

Spinal Cord Venous Infarction Presumed to be Caused by a Lumbar Vertebral Body Malformation after Vertebral Compression Fracture

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Summary

We report a case of a 81-year-old woman with a subacute bilateral legs palsy due to venous congestion of the spinal cord caused by an arteriovenous fistula in the first lumbar vertebra which fractured previously.

We diagnosed her spinal infarction only by MRI. She recovered from leg palsy at first. So we waited and saw her symptom. But after that leg palsy repeated with short interval and gradually her palsy was getting worse and became completed palsy. We performed selective spinal angiography and found out arteriovenous fistula in first lumbar vertebra fractured previously.

We estimated that vertebral fracture might make arteriovenous fistula in vertebral body and this fistula caused venous congestion spinal cord.

Spinal cord venous infarctions due to venous congestion with lumbar vertebral body malformation is a very rare.

Therefore we delayed to diagnose and this delay caused to permanent neurological deficits.

Keywords: Vertebral compression fracture; Spinal cord venous infarction; Arteriovenous fistula

Introduction

In general, anterior cord syndrome is the most common type of spinal cord infarction, and most cases are caused by an interruption in the arterial blood circulation. As grey matter signal changes supplied by an artery are clearly observed by magnetic resonance imaging (MRI) in cases of arterial infarction, this disease may be suspected on the basis of imaging findings alone [1,2]. However, as there are few characteristic imaging findings for a spinal cord venous infarction, it is difficult to diagnose this disease based on imaging alone [3]. Most previous reports have diagnosed spinal cord venous infarctions on the basis of anatomical examinations of fatal cases. These reports stated that most venous infarctions are caused by thrombosis, but few reports are available on lumbar vertebral body malformation in combination with a spinal cord venous infarction [4].

We treated and report here a case of what appeared to be a lumbar vertebral body malformation complicated by a spinal cord venous infarction.

Case

81-year-old female.

Medical history

None in particular. The patient was recuperating at home for lower back pain after a fall. Subsequently, mild paralysis in the right lower limb set in.

History of present illness

Un-triggered paralysis in both lower limbs occurred, and the patient was unable to stand or walk. She was transported by an ambulance to her previous doctor and was diagnosed with a spinal cord infarction on the basis of MRI findings. Lower limb capacity improved gradually with conservative treatment, and she was able to stand. Although she continued to undergo rehabilitation, she fell again, aggravating the paralysis in both lower limbs. Because the paralysis progressed after she fell, her previous doctor assumed that the paralysis may have been

caused by an external injury and referred her to our hospital for detailed examination.

Present status

Hypoesthesia was noted throughout both lower limbs, and right-side predominant muscle weakness was observed. The deep tendon reflexes in both lower limbs were enhanced, and the patient was unable to stand and had bladder and rectal disturbances.

Examination

Plain X-ray imaging revealed an obsolete compression fracture of the first lumbar vertebra (Figure 1). MRI revealed high intensity changes on T2-weighted images in the spinal cord grey matter from the seventh thoracic vertebra to the high reaches of the second lumbar vertebra (Figure 2). No clear occupying lesion was observed. Blood tests did not reveal any obvious abnormalities.

MRI was repeated, which revealed increased high intensity changes in the grey matter on T2-weighted images (Figure 3).

Course

As tests were conducted, muscle weakness in both lower limbs progressed gradually until they were completely paralysed.

The patient was referred to hospital by a resident radiologist, and a chest and spine computed tomography scan was conducted, which

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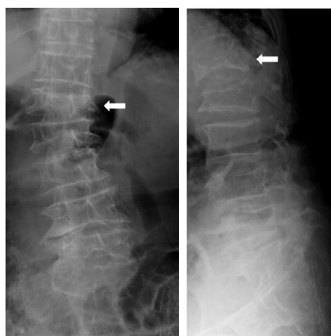


Figure 1: Plain X-ray photograph: Obsolete compression fracture observed in the first lumbar vertebra (arrow).

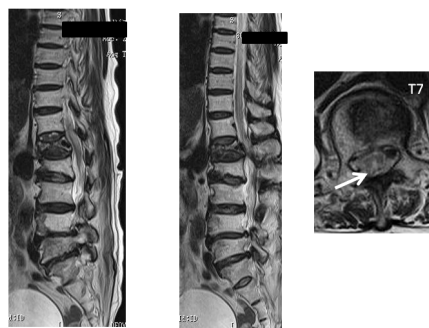


Figure 2: High signal areas observed in the spinal cord grey matter on T2-weighted images from the seventh thoracic vertebra to the high reaches of the second lumbar vertebra (arrow).

