Surfer's Myelopathy: A Rare Form of Spinal Cord Infarction in Novice Surfers: A Systematic Review

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Received, February 10, 2015.
Accepted, September 11, 2015.
Published Online, October 30, 2015.

BACKGROUND: Surfer's myelopathy is a rare, acute, atraumatic myelopathy that occurs in novice surfers.

OBJECTIVE: To review the literature and to present an illustrative case.

METHODS: Medical literature was queried for all reports of this condition, systematically abstracted, and analyzed. An illustrative case that provides the most definitive support for a vascular cause is presented. Treatment considerations based on prior cases and expert opinions are provided.

RESULTS: Sixty-four cases of surfer's myelopathy have been reported to date. This atraumatic thoracic/conus medullaris myelopathy with only a 42% neurological recovery rate almost uniformly affects young, healthy, novice surfers who have no pre-existent spinal disease. Symptoms usually start with back pain and rapidly progress to complete or incomplete myelopathy. T2 magnetic resonance images show increased signal in the central spinal cord within 24 to 72 hours. Gadolinium enhancement and diffusion-weighted imaging are not helpful. Angiography has been underused. Angiogram in our case showed the absence of a right T12 radicular artery and no artery of Adamkiewicz, which, along with clinical findings, support the vascular origin theory. Incomplete cases often improve within 24 hours of onset, whereas no improvement has been reported for American Spinal Injury Association class A cases. Several acute interventions have been tried. Steroids are most common, and patients receiving steroids improved 55% of the time with no reported adverse effects.

CONCLUSION: Surfer's myelopathy is a clinical entity associated with complete deficit in >50% of cases. Its prognosis is almost exclusively dictated by severity at presentation/nadir. Thus, publicizing this rare but serious condition (within and outside the medical literature) may be an effective intervention.

KEY WORDS: Spinal cord injury, Surfer myelopathy, Surfer's myelopathy, Surfing

Neurosurgery 78:602–611, 2016 DOI: 10.1227/NEU.0000000000001089 www.neurosurgery-online.com

The term surfer's myelopathy was first introduced by Thompson et al[1] in the report of 9 patients with this condition. All were young, healthy, novice surfers who acutely developed back pain and, within an hour, progressive neurological deficits. Fortunately, all but 1 of these patients had good or complete recovery of strength. However, since then, there have been 55 additional cases (including the case here) reported in 12 articles, for which prognosis was less optimistic (Table 1).[2-13]

ABBREVIATIONS: ASIA, American Spinal Injury Association; **NASCIS,** National Acute Spinal Cord Injury Studies; **SCI,** spinal cord injury; **tPA,** tissue-type plasminogen activator

Authors on this subject have concluded, on the basis of mounting circumstantial evidence, that the most likely cause of this condition is ischemic compromise to the spinal cord. First, surfer's myelopathy and spinal cord ischemia/infarction associated with aortic disease/surgery are very similar in terms of their atraumatic nature, acute onset, and the severity and distribution of symptoms. Second, magnetic resonance imaging (MRI) findings in surfer's myelopathy are most closely related to those seen in spinal cord infarct. Third, those afflicted are young individuals in good states of health who have no underlying spinal pathology. Lastly, reported

TABLE 1. Summary of the Literature

Author, Year	Cases, n	Mean Age, y/Male Sex, n/N	Rapid Deficit, n/N[a]	Motor Complete, n/N[b]	Early Recovery, n/N[c]	Steroids Used, n/N[d]	Steroids Helped, n/N[e]
Thompson et al,[1,f] 2004	9	24.8/8/9	1/2[g]	1/9	5/9	2/2	1/2
Avilés-Hernández et al,[2] 2007	1	37/1/1	1/1	1/1	0/1	NS[h]	NS
Shuster and Franchetto,[3] 2011	1	23/1/1	1/1	1/1	0/1	NS	NS
Chung et al,[4,i] 2011	1	24/1/1	1/1	1/1	1/1	NS	NS
Dhaliwal et al,[5] 2011	1	29/1/1	1/1	1/1	1/1	1/1	1/1
Karabegovic et al,[6] 2011	1	29/1/1	0/1	0/1	0/1	1/1	0/1
Lieske et al,[7] 2011	1	15/0/1	1/1	0/1	1/1	1/1	1/1
Lin et al,[8] 2012	1	19/1/1	1/1	1/1	NS	1/1	NS
Chang et al,[9,f] 2012	19	26.8/14/19	16/19	13/19	10/19	13/19	8/13
Fessa and Lee,[10,i] 2012	1	19/1/1	1/1	1/1	0/1	1/1	0/1
Nakamoto et al,[11] 2013	23	26.3/19/23	NS	8/23	7/20	7/23	NS[j]
Takakura et al,[12] 2013	3	26.7/2/3	2/3	3/3	0/3	1/2	1/1
Aoki et al,[13,i] 2013	1	26/1/1	0/1	1/1	0/1	1/1	0/1
Current case[j]	1	19/1/1	1/1	1/1	0/1	1/1	0/1
Total	64	25.9 (15-46)/80% male	27/34 (79%)	33/64 (52%)	25/60 (42%)	30/53 (57%)	12/22 (55%)

