Exercise Related Spinal Cord Infarction: A Case Report

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Abstract-

Purpose: A sudden elevation of the intervertebral disc pressure could result in injection of cartilaginous material into spinal small caliber vessels causing embolism of the underlying vessels is well documented but rarely occurs.

Case report: Here we present a patient with sudden onset of weakness over left upper extremity several minutes after neck rotation exercise. The symptoms were progressive with quadra-paresis and urine incontinence without cranial nerve or sensory function deficit. The cervical spine MRI showed cervical spondylosis with degenerative osteophytes of lower cervical-spine with mild indentation of the thecal sac. There was also focal T2-high intensity edema of "H" gray matters of C3 spinal cord. According to the preliminary examination and clinical course, the symptoms of the patient can be explained by the ischemia of spinal cord secondary to fibrocartilaginous embolism(FCE) derived from intervertebral discs. After aspirin and IV dexamethasone treatment, the clinical condition of the patient improved significantly.

Conclusion: Neck rotation exercise is common among all kinds of relaxation or physical rehabilitation activities. However, the potential unwanted effects of this exercise suggest that one should be aware of the potential adverse neurologic outcomes especially inappropriate non- professional method.

Key Words: fibrocartilaginous embolism, neck rotation exercise, spinal cord infarction

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INTRODUCTION

Hyperextension or rotation of the neck from a wide variety of activities (including coughing, sports, lifting, ceiling painting, yoga, archery, and falls) has been associated with neurovascular syndromes due to dissection or occlusion of the vessels. Such neck movements, particularly when they are sudden, may injure the artery as a result of mechanical stretching[(1)]. Because of the neck anatomy, the vertebral arteries are particularly susceptible to injury resulting from head or neck torsion[(2)]. A sudden elevation of both the venous pressure in the craniospinal axis and the intervertebral disc pressure could result in injection of cartilaginous material into spinal small caliber vessels causing embolism of the underlying vessels[(3-4)]. Such fibrocartilaginous embolism(FCE) is an uncommon cause of anterior spinal artery infarction that is generally diagnosed only

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at autopsy. Several researches also found that chiropractic neck manipulation has been associated with carotid artery or vertebral artery dissection resulting in progressive brainstem infarction[5,6]. Here we present a patient who had sudden onset of limbs weakness with the initial impression as brain stem infarction. After further survey and imaging findings the diagnosis was changed to spinal cord infarction. The patient reported that the symptom occurred right after he performed successive rotation of head. Since there was no other significant predisposition or risk factors for him, we concluded that our patient may be the first reported case of anterior spinal artery occlusion occurring in association with neck rotation resulting in fibrocartilaginous embolization.

CASE REPORT

A 58-year-old man came to emergency room because of an acute onset of four limbs weakness for two hours. He has not been diagnosed with any systemic diseases, and did not have any operations before. He smoked one pack per day for almost thirty years and he drank around two bottles of beer or wine three times a week. According to the patient's statement, the previous afternoon at approximately one o'clock in the afternoon after drinking one bottle of light wine(Paolyta-P liquid), he performed successive rotation of neck near vertical angle around 360˚, and repeated another cycle soon later for reliving stiffness of nuchal region. Several minutes after the exercise, he experienced a sudden onset of weakness over left upper extremity. He had an idea of possibility of stroke and repeated the same exercise two cycles in order to relieve the symptom. However the condition worsened gradually and weakness extended to four limbs twenty minutes later.

He was brought to our emergency room where CT of brain performed under tentative impression of brains stem stroke did not show any lesions. At ER, the muscle strength of four limbs partially improved. Three hours late he developed acute urinary retention and received urinary catheterization. Under impression of brain stem infarction or cervical spine myelopathy, the patient was admitted to our neurology ward. The initial vital signs were stable except elevation of the blood pressure 140/100 mm Hg (rechecked to be 129/88mm Hg). Neurological examination showed an awake but lethargic man with normal speech, cognition, and cranial nerve function. Motor examination disclosed incomplete paralysis of the four limbs with muscle strength grade 3 in left upper limb, grade 4- in right upper limb and grade 4 over bilateral lower limbs. The deep tendon reflexes were absent over four limbs. Sensory examination unfolded normal pinprick, light touch and vibration test.

During the first day of hospitalization, quadiparesis and urine incontinence persisted without cranial nerve or sensory function deficit. Cervical spine MRI was obtained showed cervical spondylosis with degenerative osteophytes of lower C-spine with mild indentation of thecal sacs. There was also focal T2-high intense edema of "H" gray matters of C3 spinal cord consistent with an impression of acute transverse myelitis or ischemic infarcts (Fig. 1A-B). According to preliminary examination and clinical course, the condition of the patient was more consistent with spinal cord infarction. The patient was given four doses of aspirin, IV dexamethasone and underwent series of diagnostic work up. The laboratory studies revealed mild elevated cholesterol (220mg/dl), sugar (119mg/dl), and normal ANA, non-reactive VDRL test. The patient also had full evaluation for coagulation profile, which showed normal results of anti-thrombin III, homocysteine and protein S, except mild elevated protein C(153.1%). Brain and neck 64 multi-detector computed tomographic showed minimal calcified plaques of bifurcations of bil CCAs with less than 10% narrowing of lumen without significant abnormalities of bilateral ICAs and vertebral arteries, and intracranial circle of Willis. Aside from those, the cardiac echogram and ophthalmological evaluation of his fundus revealed no strong evidence of hypertensive cardiovascular disease.

