

Traumatic carotid artery dissection caused by bungee jumping

Wei Zhou, MD, Tam T. Huynh, MD, Panagiotis Kougias, MD, Hosam F. El Sayed, MD, and Peter H. Lin, MD, *Houston, Tex*

Bungee jumping is a popular recreational activity in which participant experiences transient freefall while connected to a bungee cord. The rapid freefall and the resultant rebound force created by the bungee cord can result in a variety of bodily injuries. We report herein a case of traumatic carotid artery dissection caused by bungee jumping. The symptoms related to carotid artery dissection were successfully treated with anticoagulation. The etiology of carotid dissection related to bungee jumping is discussed. Physicians should be cognizant of this potential injury due to the force created by the freefall and rebound motion associated in this recreational sport. (*J Vasc Surg* 2007;46:1044-6.)

Bungee jumping is a popular recreational activity as participants connected to a safety bungee cord leap off a platform high above the ground to experience a transient sensation of freefall. It originated as a rite of passage into manhood in the Pentecost Islands of the Southern Pacific.¹ The rite is to test male courage by jumping off a bamboo tower with vines attached to their ankles. The concept was introduced into the United Kingdom in 1979 by the Oxford Dangerous Sports Club.² Since that time, this recreational activity has evolved significantly with participants leaping from far higher ground to experience the freefalling thrills. Obvious bodily injuries and even death have occurred due to miscalculation of cord length, break in the bungee cord, or improper placement of safety harness. Additionally, physical injuries related to the high impact force of this recreational sport have been described, which included ocular injury,³ orthopedic trauma,⁴ and even intracranial hemorrhage.² We report herein the first known case of traumatic carotid dissection as a result of bungee jumping. Potential etiological factors related to this injury and management is discussed.

CASE REPORT

A 28-year-old male participated in a bungee jumping event in which he leaped from a bridge situated approximately 400 feet above an underlying river. Within 10 minutes following this uneventful bungee jumping, he developed fluctuating dull pain in his left neck, left ear, and left occiput. Approximately 2 hours later, he developed right arm paresthesia. He did not experience visual disturbances or loss of consciousness. He was brought urgently to a regional hospital for further evaluation. A physical examination revealed mild right arm paresthesia without lower extremity symptoms or any motor weakness. The patient did not suffer any

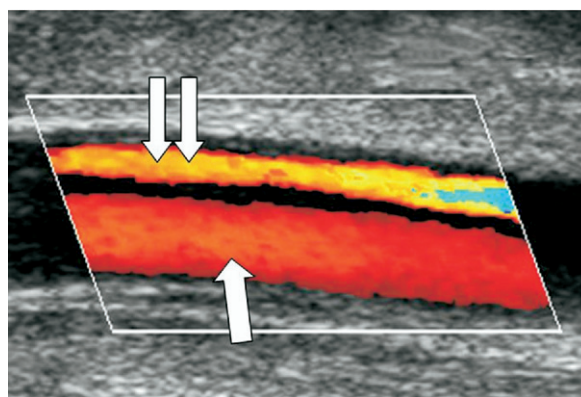


Fig 1. A carotid duplex revealed evidence of carotid dissection in the common carotid artery just proximal to the carotid bulb. Flow in the true lumen (*double arrows*) was distinctively separate from the flow in the false lumen (*single arrow*).

ophthalmologic or Horner's syndromes. No orthopedic or other neurological symptoms were noted in this patient. An ultrasound examination of the carotid arteries was suggestive of left carotid artery dissection (*Fig 1*). A magnetic resonance imaging (MRI) of the brain showed no evidence of cerebral infarction. A concomitant MR angiogram showed a partially flow-void lumen surrounded by the high-intensity signals of intramural hematoma, which was consistent with a carotid dissection (*Fig 2*). The dissection began in the common carotid artery and extended to the intracranial siphon. He was admitted to the hospital and received intravenous heparin, which was followed by oral coumadin for 6 months. His headache and extremity paresthesia gradually resolved within 2 days following anticoagulation. He remained symptom-free following discharge from the hospital 1 week later. A follow-up ultrasound examination at 1 year showed complete healing of the left carotid artery without evidence of carotid dissection (*Fig 3*).

DISCUSSION

Bungee jumping has been associated with a variety of injuries. The most common reported injury relates to ophthalmologic trauma such as retinal hemorrhage and ocular

From the Division of Vascular Surgery and Endovascular Therapy, Michael E. DeBakey Department of Surgery, Baylor College of Medicine and Michael E. DeBakey VA Medical Center.

Competition of interest: none.

Reprint requests: Peter H. Lin, MD, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston VAMC (112), 2002 Holcomb Blvd, Houston, TX 77030 (e-mail: plin@bcm.tmc.edu).