[a]Rapid deficit is defined as development of motor deficit ≤1 hour after the initial back symptom.
[b]American Spinal Injury Association A or B at nadir of neurological deficit, before discharge or transfer from presenting hospital.
[c]Early recovery is defined as a ≥1-grade improvement in American Spinal Injury Association score from nadir to discharge/transfer from initial treating facility. This definition does not acknowledge incremental improvements in strength that occurred within the American Spinal Injury Association D zone (ie, 3/5-4/5 or 5/5), and it includes a follow-up period that is available for all 64 cases.
[d]Steroids of any dose were reported as being given acutely, most commonly methylprednisolone under National Acute Spinal Cord Injury Studies III protocol.
[e]"Steroids helped" means that the patient reported as receiving any steroid acutely demonstrated a ≥1-grade American Spinal Injury Association improvement.
[f]Thompson et al[1] and Chang et al[9] reported angiography use in 2 patients.
[g]Thompson et al[1] provided detailed case histories on only 2 patients; the others were described in table form.
[h]Not stated. No follow-up examination after the initial examination was reported, or there was no mention of steroid use.
[i]These prior articles reported use of angiography in 6 cases (3 angiograms, 2 computed tomographic angiograms, 1 magnetic resonance angiograms); all were normal.
[j]Not stated. Nakamoto et al[11] reported only that steroid use did not significantly improve outcome.

workups have excluded other rare potential causes such as transverse myelitis, infection, neoplasm, vascular malformation, and inflammatory disorder.

To date, no case report has included angiographic evidence of the compromised flow. The only reported prior angiograms have demonstrated normal findings.[1,4,9,10,13] The case reported here includes angiographic and long-term MRI evidence of an ischemic source for this condition, which, taken with the clinical and imaging findings reported to date, reinforces the ischemic nature of this condition. The purpose of this work is to report this illustrative case, to systematically review the existent literature, and to provide considerations for prevention and interventions to mitigate the adverse effects of surfer's myelopathy.

CASE MATERIAL

Systematic Review

This study is a case report and systematic review of the literature. On August 25, 2014, we queried PubMed for the following search terms: surfer's myelopathy, surfers myelopathy, surfer myelopathy, surfing and myelopathy, and surfing and spinal cord injury. The 15 individual articles found were all reviewed. The references for each were also reviewed, which identified a single additional article; however, reading of this article demonstrated that it was likely the same patient as reported by Dhaliwal et al.[5] The 13 articles that included at least 1 clinical case of surfer's myelopathy were systematically reviewed in this study. Clinical information was categorized into examination, imaging, treatment, and outcome findings. Each full article was reviewed, and the specified clinical information was abstracted. The primary findings were summarized in tabular form (Table 1). In addition to this systematic review, we provide an additional illustrative case that embodies the prototypical elements of this condition. The subject in this case provided consent for use of his health information.

Case Report

A healthy 19-year-old male, elite high-school athlete with opportunities to play football at the collegiate level was on vacation with his family in Hawaii in 2008 taking his first surfing lesson. About halfway through the lesson, without a traumatic event, he developed acute, relatively severe pain and spasms in his back. Within a few minutes, he developed tingling in his bilateral lower extremities, followed by rapidly progressive weakness. He was taken to the local emergency room and evaluated. He presented with a complete (American Spinal Injury Association [ASIA] class A) spinal cord injury (SCI) with absent bowel and bladder function.

His sensory level was located halfway between the umbilicus and the inguinal crease (T11). Similar to most reported cases, he did not have a dissociated sensory loss (ie, absent anterolateral and preserved dorsal column sense), which would indicate a true anterior spinal cord syndrome; rather, he had a complete loss of both spinothalamic (pain, temperature, and light touch) and dorsal column sensation (2-point discrimination, vibration and proprioception). MRIs were obtained early (2008) and late in follow-up (2012), and they demonstrated characteristic evidence of spinal cord infarction from the T9 level to the conus medullaris (Figure 1). These findings included longitudinal T2 hyperintensity and spinal cord swelling acutely (Figure 1A and 1B), followed by atrophy at the final follow-up (Figure 1C and 1D) from the midthoracic spine to the conus medullaris. The patient was admitted to the intensive care unit for close evaluation. He was afebrile, and his laboratory findings were unremarkable, including inflammatory markers and at a later date hypercoagulable studies (ie, protein C and S levels). A lumbar drain was placed and high-dose intravenous steroids (according to National Acute Spinal Cord Injury Studies [NASCIS] III)[14,15] were administered. He demonstrated no immediate neurological recovery and was transferred to a SCI rehabilitation center closer to his home. At discharge from the rehabilitation center and final follow-up (>4 years from injury), the patient remained motor complete/ASIA A (neurological level T11 with recovery of 1/5 iliopsoas strength in the zone of partial preservation). The patient had no history of spinal, vascular, or venothrombotic disorders, nor were any vascular malformations identified on advanced imaging. During his evaluation, the patient was referred to a tertiary center

