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Spinal Cord Infarction: Report of Two Cases

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Spinal cord infarction is a rare disease. It is even more uncommon that spinal cord infarction occurs in a patient without any cardiovascular disease. Anterior spinal artery syndrome is much more common than the posterior spinal artery syndrome due to the difference in collateral supply. We present two patients with spinal cord infarction. In both patients, the clinical picture was stereotyped: sudden onset of weakness of limbs, sensory loss below the level of infarction and sphincter dysfunction. Treatment for the two patients was supportive and they had a good recovery over a period of weeks. (J Rehab Med Assoc ROC 2002; 30(4): 241 - 249)

Key words: spinal cord infarction, anterior spinal artery syndrome, posterior spinal artery syndrome

INTRODUCTION

Spinal cord infarction is an uncommon disease which is much less frequent than cerebral vascular disease. However, the true incidence of spinal cord infarction is not known.[1,2] The spinal cord can be affected at cervical, thoracic, or lumbar level and the clinical picture of spinal cord infarction is various, such as abrupt onset of flaccid paresis or quadriplegia, local or radicular back pain, dissociated sensory loss below the level of infarction and sphincter dysfunction.[3-6] Common etiologies include atherosclerosis of the aorta, dissecting aortic aneurysm, embolism, aortic surgery and traumatic injury to the arteries supplying the spinal cord.[4]

Arterial supply to the spinal cord comprises three major vessels, including one anterior (ASA) and two posterior spinal arteries (PSAs).[3] Anterior spinal artery infarction is much more common than the posterior spinal artery infarction because they differ in collateral supply.[6]

Here we report two cases of spinal cord infarction. One patient is a case of anterior spinal artery syndrome (ASAS) resulting from an infarction of the anterior aspect of the spinal cord at the C2 to T3 levels and the other is a case of posterior spinal artery syndrome (PSAS) at T9 to T12 level.

CASE REPORT

patient 1

A 46-year-old man presented at the emergency department with total paralysis of both upper extremities and right lower extremity. He reported that chest tightness and cold sweating were experienced on the morning of 15 January 2001. Sudden onset of bilateral upper limbs with progressive right leg weakness developed later. On
arriving emergency room, electrocardiogram (ECG) revealed normal. Neurological examinations showed a
46-year-old, well-developed, well-nourished man who was alert and oriented with normal cranial nerves. He had
total paralysis of both upper limbs and right lower limb. Tendon reflexes were diminished with bilateral absence
of Babinski sign. Pinprick and thermal sensations were
decreased below C4 dermatome with preservation of the
sensations over right lower extremity below L1 derma-
tome. Vibratory, light touch and proprioceptive sensa-
tions were preserved in the trunk and four limbs. Al-
though the sense of bladder distension was preserved, he
had difficulty of micturition. Defecation and perianal
sensations remained normal.

Figure 1. MR images of patient 1 within 6 hours of the event. (A) The sagittal T1-weighted image had no
demonstrable abnormality. (B) The sagittal T2-weighted image showed abnormal increased signal
intensity in the ventral part of the spinal cord from the C2 level to the C7 level.
Intravenous administration of 5mg decadron was given immediately, and a foley catheter was inserted. Hematologic and biochemical data and urinalysis findings were within normal limits. Magnetic resonance imaging (MRI) of cervical spine showed no abnormality on the T1-weighted image (Figure 1A), and an abnormal increase in signal intensity from C2 to C7 on the T2-weighted image (Figure 1B).

Cerebrospinal fluid (CSF) examination showed no blood, no organisms, no WBC and no increase of protein. The blood laboratory data for infectious agents, such as cytomegalovirus, EB virus, herpes simplex, varicella-zoster, syphilis and HTLV-1, were negative. Methylprednisolone (1000mg/day intravenously) was given for 5 days. Chest computed tomography (CT) for aortic conditions disclosed normal results. MRI of thoracic spine followed up 5 days later revealed T2 high-intensity lesion at the levels of low cervical cord to T3 cord (Figure 2).

The patient was transferred to the physical medicine and rehabilitation (PMR) service 3 weeks later. Urodynamic studies were as follows: bladder capacity at first desire to void was 414ml, at maximum desire to void, 589ml. Normally compliant, areflexic bladder was diagnosed. Intermittent catheterization every 4 hours was performed for training of his bladder functions. Muscle strength in both lower limbs had improved greatly and he could walk independently after being in hospital for 6 weeks.

