A rare cause of Brown-Sequard syndrome: A case report and review of literature

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ABSTRACT

The incidence of spinal strokes is 1% of all strokes and spinal strokes presenting as Brown-Sequard syndrome is very rare. The management of spinal strokes needs early investigation which includes magnetic resonance imaging with diffusion-weighted imaging sequence. Patients presenting within 3–5 h of the onset of symptoms have been treated with thrombolysis. For late presentations, early spinal rehabilitation should be initiated to prevent complications. We are reporting the case of a 49-year-old female patient who presented with acute-onset right lower limb monoplegia with sensory symptoms and diagnosed as incomplete Brown-Sequard syndrome due to sulcal artery infarction.

Key words: *Brown-Sequard syndrome, Monoplegia, Spinal strokes*

Spinal stroke is a rare entity accounting for 1% of the all strokes. Based on the site of occlusion in the spinal arterial system, it can be classified as anterior spinal artery syndrome (most common), posterior spinal artery syndrome, Brown-Sequard syndrome (sulco-commissural artery syndrome), central cord syndrome, and complete transverse syndrome. Brown-Sequard syndrome occurring due to spinal sulcal artery occlusion is very rare and only a few cases have been reported in the world literature. It is characterized by ipsilateral upper motor neuron weakness, ipsilateral loss of vibration, proprioception and contralateral loss of pain, and temperature below the level of spinal cord lesion.

Spinal cord trauma is the most common cause of Brown-Sequard syndrome (hemi-cord syndrome). The exact incidence and prevalence of spinal strokes resulting in Brown-Sequard syndrome are not known as it is rare [1]. There is a delay in diagnosing sulcal artery occlusion due to its rarity and its unfamiliarity. We report a 49-year-old female who presented to us with incomplete Brown-Sequard syndrome without the involvement of the dorsal column and evaluated to have sulcal artery stroke.

CASE REPORT

A 49-year-old female admitted with the complaints of sudden onset of inability to move her right lower limb and benumbed sensation over the left lower limb extending up to umbilicus of 1-day duration. There was no history of trauma, fever, headache, and seizures. There was no history suggestive of other limbs, cranial nerve, bowel, bladder, or cerebellar involvement.

On examination, the patient was conscious and oriented. Cranial nerve examination was normal and the spino-motor system revealed right lower limb monoplegia of Grade 0 power with the decreased tone, absent knee, and ankle reflex. Superficial reflexes were intact except abdominal reflex was absent in the right quadrants. Pain and temperature were diminished over the left lower limb up to T10 and other modalities of sensations were normal. The patient was diagnosed as incomplete Brown-Sequard syndrome and evaluated for etiology.

Her routine investigations were within normal limits except elevated blood sugar. Magnetic resonance imaging (MRI) thoracolumbar spine with contrast showed ill-defined T2 hyperintensity with diffusion restriction involving T3 to T5 involving the right hemi-cord without contrast enhancement suggestive of spinal cord infarction (Figs. 1 and 2). She was evaluated for spinal stroke and its mimics. Chest X-ray, ultrasonogram abdomen and pelvis, echocardiogram, and MRI brain were normal, and vasculitic workup (antinuclear antibody and antineutrophil cytoplasmic antibody) was negative. Digital subtraction angiography revealed no arteriovenous malformation or fistula, normal anterior spinal artery, and posterior spinal arteries.

The patient was treated with intravenous methylprednisolone 1 g daily for 5 days along with aspirin 75 mg and atorvastatin 40 mg. The patient also received insulin for glycemic control and daily physiotherapy was given. The patient showed improvement in motor power from Grade 0 to Grade 2 after 10 days of illness; however, sensory symptoms did not improve.

