Steroid-induced Paraparesis: Spinal Epidural Lipomatosis Complicated by a Wedge Deformity of the Middle Thoracic Vertebrae

Toshitada Miwa, Tomoya Yamashita, Hironobu Sakaura, Kenji Ohzono and Tetsuo Ohwada

Abstract

Steroid therapy is commonly prescribed, although a variety of complications have been reported. Among such complications, spinal epidural lipomatosis is rare and difficult to diagnose before paraparesis occurs. The purpose of this report is to present a rare but catastrophic complication of steroid therapy. A 64-year-old woman undergoing long-term steroid therapy suffered from an osteoporotic vertebral compression fracture and was unable to walk due to paraparesis. Magnetic resonance imaging (MRI) demonstrated a D7 compression fracture and stored epidural adipose tissue between D5 and D8. After surgery, the patient was able to walk with double canes. This case indicates that long-term steroid use has the potential to induce paraparesis.

Key words: steroid therapy, spinal epidural lipomatosis, vertebral fracture, paraparesis

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Introduction

Spinal epidural lipomatosis is a rare complication associated with exogenous steroid use (1). Excessive deposition of adipose tissue in the epidural space compresses the spinal cord, resulting in progressive paraparesis. Long-term steroid therapy induces osteoporosis and can result in insufficiency vertebral fractures, which generally do not cause neurological deficits. We herein report a unique case of paraparesis triggered by a simple wedge deformity caused by an osteoporotic vertebral fracture associated with spinal epidural lipomatosis.

Case Report

A 64-year-old woman complained of back pain after lifting a watermelon. An X-ray revealed a D7 compression fracture. A corset and non-steroidal anti-inflammatory drugs (NSAIDs) were prescribed. Two weeks later, the patient suffered from intercostal neuralgia, followed by gait disturbance due to weakness in both legs and was admitted to our hospital. Her medical history included autoimmune hemolytic anemia, interstitial pneumonia, chronic renal failure and atrial fibrillation. Three years before the onset of paraparesis, treatment with 20 mg/day of prednisolone had been initiated for interstitial pneumonia. The prednisolone dose was reduced every two months, and after one year of treatment, the prednisolone treatment was completed. However, three months before the onset of paraparesis, treatment with 40 mg/day of prednisolone was again initiated for autoimmune hemolytic anemia. The prednisolone dose was reduced to 20 mg/day at the time of paraparesis onset.

On admission, the patient presented with moon face and central obesity. Her height was 151 cm and her weight was 56 kg. Her blood pressure was 116/60 mmHg and her body temperature was 36.8°C. She had chest neuralgia and decreased sensation below the D6 dermatome. Her muscle strength in the lower extremities was grade 3/5. She also had urinary retention. The laboratory data were as follows: WBC, 9,200/mm³ (93.1% neutrophils, 5.7% lymphocytes, 1.0% monocytes, 0.0% eosinophils, 0.2% basophils); RBC, 2.97×10¹²/mm³; hemoglobin, 9.4 g/dL; platelet count, 16.1×10⁹/mm³; total bilirubin, 0.3 mg/dL; alanine aspartate aminotransferase (AST), 14 U/L; alanine aminotransferase (ALT), 15 U/L; total protein, 5.1 g/dL; creatinine, 3.84 mg/
We herein presented a case of subacute paraparesis caused by a D7 osteoporotic vertebral fracture with spinal epidural lipomatosis. In this case, the vertebral fracture alone was unlikely to have caused the paraparesis because the retropulsed bone fragment was likely too small to cause neurological deficits. We speculate that impending paraparesis caused by spinal epidural lipomatosis became apparent due to the vertebral fracture. To the best of our knowledge, there are only six reported cases of paraparesis caused by vertebral fractures with spinal epidural lipomatosis (2-7). All six patients received steroid therapy and developed paraparesis after sustaining middle thoracic vertebral fractures. None of the patients had retropulsed bone fragments in the spinal canal and instead had anterior wedge fractures. It has been reported that local kyphosis in the middle thoracic spine is a risk factor for paralysis (8, 9). In addition, the blood supply in the spinal cord is the least rich at the middle thoracic vertebral level (from D4 to D9), and injury at this level is most likely to result in paraparesis (10). Both local kyphosis and spinal epidural lipomatosis in the middle thoracic spine contributed to paraparesis in the previous and present cases.

