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DIAGNOSTIC COMPLEXITY IN ANTERIOR SPINAL ARTERY INFARCT: MONOPARESIS AND WASTING OF LEFT UPPER LIMB MASQUERADING AS ANTERIOR HORN CELL DISEASE

Neurology

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ABSTRACT

Spinal cord infarction is a rare condition, accounting for about 1% of all hospital admissions related to vascular issues in the nervous system. Patients may exhibit signs and symptoms akin to acute myelitis, making a history of sudden onset critical for diagnosis. We present a case of cervical spinal cord infarction in a 30-year-old male who experienced sudden-onset quadriparesis, sensory deficits below the neck, as well as breathlessness and palpitations. Initially treated for atrial fibrillation elsewhere, he did not seek further medical care. Although his symptoms improved over the following days, three months later he came to us with persistent weakness in his left upper limb, along with progressive atrophy and thinning of that limb, but without any sensory disturbances. This case illustrates that motor symptoms resembling anterior horn cell disease can occur during an anterior spinal artery infarction.

KEYWORDS

Spinal Cord Infarction, Anterior Spinal Artery, Acute myelitis, Anterior Horn Cell

INTRODUCTION:

Spinal cord infarction is significantly less common than cerebral stroke, but recognizing it early is crucial as it can indicate serious aortic issues. Among spinal cord infarction, the most common type is anterior spinal artery syndrome, which typically manifests as bilateral weakness (often paraparesis), loss of spinothalamic sensation, and intact posterior column sensation. Depending on the location, it may also lead to respiratory problems. Less commonly, posterior spinal infarcts can occur, preserving spinothalamic sensation while affecting only proprioception and vibration sensation. Unilateral, central, or transverse infarcts may also be observed, likely due to various mechanisms. Other rarer types of spinal ischemia include spinal TIAs, venous infarction, fibrocartilaginous embolism, and decompression sickness.

Case Report:

A 30-year-old male with no known comorbidities woke up with decreased sensation below his neck. He managed to get out of bed and walk to the fridge to get water but could not lift his arms to open it. He returned to bed and soon lost movement in both his lower limbs. Simultaneously, he experienced sudden breathlessness and palpitations. He was promptly taken to a nearby hospital, where an ECG revealed atrial fibrillation. He was treated with intravenous amiodarone, which alleviated his breathlessness and palpitations. He also experienced difficulty urinating and required catheterization. He has no recent trauma and no history of neck, chest, or back pain. Although the motor symptoms of the right upper limb and bilateral lower limbs, sensory, and bladder symptoms gradually improved over the next 5 days, there was persistent weakness in his left upper limb without accompanying sensory disturbances. His chest X-ray, echocardiogram, and repeat ECG were normal. MRI Brain **(Figure 1)** including diffusion imaging done was normal. The cervical spine MRI **(Figure 2- A,B)** with whole spine screening taken on second day of symptom onset reveals T2 hyperintensity extending from the C3 to T1 levels, with a predominantly anterior segment resembling a pencil-like hyperintensity.

He had been taking native medications and was not following up with any medical facility. Over the next three months, he experienced persistent weakness in his left upper limb, progressive thinning of the left forearm, and twitching in the left arm. At this point, he sought further investigation and management at our hospital.

On examination, he was conscious, and oriented, with normal eye movements, pupillary reflexes, and facial sensations. There was noticeable wasting over the medial aspect and distal third of the left forearm, left anterior chest, and left anterior axillary fold. Fasciculations were present in the left arm. No signs of Horner syndrome were observed. The tone was normal in the right upper limb, distal hypotonia was noted in the left upper limb and spasticity was noted in both lower limbs.

A power examination of the left shoulder revealed normal strength (5/5) in flexion, extension, adduction, abduction, and internal and external rotation. However, elbow flexion was 4+/5, elbow extension was 2/5, palmar flexion was 3/5, wrist dorsiflexion was 4-/5, and the intrinsic muscles of the left hand were weak. Strength in the right upper and both lower limbs was normal (5/5). Sensory examination, including the lateral spinothalamic tract, posterior column, and cortical sensation, was intact in all limbs. Extensor plantar responses were observed in both lower limbs.

Deep tendon reflexes were brisk in the bilateral biceps, bilateral supinator, right triceps, bilateral knees, and ankles. An inverted triceps reflex was noted in the left upper limb, along with positive finger flexor, Hoffman, and Wartenberg reflexes on both sides.