**Patient 2**

A 75 year-old woman experienced sudden onset of weakness and numbness of both lower extremities on June 10, 2000. Past history included hypertension with regular control for 20 years, transitional cell carcinoma (TCC) of urinary bladder in stage A of Marshall’s staging system for 4 years, and had received transurethral resection of bladder tumor (TURBT) and intravesicle instillation with mitomycin-C twice on September 1, 1997 and August 10, 1998. Neurological examinations revealed an alert patient with muscle power of grade 3/5 over bilateral lower extremities. Reduced ankle jerks with obtainable plantar response were noted. Vibratory sensation, light touch and proprioception were lost below the level of the umbilicus (T10). Sensations to pinprick and temperature were preserved. Cranial nerves were all intact. Urine retention was noted in the first two days. ECG demonstrated normal sinus rhythm. Analysis of CSF showed no blood, no organisms, no WBCs and no increase of protein. CT of chest and abdomen failed to demonstrate any abnormalities such as aortic aneurysm or dissection.

![Figure 2](image-url) **Figure 2.** MR image five days after the onset of patient 1. The T2-weighted image also showed hyperintense lesion from low cervical through upper thoracic level on the sagittal view.
A MRI examination of the spinal cord was performed on the following day. There was no abnormality seen on the T1-weighted image (Figure 3A). However, a high signal from the T9 to T12 levels was seen on the T2-weighted image (Figure 3B). Six weeks later, the same hyperintense lesion stood out on the T2-weighted image (Figure 4).

Figure 3. MR images of patient 2 three days after onset. (A) The T1-weighted image was normal on the sagittal view. (B) However, there was hyperintense signal in the posterior part of the cord from the T9 level to the T12 level on the sagittal T2-weighted image.
The patient required intermittent bladder catheterization throughout her hospitalization. She regained most of her motor power of both lower limbs and she was able to walk with a walker at the time of discharge.

She was regularly followed up at the clinics of Neurology and Rehabilitation. Due to incontinence and frequency on urination, urodynamic study was performed on February 9, 2001 and showed bladder capacity at maximum desire to void was 192ml with hyperreflexic bladder with high residual urine (77%). She was admitted to our PMR ward for intermittent catheterization training and further evaluation on February 28, 2001.

**DISCUSSION**

**Vascular anatomy of the spinal cord**

The blood supply of the spinal cord is through the anterior and posterior spinal arteries. These spinal arteries are supplied by radicular and medullary arteries which originate in the vertebral, thyrocervical, costocervical, intercostal, and lumbar arteries. The spinal cord infarct is more likely in the ASA than the PSA distribution because the PSAs have many cross-anastomoses and are supplied by more radiculomedullary arteries. The ASA is the principal blood supply to the spinal cord vascularizing the anterior two thirds of the cord and the PSA supplies the posterior third of the cord. Thus, cord infarcts are more likely in the central gray matter and anterior white matter than in the lateral gray matter and posterior white matter.

In the cervical region, the ASA is supplied by one or more anterior radicular arteries from vertebral arteries proximal to their union to form the basilar artery or other branches of the subclavian arteries; the blood supply is rich in collateral branches. However, the blood supply to the midthoracic cord is relatively tenuous, often consisting of only one significant radicular artery which commonly occurs near T7. Therefore, the midthoracic region of the spinal cord is considered to be the most vulnerable to vascular insufficiency, and spinal cord infarction is most likely to occur at T4 but recent study
(Cheshire et al. 1996) reports that the lower thoracic cord is at great risk.\[4\]

In the lumbar and sacral regions, the major radicular artery, the arteria radicularis magna or great artery of Adamkiewicz, which usually arises on the left and variably from T9 to T12 (occasionally as high as T5 or as low as L4), provides a major portion of the blood supply to the lower thoracic cord, lumbar cord and conus medullaris.\[12\] The conus and cauda equina are also supplied by the sacral artery arising from the internal iliac, lateral sacral, or obturator artery.\[9\]

The central (sulcal) arteries which originate in the ASA course backwards deep into the anterior median fissure and give rise to multiple perforating arteries on one or the other side of the cord. These arteries supply the anterior two thirds and central area of the spinal cord. The posterior third of the spinal cord is supplied by penetrating branches from the PSAs. The superficial white matter also receives blood flow via the circumflex anastomoses from the ASA and PSAs.\[4,7\]

The venous system of the spinal cord parallels the arterial supply and venous drainage occurs via the median posterior and the anterior spinal veins. Radicular veins are more numerous than radicular arteries and drain into the epidural venous plexus, which in turn communicates with the inferior vena cava and azygos system through the paravertebral and intravertebral plexi.\[4,7\] Besides, the lack of venous valves may be a factor in the pathogenesis of some spinal cord vascular disease.\[7\]

