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GRAND ROUNDS

Acute complete paraplegia of 8-year-old girl caused by spinal cord infarction following minor trauma complicated with longitudinal signal change of spinal cord

Kosei Nagata[1] · Yuji Tanaka[1] · Hiroyuki Kanai[1] · Yasushi Oshima[2]

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Abstract

Introduction Spinal cord infarction followed by minor trauma in pediatric patients is rare and causes serious paralysis. Fibrocartilaginous embolism (FCE) is a possible diagnosis and there have been no consecutive magnetic resonance imaging (MRI) reports. Here, we report a case of an acute complete paraplegia with spinal cord infarction and longitudinal spinal cord signal change following minor trauma in an 8-year-old girl.

Case description An 8-year-old girl presented to our hospital emergency services with total paraplegia 2 h after she hit her back and neck after doing a handstand and falling down. She completely lost pain, temperature sensation, and a sense of vibration below her bilateral anterior thighs. Four hours later on MRI, the T2-weighted sequence showed no spinal cord compression or signal change in vertebral bodies. The patient was treated with rehabilitation after complete bed rest. A week after the trauma, the T2-weighted sequence indicated longitudinal extension of the lesion between T11 and C6 vertebral level with ring-shaped signal change. In addition, the diffusion-weighted MRI showed increased signal below C6 vertebral level. Two weeks after the trauma, we performed the T2 star sequence images, which showed minor bleeding at T11 vertebral area and spinal cord edema below C6. Four weeks after the trauma, MRI showed minor lesion at C6 vertebral level, but spinal cord atrophy was observed at T11 vertebral level without disc signal change. Thirteen weeks after the trauma, her cervical spinal cord became almost intact and severe atrophy of the spinal cord at T11 vertebral level. At 1 year following her injury, complete paraplegia remained with sensory loss below T11 level.
Conclusion Her clinical presentation, lack of evidence for other plausible diagnosis, and consecutive MRI findings made FCE at T11 vertebral level with pencil-shaped softening the most likely diagnosis. In addition, consecutive cervical MRI indicated minor cervical spinal cord injury. This Grand Round case highlights the consecutive MRI in a case with double spinal cord lesion with longitudinal spinal cord signal change.

Keywords Spinal cord infarction · Fibrocartilaginous embolism · Pencil-shaped softening · Magnetic resonance imaging

✉ Kosei Nagata
knagata-tky@umin.ac.jp

[1] Department of Orthopaedic Surgery, Tokyo Metropolitan Bokutoh Hospital, 4-23-15 Kotobashi, Sumida-ku, Tokyo 130-8575, Japan

[2] Department of Orthopaedic Surgery, The University of Tokyo Hospital, Tokyo, Japan

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Case presentation

An 8-year-old girl fell down while doing a handstand and hit her back and neck with hyperflexion. When she got up and was able to walk soon after the trauma, she felt minor back pain with weakness in both legs. Two hours later, she was admitted to the hospital with total paraplegia. Her medical history was unremarkable, and family history was negative except for an uncle with complete paralysis caused by spina bifida. Sensory examination revealed the absence of sensation to pinprick, light touch, pressure, and vibration bilaterally below the T11 level. Four hours later on magnetic resonance imaging (MRI), the T2-weighted sequence showed no spinal cord compression or signal change in vertebral bodies. We found no signal change of the spinal cord in sagittal images (Fig. 1a, b). She denied any upper extremity symptoms, but she had bradycardia (heart rate 20–40). Because we did not exclude cervical spinal cord minor lesion and spinal shock, the patient was transported to a pediatric intensive care unit to prepare for expansion of paralysis, including respiratory muscle.

The next day, neurological findings persisted. On MRI of the lumbar spine, the T2-weighted sequence showed increased signal within the cord from the T11 to cranial level. Gadolinium-enhanced MRI did not reveal tumors or inflammatory disorders (Fig. 2). On further investigation, CT angiography did not reveal any vascular malformation. Laboratory investigation, including cerebrospinal fluid test, showed no evidence of infections, autoimmune, inflammatory, or coagulation abnormality.