Two days after hospitalization the patient's muscle power improved gradually. On the third day, his lower limbs weakness as well as sphincter dysfunction returned to nearly normal level but his upper limbs still showed partial paralysis with grade 4 + over right upper

limb , grade 4- in left upper limb distal muscle group and grade 3 in the proximal muscle group. After all survey the final diagnosis was anterior spinal artery infarction related to rotation of the neck.

The patient returned to neurology OPD six months after the attack, and his muscle power of four limbs returned to normal function except left upper limb with muscle power of grade 4 in distal muscle group and grade 4- in the proximal muscle group. The follow-up C-spine MRI was also correlated to the clinical symptoms of the patient with old ischemia infarction of left C3 cord with residual irregular longitudinal hyperdense lesion and absence of edematous change. (Fig. 2A-B)

DISCUSSION

Infarction of the spinal cord is rare. The classic description of the clinical syndrome of anterior spinal artery infarction was given nearly 100 years ago by Preobrashenski[7]. Due to its rarity, reliable estimates of incidence are scarce; however one study noted spinal cord infarction accounted for 1.2% of all strokes[8]. Of

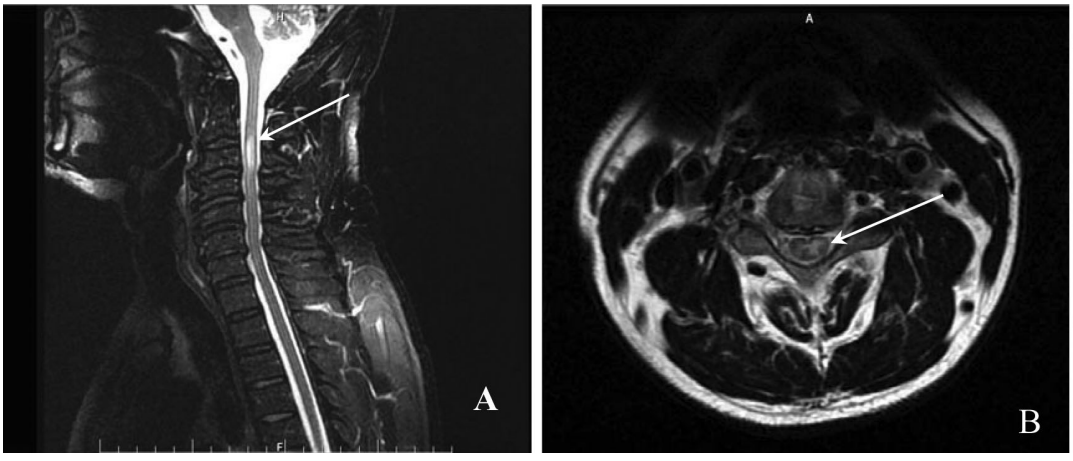

Figure 1. C-spine MRI. (A) & (B) T2-high intensity edema of the "H" gray matter of the C3 spinal cord with minimal enhancement.

