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Fibrocartilaginous embolism after mountain cycling: a case report with clinical and radiological follow-up and almost complete recovery

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ABSTRACT

Introduction Fibrocartilaginous embolism (FCE) is a rare spinal cord infarction due to embolism of fibrocartilaginous material with consecutive arterial infarction of the anterior spinal artery. Physical activity with increased axial pressure is the underlying mechanism of the retrograde migration of primarily nucleus pulposus material into the arterial system of the spinal cord. The initial severity of the clinical symptoms is supposed to be a prognostic predictor of recovery and so far, no specific treatment recommendation exists.

Methods We present a case of spinal cord infarction due to FCE after long and sporty mountain cycling (during 6 hours and 2500 altitude difference) with detailed clinical and radiological follow-up.

Results The clinical and radiological follow-up at month 4 showed an unexpected almost complete recovery despite the extensive initial clinical impairment.

Conclusion Mountain cycling has not yet been described as a specific trigger of FCE with spinal cord infarction. Further observation is necessary to show if the prolonged bent posture and core muscle imbalance in cycling, in addition to the Valsalva manoeuvre during physical effort, may contribute to FCE. It is unknown if prognosis of spinal cord infarction due to FCE differs from other causes of spinal ischaemia and if anticoagulation treatment presents a therapeutic option.

INTRODUCTION

Spinal cord infarction represents 1%–2% of all neurovascular events and is a rare cause of myelopathy.[1]

The most common causes are aortic diseases, probably due to the vulnerability of the thoracic and lumbar spinal cord to hypoperfusion.[1]

Fibrocartilaginous embolism (FCE) represents only 5.5% of all spinal cord infarctions.[2] This embolism is caused by the migration of fibrocartilaginous material, mostly of the nucleus pulposus[2] into the arterial or venous spinal cord system. Physical activity with increased axial pressure is the underlying mechanism of retrograde migration of nucleus pulposus material in the anterior spinal artery.

Clinical features include pain, sudden motor weakness, loss of sensitivity (spinothalamic deficits with preservation of pallesthesia and position sense if the anterior spinal artery is affected) and loss of sphincter control, inducing incontinence.

The initial severity of the motor impairment has consistently shown to correlate with poor functional outcome in acute spinal cord ischaemia.[3] The prognosis of functional outcome after fibrocatilaginous embolic myelopathy may differ and be more favourable.[4]

We present the case of a 56-year-old patient with spinal cord infarction due to FCE, who made a nearly complete recovery, documented by clinical and MRI follow-up, after initially severe symptoms of an anterior spinal artery syndrome. The patient's consent has been obtained before the publication of this case.

CASE PRESENTATION

A healthy and athletic 56-year-old man arrived at the emergency department with acute back pain, localised between the shoulder blades, and bilateral lower extremity weakness. The day before, he drove to the mountains and lifted his bicycle out of the back of his car. He cycled up on his own the highest mountains of the Vosges (about 2500 m of altitude difference) for 6 hours without interruption or special incidents. It was his only physical activity on this day. The patient was well trained and used to cycle three times a week. He was a responsible sportive, practising a warm-up and cool down before and after his rides.

In the second part of the following night, he woke up due to acute back pain between the shoulder blades and stayed in bed, but

was not able to sleep anymore. In the morning, while trying to get up, he noticed a bilateral weakness of the lower extremities and sought care.

On his admission, his vital signs were normal and without fever. The neurological examination showed dorsal pain localised at the T4–6, sensory level T8 with analgesia and thermoanaesthesia. Deep tendon reflexes were absent on the lower extremities except for the right patellar reflex, there was no Babinski sign, paraparesis 1/5 on muscle strength scale, loss of control of the anal sphincter with faecal incontinence. He presented a typical anterior spinal artery syndrome.

Biological findings did not show inflammatory signs (normal C-reactive protein), D-dimers were slightly elevated 669 µg/L (max 500 µg/L) and the ECG was rhythmic at admission.

Spinal cord infarction was suspected, the spinal MRI did not show an acute spinal compression, an aortic aneurysm was excluded by a thoracic–abdominal–pelvic scan, no lumbar puncture was performed. The patient was treated with 250 mg aspirin (ascorbic acid) and admitted into our neurological stroke unit.

ECG monitoring during the night showed a brief episode of atrial fibrillation and anticoagulation was started immediately with apixaban (5 mg two times per day).

Radiological follow-up with T2-weighted MRI on day 3 after admission showed a mild hyperintensity of the ventral spinal cord T6 with an adjacent intravertebral disc herniation (Schmorl node) (figure 1A,B) and a point-like hyperintensity in the diffusion-weighted axial imaging sequence (figure 1C).

Another MRI on day 10 after admission confirmed a large spinal cord infarction from T4 to T8 on T2-weighted images (figure 2).

A diagnosis of spinal infarction due to FCE was made, with regard to the typical clinical history with physical effort, acute back pain, progressive paraparesis, anterior spinal artery syndrome on clinical examination, spinal infarction marks on the MRI follow-up with an adjacent typical Schmorl node.

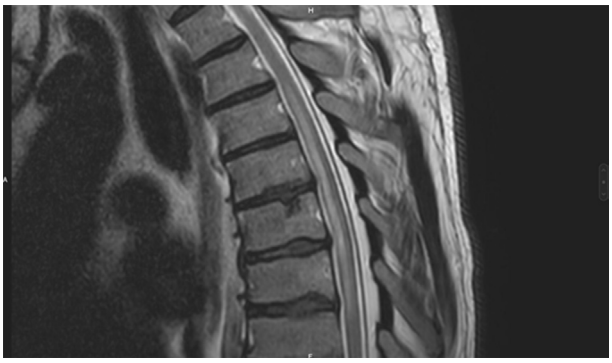

Figure 2 The MRI on day 10 after admission confirmed a large spinal cord infarction from T4 to T8 on T2-weighted images.