On the cervical MRI on day 7, the T2-weighted sequence showed longitudinal spinal cord signal change from caudal to C6 vertebral level with ring-shaped signal change. In addition, the diffusion-weighted MRI (DWI) showed increased signal below C6 vertebral level. Her general condition was good except for nighttime bradycardia. Her vital sign became stable on day 11. Two weeks after the trauma, we performed the T2 star sequence images, which showed minor bleeding at T11 vertebral area and spinal cord edema below C6 (Fig. 3). A follow-up MRI 4 weeks after the trauma showed minor lesion at C6 vertebral level (Fig. 1b), but spinal cord atrophy was observed at T11 vertebral level without disc signal change. Thirteen weeks after the trauma, her cervical spinal cord became almost intact and severe spinal cord atrophy was revealed

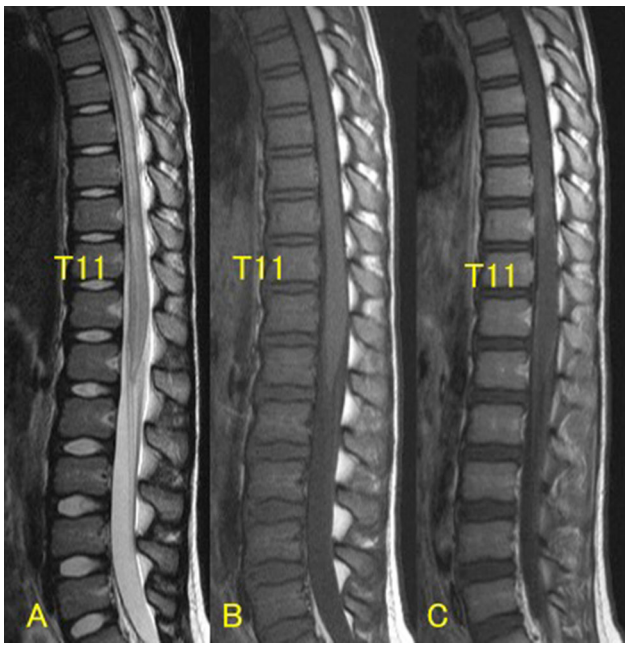

Fig. 2 Magnetic resonance images (MRI) of the lumbar spine 1 day after trauma. T2-weighted MRI (**a**) shows increased signal change at T11 vertebral level with longitudinal signal change to cranial level. T1-weighted (**b**) and gadolinium-enhanced MRI (**c**) showed no signal change in the spinal cord, vertebral bodies, or intervertebral discs

