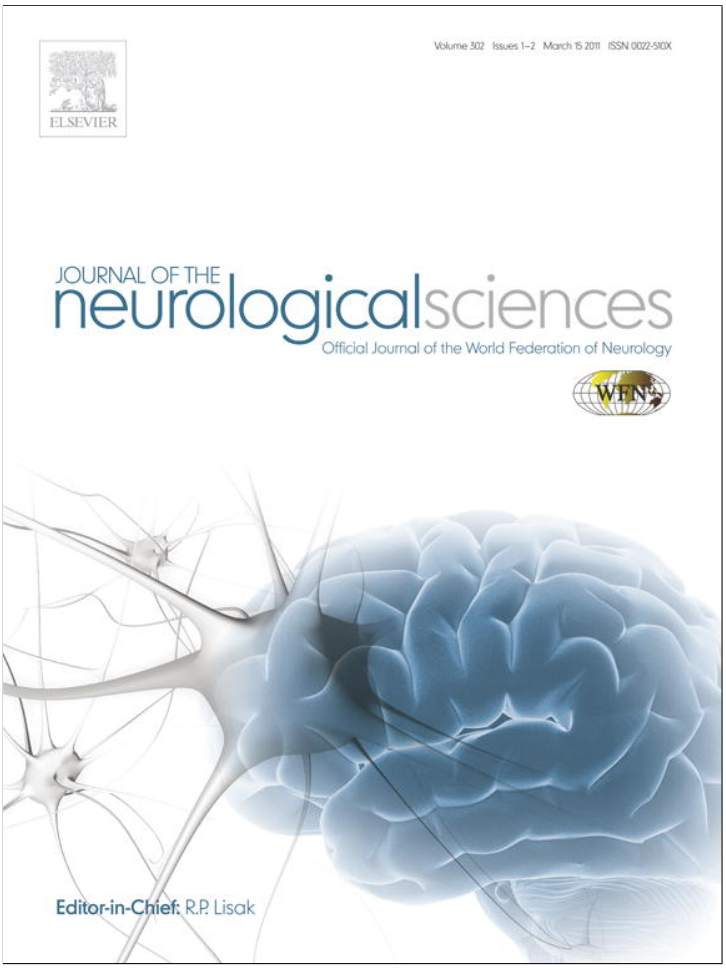

Journal of the Neurological Sciences 302 (2011) 118–120

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Short Communication

Non-traumatic anterior spinal cord infarction in a novice surfer: A case report

Hsin-Yeh Chung [a], Shu-Fen Sun [a,b], Jue-Long Wang [a], Ping-Hong Lai [b,c], Chiao-Wen Hwang [a,*]

[a] Department of Physical Medicine and Rehabilitation, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan
[b] National Yang-Ming University School of Medicine, Taiwan
[c] Department of Radiology, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan

ARTICLE INFO

Article history:
Received 17 August 2010
Received in revised form 24 November 2010
Accepted 30 November 2010
Available online 31 December 2010

Keywords:
Spinal cord ischemia
Anterior spinal cord syndrome
Surfer's myelopathy
Valsalva maneuver
Fibrocartilaginous embolism
Stroke

ABSTRACT

We present a case involving a 24-year-old woman who developed anterior spinal cord infarction with paraplegia but preserved posterior column sensation shortly after her first surfing lesson. The clinical presentation and magnetic resonance imaging findings were compatible with anterior cord infarction from T11 to conus medullaris. However, a thorough diagnostic workup revealed no predisposing factors of ischemia. Like previously reported cases of surfer's myelopathy, our patient was inexperienced and lacked trained back muscles, though none of the previously related cases were reported to have anterior cord infarction. In this case report, we discuss the probable relationship between surfing and ischemic myelopathy. Non-traumatic myelopathy in surfing is a relatively new entity thought to occur in surfers while paddling their boards in a hyperextended position and perform the Valsalva maneuver while attempting to stand up on the boards, which may increase intra-spinal pressure. Increased awareness of this injury may make it possible to recognize its early symptoms and prevent further injury.