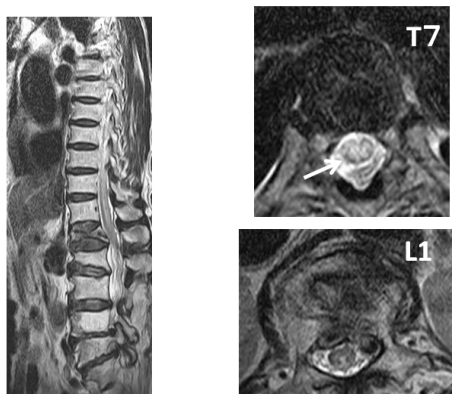


Figure 3: Spinal cord grey matter signal changes on T2-weighted images were high from the time of initial examination, and these changes expanded in range (arrow).

revealed a vascular malformation in the first lumbar vertebra (Figure 4).

A vasodilation image was observed in the vertebral body when angiography was conducted from the first lumbar artery. A shunt had formed between the lumbar artery and the area of vasodilation (Figure 5). The vascular malformation was diagnosed as a dural arteriovenous fistula.

The spinal cord venous infarction was presumed to occur because of spinal venous stasis caused by the arteriovenous fistula. Although

procedures such as vascular embolisation were considered, it was determined that surgery would have little effect because the limbs were already paralysed completely, and no additional procedures were conducted. At present, the patient has been discharged from hospital and is undergoing therapy at a medical facility.

Discussion

Spinal cord infarction is the necrosis of the spinal cord due to vascular occlusion. It occurs less frequently than cerebral infarction, and most cases are said to occur because of infarction of the anterior spinal artery [5]. It is rarely caused by isolated blockage of the posterior spinal artery [6]. Mechanisms of the onset of the spinal cord venous infarction have been reported as thrombosis and the presence of a spinal dural arteriovenous fistula in the thoracolumbar region. Most reports on the spinal cord venous infarction involve pathologic autopsy cases, and only a limited number of reports involve diagnostic imaging [7,8]. This is thought to be because there is little diagnostic basis for confirming the venous origin of an infarction with diagnostic imaging alone. The present case could not be confirmed histologically as a spinal cord venous infarction.

Because vasodilation from the first lumbar vertebra was noted in



Figure 4: Contrast computed tomography scan revealed an osseous defect in the vertebral body of the first lumbar vertebra, and vascular malformation was revealed internally.

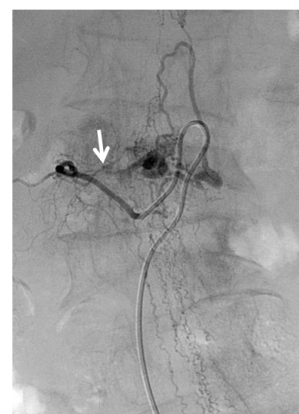


Figure 5: Image of vasodilation observed on angiography from the first lumbar artery. A shunt had formed between the lumbar artery and the area of vasodilation (arrow).

the present case, we presumed that blood stasis occurred in the same region, which increased venous pressure in the spinal cord, causing ischemia in the spinal cord and leading to the spinal cord infarction symptoms. We speculate that this situation continued for a long period of time, resulting in wide-ranging grey matter signal changes of the spinal cord on T2-weighted images and the MRI results that caused spinal cord infarction to be suspected. Kousyun et al. investigated venous congestion associated with dural arteriovenous fistula cause myelopathy [9]. We consider same mechanism work in this case.

The vasodilation image observed in the vertebral body in the present case may have been a congenital vascular malformation or an arteriovenous fistula formed due to a vertebral compression fracture from an external injury. However, because the patient was not examined at a medical facility prior to onset, there was no way of knowing her previous condition. Therefore, we were unable to identify whether this case was congenital or acquired. Most reports to date have involved congenital cases. To the best of our understanding, only one other venous infarction case has been reported after a vascular malformation due to external injury [10]. Nevertheless, it appears highly likely that vasodilation is an important factor in the onset of a spinal cord infarction [11].

If the spinal cord infarction is induced by vertebral body vasodilation, embolisation of the dilated blood vessels based on treatment for spinal arteriovenous fistula should be considered [12]. In the present case, a set of detailed examinations was initiated keeping mechanical compressive myelopathy caused by a compression fracture in mind, which delayed a definitive diagnosis. Complete paralysis was observed, and we missed the opportunity to treat it. If diseases other than compressive myelopathy that cause spinal cord infarction symptoms had been considered as detailed examinations were conducted and an early diagnosis had been reached, appropriate treatment could have been conducted.

Rubin and Rabinstein reported that effective treatment of spinal cord infarction depend on rapid diagnosis [13].

Diffusion MRI can be helpful, but its sensitivity is not as high as in the brain.

We have to consider this diagnosis when patients suffer from acute weakness, sensory defect, and sphincter dysfunction.

The mainstay of treatment is raising spinal cord perfusion pressure. In this case we had to performed angiography to diagnose and to get perfusion of spinal cord.

When examining spinal cord diseases, tests should be conducted considering the possibility of diseases other than compressive spinal cord disorders.

Acknowledgement

Each author certifies that he or she has no commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, etc) that might pose a conflict of interest in connection with the submitted article. Each author certifies that Misasa Onsen Hospital has approved the reporting of this case report, that all investigations were conducted in conformity with ethical principles of

research, and that informed consent for participation in the study was obtained. This work was performed at Misasa Onsen Hospital.

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