In our case, MRI revealed spinal cord swelling above and below the compressed site. In patients with acute spinal cord trauma, edema of the cord is the most commonly reported MRI finding and has a poor prognosis, especially when accompanied by hemorrhage (11, 12). However, the spinal cord damage observed in our case developed gradually after the patient sustained a spinal compression fracture, not at the same time as the fracture. The significance of spinal cord swelling in our case is different from that observed in patients with acute spinal cord trauma. Lee et al. suggested that spinal cord edema caused by chronic spinal cord compression is reversible. They also suggested that venous circulation disturbance caused by spinal cord compression results in local venous hypertension at the affected level that...
Figure 3. Transience of the volume of epidural fat at the D7 vertebral level. (a) Before the first round of steroid therapy. (b) Three months after the initiation of steroid therapy for interstitial pneumonia. (c) The time free from steroids and before steroid therapy for autoimmune hemolytic anemia. (d) Paraparesis developed when the volume of epidural fat increased during high-dose steroid therapy and decreased on the steroid-free days.

Figure 4. X-ray after surgery.

evitably leads to venous ischemia of the intramedullary vessels with consequent spinal cord edema at the compression site and adjacent spinal cord parenchyma (13). The neurological improvement observed after surgery in this case suggested that the spinal cord swelling was the result of a venous circulation disturbance.

Spinal epidural lipomatosis is a rare condition characterized by pathological overgrowth of normal adipose tissue within the epidural space of the spine, which can cause compression of the spinal cord or nerve root. Lee et al. reported the first case of epidural lipomatosis in a kidney transplant recipient treated with exogenous steroid therapy in 1975 (14). Most cases are associated with long-term systemic steroid therapy. Long-term steroid therapy causes excessive deposition of body fat in a centripetal fashion about the head, neck and trunk, which is the characteristic bodily habitus of patients with Cushing’s syndrome. Excessive deposition of fat is also seen in the spinal epidural space (7). Mostly epidural lipomatosis is associated with high-dose, prolonged steroid therapies, although epidural lipomatosis has also been reported to occur with prednisolone doses as low as 15 mg/day for as short a duration as four months (15). Thoracic spine involvement is most common, followed by lumbar involvement (15). The common clinical manifestations include back pain, radicular pain and
progressive paralysis. Back pain is the most common presenting symptom. Therefore, new onset or worsening back pain in patients receiving chronic steroid treatment is a warning sign of epidural lipomatosis (16). The diagnostic modality of choice is MRI. Quint et al. measured the thickness of epidural fat in 28 healthy subjects and six patients suffering from spinal epidural lipomatosis. The mean sagittal thickness of epidural fat in the former group was 4.6 mm, whereas that in the latter group was 8 mm (17). The thickness of epidural fat in this case was 8 mm, which is consistent with abnormal deposition of epidural adipose tissue. Treatment depends on the severity of symptoms. Surgical intervention is required in patients with progressive paralysis or those who fail to improve with conservative treatment. Although laminectomy and fat debulking are the most popular procedures (18), employing the fusion technique is necessary in patients with local kyphosis and/or vertebral fractures.

It is very interesting to note that the periodic CT scans of the thoracic spine used to follow the present patient’s interstitial pneumonia revealed that the thickness of the epidural fat increased and decreased in direct proportion to the dose of steroids (Fig. 3). To our knowledge, this is the first report to observe a relationship between the thickness of epidural fat and the dose of steroids. These data suggest that reducing the steroid dose, if possible, may be an effective treatment for epidural lipomatosis. The chest CT scans of patients receiving long-term steroid therapy may be helpful for monitoring the depth of spinal epidural fat.

Steroid therapy is a risk factor for both spinal epidural lipomatosis and osteoporotic vertebral fractures. Therefore, paraparesis in patients who receive steroid therapy may be caused by vertebral fractures combined with spinal epidural lipomatosis in the middle thoracic spine.

The authors state that they have no Conflict of Interest (COI).

References