DISCUSSION

In 1949, Dr. Charles-Édouard Brown-Séquard described Brown-Sequard syndrome which results from hemisection of the spinal

cord [2]. Brown-Sequard syndrome is caused either due to traumatic or non-traumatic etiology. The traumatic causes, being the most common, include penetrating injury from one side (gunshot injury or stabbing with a knife) and vertebrae fracture. Non-traumatic causes include multiple sclerosis, malignancies, vascular causes, cysts, cervical spondylosis, meningitis, syphilis, tuberculosis, and transverse myelitis [3].

The symptoms, include ipsilateral loss of motor power, loss of joint sense, position sense, and vibration sense (descending tract – pyramidal tract and ascending tract – posterior column), contralateral loss of pain and temperature (ascending tract – spino-thalamic tract) (below the level of the lesion), and tracts of the spinal cord, are shown in Fig. 3a. In our case, the patient presented with acute onset of the right lower limb monoplegia and diminished pain and temperature over the left lower limb up to T10. These features are suggestive of incomplete Brown-Sequard syndrome sparing posterior column.

Our patient presented to us acutely without a trauma history. Hence, our initial differentials were vascular myelopathy, inflammation/demyelination, and disc prolapse. Our patient underwent MRI which showed no compression and revealed ill-defined T2 hyperintensity with diffusion restriction involving T3 to T5 right hemi-cord without contrast enhancement suggestive of spinal cord infarction. From a previous literature review, spinal infarction presenting as incomplete Brown-Sequard syndrome is due to the involvement of the sulco-commissural artery of the spinal cord. Sulco-commissural artery is a small branch of the anterior spinal artery which supplies anterolateral part of hemi-cord.

As compared to a cerebral stroke which presents with acute hemiplegia with little differentials, spinal stroke presenting as monoplegia/paraplegia has a lot of differentials. Added to this, imaging techniques also possess a problem in a spinal stroke. Diffusion-weighted imaging (DWI) of the spinal cord is less sensitive compared to DWI of the brain, due to technical difficulties produced by the csf flow, swallowing and respiration artifact which can distort the images [4]. Hence, it is important to know different presentations of the spinal cord infarction and also its risk factors.

The spinal cord is supplied by an unpaired anterior spinal artery, paired posterior spinal arteries, and radicular arteries [5]. The anterior spinal artery runs in the anterior median fissure and supplies anterior two-third of the spinal cord, whereas the posterior spinal arteries run in the posterolateral sulcus and supply the posterior one-third of the spinal cord. The anterior spinal artery gives two branches, namely, anterior sulcal artery and circumflex artery. Anterior sulcal artery enters into the anterior median fissure and supplies anterolateral part of hemi-cord and occlusion of this artery causes incomplete Brown-Sequard syndrome. The blood supply of the spinal cord is not equal throughout and they are reinforced at multiple levels by radicular arteries arising from segmental arteries (branches of the descending aorta). The artery of Adamkiewicz is the largest radicular artery which supplies lower thoracic and lumbar segments, but this vessel varies in anatomy [5]. Various tracts and blood supply of the spinal cord are described in Fig. 3b.

Spinal cord infarction can present in five different ways: (a) Anterior spinal artery syndrome (most common) due to anterior spinal artery occlusion. The anterior spinal artery supplies the anterior two-thirds of the spinal cord and occlusion causes' bilateral pyramidal tract involvement and bilateral spino-thalamic tract involvement. The clinical features include lower motor neuron type of weakness at the level of the lesion and upper motor neuron type of weakness below the level of lesion bilaterally, and

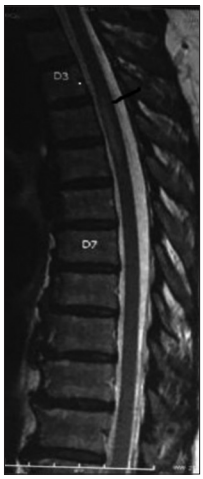

Figure 1: MRI thoracic spine sagittal image shows T2 hyperintensity noted from D3 to D5 level