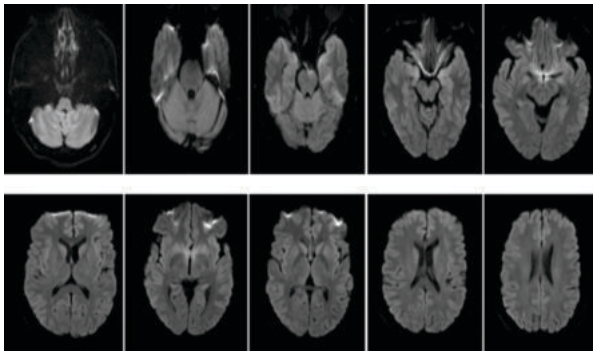

Figure 1- displays an axial section of an MRI brain scan, showing normal diffusion-weighted images.

Blood analysis results were unremarkable, with negative HIV and VDRL tests. Autoimmune workup was also negative, including ANA, anti-dsDNA, APLA, ENA profile, and cANCA. Chest X-ray, ECG, Holter ECG, and echocardiogram were normal. Cerebrospinal fluid (CSF) analysis showed normal protein, sugar, and cell count levels. Serum tests for NMO and MOG were negative. A repeat spine MRI **(Figure 3-A,B)** showed T2 hyperintensity involving the C4, C5, and

C6 segments, particularly affecting the anterior horn cells on the left side. MRI of the brachial plexus **(Figure 4)** revealed C4, C5, C6, and C7 root enhancement on both sides with more enhancement of the C6 root on the left side. Since the patient presented three months after symptom onset, diffusion-weighted imaging (DWI) of the spinal cord was not performed.

Based on the acute onset of symptoms, atrial fibrillation at symptom onset, anterior pencil-like hyperintensity on MRI, and nerve root enhancement on follow-up MRI, he was diagnosed with cervical cord infarction. He was treated with anticoagulants and underwent physical and occupational rehabilitation.

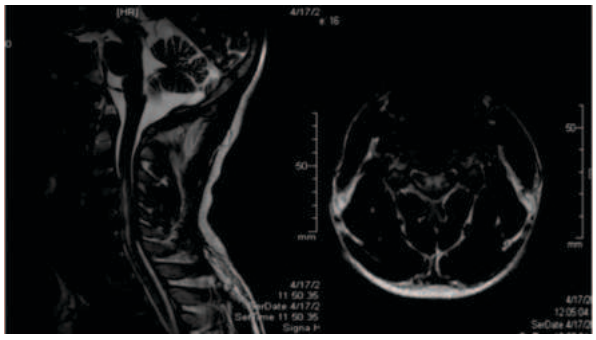

Figure 2. A and B - MRI CERVICAL SPINE- taken second day of the onset of symptoms shows T2 hyperintensity extending from the C3 to T1 levels, with a predominantly anterior segment resembling a pencil-likehyperintensity.

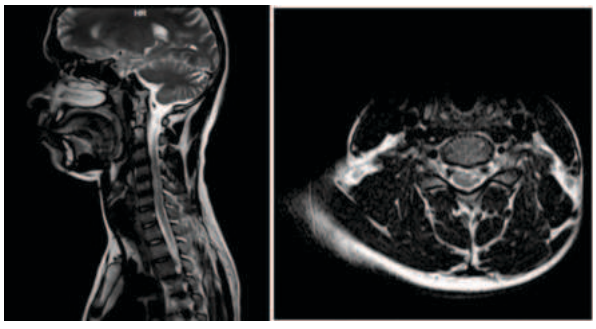

Figure 3-a,b – MRI CERVICAL SPINE- shows T2 hyperintensity involving the C4, C5, and C6 segments, particularly affecting the anterior horn cells on the left side

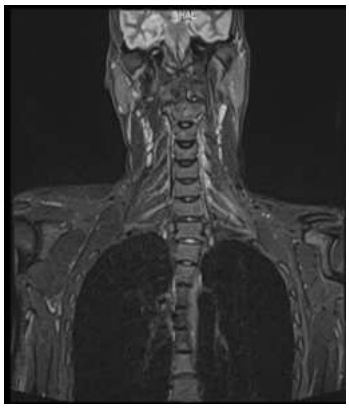

Figure 4. MRI of the brachial plexus shows enhancement of the C4, C5, C6, and C7 roots on both sides, with greater enhancement of the C6 root on the left side.