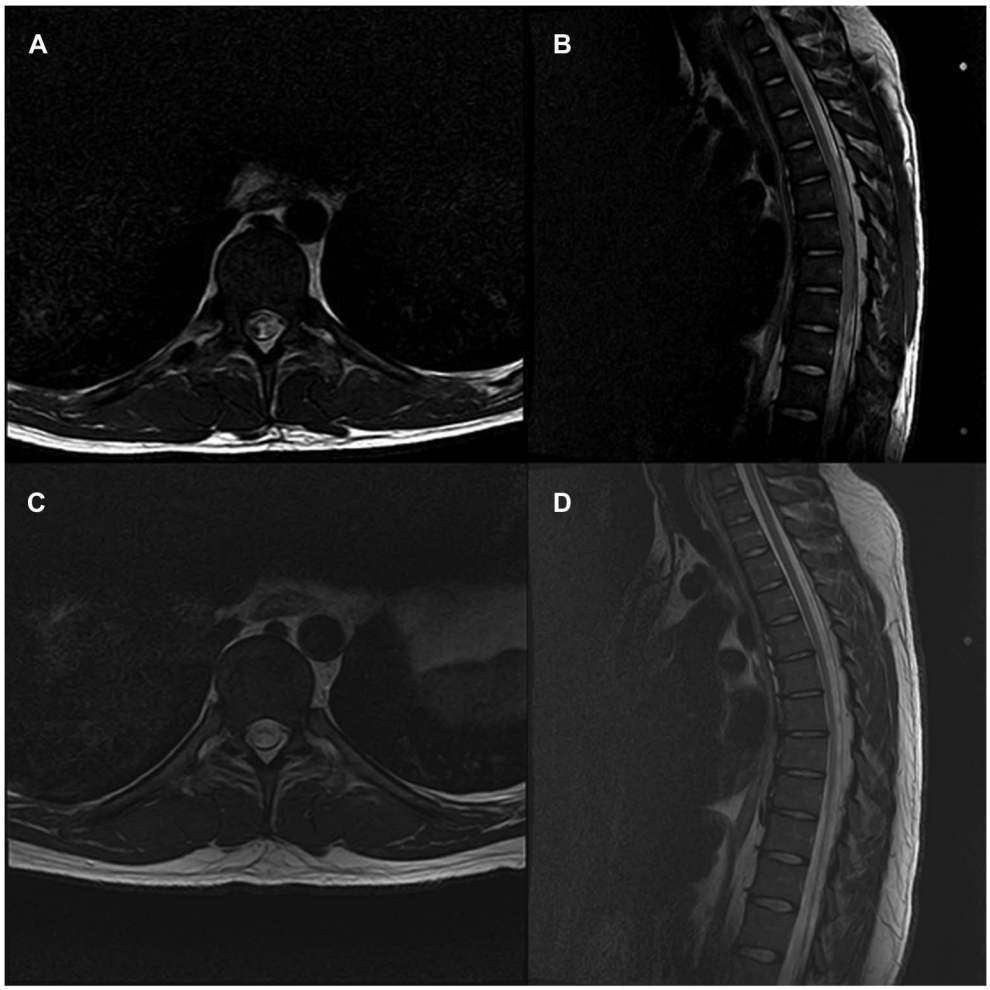

FIGURE 1. *T2-sequence axial (A) and sagittal (B) images from the 2008 and (C and D) 2012 magnetic resonance imaging scans. The earlier images show hyperintense signal within the central portion of the spinal cord, extending several levels, without intrinsic spinal column disease. At 4 years after injury (C and D), the T2 images show no more signal hyperintensity within the cord, but there is marked "pencil-like" longitudinal atrophy of the cord over >4 levels.*

for a second opinion. Spinal angiography was performed 4.5 months after injury by an experienced cerebrovascular/endovascular neurosurgeon at a center experienced with spinal angiography and equipped with state-of-the-art angiographic equipment. The angiogram failed to demonstrate the right T12 radicular artery (Figure 2). In addition, no artery of Adamkiewicz was visualized. Long-term follow-up T2 sagittal MRI demonstrated >50% atrophy of the spinal cord over >5 segments. The absence of the right T12 radicular artery and the artery of Adamkiewicz does not confirm that T12 was the primary radicular feeder to the spinal cord or that it was acutely occluded (it may have been congenitally/developmentally absent). An alternative explanation is that in the absence of a dominant radicular artery, the mechanics of the surfing lesson induced a period of hypoperfusion through multiple thoracic radicular arteries and collaterals, precipitating the ischemia. Although T2 signal change with subsequent atrophy on MRI are characteristic findings after many causes of SCI, it is our opinion that the absence of severe trauma, the acuity and intensity of symptom onset in an otherwise healthy spinal column, and the late angiographic findings in this case all support vascular occlusion/insufficiency as a cause for this condition, which in this case resulted in dense, irrecoverable ischemic infarction of the lower thoracic spinal cord and conus medullaris.