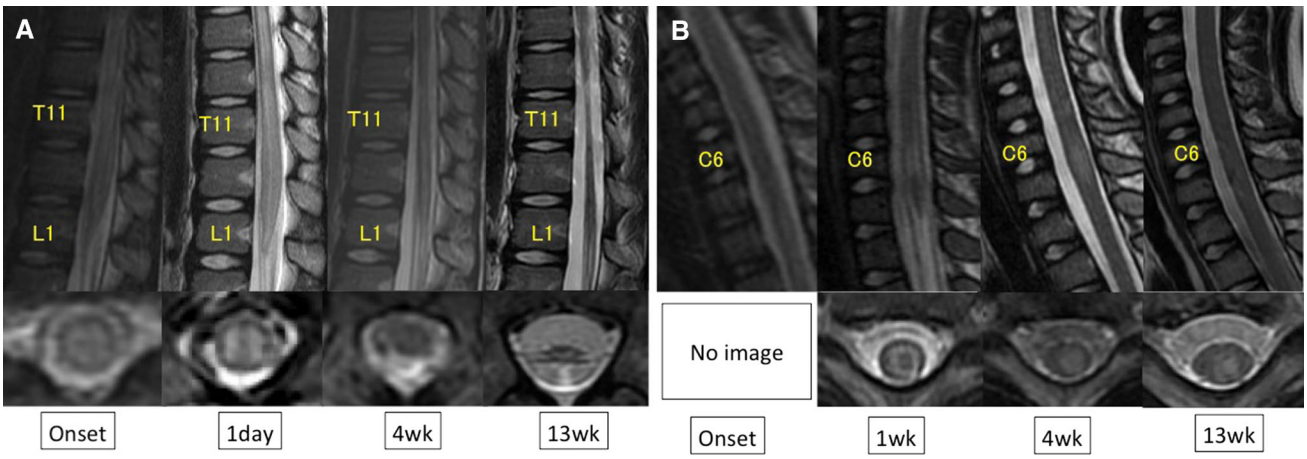

Fig. 1 Consecutive T2-weighted magnetic resonance images of lumbar and cervical spine. **a** Lumbar spine sagittal images and axial images at T11 vertebral level. **b** Cervical spine sagittal images and axial images at C6 vertebral level

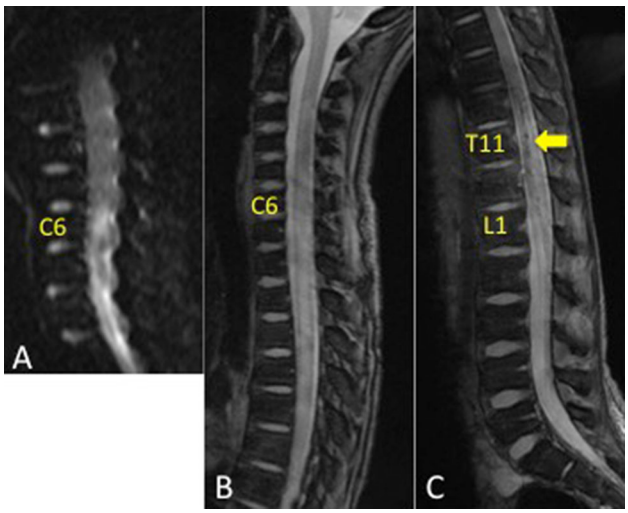

Fig. 3 Magnetic resonance images (MRI) of 1 week after trauma (**a**) and 2 weeks after trauma (**b**, **c**). Diffuse weighted MRI (**a**) shows increased signal below C6 vertebral level. T2 star-weighted sequence shows minor bleeding at T11 vertebral level (*arrow*) and increased signal within spinal cord below C6

at T11 vertebral level (Fig. 1a). At 1 year following her trauma, complete paraplegia remained with sensory loss below T11 level.

Differential diagnosis

Considering her clinical course, she suffered from transverse spinal cord infarction at T11 vertebral level after she hit her back and neck. Spinal cord infarction due to minor trauma occurs in children without vertebral fracture or dislocation [1]. Differential diagnoses of acute progressive paraplegia in children include acute transverse myelitis, Guillain–Barre syndrome, anterior spinal cord syndrome, spinal cord injury without radiographic abnormalities (SCIWORA), or FCE [2]. Acute transverse myelitis and Guillain–Barre syndrome were excluded after cerebrospinal fluid examination, including the virus detection test. Anterior spinal cord syndrome was not strongly suspected, because enhanced CT showed no embolic change and she lost sense of vibration. SCIWORA is originally defined as spinal cord injury in children without evidence of fracture or dislocation [3]. Yucesoy et al. claimed that patients should not be classed as SCIWORA if any pathology is detected on MRI [4]. Transverse cord ischemia following minor trauma was observed in this case and FCE could affect anterior and posterior circulation of the spinal cord [5]. A review of pediatric spinal cord infarction after minor trauma concluded that FCE was the principal etiological factor [6]. FCE is a rare but possible differential diagnosis of spinal cord infarction and causes serious paralysis in pediatric patients [2].