DISCUSSION:

The spinal cord is supplied by three arteries: one anterior and two posterior spinal arteries, which receive blood from radicular arteries branching off the aorta, especially in the cervical and lower thoracolumbar regions. A thin plexus links these arteries, with the anterior spinal artery giving off unilateral sulcal branches. Occasionally, this artery may be duplicated, leading to unilateral anterior infarction. The anterior spinal artery supplies the anterior two-thirds of the spinal cord, while the posterior arteries supply the remaining third.

Symptoms of a spinal artery infarct typically appear acutely, within minutes, but can also develop over hours. Anterior spinal artery infarction is the most common type, characterized by bilateral motor deficits, primarily paraparesis, and loss of thermal and pain sensations, with intact proprioception and vibratory sense due to selective involvement of spinothalamic tracts.

Acute symptoms include flaccidity and loss of deep tendon reflexes, later developing into spasticity and hyperreflexia. Autonomic dysfunction may manifest as hypotension, sexual dysfunction, or bowel and bladder issues. If the rostral cervical cord is affected, respiratory function may also be compromised.

Incomplete spinal artery syndrome can occur with localized anterior horn ischemia, presenting as:[1]

1. Acute paraplegia without sensory or sphincter dysfunction.
2. Painful bilateral brachial diplegia from cervical lesions, known as "man-in-the-barrel syndrome."
3. Progressive distal amyotrophy from chronic anterior horn lesions, possibly confused with lateral amyotrophic sclerosis.

The gold-standard diagnostic method for anterior spinal artery syndrome is MRI, including diffusion-weighted images [2]. While results may be negative in the first 24 hours, MRI is crucial for diagnosis, showing hyperintensity in the anterior horns on T2-weighted images. Acute ischemia appears as a thin, pencil-like hyperintense region across multiple spinal levels. Axial MRI reveals a central T2-hyperintense signal flanking the median fissure, indicating sparing of peripheral and posterior cords. Occasionally, a central T2-hyperintense signal may resemble a snake or owl's eyes, often associated with other conditions. Additional signs include T1-weighted hypointensity at the injury site and spinal cord expansion from early inflammation. MRI angiography or CT angiography can further delineate vascular pathology.

This case describes a patient with cervical cord infarction who developed acute quadriparesis after experiencing palpitations and breathlessness, initially treated as atrial fibrillation. Cervical cord infarction should be considered among the causes of acute tetraparesis, alongside conditions like spinal epidural hematoma and Guillain-Barré syndrome[3]. Acute neck pain, commonly seen in spinal cord infarction, was absent.

Initially treated elsewhere, the patient's quadriparesis, sensory symptoms, and bladder symptoms improved, but he continued to experience weakness and fasciculations in his left upper limb over the next three months before coming to our hospital. An MRI taken 24 hours post-presentation revealed T2 hyperintensity from C3 to T1, primarily affecting the anterior segment. A repeat MRI taken at the time of admission in our hospital showed T2 hyperintensity at C4, C5, and C6, focusing on left anterior horn cells, while brachial plexus MRI indicated C4-C7 root enhancement, particularly of the left C6 root. These findings are consistent with research by Zalewski et al.,[4] which noted similar enhancements and longitudinally extensive T2 hyperintensities.

The left-sided weakness, with maximum weakness of elbow extension and inverted triceps may relate to the left C6 root enhancement. The vascular etiology remains unclear, but the initial treatment for atrial fibrillation raises the possibility of vertebral artery ischemia. Atrial fibrillation can increase the risk of spinal artery infarcts, and it may also indicate autonomic instability following cervical spinal cord injury. However, a detailed cardiac evaluation revealed no cardioembolic source.

Monoparesis due to spinal cord infarction is rare, with few case reports [5-7]. The initial quadriparesis indicated a broad ischemic event, and the dissociative sensory loss suggested vascular involvement. While ischemia seemed transient, limited damage occurred, particularly in the left anterior horn due to its lower tolerance for ischemia[8]. The reason for this unilateral infarction may relate to hypoplasia of the left central sulcal artery,a branch of an anterior spinal artery or variations in spinal cord collateral circulation.

CONCLUSION:

This case highlights the importance of considering spinal cord

infarction even in patients with monoparesis and progressive amyotrophy.

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