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SULCAL ARTERY SYNDROME - CASE REPORT

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ABSTRACT

Spinal cord infarction presenting as sulcal artery syndrome is very rare and poses a diagnostic challenge. Anterior spinal artery gives off penetrating branches, which are sulcal arteries supplying either left or right side of spinal cord but not both. This result in incomplete Brown-Sequard syndrome, that is involvement of ipsilateral corticospinal and contralateral spinothalamic tracts with sparing of dorsal columns. This report presents a case of acute spinal cord infarction presenting as sulcal artery syndrome with acquisition of MRI to aid in diagnosis.

INTRODUCTION-

Spinal cord infarction is a rare, devastating disorder presenting as paraparesis or quadriparesis. The diagnosis of spinal cord infarction is made clinically and is confirmed by MRI after excluding other causes.

DESCRIPTION OF CASE-

50-year-old lady, diabetic on regular medication, house wife by occupation, presented to the Emergency department with complaints of difficulty in lifting her right leg for last 6 hours. She was apparently normal when she woke up in the morning. She did her routine daily activities and went to bed again around 10am. At around 12pm, when she tried to get out of the bed, she found that she was not able to move her right leg. She also had difficulty in turning her body from side to side. She was able to move her both arms and left leg and she was able to lift her head off the bed. These complaints were all spontaneous and there was no preceding history of backache or lifting heavy objects. She was able to feel the clothes on her body but gives history of numbness of her left leg. She did not complain of any band like sensation over the abdomen or disturbances in bowel and bladder function. She did not give any history of visual disturbances or drooping of eyelids. There is no history of deviation of angle of mouth, difficulty in chewing food or nasal regurgitation. No history of difficulty in shrugging of shoulders, turning head side to side or difficulty in tongue movements. She had not received any vaccinations recently. There is no antecedent history of respiratory or gastrointestinal infection.

To summarise: A 50 year old lady, presented with sudden onset of weakness of right lower limb with numbness of her left leg with no involvement of cranial nerves, autonomic nervous system and higher mental functions. Patient was suspected to have involvement of corticospinal and spinothalamic tract on right side above L1 level with possibilities secondary to spinal cord ischemia or partial myelitis were considered.

On examination, patient vitals were stable. Higher mental functions and Cranial nerve examination was normal. Fundus examination was normal. On motor examination bulk was normal in all her four limbs with flaccidity noted in her right lower limb. Upper limb power examination was normal. Lower Limb examination showed power 5/5 in her left lower limb and 0/5 power noted in her right lower limb. Deep tendon reflexes of right knee and ankle were absent with rest of the reflexes being preserved. Plantar examination showed bilateral equivocal response. Her sensory examination revealed diminished touch, pain and temperature modalities below T10 level on left side. Vibration and joint position modalities were preserved bilaterally. Upper limb coordination was normal and Lower limb coordination could not be assessed. Spine examination was unremarkable. Peripheral pulses were all felt. With possibilities of Partial myelitis and Spinal cord infarction, MRI whole spine with brain screening was done which was normal.

She was started on anti-platelets and steroids. Her ESR was normal. Vasculitic profile was negative. Routine CSF analysis was normal. CSF-OCB turned out to be negative. 2D-echocardiogram was normal. On the third day of admission, repeat MRI spine with contrast was done which showed ill-defined T2 hyper-intensity showing diffusion restriction involving the dorsal spinal cord for a length of 4cm at T4 level (from T3 inferior end-plate to T5 inferior end-plate level), more involving the right hemi-cord than left hemi-cord, which was suggestive of spinal cord infarction presenting as sulcal artery syndrome.

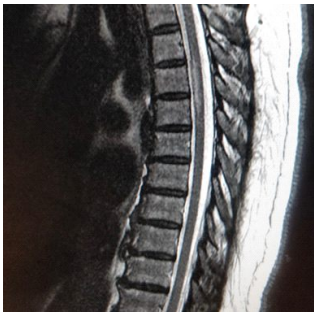

Image 1: T2 sagittal sequence showing hyper-intensity of cord from T3 inferior endplate to T5 inferior endplate

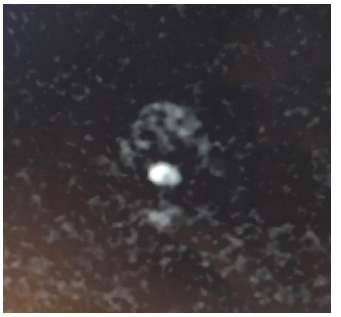

Image 2: DWI sequence with diffusion restriction seen in right half of the spinal cord at T4 level.

Patient's power started to improve. Patient was followed up, and after two months, she was able to walk with minimal deficit in her right leg.

DISCUSSION-

Spinal cord ischemia represents only 5-8% of myelopathies (Nedeltchev K, 2004). In childhood, it is mainly secondary to trauma and cardiac malformations. In adults, this disorder is usually secondary to atherosclerosis and its related complications (Vargas MI, 2014).

Spinal cord is supplied by single anterior spinal artery and paired posterior spinal arteries.

The anterior portion of spinal cord is supplied by anterior spinal artery. The anterior spinal artery is formed by anastomosis of two vertebral arteries at the level of medulla, then the anterior spinal artery courses downward to supply spinal cord. Throughout its course, many feeder arteries supply it. The anterior spinal artery communicates sporadically with the posterior spinal arteries via a pial plexus that encases the cord. The anterior spinal artery gives off sulcal artery in the anterior median fissure, which supplies either left or right side of spinal cord, not both (Bowen BC, 2008).

As reported in the case, infarction in the area supplied by sulcal artery leads to paresis on the same side with impaired sensations on the opposite due to involvement of ipsilateral corticospinal tract and contralateral spinothalamic tract with sparing of dorsal columns as they are supplied by posterior spinal artery.

The hallmark of spinal cord infarction is the presence of abnormal T2 signal within the cord, the pattern of which will depend on the territory.

Diffusion-weighted imaging is challenging in the spine, largely due to physiological CSF flow induced artefact, but can show restricted diffusion, is being increasingly used. (Quencer RM, 2000)

In the acute phase, the cord can also appear expanded due to oedema.

There are case reports which have shown initial MRI can be normal early in the course of disease where ischemic changes are not seen hence, repeat imaging helps in confirmation of diagnosis. (Masson C, 2004)

CONCLUSION-

There are few case reports published in literature of patients presenting with sulcal artery syndrome and the importance of recognising this entity clinically is important so that proper investigations and treatment can be tailored n a quick manner.

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