1. Introduction

Spinal cord infarction is a rare disorder sharing similar symptoms as other acute myelopathic syndromes. Surfer's myelopathy is a relatively new entity reported by Thompson et al. [1] in a series of nine cases involving people developing acute paraparesis during or shortly after their first experience in surfing. The only possible cause shared by all cases was no previous surfing experience, though ischemic myelopathy was suspected. Outcomes range from complete recovery to persistent paraplegia [1].

This case report involves a 24-year-old female tourist who began feeling progressive loss of strength in her lower limbs for which she was eventually sent to our emergency ward. There, clinical presentation, physical examination, and magnetic resonance imaging (MRI) findings were compatible with anterior spinal cord infarction. After six weeks in neurology and rehabilitation ward, she was discharged with partial recovery from paraplegia. Because there are no previous reports of anterior cord infarction associated with surfing, we report this case and discuss its possible causes.

2. Case report

A relatively thin (55 kgw, 158 cm, BMI: 22) 24-year-old female tourist with poor back musculature began feeling loss of leg strength when returning to shore after her first surfing lesson. After 2 h practicing how to paddle while lying in a prone position and how to stand up quickly from that position (Fig. 1), she surfed for another 2 h. Although the surfer reported performing the Valsalva maneuver each time she stood up on her board, the surfing experience was uneventful with no impact trauma. She noticed lower limbs weakness and thought she was becoming tired only and finished the practice session paddling lying prone. Upon returning to shore, she noticed continued lower limb weakness and mild back pain, rated 2 on a 10-point scale on recall. She returned to her hotel room with support from friends and rested about 3 h, but felt increasing weakness, numbness, and an inability to urinate. An ambulance was called, and she was taken to a regional hospital emergency ward. By that time she had complete paraplegia, so she was transferred to emergency department of our medical center, 8 h after the onset of her symptoms.

Her clinical presentation, physical examination, and magnetic resonance imaging (MRI) findings suggested anterior spinal cord infarction. She reported no recent illness, fever, chills, vomiting, diarrhea, cough, animal bites, vaccinations, or previous back pain. Her temperature was 37.2 °C, blood pressure 116/73 mm Hg, pulse rate 64 beats/min and respiratory rate 17 breaths/min. Her neck had no tightness or rigidity. Cardiac, pulmonary, and abdominal examinations were normal. Although she had no back deformity, her lumbar area was tender and tight. Skin was intact without rash or ecchymosis. The mental status, cerebellar, and cranial nerve examinations were normal. However, both lower limbs were totally paralyzed (MMT 0/5). She could perceive vibration and proprioception but not pain or temperature. Sensory level was T 11.Patellar and ankle reflex was diminished. Urine retention was noted.

* Corresponding author. Department of Physical Medicine and Rehabilitation; Veterans General Hospital Kaohsiung, Taiwan, No 386, Ta-Chung 1st Road, Kaohsiung 813, Taiwan. Tel.: +886 7 3422121 4223; fax: +886 7 3420243.
E-mail address: rehmedyeh@gmail.com (C.-W. Hwang).

0022-510X/$ – see front matter © 2010 Elsevier B.V. All rights reserved.
doi:10.1016/j.jns.2010.11.026

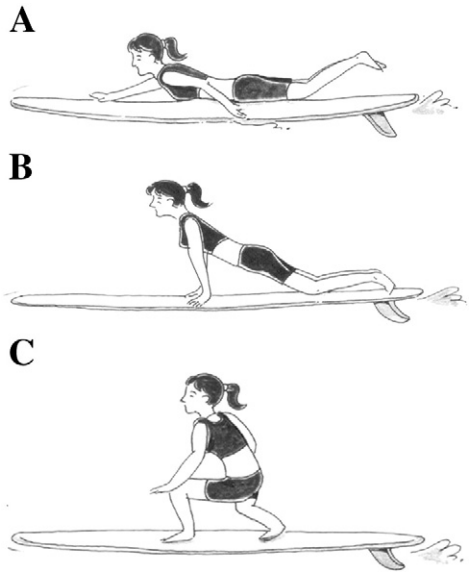

Fig. 1. Body motion in surfing. (A) Paddling (B) (C) Take off. Hyperextended back during paddling and tack off is common body motion in surfing. Valsalva maneuver is profound in inexperienced surfer during take off.

