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Clinical Observations

# Spinal Cord Stroke Presenting With Acute Monoplegia in a 17-Year-Old Tennis Player

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ABSTRACT

**BACKGROUND:** Acute monoplegia is a rare presentation for spinal cord stroke, which usually presents with paraplegia or paraparesis. **PATIENT DESCRIPTION:** We describe an athletic girl who presented after a week of heavy athletic activity complaining of back and left leg pain, followed by flaccid left leg paralysis. **RESULTS:** The prothrombotic evaluation was unremarkable. Cerebrospinal fluid studies demonstrated elevated myelin basic protein but no oligoclonal bands. Magnetic resonance imaging revealed a lesion in the anterior cord from T9 to T11 with T2 hyperintensity, contrast enhancement, and diffusion restriction, suggesting infarction. There was a herniated disc at T10-T11 contacting the spinal cord and Schmorl's nodes at T11 and T12. Magnetic resonance angiography of the spinal cord was limited by movement artifact. **CONCLUSIONS:** The combination of our patient's clinical presentation, imaging studies, and laboratory evaluation suggests that our patient had a spinal cord infarct. A fibrocartilaginous embolism was the likely mechanism of infarct due to the presence of Schmorl's nodes and disc herniation on imaging. In addition to spinal cord stroke, other possible mechanisms leading to presentation with monoplegia, such as transverse myelitis, neuromyelitis optica, and multiple sclerosis, are discussed.

***Keywords:*** stroke, spinal cord, monoplegia, fibrocartilaginous embolism, Brown-Séquard syndrome

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## Introduction

Spinal cord infarction in children and adults usually presents with paraparesis or hemiplegia.[1] The artery of Adamkiewicz supplies most of the spinal cord[2]; ischemia of this artery usually leads to bilateral symptoms.[1] Presentation with monoplegia is rare. We describe a teenage athlete who presented with pain and flaccid paralysis of her left leg. Imaging was consistent with spinal cord stroke. The differential diagnoses, the difficulty in making the diagnosis, and the likely pathogenesis of this infarct are discussed.

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## Patient Description

This 17-year-old athletic girl with no significant personal or family medical history presented at an outside emergency department, complaining of left hip pain and weakness that had evolved from pain and limited mobility of the left knee the previous evening. She was a high school tennis player who had played in four matches the week before presentation and had gone running the day before symptoms started. She came to the emergency department because that morning, she had fallen when getting out of bed because her left leg would not support her weight. Her physical examination demonstrated sensitivity to palpation on the lateral left hip and increased hip pain with movement. Her left knee and foot had decreased range of motion. Strength in the lower left leg was 3/5. Sensation in both legs was preserved. She was discharged home after a normal X-ray of the hip and told the diagnosis was likely hip strain.

The next day (symptom day 3), she complained of bilateral lumbar pain and left leg paresthesias that did not respond to ibuprofen. She saw a neurologist, who noted that she could not move her left leg except for slight ankle plantarflexion and dorsiflexion. She had decreased pinprick sensation in her left leg in a patchy distribution but intact vibration sensation. Left knee jerk and both ankle jerks were decreased at 1+, right

knee jerk was normal, and Babinski reflexes were negative. Magnetic resonance imaging (MRI) without contrast of her thoracic spine revealed a small disc extrusion with superior migration at T10-T11 causing mild anterior cord compression. There was an adjacent lesion within the anterior spinal cord with abnormal T2-weighted signal from spinal levels T9-T11 concerning for anterior cord ischemia caused by anterior spinal artery occlusion or stenosis possibly caused by disc extrusion (Fig 1). She was administered prednisone 20 mg twice daily, tapering by 5 mg every other day.

Her left leg weakness did not improve with prednisone. She developed urinary incontinence on symptom day 5 and was referred to our emergency department. She had flaccid paralysis of the left leg. Her right leg strength was 4+/5 with normal tone. Several examiners noted decreased temperature sensation in the right leg, but proprioception and vibration were intact in both legs; there was some variation among examiners. Her left knee jerk was 1-2+ and her right knee jerk was 2-3+, with no Babinski signs. Lumbar spine MRI without contrast that day (symptom day 5) demonstrated increased T2 signal in the anterior cord along with Schmorl's nodes at the T11-12 and T12-L1 levels. The next day (symptom day 6) a third whole spine MRI was performed with three-dimensional T2 images, postcontrast, and diffusion-weighted images. The disk extrusion at T10-T11 with associated spinal cord lesion was redemonstrated. Axial T2 images showed the lesion enhanced with contrast and was confined to the anterior half of the spinal cord, more on the left. There was focal restricted diffusion within the spinal cord. Small Schmorl's nodes were noted at the inferior end plates of T11 and T12 as well as another disk herniation at T8-T9 (Fig 2). Magnetic resonance angiography was attempted, but the anterior spinal artery could not be evaluated due to movement artifact. Brain MRI was unremarkable.