**Etiology**

The causes of spinal cord infarction include aortic atherosclerosis, infection, vasculitis, embolic events, sickle-cell disease, neurotoxic effects of iodinated contrast material used in angiography, compression of spinal arteries by tumor, aortic surgery, spontaneous aortic, carotid or vertebral dissection, cervical or thoracic spondylosis, an episode of hypotension, thrombosis of a spinal arteriovenous malformation, disc disease and spinal trauma.\[4,9-11\] In a ten-year study of Cheshire et al. at two university hospitals, 44 cases of ischemia and infarction of the spinal cord were reported.\[9\] 15 of 44 patients had aortic aneurysm repair (ten) or traumatic aortic rupture (five) as the cause of spinal cord infarction. Cardiac arrest (four cases) was the next most common cause. The other causes included transient ischemic attack (three), arteriovenous malformation (three), aortic dissection (two), aortic thrombosis (two), systemic lupus erythematosus (two), etc.

However, the cause in our two patients was unknown. The first patient was a healthy man before. The investigation of him included MRI of spine, CSF examination and laboratory tests for infectious agents but the cause of spinal cord infarction was not found. The second patient had diagnoses of hypertension and TCC of urinary bladder. CT scan of the chest & abdomen and examination of CSF were all normal. In our patients, the spinal angiography was not performed due to the possibility of spasm of the artery of Adamkiewicz and further interruption of the circulation.\[12\]

**Clinical presentation**

Abrupt onset of weakness, flaccid paraplegia or quadriplegia, disturbance of pain and temperature sensation, bladder dysfunction and absent deep tendon reflexes are common clinical presentations of spinal cord infarction.\[3-5\] The symptoms occur in a time frame of minutes to several hours after the onset of spinal cord infarction. Usually, the first symptom may be transient local or radiating sharp back pain. Numbness, burning, aching and tingling in the lower trunk and extremities are other common sensory symptoms and these are followed by acute onset of weakness of extremities. The weakness may progress gradually and the patient may be unable to walk soon.\[8-13\]

In general, the symptoms vary with the level of the lesion.\[4\] In high cord lesion (the lower cervical/upper thoracic level), the patient will present with variable involvement of the upper extremities; the lower-extremity signs and symptoms are often obvious, and bladder and bowel paralysis are often noted. Initially, the limbs are usually flaccid and absent deep tendon reflexes are noted. However, it may later progress to spasticity, hyperactive tendon reflexes and the presence of Babinski signs.\[7,26-13\] In the occlusion of anterior spinal artery, loss of pain and temperature sensation below the lesion is noted, whereas in a PSAS light touch, vibration and proprioceptive senses below the lesion are impaired due to damage of the posterior column.\[14\] Clinically, a PSAS only occurs rarely.\[9\]
The infarction occurs preferentially in the midthoracic portion of the cord because of the relative hypovascularity of the region of the spinal cord.\textsuperscript{[4]} The patient may present with paraparesis or paraplegia, sensory loss below the lesion and urinary dysfunction. Sacral sparing of cutaneous sensation may also be noted.\textsuperscript{[4,7,24-26]} However, obstruction of the blood supply to the lumbar portion of the spinal cord may cause sphincter dysfunction, weakness of both lower limbs, and loss of cutaneous sensation with sacral sparing.\textsuperscript{[4,7,24-26]}

The Brown-Squard syndrome may also occur. It results from the obstruction of a central (sulcal) artery and can present with ipsilateral paralysis and contralateral loss of pain and temperature sensation below the lesion.\textsuperscript{[9]} When a patient with lumbar spondylosis or stenosis has a change in posture, spinal transient ischemic attacks (TIAs) may occur.\textsuperscript{[4,7]} It may manifest as weakness and numbness of the extremities for a short time and has a good recovery.

**Investigations**

The typical investigations used, including plain radiography, myelography, CT, MRI and CSF studies, are for the purpose of ruling out other abnormalities such as a spinal cord neoplasm, myelitis or cervical spondylosis.\textsuperscript{[4]} MRI is a powerful tool for diagnosing spinal cord infarction. It provides a rapid and noninvasive method for screening the entire spinal axis. In the spinal cord infarction, the MRI examination should include T2-weighted images because it seems to be most sensitive for detecting a cord infarct.\textsuperscript{[10,11,15-17]} The T2-weighted images typically show a increased signal intensity of the spinal cord. Morphologically, the cord is often mildly enlarged in the acute stage and undergoes atrophy in the chronic stage of the spinal cord infarction. However, transverse myelitis, multiple sclerosis, and spinal cord tumor could present a similar radiographic feature.\textsuperscript{[10,11,15-17]} Hence, a diagnosis of spinal cord infarction based on MRI alone may be impossible. Correlation with clinical history and presentations, other laboratory data, and images is necessary to establish the correct diagnosis.\textsuperscript{[16]}

