Nontraumatic spinal cord ischemia is uncommon, especially at the cervical spinal cord. We describe a 30-year-old man presenting with acute onset of quadriplegia and impaired sensation for pain and temperature after backing up the car. He was diagnosed noninvasively with magnetic resonance imaging (MRI) and electrophysiological studies. The image studies had revealed a high signal intensity lesion in the cord from C5 to the upper part of thoracic spinal cord (T2) on T2-weighted images. The nerve conduction velocity study (NCV) revealed non pick-up of bilateral F waves and median nerves of upper limbs with decreased compound muscle action potentials (CMAPs) of the bilateral ulnar nerves. Motor evoked potentials (MEP) after transcranial cortical stimulation revealed low amplitudes and abnormal central conduction time to the legs and arms, which was more severe on the upper limbs and especially on the left side. The patient regained most of the muscle power except extensor muscles of the elbow and wrist after conservative treatment.

Key words: anterior spinal artery syndrome; Magnetic resonance imaging; electrophysiological study

CASE REPORT

A 30-year-old man suffered from acute onset of stabbing chest pain, which radiated to the left upper limb especially down to the distal part, after backing up the car. At that time he backed up his car by looking at the back mirror without twisting his neck. There was no history of trauma except playing tennis the day before the incident. He sought treatment at ER at once and was treated for acute myocardial infarction with oxygen and nitroglycerate. Gradually, he noticed rapid loss of muscle strength of the upper limbs followed by lower limbs within 6 hours after admission. Later he was unable to urinate and feel numbness sensation in an ascending nature.

His family history and past medical history were unremarkable. He smoked 1 pack of cigarettes per day. On initial examination his blood pressure was 130/80 mmHg; pulse rate 76 beats/min; temperature 36.6°C; respiratory rate 20/min. No abnormality was found on...
the examination of heart and abdomen. The emergent computed tomography (CT) of the brain was done under the impression of a young stroke with unremarkable findings. On neurological examination, the cranial nerves were normal. However, weakness was more prominent in the distal parts of limbs. Flaccid paraplegia was observed in his lower extremities with muscle strength of the upper limbs as follows: medical research council (MRC) grade 5/5 for shoulder flexors and extensors; 5/3 for elbow flexors and extensors; 5/3 for finger flexors and extensors. Pain and temperature sensation were impaired below the level of T4 with sparing of the sensation of touch, position, and vibration. Deep tendon reflexes were retained with absence of abdominal and cremastic reflexes. Plantar reflexes were flexor bilaterally. Other neurologic findings included a flaccid rectal tone and a neurogenic bladder. An emergent CT scan of the spine from the level of C7 to the T4-5 was arranged under the impression of a lower cervical lesion but no abnormality was found. He was treated conservatively with steroid injection immediately after a normal cerebrospinal fluid study. MRI of the cervical spine without contrast enhancement was performed 2 days later and revealed a high signal intensity stripe in the anterior portion of the cervical spinal cord from the bony level of C5 to the upper part of T2 level on T2-weighted sagittal images with swelling of the cord (Figure.1, 2, 3).

Investigative studies including peripheral blood cells, biochemistry profiles with cardiac enzymes, chest x-ray, electrocardiograph, gas analysis, urinalysis, erythrocyte sedimentation rate, prothrombin and thromboplastin time, serologic test for syphilis, glucose, cholesterol and triglyceride, were all within normal reference ranges (RR). Coagulation and collagen profiles were unremarkable. Echocardiogram of the heart revealed early sclerosis of the aortic valve with moderate mitral regurgitation and tricuspid regurgitation while transesophageal echocardiogram showed a flat detachment of the interatrial septum at the patent foramen ovale, and no right to left shunt after contrast echo. Perfusion/ventilation lung scans showed no apparent segmental perfusion defect. The patient refused an angiography for fearing a deterioration of muscle power. SSEP obtained from the stimulation of the tibial and median nerves were normal. Extracranial Doppler ultrasound investigation on admission was normal. Nerve conduction velocity (NCV) studies performed 1 week after admission showed non pick-up of F waves of the upper limbs and non pick-up of the bilateral median nerves with markedly decreased compound muscle action potentials (CMAPs) of the bilateral ulnar nerves. The sensory components were normal with sparing of the lower limbs. MEP after transcortical stimulation revealed low amplitudes and abnormal central conduction time to the legs and arms, which was more severe on the upper limbs and especially on the left side.

Figure 1. A sagittal T1-weighted MR image of the cervical spine on admission shows no enhancement of the lesion.

Figure 2. A sagittal T2-weighted MR image of the cervical spine on admission shows high signal intensity lesion extending from bony level of C5 to upper part of the T2.
After an intensive rehabilitation program, he regained most of his motor power except for the elbow and finger extensors. However, sensory impairment to pin-prick and temperature was still diminished below T4. At discharge on day 33, he could walk well but continued to have some retention of urine. Follow-up MRI of the cervical spine 3 months later revealed atrophy of the corresponding spinal cord and NCV study showed no significant improvement (Figure 4, 5). The electromyography revealed non-pick-up in sampling of the hand muscles like abductor pollicis brevis and abductor digiti minimi. Clinically, presumably acute spinal cord infarct was impressed from the imaging finding, clinical history and electrophysiological studies.

