Anterior spinal artery syndrome due to fibrocartilaginous embolism – case report and treatment options

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Introduction

Acute occlusion of the anterior spinal artery and subsequent spinal ischemic infarction lead to anterior spinal artery syndrome, characterized by back pain, bilateral flaccid paresis with loss of protopathic sensibility, whilst epicritic sensibility is usually preserved. It is commonly observed following aortic surgery or in adults thoraco-abdominal aortic disease (1). Fibrocartilaginous embolism associated with trauma, sports or unusual strain has been described (2-4).

Case report

The 11 y.o. female patient presented with neck pain, paresthesia and paresis of both arms and legs. She was unable to lift her arms, grasp, stand or walk. Deep tendon reflexes on the left side were impaired. She had urinary incontinence. The day before symptom onset, she had been exercising with a gym wheel for 3 hours.

MRI revealed a hyperintense T2 signal in the anterior spine with accompanying diffusion restriction. The adjacent annulus fibrous of the intervertebral disc showed a fissure without disc protrusion (Figure 1).

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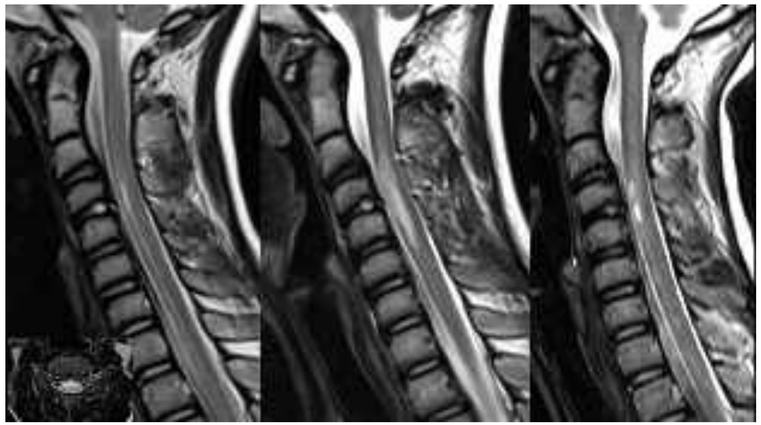
Treatment with prednisolone and enoxaparin was started within 12 hours of beginning of symptoms and continued over 6 days and 8 weeks, respectively.

On follow-up her motor function and bladder control gradually improved and after 5 months, she showed only slight residual impairment affecting elevation and abduction of the right arm.



A B C

Figure 1:

(A) MRI in the acute situation revealed a longitudinal hyperintense T2 signal in the anterior spinal cord at the level of C4 -C5 with a typical snake bite sign on axial T2 weighted images (small picture at bottom) and accompanying diffusion restriction (not shown). The adjacent annulus fibrous of the intervertebral disc showed a fissure without disc protrusion.

(B) On follow-up, the longitudinal myelopathy with edema extended from C3 to C6.

(C) Long-term follow-up showed small hyperintense defects at the level of C4-C5 in an otherwise normal cervical myelon.

Discussion

Fibrocartilaginous embolism is a rare cause of acute anterior spinal cord ischemia in children. It has been speculated, that following an increase in intra-thoracic or intra-abdominal pressure, fibrocartilaginous disc material may gain access to the anterior spinal artery via arterial or venous routes (3). Unusual exercise or lifting have been described as predisposing factors.

Symptoms include a transient neck or back pain followed by sensory deficits, bladder or bowel dysfunction and a motor deficit depending on the height of the spinal cord ischemia.

Outcome is usually poor and there are no treatment protocols. Corticosteroid treatment is frequently initiated, aiming to reduce spinal cord edema. As in our patient, antithrombotic agents are frequently added because of the uncertainty of the underlying pathomechanism. Intraventional application of fibrolytic substances has been suggested (3). However, outcome usually is poor with severe sequelae (4). In our patient, prednisolone was started within 12 hours following onset of symptoms. The short interval between onset of symptoms and treatment may have contributed to the favorable outcome.

Conclusion

The patient suffered from acute anterior spinal cord ischemia with imaging findings in line with a presumed fibrocartilaginous embolism. Unlike cases published in the literature, our patient showed almost complete recovery following treatment with prednisolone and enoxaparin. We speculate that the positive outcome may be related to the rapid treatment initiation.

In patients presenting with symptoms suggesting anterior spinal artery syndrome, rapid diagnostic confirmation via MRI and treatment initiation should be aimed for.

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