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Case Report

T12 Compression Fracture Complicated with Delayed Spinal Epidural Hematoma: A Case Report

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Delayed spinal epidural hematoma (SEH) after a spinal compression fracture is a very rare but important cause of symptomatic spinal cord compression. We report a patient with spinal cord compression and cauda equina syndrome due to delayed SEH after a traumatic T12 compression fracture. A 74-year-old male patient had severe back pain without neurologic deficits after a traumatic T12 compression fracture. He developed progressive bilateral lower extremity weakness, sensory loss, and sphincter dysfunction 2.5 months after the injury. Magnetic resonance imaging of the spine revealed a huge spinal epidural hematoma at the level of T12 to L5. He then received decompressive surgery half a month later. He also received rehabilitation training after surgery. His neurologic deficits improved gradually, and he could walk independently with a quadricane 1.5 years after the injury. Education about the possibility of complications and close follow-up of patients with spinal compression fracture enhances the chance of early diagnosis and treatment, which ensure a good clinical outcome and thus avoids medico-legal issues. (Tw J Phys Med Rehabil 2011; 39(4): 227 - 232)

Key Words: spinal fracture, spinal epidural hematoma, compression fracture, spinal cord injury, cauda equina syndrome

INTRODUCTION

Spinal compression fracture is a common disorder in the rehabilitaition clinic and ward. Acute spinal compression fracture usually leads to moderate to severe back pain, which is most intense at the fracture level. Complications of spinal compression fracture include mechanical problems due to the fracture itself and disruption of the posterior ligamentous complex, neurologic deficit due to spinal canal compromise, and associated comorbidities. Delayed spinal epidural hematoma (SEH) after a spinal compression fracture is very rare, but is also a very important condition, because neglect of this condition, or delay in diagnosis may lead to serious sequalae for the patient and result in medico-legal issues.

SEH can result from atraumatic or traumatic etiologies. The majority of SEH is atraumatic, which is spontaneous and is related to coagulopathy, vascular malformations, neoplasm, instances of increased intrathoracic pressure and pregnancy.[1-3] Traumatic SEH is relatively uncommon and has been reported to be predominant in men between the ages of 50 and 75 years with a history of ankylosing spondylitis or rheumatoid arthritis.[4] Traumatic SEH is a

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rare condition, with incidence less than 1% to 1.7% of all spinal injuries.[5-7] The causes of spine injuries associated with traumatic SEH included vertebral fracture, lumbar puncture, postoperative bleeding, obstetrical birth trauma and missile injury.[7-9] The incidence of SEH after spinal compression fracture is unknown, but should be much less than 1%. The symptoms of traumatic SEH generally present immediately after the inciting event. Delayed SEH is relatively uncommon. SEH with neurological compromise is a surgical emergency which generally needs emergent surgical decompression. However, due to the rare incidence, delayed diagnosis is common in previous reports.[1-4]

We report a case of T12 compression fracture complicated with delayed SEH and discuss the diagnosis, management and measurements for prevention of delayed diagnosis.

CASE REPORT

A 74-year-old male had an acute onset of severe low back pain three months prior to admission. He had medical history of hypertension under regular medical control and benign prostatic hyperplasia, which had been resected several years before. He suffered from acute back pain after he jumped down from a bus and fell on the ground on his back. He visited a local hospital, where plain films showed T12 compression fracture. A Jewett brace was prescribed. At that time, no neurological deficit was detected. However, progressive bilateral lower extremity weakness and numbness were noted 2.5 months after the injury. Because of muscle weakness, the patient needed a walker for walking and gradually progressed to being wheelchair-bound in the following one month. Three months after injury, urinary retention developed. So he visited another hospital for further evaluation. Physical examination 3 months after the injury showed muscle power 0/5 over bilateral lower extremities, absence of bilateral knee and ankle jerks, positive bilateral Babinski sign, paresthesia below anterior thigh bilaterally, and urinary retention. Thoraco-lumbar spine magnetic resonance imaging (MRI) examination showed T12 compression fracture and T12 to L5 spinal epidural hematoma with spinal cord compression (Figure 1). The hematoma was heterogeneously hyperintense on T2-weighted images.