0741-5214/\$32.00

Copyright © 2007 by The Society for Vascular Surgery.

doi:10.1016/j.jvs.2007.06.026

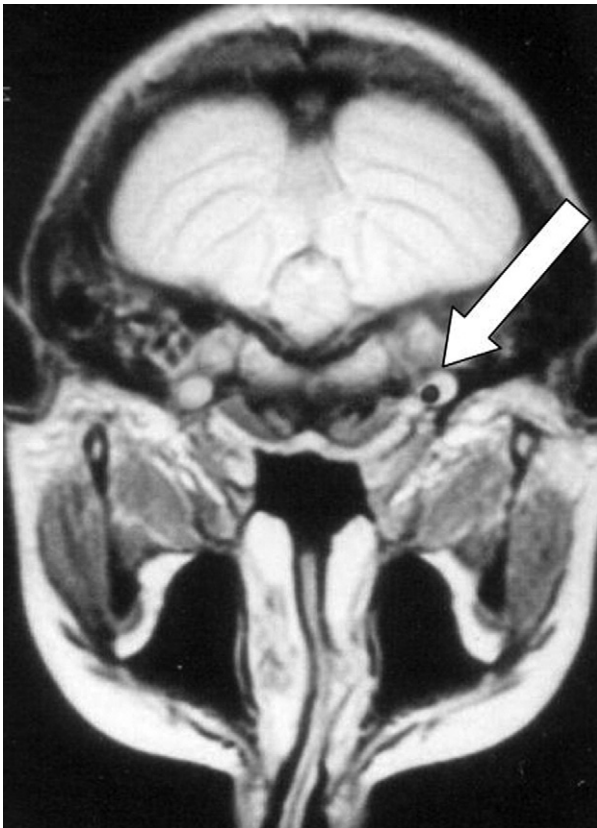


Fig 2. A MR angiogram confirmed a left carotid artery dissection (*arrow*), which depicted a partial flow-void lumen surrounded by the high-intensity signals of intramural hematoma.

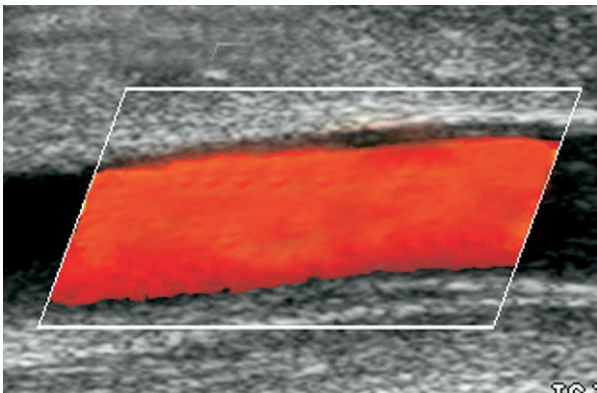


Fig 3. Follow-up duplex carotid ultrasound at 1 year showed complete healing of the carotid dissection without evidence of false lumen circulation.

emphysema.⁵ Orthopedic injuries are also common in this sport, which include symptomatic cervical spin disc herniation, humeral head dislocation, and proximal femoral fracture.⁴ Subdural hematoma has also been reported associated with bungee jumping.² To our knowledge, carotid

artery dissection due to bungee jumping has not been reported previously in the literature.

Several mechanisms related to bungee jumping may contribute to the pathogenesis of traumatic carotid dissection. During bungee jumping, the participant undergoes freefall for up to 400 feet, who is then immediately jerked back to safety by the bungee cord, which can last for up to several minutes.⁶ During the process of tightening the bungee cord, there is a tremendous deceleration force generated approaching 32 kilojoules, which may cause neck hyperextension.⁶ The force created during a vertical jumping and subsequent rebound motion may generate forces which (1) stretches the internal carotid artery over the transverse processes of the upper cervical vertebrae, (2) directly compresses the carotid artery between the angle of the mandible and upper cervical vertebrae, or (3) impinges on the carotid artery by the styloid process.⁷ The stretching force can potentially lead to an intimal tear, which can result in plaque dissection that propagates in the tunica media along the arterial wall.⁷ Even in a healthy individual who does not have significant carotid plaques, dissection of the carotid artery can develop between layers of an otherwise normal artery. Additionally, hyperextension of the neck can lead to stretching of the internal carotid artery and subsequent arterial compression against the cervical transverse process. It is noteworthy that when the neck is hyperextended, the carotid artery is more susceptible to external forces because in this position carotid artery is less protected by mandible and cervical vertebrae body.¹³ We, therefore, postulate that there is an increased risk of carotid dissection during bungee jumping when the participant's neck is hyperextended while undergoing an extreme freefalling and rapid rebound deceleration force.