RESULTS

In 13 articles plus our additional case, 64 individual cases (51 male patients [80%], 13 female patients [20%]; average age, 25.9 years [range, 15-46 years]) of surfer's myelopathy have been reported.[1-13] Nine of 13 articles (69%) were single case reports, and 4 of 13 were case series (n = 3, 9, 19, and 23). The 3 largest series were composed by different groups of authors who reported on cases occurring in 1 or more of the same 3 community hospitals in the state of Hawaii over a similar window of time; thus, it is possible that some but not all, owing to incomplete overlap of exact time periods, patient ages, and characteristics, and study sites, of the 51 cases reported in these 3 series are redundant.[1,9,11] Table 1 summarizes the relevant findings. Not included in this table is the fact that all cases were described as atraumatic nature of injury in inexperienced surfers (more than half of reports specifically indicate this was the first surfing lesson, like our case) who otherwise were healthy, young individuals with structurally normal spines whose initial symptom was some degree of acute back discomfort (95% reported pain, varying from mild to severe), and 3 patients had spasms/stiffness. All but 6 patients (91%) sustained their SCI while surfing in Hawaii. Given the atraumatic nature of this disease and the lack of a clear predisposing factor for the observed geographic proclivity, it has been proposed that recent long-haul flights are a predisposing factor. Whether this is due to dehydration (altered rheology) or stasis has not been addressed, but at least 1 case involved a local Hawaiian resident without recent air travel.[2] In all cases, a review of medical history was unremarkable except for 1 patient who had a history of spontaneous deep venous thrombus earlier in life.[5] Of the 29 patients for whom sufficient information was provided, only 9 patients had dissociated sensory deficits (31%). Complete sensory loss (anterolateral and dorsal column) is most common. Thirty-three of 64 patients (51.6%) developed complete motor

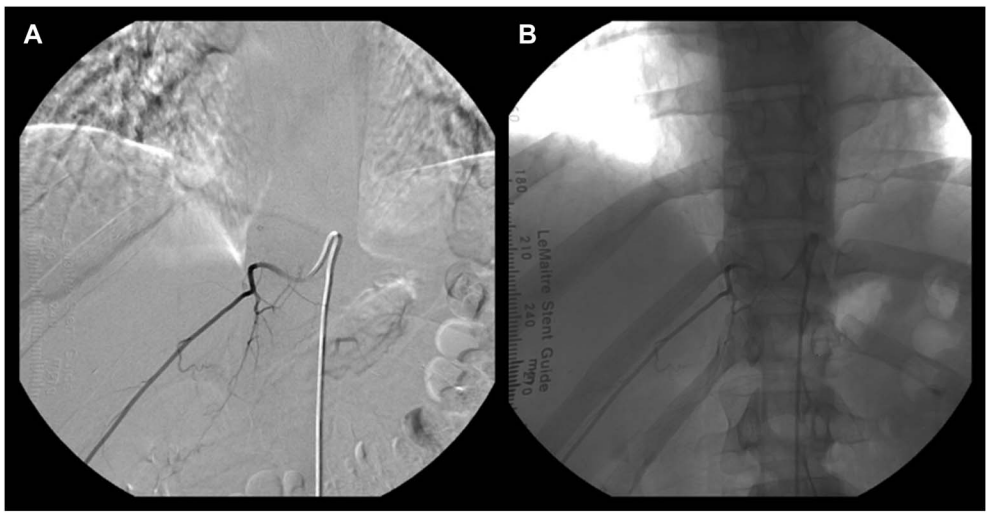

FIGURE 2. *Superselective angiogram images showing a coned down, digitally subtracted (A) and native image (B) of the right T11 contrast injection. This image shows the normal radicular anatomy, including a caudal collateral branch, which typically anastamoses with the T12 radicular vessel, but in this case, there is no evidence of flow at T12. Likewise, when supraselective catheterization of the right T12 was performed, there was no distal flow, indicating that the vessel was completely occluded. Additionally, an artery of Adamkiewicz was not detected on this examination.*

loss (ASIA A or B) acutely; 31 of these 33 had follow-up examinations; and 6 of these 31 (19.4%), all with ASIA B injury at nadir (B to C, n = 2; B to D, n = 3; B to E, n = 1), demonstrated motor recovery. Thus, ASIA A lesions have had a 0% motor recovery rate. In all but 1 case in which urinary function was reported, it was abnormal (ie, retention) at presentation (43 of 44, 97.7%). In 25 of 60 (41.7%), motor recovery of at least 1 ASIA grade occurred. Lastly, steroids were by far the most commonly used treatment, typically under the NASCIS III dosing protocol. Steroid use was discussed in 53 cases and was used in any dose in 30 of these cases (57%). In the 22 cases that reported steroid use and final neurological outcome for the patient receiving steroid, the neurological recovery rate (at least 1 ASIA grade) was 55% (12 of 22), with 7 of the other 10 patients who did not show recovery after receiving steroids having ASIA A nadir examinations. The remaining 3 who demonstrated no reported response to steroids were ASIA B (n = 2) and ASIA D with mild deficits (n = 1). Thus, with the exclusion of ASIA A complete patients, steroids had a beneficial effect on all but 3 patients with an incomplete deficit.

DISCUSSION

Acute spinal cord infarction is one of the rarest mechanisms of SCI, and it is traditionally associated with the poorest prognosis.[16-20] Most infarcts present as an anterior cord syndrome, which is manifested by bilateral paresis/paralysis and absent pain/light touch but preserved position/vibration/deep sensation (ie, dissociated sensory deficits), resulting from vascular occlusion or hypoperfusion of the anterior spinal artery and its watershed area.[19,21] The anterior spinal artery communicates with the 2 posterior spinal arteries by a fine vascular plexus, which lies on the surface of the spinal cord. The anterior spinal artery sends 2 branches into the matter of the spinal cord, called the sulcal arteries. The arborization of these vessels provides circulation to the anterior two-thirds of the spinal cord.