Therapy consisted of initial antiplatelet treatment and oral anticoagulation (introduced for atrial fibrillation), symptomatic treatment of pain and incontinence, eschar prevention and early neurological rehabilitation.

The patient already showed initial improvement on day 2, then a progressive motor recovery of 3/5 on day 8, on day 9 he managed the transfer to the sitting position, and on day 11 he started his first walking exercise.

Within 2.5 months, he managed a stable gait without technical help. Thermal hyposensitivity persisted, as well as urge incontinence. MRI follow-up after 4 months no longer showed any spinal lesion (figure 3). The patient was asymptomatic in his daily life and had started cycling again.

DISCUSSION

FCE of the spinal cord is rare in humans, only representing 5.5% of all spinal cord infarctions.[2] FCE was first described by Naiman in 1961 for humans and by Griffith in 1973 for dogs. Fibrocartilaginous material regresses, via arterial pathways, primarily into the anterior spinal artery, but posterior spinal artery infarction has also been described.[5]

FCE has a bimodal age distribution with peaks at ages 22 and 60 years.[6] Young age is hypothesised to be a clinical

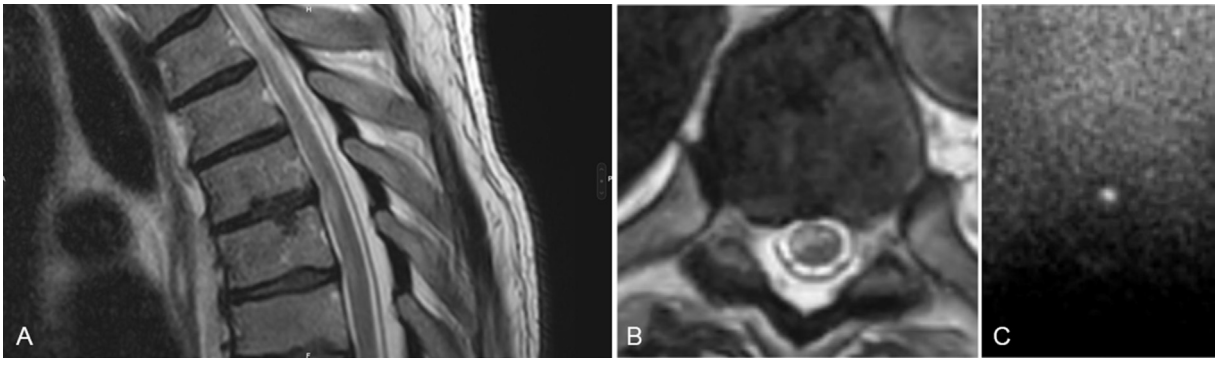

Figure 1 (A) On T2-weighed MRI images, appearance of a mild hyperintensity of the ventral spinal cord T6 with an adjacent intravertebral disc herniation (Schmorl node), (B) spinal hyperintensity on T2 axial image, (C) point-like hyperintensity on diffusion-weighted axial sequence.

Figure 3 The MRI follow-up after 4 months no longer showed any spinal lesion.

risk factor because of a larger volume and the ongoing vascularisation of the nucleus pulposus. Osteoporosis and cartilage degeneration may favour FCE in elderly patients.[6] The average age was 41 years.[2]

The pathological mechanism of FCE is mechanical, with initial increased axial intradisc and intravertebral body pressure via axial loading force (physical exercise, axial trauma) and/or the Valsalva manoeuvre. The initial trigger causes the break of fibrocartilaginous nucleus pulposus and migration into the arterial system of the vertebral disc or vertebral body.

The most common trigger event in children was intense exercise or sports.[7] In our case, the initial mechanism was the high effort of mountain cycling. FCE has not yet been described as a cycling or mountain biking complication. Cycling is increasingly becoming known as a risk factor for traumatic spinal injury.[8] Further observation is necessary to show if the prolonged bent posture and core muscle imbalance in cycling, in addition to the Valsalva manoeuvre during physical effort, may contribute to FCE.

The FCE diagnosis was established by the typical anamnesis of physical exercise, a latency of several hours before the appearance of pain and neurological deficits, clinical findings on examination with thoracic back pain, anterior spinal artery syndrome, exclusion of spinal compression and aortic disease with initial MRI and thoracic–abdominal–pelvic scanner, MRI findings with a vertebral Schmorl node adjacent to the spinal infarction on the follow-up MRI on day 3 and day 10.

The initial severity of the neurological impairment is supposed to be a clinical predictor of clinical outcome in spinal cord infarction.[3] It is not yet known whether the clinical outcome of spinal infarction differs if the embolism is due to fibrocartilaginous material or to blood thrombus. So far, there is no specific treatment recommendation such as systemic anticoagulation, antiplatelet drugs or even thrombolytic therapy.

In our case, the initial clinical presentation was severe, so the largely complete recovery was unexpected.

CONCLUSION

Spinal cord infarction due to FCE has been reported after intense sport exercise. In our case, the patient practised mountain cycling, not yet described as a specific trigger of FCE. Despite the initially severe symptoms, his clinical outcome was unexpectedly good, and a complete regression of the spinal lesion was documented on the follow-up MRI. Further studies are necessary to investigate whether prognosis of spinal cord infarction due to FCE differs from other causes of spinal ischaemia and if anticoagulation treatment presents a therapeutic option.

Contributors After having identified the uniqueness and interest of the case for neurologists, the authors jointly gathered necessary data and interpreted them. SSH and BH drafted the first version of the case report that has been completed with radiologist information by FH. All authors critically reviewed the case and agree to appropriately investigate and resolve all issues related to the work.

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