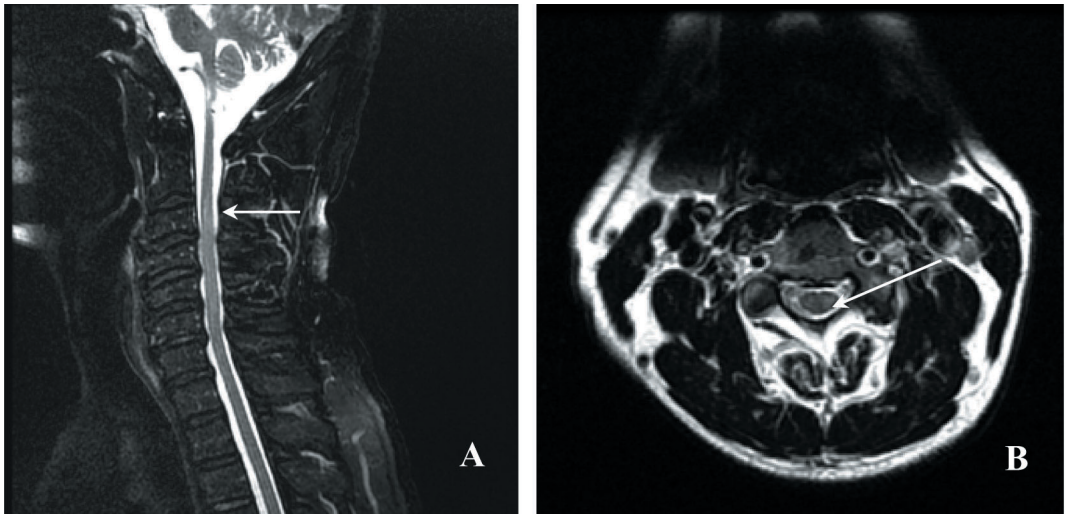

Figure 2. C-spine MRI six month after the attack. (A) Old ischemic infarct of left C3 spinal cord with residual irregular longitudinal hyperdense lesion of the left C3 region. (B) Edematous change of "H" gray matter of the C3 spinal cord recovered.

the several potential causes for spinal cord infarction (SCI), aortic surgery for aortic aneurysm is the leading cause, followed by atherosclerosis of the aorta and spinal arteries[7]. However FCE is a rarely recognized, but important cause of spinal cord infarction which occurs after minor trauma. In some patients, symptoms began on awakening, or after a Valsalva maneuver with straining at defecation or a suppressed sneeze[9-10]. Patient with hypertension, obesity, diabetic mellitus, coagulopathies, episodic hypotension, and degenerative disease of the spine are at increased risk[11].

Because the similarity of the retinal vessels and cerebral vasculature both embryologically and anatomically, abnormalities of these vessels can reflect cerebral vascular damage[12]. It has been reported that many retinal signs, in particular hypertensive retinopathy signs (eg, focal retinal arteriolar narrowing, arterio-venous nicking), may be markers of stroke risk and mortality, independent of other stroke risk factors such as hypertension or diabetic mellitus[13]. Hence one can also use retinal examination as a stroke risk assessment.

This 58-year-old man was in his good health previously. Although he had history of smoking, alcohol use with mild hyperlipidemia there was no hypertension neither hypertension related small artery lesion such as hypertensive retinopathy was found. The unfolding history of neck rotation, specific physical findings and neuroimage studies allow a narrowing of the differential diagnosis to the etiology of spinal cord infarction. Thus neck rotation might be a contributing factor to the spinal cord infarction in this patient.

Spinal cord infarction due to occlusion of the anterior spinal artery, the principal vascular supply to the spinal cord, characteristically presents with an abrupt onset of bilateral weakness, especially the lower limbs, sudden back pain that radiates caudally, flaccid paraplegia, areflexia, loss of pain and temperature sensations below the level of the lesion, sparing of proprioception and vibration sense and autonomic dysfunction involving the bladder and bowel[11]. The diagnosis should be suspected based on physical examination and confirmed by MRI[14].

Our patient presented with sudden onset of weakness over left upper extremity several minutes after performing neck rotation exercise. The condition gradually extended to four limbs after he performed another two cycles of neck rotation and eventually autonomic dysfunction occurred. The progression of symptoms can be explained by the ischemia of spinal cord secondary to FCE arising from intervertebral discs. It may be due to successive mechanical over stretch and torture resulting in disc rupture especially in degenerating discs as in our patient. The penetration of disc material finally caused spinal artery occlusion and clinically the patient would present with intervening symptom-free intervals and progressive "stroke-in-evolution" course. However, the reason of no detectable sensory deficit observed in our patient may be due to his infarction of the spinal cord mainly involving in the motor area as seen on MRI result.

The nature and severity of traumatic events which have been aetiologically associated with FCE very, and there is no established temporal course for the postulated process of disc injury and cord infarction[9]. Currently there is no treatment noted to facilitate improvement in patients with anterior spinal artery infarction caused by FCE. Treatment remains supportive and rehabilitation. Prognosis is predicted based on the MRI appearance of the spinal cord and clinical presentation.

Although several potential causes for spinal cord infarction are mentioned in the literature. In one retrospective study, the etiology and prognosis of those patients suffering from acute spinal cord ischemia were evaluated. Of 36 cases analyzed, there were still 36.1% of spinal cord infarction cases categorized as idiopathic patients[3]. In serial literature review, there are approximately 40 cases of spinal artery infarction related to FCE from intervertebral disc tissue[10,15] However, diagnosis of spinal cord infarction caused by FCE still needs histological confirmation. Nowadays neck rotation exercise especially in the elderly or people with degenerative discs may contribute a precipitating event as in our case presented here. Despite being very uncommon, cord ischemia of such an etiology should be included in the differential diagnosis of acute spinal syndromes, especially in patients who undergo trauma and have abnor-

mal signal change in the intervertebral disc shown on MRI. Moreover, detailed history taking including neck exercise is also strongly recommended.

Neck rotation exercise is commonly practiced among all kinds of relaxing or physical rehabilitation activities. However the potential unwanted effects of this exercise suggest that one should be aware of the potential adverse neurologic outcomes especially after inappropriate non- professional methods.

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