In 2012, Robertson et al[20] reviewed the experience at Mayo Clinic over a 17-year period (1990-2007) and identified the largest cohort of spinal cord infarctions reported to date. This epidemiological article details the clinical characteristics of spinal cord infarction and prognosis. Aortic disease or surgery and systemic hypoperfusion accounted for 66% of the cases. As with all SCIs, the most prognostic finding was maximal motor deficit. Forty-nine percent of the patients developed a complete motor deficit (ASIA A/B). Like surfer's myelopathy, deficits emerge rapidly, with maximal deficit reached within 1 hour of symptom onset in 68% of patients. Eighty-one percent of patients required a wheelchair to ambulate in hospital, and 89% required bladder catheterization. This study, which is skewed toward an elderly population (average age, 64 years), identified a significant mortality risk with a 5-year survival of 55% and 10-year survival of 42%, with advanced age and diabetes mellitus significantly worsening these odds. This study showed that despite their very serious pathology, about half (48%) of the initial wheelchair ambulators no longer required a wheelchair at >6-month follow-up and one-third no longer required catheterization. The ability to recover from ischemic insult to the spinal cord suggests that there is some room for improving outcomes for surfer's myelopathy. This will require early recognition of signs/symptoms on the part of patients (ie, through education of the public and surfing instructors) and emergent interventions aimed at restoring perfusion to the cord.

In 2004, Thompson et al[1] identified a new type of anterior spinal cord syndrome, which they called surfer's myelopathy. Nine cases were reported, 2 sporadic cases (in 3 years, 1998-2001) that sparked the author to create an internally maintained registry that detected 7 subsequent cases over the next 18 months. This study defined the prototypical characteristics of surfer's myelopathy. Patients are novice surfers, commonly on their first lesson, who are otherwise young and healthy without prior spinal or vascular problems. Thompson et al and others have stated that patients are typically thin with underdeveloped back musculature; however, this physical feature has been least consistent across other reported cases and was not a factor in the case reported here.[2] Without any antecedent trauma, about halfway through a typical 90-minute surf lesson, acute back pain, spasms, or paresthesias developed. The back pain can be mild or intense. Soon after (<1 hour in essentially all cases, similar to the acuity of deficit that occurs in anterior artery syndrome), the patients developed new or progressive paresthesias and then paresis. By the time they reached the hospital, their neurological deficit had reached its nadir. The maximal deficit of 7 of 9 patients (78%) was ASIA D, and all of these patients demonstrated some recovery. Those who experienced full recovery (ASIA E) did so within 24 hours of presentation. Bladder control, which was abnormal in all patients, returned to normal in 6 of 9 patients in this series. In addition to being the first to characterize this condition, the authors were the first to propose that this was an ischemic injury to the spinal cord. Thus, they speculated whether measures used in hemorrhagic cerebral infarct (ie, triple H therapy [hypertension, hypervolemia, and hemodilution]) could be used to improve outcomes in spinal infarct. These authors also provided high-dose steroids in some cases.

In response to the Thompson et al original work, other groups have reported similar cases of surfer's myelopathy, but unlike the series reported by Thompson et al, the outcomes have been worse (Table 1). This is likely due to the sporadic nature of these cases (69% were single case reports) as opposed to a directed search for cases by the Thompson group. Thus, one can infer that for every surfer's myelopathy with permanent severe or complete motor deficits, there are several less severe cases and that this condition presents sporadically in areas of the world where surfing is prevalent. Given the rarity of this pathology and the rapidity with which neurological recovery occurs (typically <24-72 hours), it is likely that most of the less severe cases are diagnosed as transient neurological events of unknown origin or occur in patients who never seek medical attention.

In regard to diagnosing surfer's myelopathy acutely, Nakamoto et al[11] have provided the most comprehensive review of the MRI characteristics of surfer's myelopathy in their report of 23 cases occurring in Hawaii from 2003 to 2012. These authors found in this largest reported series to date that all patients demonstrated T2 hyperintensities, typically in the central portion of the spinal cord (ie, the watershed location in the axial plane for the spinal cord) and in a "pencil-like" longitudinal extension on sagittal images with cord swelling that extended from the midthoracic (T5-10) level to the conus. In fact, in all but 2 cases reported to date, standard T2 MRI sequences obtained within the first 24 hours of symptom onset have shown characteristic ischemic changes.[6,11] In 1 outlier, changes were seen on a repeat MRI obtained the day after presentation, and in the other, MRI of the lumbar spine was not obtained until 2 weeks after injury in a patient with ASIA D deficits. Eleven of 23 cases had gadolinium contrast added. This provided no diagnostic benefit in all cases. Although gadolinium does not contribute to the diagnosis of surfer's myelopathy, it can be helpful for ruling out other pathologies. Nakamoto et al[11] did not have diffusion-weighted MRI to review, but others have. Diffusion-weighted imaging, which is an MRI technique that is highly sensitive for detecting regions with restricted diffusion of water related to acute ischemic cellular injury, has demonstrated areas of hyperintensity and restricted diffusion in most (10 of 15, with the other 5 studies being negative or equivocal) but not all cases.[2-4,7-9] Similar to intravenous contrast, the addition of diffusion-weighted imaging sequences has not enhanced the diagnostic workup; thus, routine MRI is the preferred method.