Echocardiogram was unremarkable. Serum testing was unremarkable and included complete blood cell count, sedimentation rate, international normalized ratio, activated partial thromboplastin time, protein C and S activity, functional antithrombin, homocysteine, antiphospholipid antibody screen, lipid panel, lipoprotein a, and genetic screen for the factor V Leiden, Prothrombin 20210, and methylene tetrahydrofolate reductase gene abnormalities. Vitamin $B_{12}$ was in the low normal range. Urinalysis demonstrated nitrites. Cerebrospinal fluid demonstrated glucose of 80 mg/dL (normal 40-70 mg/dL); protein 31 mg/dL (normal 15-45 mg/dL); white blood cell count 2/mm$^3$; red blood cell count 249/mm$^3$; myelin basic protein 74.7 ng/mL (normal 0-5.5 ng/mL); and negative oligoclonal bands. Both serum and cerebrospinal fluid were negative for aquaporin-4 immunoglobulin G.

She was admitted and given prophylactic cephalexin and a Foley catheter for bladder decompression. She initially received both steroids and aspirin because of the difficulty in determining whether she had transverse myelitis or stroke. She was administered intravenous dexamethasone 4 mg every 6 hours for four doses, then switched to 1 g of intravenous methylprednisolone a day for 5 days due to concern for cord compression then transverse myelitis. She was given 81 mg of aspirin the second day of admission as spinal stroke was a possibility. She was administered sublingual vitamin $B_{12}$ 1000 μg daily on the third day of admission. A fourth spine MRI with and without contrast and diffusion-weighted imaging on symptom day 10 demonstrated stable T2 hyperintensity and the earlier noted disc extrusions. Plain lateral radiograph of the thoracic spine demonstrated mild kyphosis and anterior wedging of T9, T10, and T11 of greater than 5 degrees (Fig 3).

She slowly regained strength in her left leg with physical therapy and was transferred to inpatient rehabilitation on hospital day seven. She could ambulate using crutches with strength of 3+/5 in the left lower extremity by the time of her discharge, 14 days after admission.

She was able to walk without crutches five weeks after her stroke, and her gait was only mildly hemiplegic. She had difficulty running and would tire easily. At 3-month follow-up, she was noted to have 5-/5 strength for left hip flexion and extension. Left knee flexion was 4/5 and extension was 5/5. She complained of right hip pain and said her right leg tired easily. This condition was attributed to her right leg doing more work than her left leg.

Thirteen months later, she was seen at the emergency department, complaining of back pain. She had bilateral paraspinal spasms in the thoracic and lumbar spine on physical examination. She complained of slight persistent difficulties with gait on her left side, including a mild foot drop, although her left leg strength was rated at almost a 5/5, which was an improvement since discharge. She reported numbness in her right leg in a stocking-glove distribution up to her right hip, which had started approximately 5 months earlier. MRI with and without contrast and diffusion-weighted imaging showed gliosis in the previous region of spinal cord abnormality and no new evidence of stroke. Her back pain was attributed to either spasticity from her spinal cord stroke the previous year or from musculoskeletal strain due to her changed gait since the stroke. Eighteen months later, she reported ongoing decreased pain, temperature, and pressure sensation in her right leg, and nearly baseline strength of her left leg. She returned to modified athletic activity and started college.

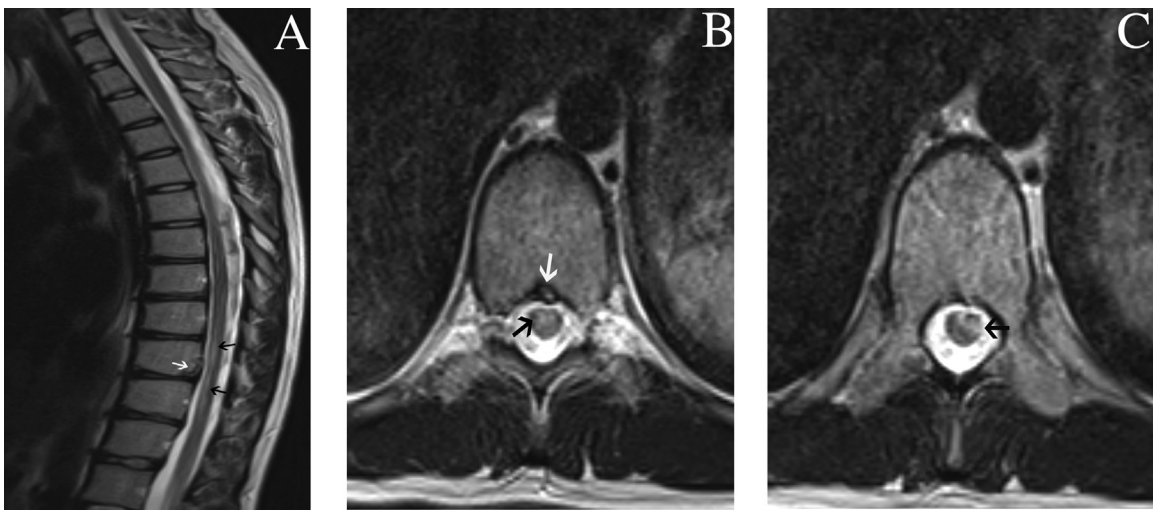


**FIGURE 1.**
Patient's third day of symptoms. Sagittal T2 image of the thoracic spine (A) shows a disk herniation with cranial migration at T10-T11 (white arrow) with associated anterior spinal cord lesion spanning T9-T11 levels (black arrows). Axial T2 image just above the T10-T11 disk level (B) shows the disk extrusion (white arrow) and the anterior involvement of the spinal cord lesion (black arrow). Just below the disk level (C) there is asymmetric involvement of the spinal cord on the left (black arrow).