic surfing injuries in 1348 respondents and found only four vertebral fractures.<sup>4</sup>

In addition to the traumatic forces of the waves inflicted on a surfer, the nontraumatic events of surfing exert some unique forces on the surfer and the spine. A large component of surfing is paddling out. Surfers lay prone, with the back and neck extended to paddle. The hyperextended posture involved with paddling can exacerbate underlying spondylosis and precipitate a radiculopathy. Our patients are unique, because they had no underlying pathology such as spondylosis or spondylolisthesis. None of our patients had overt trauma associated with the onset of their symptoms. The most common trait is their inexperience. None of our patients had prior experience surfing. Although most patients obtained a complete or near-complete recovery, complete paraplegia has occurred. Although eight of the nine patients were Japanese, it is unclear that ethnicity is a contributing factor. The patient group likely reflects the demographics of tourism in Hawaii.

The term "myelopathy" encompasses a diverse group of conditions and etiologies. Broadly defined as any pathologic condition of the spinal cord, the differential diagnosis of myelopathy includes congenital (Chiari malformations, tethered cord), acquired (stenosis, trauma), neoplastic, vascular (hemorrhage, infarction, vascular malformations), autoimmune (multiple sclerosis, postviral), metabolic/toxic (combined system disease), infectious, and degenerative (amyotrophic lateral sclerosis) disorders.

Myelitis is an incorrect term for the patients we describe. Although many disorders of the spinal cord have previously been classified as myelitis, the term should be reserved for true inflammatory lesions. The presentation of illness in our patients could be mistaken for transverse myelitis (TM). Patients with transverse myelitis typically have an acutely evolving, asymmetric, and incomplete demyelinating process.<sup>5</sup> Although our patients' symptoms evolved rapidly and typically resulted in incomplete spinal cord injury, they tended to be symmetric. One patient did have a lumbar puncture at the time of admission. Similar to TM, the protein level and lymphocyte count were mildly elevated (cerebrospinal fluid 12 white blood cells/ $\mu\text{L}$ , 824 red blood cells/ $\mu\text{L}$ , 70 mg/dL protein, 89 mg/dL glucose), patients developed motor/sensory/and urinary symptoms. Oligoclonal bands were not evaluated. No patient had a history of optic neuritis or other demyelinating symptoms.

Surfer's myelopathy is likely the result of ischemia. A watershed zone occurs within the parenchyma of the cord between the capillaries of the anterior spinal circulation and the posterior spinal circulation. A second watershed zone occurs at the junction of the vertebral contribution to the anterior spinal artery and the aortic contribution, usually found at the T2–T3 vertebral level.<sup>5</sup> Most ischemic lesions of the spinal cord occur caudal to this junction. Infarction is known to occur in the watershed zone with hypotensive episodes related to aortic crossclamping, anesthesia, and diabetic ketoacidosis.<sup>6</sup> All of our patients developed MRI changes in the lower thoracic spinal cord. We postulate that the prone hyperextended posture associated with surfing contributes to an infarction of the watershed zone. Although infarction is usually the result of thrombosis or embolism, it is not clear why the hyperextended posture leads to infarction.

Possible vascular mechanisms of injury include avulsion of perforating vessels, vasospasm of the artery of Adamkiewicz, or transient ischemia in areas of borderline perfusion as a result of tension on the spinal cord with hyperextension. Potential risk factors that could predispose surfers to an ischemic injury include body habitus (most patients were relatively thin, with underdeveloped back musculature), dehydration as occurs in travelers and those on the beach, and long-distance travel (associated with hypercoagulation and deep venous thrombosis). Although arterial insufficiency is the most likely etiology, a venous infarction is also a possibility.

A concussive injury is less probable than an ischemic injury given the atraumatic history of our patients. By comparison, many other athletes sustain equivalent or greater forces to the spine but do not sustain thoracic paraparesis or paraplegia without overt trauma. Air

Force physicians have observed cases of transient paraplegia/paraparesis in paratroopers (personal communication, Gerald W. Mayfield, MD, August 2002). The free fall of parachuting assumes a prone and hyperextended posture much like surfing. Landing involves forces distinct from surfing.

Because surfer's myelopathy could result from compromised blood flow, we recommend aggressive hydration and perhaps induced hypertension. Based on our early experience, we are developing an intensive care protocol that involves rapid MRI, empiric steroids, spinal angiogram, hemodynamic management similar to that used for vasospasm after subarachnoid hemorrhage (hemodilution, hydration, hypertension), and urologic evaluation with urodynamic studies.<sup>7</sup>

## ■ Conclusion

Surfer's myelopathy is an increasingly recognized paraparesis/paraplegia typically affecting the lower thoracic spinal cord. The condition is possibly related to concussive or ischemic injury and appears to be induced by the hyperextension associated with paddling. Most patients have made a significant recovery, but complete paraplegia has occurred. We add our experience to the literature with hopes that this entity will be better recognized and defined in the future.

## ■ Key Points

- Surfer's myelopathy is a nontraumatic spinal cord injury affecting the lower thoracic spinal cord of inexperienced surfers.
- The pathophysiology of surfer's myelopathy is likely a secondary ischemic event resulting from hyperextension and spinal cord traction.
- Although most patients achieve significant improvement, complete paraplegia can occur.

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