Although routine MRI of the spine is the preferred screening imaging modality, we believe that spinal angiography, which is the gold standard for confirming vascular pathology of the named vessels feeding the spinal cord and a treatment platform that allows direct intra-arterial delivery of medications/interventions, can be considered in cases of surfer's myelopathy with severe neurological deficit. Angiography has been rarely used in prior cases. Some of the reason for the limited use of angiography likely represents lack of resources or capabilities at the initial treating facility, but some also represents a decision to exclude this confirmatory imaging modality. Because the leading hypothesized cause of surfer's myelopathy is acute ischemic insult from occlusion (embolic or intimal flap), spasm, external compression (possibly from psoas spasm), or avulsion, spinal angiography represents the best and possibly only option to confirm the exact mechanism of the ischemic insult.

We believe that the angiographic findings in this case (the absent right T12 radicular artery and lack of an artery of Adamkiewicz) implicate vascular occlusion as the cause of this severe case of surfer's myelopathy.[16] Interestingly, Chung et al[4] performed a computed tomographic angiogram on their patient and identified a patent artery of Adamkiewicz at right T12. Chung et al noted that the limitations of computed tomographic angiogram prevented them from evaluating the patency of this vessel to the level of the spinal cord. The angiogram in the case reported here was performed late; therefore, there was no opportunity to provide intra-arterial treatments to affect outcome. Given these facts, we recommend that spinal angiography be considered in the acute evaluation and management of surfer's myelopathy (Figure 1). Lastly, in agreement with most authors, we also postulate that the prolonged prone hyperextension that is typical of novice surfers is a mechanical event that is capable of instigating the vascular insult. Hyperextension can distract the radicular vessels, which can compromise flow. Alternatively or in concert, the altered venous return that occurs from lying prone for prolonged periods of time on a surfboard can contribute to vascular insufficiency. We believe that the intravascular mechanism that leads to the disrupted perfusion of the spinal cord in these novice surfers is occlusion by embolus or vasospasm induced by prolonged hyperextension. We base this belief on the facts that none of the cases reported to date had MRI evidence of vessel avulsion (ie, hemorrhage) and that there have been no reported cases of a similar acute myelopathy in novice or elite butterfly stroke swimmers, who repetitively and violently hyperextend their flexible trunks for brief periods of time, often while performing a Valsalva maneuver. Therefore, it is more likely that prolonged hyperextension plays the putative role, which has been a previously reported mechanism for SCI.[22] Furthermore, given the rarity of this condition, it likely takes a perfect storm of events for novice surfing to produce a vascular insult to the spinal cord. Things like altered rheology (dehydration from long-haul flight) and baseline coagulability probably also contribute to this event. Unless urgent angiography becomes a standardized approach to surfer's myelopathy, we will likely never know the anatomic mechanisms that alter spinal cord blood flow. However, on the basis of our case and the evidence to date, we believe that surfer's myelopathy results from an acute transient or prolonged disruption of the anterior circulation to the spinal cord.

The purpose of this report is to enhance the understanding of this condition, especially in endemic areas, beyond simple case identification. We feel this effort must be manifested in 2 ways. First, providers should become more cognizant of this condition and of potential interventions. These patients tend to be young and healthy; their physiology can better support induced hypertension, cerebrospinal fluid (CSF) drainage, steroid administration, and thrombolytic therapy (with adequate informed consent from the patient) to provide an improved chance for recovery. Because there is no expectation of meaningful recovery in untreated patients with complete surfer's myelopathy and because there is no evidence of spinal cord hemorrhage in this disease process, therapeutic interventions suggested here may be considered for those with severe or complete SCI, whereas expectant observation is appropriate in patients who present with preserved antigravity strength in the majority of their lower extremities.[23]

Second, this work aims to establish a persuasive evidence base for surfing oversight bodies and publications. These groups need to recognize surfer's myelopathy as a serious risk to surfers,

especially novices. This realization should prompt efforts on their part to publicize this risk and to inform recommendations for shortening/modifying beginner lessons and teaching techniques that discourage prolonged, repetitive hyperextension (Figure 3). Karabegovic et al[6] included in their case report recommendations proposed by the Surfer's Myelopathy Foundation (www.smawareness.org) that are simple, common-sense measures that can mitigate the presumed causative factors of this condition. In addition, we recommend that surfers flying long distances should rest, rehydrate, and stretch before engaging in surfing lessons. Although surfer's myelopathy is rare, it is more common than a fatal shark attack or being struck by lightning while surfing. For instance, in the same period of time in which the reported cases of surfer's myelopathy have occurred (ie, 1999-2014), there were 23 reported fatal shark attacks involving surfers.[24] Although surfing organizations and publications clearly acknowledge the risk of and implement and recommend methods to mitigate shark attack, these same groups have been silent on surfer's myelopathy.