Laboratory workup showed a white blood cell count of 11,340/μl, hematocrit 37.7%, platelets 266×10^3/μL, normal electrolytes, an erythrocyte sedimentation rate 12 mm/h, C-reactive protein 0.23 mg/dL, and prothrombin (PT) and partial thromboplastin times (PTT) of 10.4 and 28.3 s, respectively. The lumbar puncture was not done since the patient's clinical course and general laboratory workup did not favor systemic inflammatory disease or infectious process.

Radiographs showed normal thoracic, lumbar and sacral spine; chest radiography was normal. Initial magnetic resonance imaging (MRI) of the thoracic and lumbar spine performed 10 h after the beginning of symptoms showed no significant findings. There was no compressive myelopathy or hemorrhage of the spinal cord. No traumatic injuries of the spine or other abnormalities,(e.g., Schmorl's nodes) were noted. Based on preserved perception of vibration and proprioception and impaired perception of pain and temperature as well as loss of lower limb strength, the patient was suspected as having anterior spinal cord syndrome and transferred to the neurology ward.

MRI examinations three days later revealed signal hyperintensities of the anterior central cord on T2 weighted image (T2WI) at the T6 to conus medullaris levels of the spinal cord. Hyperintensities was noted in the sagittal plane. The diffusion-weighted image (DWI) series findings suggested acute infarction.(Fig. 2). There was no flow-void phenomenon on T2WI, no intramedullary hemorrhage, and no gadolinium enhancement.

Additional laboratory studies for hypercoagulability and autoimmune diseases, including protein C and S, anticardiolipin antibody, rheumatoid factor, and serum IgG/IgA/IgM, antinuclear antibodies were all normal. She was diagnosed with ischemic myelopathy resulted from anterior cord infarction and started on clopidogrel as secondary prevention.

CT-angiography 6 days after initial symptoms to screen for vascular malformations showed intact proximal Adamkiwicz artery supplied by right side T12 lumbar artery. Whether distal Adamkiwicz artery or anterior spinal artery was patent could not be determined due to technical limitations of CT-angiography. Transthoracic echocardiography showed no embolic source nor patent foramen ovale. Electrocardiogram revealed normal sinus rhythm. Detailed history eliminated artheroscelerosis risk factors like smoking or family history of premature vascular events. She had no medication history.

The patient was transferred for spinal cord injury rehabilitation two weeks after admission. She had minimal recovery of proximal lower limbs muscle strength in both legs (American Spinal Injury Association Class B with sensory neurological level at T11). One month later, the patient had improved mostly in proximal lower limbs muscle strength (bilateral MMT L2/L3/L4/L5/S1:3/3/2/2/1) and had partially recovered her ability to urinate, and was discharged.