Under the impression of spinal epidural hematoma with spinal cord compression and cauda equina syndrome, the patient received right T12 to L4 hemilaminectomy and removal of T12-L5 epidural hematoma for decompression. T11-12 laminectomy with T10-L2 posterolateral fusion with transpedicle screws was performed half a month after the first operation.

A nerve conduction study (NCS) and needle electromyogram (EMG) examination was performed 170 days after the injury (56 days after the second operation). The NCS showed absence of bilateral H-reflex, relatively reduced compound muscle action potential (CMAP) amplitude of the left common peroneal nerve, and borderline motor conduction velocity of the bilateral tibial nerve (recorded in the abductor hallucis muscle). Sensory nerve conduction study was within normal limit. EMG revealed active denervation, increased polyphasic motor unit potentials, and reduced interference in bilateral tibialis anterior, medial gastrocnemius, rectus femoris, and right gluteus medius muscles. There was also reduced recruitment in the right tibialis anterior, rectus femoris, and gluteus medius muscles. Involuntary muscle contraction was noted in the left tibialis anterior and the left gastronemius muscles during muscle sampling.

One month after the second operation, the patient received rehabilitation programs including passive exercise for range of motion, progressive resistive exercise, balance training, postural training, and standing training with a walker. Two months after the second operation, the muscle strength over bilateral lower extremities improved from 0/5 to 2~3/5 (Table 1). The Foley catheter was removed for recovery of voiding function. The patient could stand with a walker but could not walk independently. One and a half years after operation, he could walk with a quadricane independently.

DISCUSSION

Spinal compression fracture is an isolated wedge-type fracture of the anterior and middle aspect of the vertebral body, and occurs mostly in the thoracolumbar junction, as there is a transition from the kyphotic thoracic spine above to the lordotic lumbar spine below.[10] Spinal compression fracture may be asymptomatic. If

Table 1. Time course about change of muscle strength of lower extremities

Time after trauma (days)	Muscle strength of lower extremities (right/left)
Immediate	5/5
77	4/4
96	0/5
98	Right T12 to L4 hemilaminectomy for decompression and removal of SEH
99	Hip flexor: 0/5 Knee extensor and ankle dorsiflexor: 1/5
112	1/5
114	Fixation of unstable spine
131	2/5
151	Hip movement: 2/5; Knee and ankle movement: 3-/5
173	Hip movement: 2/5; Knee and ankle movement: 3-/5

SEH: spinal epidural hematoma

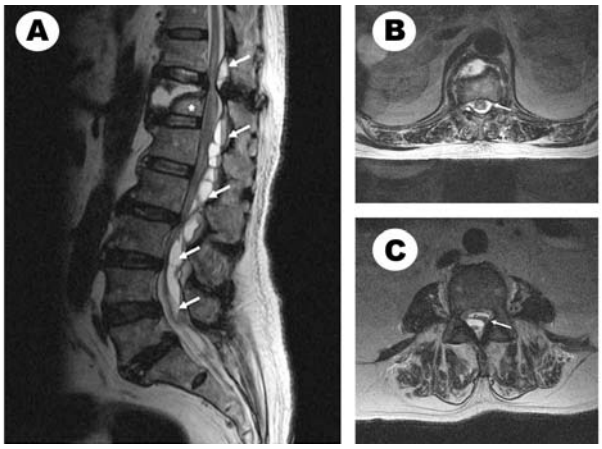

Figure 1. (A) T2-weighted sagittal MR image of the thoracolumbar spine shows a compression fracture at T12 (asterisk) and a huge collection of epidural hematoma (arrow) at the level of T10 to sacrum with spinal cord and cauda equina compression. The heterogeneous intensity is compatible with delayed onset of SEH which was examined at least 36 hours after symptom onset. (B) T2-weighted axial MR image of the thoracic spine at T12 level showing an eipdural hematoma (arrow) with spinal cord compression and (C) the lumbar at L4 level showing a epidural hematoma (arrow) with cauda equina compression.

symptomatic, the presentation is typically sudden onset of severe pain at the fracture level with or without radiating pain. Complications of spinal compression fracture include spinal instability, and neurologic deficit due to compression from posterior bony retropulsion, disc herniation, or hematoma. However, spinal compression fractures are generally mechanically stable and are rarely associated with neurological deficit.[11] As far as we know, delayed SEH after spinal compression fracture has rarely been reported in the literature.