FIGURE 3. *Photograph of a surfer demonstrating the hyperextended prone waiting posture (A) that is typical of novice surfers and is postulated to be a predisposing factor for surfer's myelopathy. From this position, the novice surfer must forcefully and rapidly "pop up" to a crouched and then upright surfing position. Novices perform the pop-up over and over as they learn the art of transitioning from waiting for a wave to surfing one. B, photograph showing the recommended seated posture for waiting for a wave.*

Realizing the importance of enlisting these groups in efforts to prevent and mitigate surfer's myelopathy and acknowledging the fact that prior calls in the medical literature for better publicity and preventive measures have failed to spontaneously evoke response from these groups, we are directly seeking to collaborate with them. Similar to shark attack and lightning strike, surfer's myelopathy is a rare but serious adverse event that should be mitigated, and the most effective mitigation strategy will be education and training modification.

Regarding medical intervention, we acknowledge that there is no available evidence base that directly informs best treatment options for this rare condition and that basing treatment recommendations on case reports and expert opinion has significant inherent limitations, with the caveat that we feel that it is worthwhile to share our recommended treatments (Table 2). In the absence of literature on the subject, we borrow from the thoracic aortic surgery and stroke literature and advocate the following.[25-28] First, when resources exist, emergent aortic catheterization and spinal angiography of the radicular vessels can be considered with administration of intra-arterial tissue-type plasminogen activator (tPA) in the presence of confirmed embolic occlusion of a radicular artery and nicardipine in the presence of vasospasm. The potential risk of selective arteriography and administration of tPA or calcium channel blockers must be disclosed during preprocedural counseling, as well as the lack of validating evidence for their use in this condition. If no occluded radicular artery or artery of Adamkiewicz is identified or when emergent spinal angiography is not available, intravenous tPA can be considered.[27] Timeframes from symptom onset and dosing protocols should be based on the National Institute of Neurological Disorders and Stroke protocol for cerebral stroke.[25,27] We feel that on the basis of our case and the circumstantial evidence in prior cases, surfer's myelopathy should be treated as an acute vascular compromise to the central nervous system (ie, a stroke) and managed as such. Alternative to or after tPA infusion, heparin and/or antiplatelet therapy can be started, similar to treatment of a vertebral artery injury. Another consideration (in lieu of but not in conjunction with full-dose intravenous tPA) should be acute placement of a lumbar drain. First, this allows CSF sampling, which is part of an exhaustive workup for acute atraumatic spinal cord deficit. Second, this allows CSF pressures to be reduced to <10 to 15 mm Hg, which has been shown repeatedly to be neurologically protected.[29-32] Furthermore, it is commonly accepted among SCI centers that intentionally elevating the mean arterial pressures (>85-90 mm Hg) after acute SCI is beneficial. In fact, Hawryluk et al[33] have contributed the most compelling evidence to date on this subject. They demonstrated that maintaining some degree of hypertension (average mean arterial pressure >85 mm Hg) for the initial 5 to 7 days after acute SCI yielded better neurological recovery after acute SCI. Dropping CSF pressure and increasing mean arterial pressure work in concert to improve perfusion to the sulcal arterial bed and spinal cord parenchyma.[31,32,34,35] In addition, patients presenting within 3 hours of SCI may be considered for the NASCIS III methylprednisolone protocol.[14,15] Steroids have a demonstrated beneficial efficacy in non–ASIA A cases of surfer's myelopathy without reported adverse events. Whether they had a beneficial effect is unknown, but the detrimental impact of high-dose steroids associated with SCI in the setting of major trauma is less likely to complicate this situation because these are atraumatic, nonpenetrating isolated injuries in otherwise healthy patients. This is perhaps the singular clinical situation in which the risk of high-dose steroid therapy is maximally minimized, making it appropriate to consider administrating the protocol to achieve its questionable benefit. In this regard, surfer's myelopathy may be more akin to transverse myelitis, for which acute steroid administration is an accepted standard of care, although the exact mechanism of action is not entirely certain. This opinion is not in contrast to the recommendations of leading bodies against the use of the NASCIS protocol in acute traumatic SCI. We feel that this is a different condition for which surgery is not indicated, and the physiology of those who typically acquire the condition is most capable of tolerating the stress of high-dose steroid exposure. If presented with a similar acute (<3 hours from symptom onset) case of complete or near-complete surfer's myelopathy, we would take the patient for emergent angiography, at which time we would be prepared to administer intra-arterial thrombolytics (in cases of embolism such as postulated to

TABLE 2. Prioritized Treatment Recommendations for Acute Surfer's Myelopathy (ie, Presentation <3 Hours From Symptom Onset)[a]

1. Confirm diagnosis by clinical history, physical examination, and emergent routine magnetic resonance imaging of the spine (rule out underlying spinal disease)
2. Consider 1 of these interventions
Emergent aortic spinal angiography and superselective catheter–delivered tPA or nimodipine (rule out underlying aortic disease), or
Administer intravenous tPA, National Institute of Neurological Disorders and Stroke protocol dose, or
Place lumbar drain and maintain cerebrospinal fluid pressure <10-15 mm Hg and send sample
3. Admit to intensive care unit for close monitoring; place Foley catheter; record postvoid residual
4. Elevate mean arterial pressures >85 mm Hg for at least 24 h, first with intravenous fluids, judicious use of narcotic pain medications, and then vasopressors
5. Consider NASCIS III methylprednisolone protocol (24-h dose; 30-mg/kg loading dose; 5.4-mg·kg^{-1}·h^{-1} infusion × 23 h)
6. Bedrest in a position of comfort for 24 h; Immediate passive and active range of motion for all 4 extremities; physical medicine and rehabilitation/physical therapy/occupational therapy consults
7. Evaluate outcome at 24-48 h; those with severe deficits present at 48 h will require transfer to a spinal cord injury rehabilitation center and urological consultation/urodynamic study