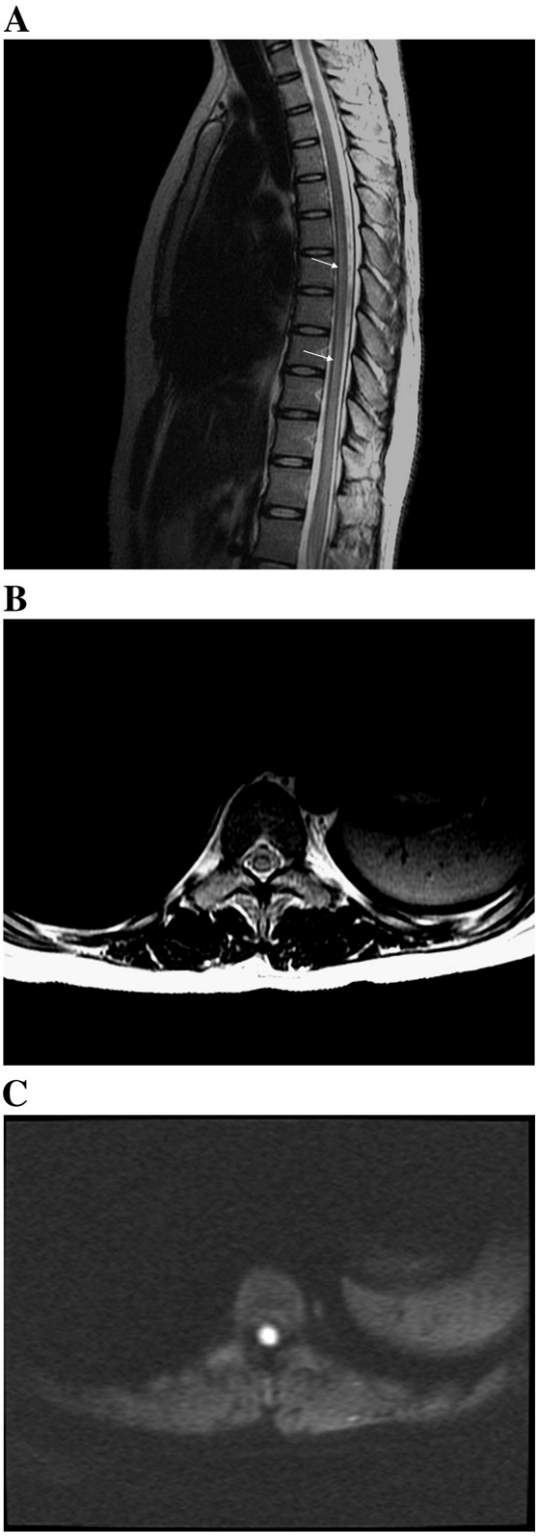

Fig. 2. Throacic MRI 3 days after symptoms onset. (A) Sagittal T2WI. (B) Axial T2WI. (C) Diffusion-weighted image. Presence of diffuse high signal intensity noted within the anterior-central spinal cord on T2WI(B) and DWI(C) noted from T6 through conus medullaris (arrows, A). T1WI of the region showed isoindensity and postcontrast study shows no obvious enhancements. These findings were compatible with spinal cord infarction, localized within the anterior and central spinal cord.

At six months, her lower extremity muscle strength improved to MMT:4 (L2 to S1). She felt persisted paresthesia below L3 dermatome. She walked with the use of bilateral Lofstrand crutches and urinated by reflex voiding. MRI of thoracic and lumbar spine showed the spinal cord hyperintensities had subsided. At one year, she could walk with single cane. Marked spasticity of lower extremities was noted.

3. Discussion

Most surfing injuries are either caused by impact with surfboard, ocean floor, or wave or surfer's own body motions, causing sprains, strains, lacerations, contusions and fractures [2,3] . In the first case series of surfer's myelopathy, Thompson et al. found the symptoms to be back pain, urinary retention, sensory deficit and lower limb weakness during or shortly after surfing [1]. They found MRI signal changes in the lower thoracic spinal cord, but spinal angiography findings, which was performed for two patients, were negative [1]. The only similarity they found in their patients was inexperience surfing and a lack of trained musculature [1]. Avilés-Hernández reported one case involving a man with trained musculature [4]. Outcomes vary from complete recovery to incomplete recovery with persistent paraparesis and urine retention to persistent paraplegia.

Thompson et al. suggested the injuries were caused by avulsion of perforating vessels, vasospasm of the artery of Adamkewicz, and transient ischemia in areas of borderline perfusion resulting from tension in a hyperextend spinal cord of a surfer lying prone while paddling. They suggested that potential risk factors included body habitus, dehydration, and long distance air travel, associated with hypercoagulable state and deep venous thrombosis. Avilés-Hernández suggested that inferior vena cava compression might lead to increased intraspinal pressure and fibrocartilaginous embolism (FCE) myelopathy [4].