Carotid artery dissection may present with an array of symptoms from asymptomatic to massive cerebral infarct. Many patients may present with nonspecific symptoms such as headache or neck pain, which were initial symptoms in our patient. Ischemic symptoms may be caused by hematoma in the tunica media compressing the true lumen and reducing cerebral blood flow. More commonly, it is believed that the symptoms are due to a distal embolization of thrombus that has developed at the site of the intimal tear or within an aneurysmal dilation. Hematoma may also compress the sympathetic nerve fibers that travel along the internal carotid artery causing Horner's syndrome.⁸ There is frequently a delay between the episode of trauma and the onset of symptoms. Bioussé and associates studied 80 patients with angiographically diagnosed carotid artery dissection and followed these patients to determine the time elapsed between the onset of the first symptoms and the onset of any ischemic event.⁹ These researchers concluded that completed stroke usually occurs in the first few days after the onset of the first symptoms, but it can occur as much as 1 month later. This delay provides a window of opportunity where early detection and intervention may significantly improve outcome and prevent lifelong morbidity or mortality in a generally young population of patients. In our patient whose hemiparetic symptoms oc-

curred within 2 hours following the bungee jumping event, we postulate that this was a cerebral ischemic phenomenon due to flow cessation in the internal carotid artery. This was further supported by the findings in the MR angiogram.

Due to the rarity of traumatic carotid dissection, there is no evidence-based guideline for the best management of this entity. Carotid duplex ultrasound should be the first diagnostic evaluation in patients suspected of carotid injury following bungee jumping or other recreational misadventures. Aggressive surgical or interventional treatment is often not helpful for an established neurological deficit, particularly in patients who have sustained blunt trauma. Surgical or interventional treatments may be appropriate in selective patients who suffer carotid dissection due to penetrating or iatrogenic catheter-related injuries. In contrast, medical therapy with systemic anticoagulation therapy is believed to be helpful in preventing progressive thrombosis and arterial occlusion or distal arterial embolization with resultant cerebral ischemia. It is noteworthy that systemic heparinization should be administered only after possibility of cerebral hemorrhage is excluded by means of computed tomography (CT) or MRI of the brain, particularly in patients with symptoms of neurological defects. In a registry report, which examined the outcome of 683 patients with carotid dissection, Lyrer and associates reported that there was a lack of evidence to support treatment superiority using either antiplatelet or anticoagulant therapy in carotid dissections.¹⁰ However, anticoagulation with heparin followed by oral coumadin therapy remains the most widely accepted treatment strategy in this condition. Follow-up duplex ultrasound is important in patients who sustained carotid dissection to ensure carotid healing and possible early detection of other sequelae such as pseudoaneurysm formation. Our patient responded clinically once

systemic anticoagulation was initiated and had no residual sequelae in follow-up visits.

With the increased popularity of bungee jumping, health care providers such as emergency room physicians or vascular surgeons may encounter vascular complications such as carotid dissection as a result of this recreational sport. It is important that physicians have a heightened index of suspicion of this injury so that prompt diagnosis can be made, followed by appropriate treatment to avoid potential neurological sequelae of this injury.

REFERENCES

1. Harries M. The ups and down of bungee jumping. *BMJ* 1992;305:1520.
2. FitzGerald JJ, Bassi S, White BD. A subdural hematoma following 'reverse' bungee jumping. *Br J Neurosurg* 2002;16:307-8.
3. Chan J. Ophthalmic complications after bungee jumping. *Br J Ophthalmol* 1994;78:239.
4. Huang KW, Huang SJ, Lin TH, Yen ZS, Chen SC. Anterior dislocation of the humeral head from bungee jumping. *Am J Emerg Med* 2001;19:322-4.
5. Vanderford L, Meyers M. Injuries and bungee jumping. *Sports Med* 1995;20:369-74.
6. Shapiro MJ, Marts B, Berni A, Keegan MJ. The perils of bungee jumping. *J Emerg Med* 1995;13:629-31.
7. Schievink WI, Atkinson JL, Bartleson JD, Whisnant JP. Traumatic internal carotid artery dissections caused by blunt softball injuries. *Am J Emerg Med* 1998;16:179-82.
8. Baumgartner RW, Bogousslavsky J. Clinical manifestations of carotid dissection. *Front Neurol Neurosci* 2005;20:70-6.
9. Biousse V, D'Anglejan-Chatillon J, Touboul PJ, Amarenco P, Boussier MG. Time course of symptoms in extracranial carotid artery dissections. A series of 80 patients. *Stroke* 1995;26:235-9.
10. Lyrer P, Engelter S. Antithrombotic drugs for carotid artery dissection. *Cochrane Database Syst Rev* 2000:CD000255.

Submitted May 13, 2007; accepted Jun 6, 2007.