**DISCUSSION**

Infarction of the spinal cord is rare compared with cerebral infarction. There are only very limited epidemiological data on the prevalence of the spinal cord infarction. Spiller et al. provided the classic description of anterior spinal artery in 1909 [4]. The dramatic clinical picture of acute loss of segmental spinal cord function without obvious external cause requires urgent action to diagnosis potentially treatable conditions. Nontraumatic spinal cord ischemia is uncommon, especially when the cervical cord is involved as in our patient. There were literatures discussing minor trauma relating to spinal cord injuries in children but not in adult [5]. The most common cause of acute intrinsic spinal cord lesion is aortic disease [6]. Other causes include myocardial infarction, dissecting aortic aneurysm, hematomyelia, arteriovenous malformation, acute transverse myelitis, remote...
effects of malignancy, cardiogenic embolism, sickle cell anemia, surgery, radiation, trauma, fibrocartilaginous embolism, bulging intervertebral disks, cervical spondylosis, hypercoagulable state and multiple sclerosis [6,7-15]. All of these conditions were excluded in our patient by history, normal laboratory data, CSF study, perfusion and ventilation scan, echocardiograms and MRI finding. Another consideration is vertebrobasilar artery insufficiency, which is unlikely, however, in patients with normal extra-cranial Doppler study [14].

In our patient, the lesion appeared as a high signal intensity involving the center of a long segment of cervical spinal cord on T2-weighted sagittal images and as cord swelling on T1-weighted axial images. Follow-up MRI showed atrophy of the cord, which was compatible with ischemia of the territory of the anterior spinal artery. According to the literature review of ASAS, we found that ASAS in our case might have been a cryptogenic in origin. Foo and Rossier in a review of 60 patients with ASAS found no clear cause in almost 25% of the cases [16].

The diagnosis of ASAS is made from the characteristic clinical presentation. MRI is a useful modality in detecting spinal cord infarction and the associated vascular and bony changes. The patterns of bone marrow abnormalities reflect the underlying pathophysiology of the blood supply to the spinal cord and bone [17]. Mawad et al. showed that in the early stage of the lesion the anterior horns are predominantly affected, leading to an “owl’s eye” pattern on the T2-weighted axial images. Four different patterns of T2 signal intensity abnormality on the axial images were described, which are related to the degree of involvement of the gray matter; the type A category includes lesions confined to the anterior gray matter (“owl’s eye” pattern), the type B category indicates the entire gray matter involved, the type C pattern indicates the gray matter and adjacent central white matter are involved, and the type D pattern indicates the signal abnormality spreads to involve the majority of the cross-section area of the cords [18]. In recent years there have been several studies of serial MRI in patients with ASAS [19,20]. The abnormal electrophysiological studies mentioned above are compatible with the clinical course. In ASAS, the ischemic process involves neither the spinal ganglion nor the dorsal columns; thus, the abnormal sensory nerve conduction studies and SSEP finding are absent as in our patient [21]. He also had abnormal MEP, which can detect anterior horn cell involvement in case of ischemia of the spinal cord [22]. EMG study also confirmed anterior horn cell lesion. So the patient was diagnosed with the aid of MRI and electrophysiological findings like other studies[2,23].

The symptoms described by our patient included abrupt onset with maximal deficit occurring after a few hours, loss of all sensory modalities, with preserved proprioception and vibration sense. The specific electrophysiological findings of normal SSEP and sensory findings, abnormal MEP and motor component of NCV studies may suggest anterior spinal artery occlusion affects the bilateral anterior horn cells of the upper cervical cord (C5-C7), which is verified with MRI. In conclusion, MRI and electrophysiological are valuable tools to further confirm both the location and extension of the spinal lesion in patients with ASAS.

**REFERENCES**

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前端脊動脈症候群：病例報告

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非外傷性的脊髄缺血是不尋常的現象，特別是發生在包含頸部脊髄的情形更是少見。我們描述為一位三十歲男性在倒車之後，出現四肢偏癱併有疼痛及溫度感覺受損之情況。利用非侵入性的核磁共振攝影（MRI）及電生理檢查來協助診斷。影像學的研究顯示：在T2-weighted的影像下，一個高信號強度的病灶出現於第五頸椎至第二胸椎的上端部位。而神經傳導速度研究（NCV）則發現，上肢正中神經及雙側F波無反應以及在雙側尺神經的運動動作電位（CMAP）降低。經顱大腦皮質刺激的運動誘發電位（MEP）則出現對於四肢神經之振幅下降且中央傳導時間異常，此一情況在上肢及左側特別嚴重。在保守治療的數個月後，除了肘部及手腕的伸展肌肉外，其他大部份肌力都獲得改善。

關鍵詞：前端脊動脈症候群；核磁共振攝影；電生理學研究