[a]NASCIS, National Acute Spinal Cord Injury Studies; tPa, tissue-type plasminogen activator.

have occurred in the case reported here on the basis of the angiographic findings) or nicardipine (in cases of vasospasm) to acutely restore anterior circulation to the spinal cord, and then we would admit the patient to the intensive care unit for close monitoring and induced hypertension. In addition, we would complete the NASCIS III protocol (24-hour dose), but with the understanding and after informing the patient that the evidence supporting the efficacy of this intervention is circumstantial and inconclusive whereas the risk associated with this medication is unquestionable.

CONCLUSION

Surfer's myelopathy is a rare but serious condition that affects young, novice surfers. Patients who present with complete deficits have demonstrated no significant neurological recovery. Those with preserved motor function have demonstrated good to excellent recovery. In this condition, for which various treatments attempted to date have not influenced outcome, we must consider 2 approaches to reduce the impact of this disease.[9] First, the risk and warning signs of this condition need to be publicly available to novice surfers. As with many injury patterns seen in the trauma setting, maximal effect on outcome will come only with a focus on preventive measures. Second, treatment of this spinal cord ischemic event should be aggressive in cases of severe or complete deficit. Implementation of these 2 aims will, we hope, mitigate the impact of this rare event.

Disclosures

The views expressed in this article are those of the authors and do not reflect the official policy of the Department of the Army, the Department of the Navy, the Department of Defense, or the US government. This work was prepared as part of their official duties and thus there is no copyright to be transferred.

Dr Rasmussen reports the following conflicts: Blockade Medical, Strategic Advisory Board, investor, and stockholder; Medtronic Neurovascular, consultant and honorarium; Stryker Neurovascular, consultant and honorarium; Perflow Medical, Strategic Advisory Board and stockholder. The other authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENTS

The authors provide a very illustrative case of a rare entity and provide an excellent literature review. Although most will likely not ever see surfer's myelopathy, this report is educational and especially relevant where surfing is a regular sporting and recreational activity.

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Freedman et al have provided an interesting case report and review of the literature of surfer's myelopathy, a rare clinical syndrome characterized by sudden-onset, nontraumatic thoracolumbar spinal cord injury while surfing. The underlying cause of this syndrome remains unknown. The several cases reported in the literature share similar commonalities: lack of direct spinal trauma; frequent occurrence in novice surfers; and prevalence of cases in Hawaii, perhaps suggesting tourists who have recently undergone long-distance air travel. This has led the authors to hypothesize an underlying vascular pathogenesis. In novice surfers, prolonged lumbar hyperextension while paddling on a surfboard combined with dehydration and vascular stasis after a long airline flight may culminate in a perfect storm of thromboembolic occlusion or vasospasm of critical thoracic segmental arteries.

The authors describe a delayed angiogram performed late after surfer's myelopathy that fails to demonstrate a T12 radicular artery or an artery of Adamkiewicz. The authors postulate that at the time of injury, sudden occlusion of a thoracic radicular artery (and Adamkiewicz hypoperfusion) was the inciting event that led to paralysis. Although this hypothesis certainly seems plausible, it leaves a few unresolved issues. First, catheter angiography can fail to identify Adamkiewicz in as many as 29% of individuals without arterial occlusion,[1] thereby bringing into question the significance of this radiographic finding.

Second, presumably countless novice surfers venture for the first time in remote locations, yet the literature suggests that surfer's myelopathy is a rare event. If lumbar hyperextension combined with dehydration and vascular stasis can result in thoracic radicular artery occlusion, then one might expect that surfer's myelopathy or even a transient, milder syndrome would be more commonly reported. Alternatively, the thoracic aortic aneurysm and spinal oncology surgery literature indicates that many individuals can safely tolerate sacrifice of multiple thoracic segmental arteries. A small percentage, however, demonstrate neurological changes with occlusion of even a single thoracic segmental artery (likely the primary feeding artery to Adamkiewicz), hence the common use of intraoperative neuromonitoring during these procedures. Perhaps, this same subset of the population is susceptible to spinal cord ischemia from thoracic segmental artery sacrifice that is also at risk for surfer's myelopathy.

Finally, if surfer's myelopathy is indeed due to vascular occlusion, then the fundamental issues are how to prevent occurrence and how best to treat those who are affected. With regard to treatment, the authors propose that emergent angiography with systemic tissue-type plasminogen activator, intra-arterial thrombolysis, or angioplasty may be of benefit. Unfortunately, there is a lack of evidence describing this approach for surfer's myelopathy; therefore, the recommendations made by the authors must be interpreted cautiously. With regard to prevention, predetermining those who are at high risk remains unsolved. Therefore, the authors suggest that heightened public awareness may be the most important first step toward prevention. Admittedly, I was unaware of surfer's myelopathy until I read this manuscript. I suspect that with more case reports and case series, the authors will succeed in drawing greater attention to this potentially devastating condition from both the surfing and neurosurgical communities. For this, the authors should be commended.

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