Our case was matched with schematic approach to diagnose FCE [7]. After excluding traumatic, compressive, and inflammatory etiologies of myelopathy, this case met one 'Major' criterion; clear vascular distribution on imaging modalities of MRI and three 'Minor' criteria; accompanying new onset neck or back pain, symptom progression to nadir in less than 4–8 h, and the initial unremarkable MRI of the spinal cord with subsequent evolution of an intra-parenchymal lesion [7]. Because there are no noninvasive tests to confirm FCE [8], consecutive MRI may be useful for understanding its pathology.

Diagnostic imaging and pathology

The absence of T2 abnormalities with acute ischemia may be due to an abnormal increase of T2 due to cytotoxic edema, which takes some hours to develop, and repeated MRI revealed abnormalities [9]. In the subacute phase, spinal cord ischemia typically manifests in MRI as focal cord swelling and 'pencil-like' hyperintensities on T2-weighted images [9, 10]. As for FCE, there is general agreement that the emboli originate from the intervertebral disc, but the mechanism whereby disc fragments enter the vessels is difficult to understand. One hypothesis is that an intervertebral disc had ruptured laterally, causing damage to an adjacent radicular artery with entrance of disc fragments into the arterial circulation. An alternative hypothesis was that high axial loading had caused increased pressure but not ruptured disc, with injection of semifluid nucleus pulposus material into small arteries and retrograde spread to a radicular artery and caused ischemia [11]. Therefore, disc signal change or Schmorl nodes in MRI were reported to be characteristic for FCE [8]. However, Abdel Razek et al. indicated that degenerative disc diseases and Schmorl nodes were observed in only 32 and 17% FCE cases [7]. All MRI did not show disc signal change or Schmorl nodes on our case.

At 1 week after the trauma, we considered that her lesion at T11 level was spinal cord infarction and we did not perform DWI of thoracolumbar spine. However, we were not able to make a diagnosis of her cervical spinal cord and we performed DWI of cervical spine. Although DWI of cervical spinal cord showed high signal in our case, her cervical spinal damage recovered 4 weeks after the trauma, when her bradycardia was relieved. The early restricted diffusivity on DWI is reported to allow a more confident diagnosis of spinal cord infarction detecting cytotoxic edema in infarction [10], but its application in spinal cord lesions has been limited for technical reasons; for example, artifacts due to the movement and small size of the spinal cord [9].

Considering her injury pattern and consecutive MRI, the main lesion associated spinal cord infarction followed by spinal cord atrophy was at T11 vertebral level and minor cervical injury happened simultaneously. When double-level spinal cord lesion is suspected, total spinal cord MRI can be feasible to understand the pathology. The T2 hyperintense ring of the cervical cord may be an ascending edematous or necrotic tissue from T11 vertebral level via central cord. This pathology may be described as pencil-shaped softening. Pencil-shaped softening of the spinal cord is considered as an expansion of necrotic tissue possibly caused by spinal cord infarction; However, it does not always result in neurological abnormalities [12, 13]. In our case, it diminished 4 weeks after the trauma.

Outcome and follow-up

Our patient was treated in an intensive care unit to prepare for expansion of paralysis, including respiratory muscle, because there were some mortality cases from thoracolumbar lesion [14] or with damage expanding to entire spinal cord from minor trauma [15].

Most children with complete and severe lesions on admission do not recover [16]. Complete paraplegia, loss of bladder control, and loss of sensory below T11 level remained at the follow-up 1 year after treatment and follow-up MRI showed severe atrophy of spinal cord. Resiner et al. revealed that follow-up MRI of pediatric patients with FCE showed minor atrophy and cystic myelomalacia of the spinal cord in patients and their AISA Grade recovered from B to D [2]. Severity of spinal cord atrophy may be associated with patients' neurological outcome. We reported consecutive MRI in a case with double spinal cord lesion with longitudinal spinal cord signal change and further case series are needed to improve diagnostic accuracy.

Compliance with ethical standards

Conflict of interest None declared.

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