CT scan may be helpful in detecting vertebral fractures, aortic aneurysm or dissection, and major atheromatous disease.\textsuperscript{[4]} The spinal angiography is seldom done because of a risk of further dysfunction of the circulation of the cord unless a spinal arteriovenous malformation is highly suspected.\textsuperscript{[12]}

CSF and cytologic studies are also valuable in ruling out infection, multiple sclerosis, and presence of tumor cells. In the spinal cord infarction, the CSF protein is slightly elevated, but the gamma globulin level is normal. However, in multiple sclerosis and transverse myelitis, the gamma globulin content in CSF is elevated.\textsuperscript{[10]} Although multiple sclerosis and spinal cord tumor may simulate spinal cord infarction, neither of these has the sudden onset of clinical presentations.

**Treatment and prognosis**

The therapy of spinal cord infarction is supportive, including maintenance of adequate blood pressure, early bed rest and reversal of proximate causes such as hypovolemia or arrhythmias.\textsuperscript{[7]} There has not been a good study that has demonstrated a specific therapeutic regimen in patients with spinal cord infarction. In the Second National Acute Spinal Cord Injury Study (NASCIS-2), high dose steroid treatment in patients with lesions resulting from blunt trauma seems to improve in neurologic function.\textsuperscript{[18]} Steroids have some advantages such as protecting cell membranes and cell function, increasing tissue oxygen solubility and reducing the production of free radicals that occurs after injury.\textsuperscript{[12,13]} As steroids are to be used, they should be started as early as possible to get the maximum benefit.\textsuperscript{[13,18]}

Naloxone has been used experimentally to treat spinal cord infarction to reduce neurologic injury because these drugs appear to increase blood flow to the spinal cord and may prevent calcium entry into nerve cells.\textsuperscript{[19]}

Baba et al. reported that direct perfusion of dexamethasone sodium phosphate and urokinase into the artery of Adamkiewicz is the treatment of choice in the acute phase of the ASAS.\textsuperscript{[12]} In their study, three patients with ASAS received three consecutive injections with an interval of a week between each. All the patients made a good recovery.

In the study of Cheshire et al., the overall mortality of the patients with spinal cord infarction was 20%.\textsuperscript{[8]} Besides, 46% remained essentially unchanged, 17% improved, and 17% markedly improved. Although the extent of recovery from spinal cord infarction is highly variable, the physical and occupational therapy are useful.
in promoting functional recovery and most patients can return to home and live without professional assistance.\[^7\]

### CONCLUSION

The clinical diagnosis of spinal cord infarction is made on the basis of clinical history and presentations, detailed neurological examination, MRI findings and other laboratory data. Our two patients commonly presented with a history of sudden onset of weakness of limbs. In patient 1, ASAS was diagnosed and the loss of pain and temperature sensation was noted. However, in patient 2 of PSAS, loss of light touch, vibratory sensation and proprioception was noted. Luckily, the two patients had a good recovery in motor function. Patient 1 could walk independently before discharge and patient 2 was able to walk with the walker.

### REFERENCES

脊髓梗塞：兩病例報告

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脊髓梗塞是一種罕見的疾病。臨床上，發生在脊髓前三分之二部分的前脊髓動脈症候群比發生在後
三分之一部分的後脊髓動脈症候群的機會還要高，這與後脊髓動脈有豐富的側枝循環及其擁有兩條動脈
有關。造成脊髓梗塞的致病因子，根據文獻上的報告以動脈硬化、動靜脈血管異常、主動脈瘤手術、主
動脈剝離及心跳停止等較為常見。診斷脊髓梗塞主要是根據病患臨床上的症狀表現，患者常會有突然的
肢體無力、深部肌腱反射消失、對疼痛及溫度的感覺喪失、或振動感覺及本體感覺的消失、甚至排尿功
能障礙等。另外，再藉由一些實驗室的檢查，可幫助我們排除其它可能的致病因素；而核磁共振造影的
檢查，對診斷脊髓梗塞及確定梗塞位置、大小有極大之用途。

本研究報告兩位脊髓梗塞的病患，分別為前脊髓動脈症候群及後脊髓動脈症候群，依據其梗塞位置
而有不同的臨床表現。在治療方面，仍是以支持療法為主，兩位病患經過幾週的復健治療後，下肢肌力
恢復情況良好，且在生活功能上皆能達到不錯的進步。（中華復健醫誌 2002; 30(4): 241 - 249）

關鍵詞：脊髓梗塞(spinal cord infarction)，前脊髓動脈症候群(anterior spinal artery syndrome)，
後脊髓動脈症候群(posterior spinal artery syndrome)