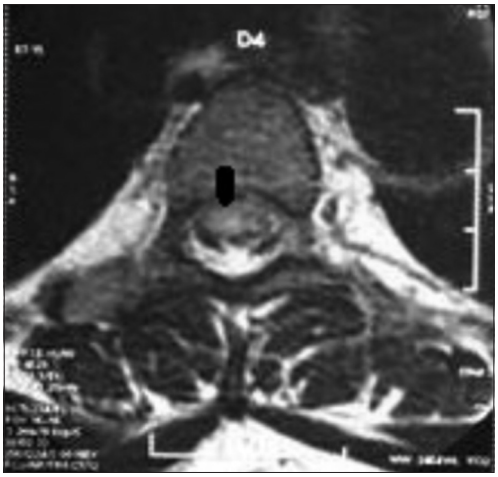

Figure 2: MRI thoracic spine axial image shows T2 hyperintensity in the right hemi-cord at D4 level

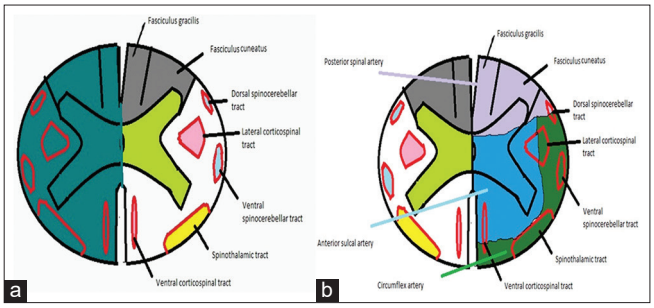

Figure 3: (a) Brown-Sequard syndrome; (b) blood supply of the spinal cord

bilateral loss of pain and temperature at the level of the lesion and below the level of lesion [6]; (b) the posterior spinal arteries infarct results in bilateral loss of vibration sense and position sense due to bilateral posterior column involvement; (c) sulco-commissural (anterior sulcal artery) syndrome which results in partial Brown-Sequard syndrome without posterior column involvement; (d) central spinal infarction causes lower motor neuron weakness of bilateral upper limbs sparing legs (man-in-the-barrel syndrome) and dissociated sensory loss of pain and temperature in segmental distribution [7]. It mostly occurs following trauma in the cervical region which results in localized ischemia to the central part of the spinal cord, leading to ischemia of the anterior horn cells of the upper limbs and crossing spino-thalamic fibers; and (e) complete transverse myelopathy occurs in the setting of hypotension, leading to whole spinal cord infarction.

Infarctions in the spinal cord represent 5–8% of all acute myelopathies [1]. It has various causes and the most common being aortic surgery. Aortic aneurysm repair, vascular risk factors (diabetes, hypertension), prolonged hypotension following cardiac arrest, arteriovenous (AV) malformations, and dural AV fistula are the important causes for spinal stroke [8]. MRI with DWI is the investigation of choice. When the diagnosis is in doubt even after MRI, catheter angiography is done to rule out vascular insult and also therapeutic intervention can be done in the same settings.

If the patient presents within a window period (<4.5 h), intravenous tissue plasminogen activator (tPA) can be tried and it is contraindicated in aortic dissection [9]. The role of thrombolysis in the sulco-commissural artery infarct is also not well established in the literature. There is no role of steroids unless it is caused by vasculitis, but in many studies and many centers, steroids are given in short duration to reduce cord edema [10]. Our patient was also treated with steroids along with antiplatelets. If infarction is caused by decompression sickness, hyperbaric oxygen can be used [11]. Secondary complications related to the spinal cord infarction include pressure sore, deep venous thromboembolism, urinary tract infections, aspiration pneumonitis, and autonomic dysfunction. Rehabilitation and treatment of complications are more important to prevent the fatal outcome [12].

CONCLUSION

Incomplete Brown-Sequard syndrome due to sulcal artery occlusion is very rare. Familiarizing with this presentation of spinal stroke avoids unnecessary investigations and treatment. Rehabilitation remains the mainstay of treatment in spinal stroke.

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