Our case is the first reported case of surfing-related non-traumatic myelopathy resulting in anterior cord infarction. The anterior spinal cord is more vulnerable to infarction in the lower thoracic region than posterior spinal cord, because the radicular artery (Adamkiewicz) supplies the anterior spinal artery which has a less extensive collateral blood supply [5]. Anterior spinal cord syndrome has been associated with atherosclerosis and aortic disease [6,7], hypercoagulation [8], cardiovascular embolic events [9], and aortic surgery complications [6,10]. Novy et al. [5], studying 27 cases of spinal cord infarction classified them into (1) radicular artery territory infarct (bilateral anterior or posterior spinal artery infarcts and unilateral infarcts which occurred after mechanical triggering) and (2) central and transverse infarcts related to extensive spinal cord hypoperfusion. Our patient presented preserved dorsal column sensation with paraplegia. Her MRI findings showed high T2WI signals and DWI in the anterior-central cord, as is found in anterior spinal cord infarction. This began while surfing and progressed to paraplegia within 3 h.

Another possible cause for the spinal cord infarction is fibrous cartilaginous embolism (FCE), a fatal or debilitating disease. Its first symptoms are sudden onset of back pain, followed by a relatively rapid progressive neurologic deficit, indicating a “spinal stroke in evolution” [11]. Histological examination is needed to confirm this diagnosis, but many clinically suspected FCE myelopathy cases do not provide it, relying on neuroimaging only [11,12]. FCE is thought to be forced into the radicular artery by retrograde flow from the nucleus pulposus blood vessels or from vertebral endplate sinusoids [11]. This could be triggered by Valsalva maneuver. Our patient shared the same symptoms and similar triggering event as those previously reported FCE cases though we also do not have histological proof. However, absence of Schmorl's nodes in the imaging studies of our patient might be a clue against FCE as some authors considered that it might be a potential mechanism of FCE [12].

4. Conclusion

Surfing is a popular exercise, but requires trained back muscles and several skills. Trainers and trainees should be informed about the possibility of the injuries described in this report and should remain alert for the initial symptoms like back pain and lower limb weakness. Stopping surfing immediately might reduce further injury. Valsalva maneuvers should be avoided during the surfing course to prevent an exaggerated increase in intra-spinal pressure. Physicians should be alert of surfer's ischemic myelopathy and arrange diagnostic tools including MRI with DWI for early diagnosis.

References

[1] Thompson TP, Pearce J, Chang G, Madamba J. Surfer's myelopathy. Spine 2004;29 (16):E353–356 (Phila Pa 1976).
[2] Nathanson A, Haynes P, Galanis D. Surfing injuries. Am J Emerg Med 2002;20(3): 155–60.
[3] Nathanson A, Bird S, Dao L, Tam-Sing K. Competitive surfing injuries: a prospective study of surfing-related injuries among contest surfers. Am J Sports Med 2007;35 (1):113–7.
[4] Avilés-Hernández I, García-Zozaya I, DeVillasante JM. Nontraumatic myelopathy associated with surfing. J Spinal Cord Med 2007;30(3):288–93.
[5] Novy J, Carruzzo A, Maeder P, Bogousslavsky J. Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients. Arch Neurol 2006;63 (8):1113–20.
[6] Cheng MY, Lyu RK, Chang YJ, Chen RS, Huang CC, Wu T, et al. Spinal cord infarction in Chinese patients. Clinical features, risk factors, imaging and prognosis. Cerebrovasc Dis 2008;26(5):502–8.
[7] Nedeltchev K, Loher TJ, Stepper F, Arnold M, Schroth G, Mattle HP, et al. Long-term outcome of acute spinal cord ischemia syndrome. Stroke 2004;35(2):560–5.
[8] Hakimi KN, Massagli TL. Anterior spinal artery syndrome in two children with genetic thrombotic disorders. J Spinal Cord Med 2005;28(1):69–73.
[9] Mori S, Sadoshima S, Tagawa K, Iino K, Fujishima M. Massive spinal cord infarction with multiple paradoxical embolism: a case report. Angiology 1993;44(3):251–6.
[10] Tsai YD, Liliang PC, Chen HJ, Lu K, Liang CL, Wang KW: Anterior spinal artery syndrome following vertebroplasty: a case report. Spine (Phila Pa 1976), 2010 35 (4):E134-136.
[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):153–7.
[12] Tosi L, Rigoli G, Beltramello A. Fibrocartilaginous embolism of the spinal cord: a clinical and pathogenetic reconsideration. J Neurol Neurosurg Psychiatry 1996;60 (1):55–60.