Stable compression fracture without neurologic deficit is generally treated by conservative management. Compression fracture with less than 30% to 40% of vertebral body height loss and less than 20 degrees to 25 degrees of kyphosis is considered to be stable. However, most practitioners prefer application of a Jewett brace for 6 to 8 weeks if the vertebral body height loss is greater than 10%. Plain films should be obtained not only initially but also at regular follow-up visits to monitor fracture healing and alignment.[10] Additionally, loss of vertebral body height greater than 50%, focal kyphosis greater than 25 to 30 degrees, or evidence of posterior ligamentous disruption places the patient at higher risk of increasing kyphotic deformity or neurologic deficit. In such cases, surgical intervention to stabilize the spine is usually recommended.

Cervical spondylosis, ankylosing spondylitis, rheumatoid arthritis, and Paget's disease have been considered as risk factors for traumatic SEH.[5] The reported causes of traumatic SEH include vertebral fracture, lumbar puncture, postoperative bleeding, obstetrical birth trauma and missile injury.[6-8] The clinical presentation of traumatic SEH consists of sensory, motor or sphincter dysfunction after trauma related to spinal cord compression or cauda equina syndrome.[12] The symptoms of traumatic SEH generally present immediately after the inciting event. However, delayed onset of symptoms has been observed from days to months after the injury.[7-12]

The diagnosis of SEH can be made by using computed tomography (CT) or MRI. If clinical presentation

suggests a spinal cord compression or cauda equina syndrome, plain films of the spine are not adequate for evaluation. MRI is the best choice of modality for diagnosis of SEH, giving improved resolution of soft tissue and the ability to evaluate adjacent tissue for pathology.[5]

The exact mechanisms of the bleeding in SEH remain unknown. The origin of bleeding could arise from either an arterial or a venous source. Direct impact or shear stress to the epidural arteries could cause stretching and rupture of the arterial system, and then cause a SEH.[12] However, the most commonly accepted source of SEH is the venous plexus in the epidural space. Being composed of the venous plexus in the epidural space, the thin valveless vessels may be vulnerable to rupture with abrupt changes in venous pressure, possibly resulting from blunt trauma. Delayed SEH after spinal compression fracture in this case could be explained by injury of epidural venous plexus.

SEH generally needs to be managed by early surgical evacuation of the hematoma for most symptomatic patients.[13,14] The surgical outcome is related to the timing of surgical decompression of the spinal cord, the preoperative neurologic status and the speed of development of clinical presentation.[15-18] In the previous literature, there have been some selected cases that were treated conservatively with good neurological recovery.[5,19-21] Although most SEH needs to be managed by emergent surgery, it seems that if the patients have mild neurological deficits and demonstrate early, rapid, and progressive improvement, conservative treatment by close observation, steroid treatment, and rehabilitation may be the appropriate treatment.[21] However, if there is any worsening of neurological symptoms, the patient should receive emergent decompression.[5] If there are any sequalae remaining after the surgery, rehabilitation is also indicated for enhancing the functional status of the patient.

Our patient presented with neurological symptoms occurring 2.5 months after the injury and gradually developed to paraplegia and sphincter dysfunction in the following one month. The initial presentation and physical examination showed no evidence of muscle weakness or other neurological deficit. The diagnosis of SEH was proved by MRI of the thoraco-lumbar spine. The appearance of SEH on MRI in this case showed progressively increasing heterogeneity in signal intensity due to the degradation of hemoglobin.[22] This finding can provide an important clue about the time interval after symptom onset. The differential diagnosis of SEH includes spinal tumor, spinal abscess, acute vertebral disc herniation, cord contusion, subdural hemorrhage, subarachnoid hemorrhage, and transverse myelitis,[12,23] none of which were present in this case. Also, there was no evidence of ankylosing spondylitis, rheumatoid arthritis, Paget's disease, or use of anticoagulant in the past history of this case. From the clinical picture and imaging studies, we thought that the delayed SEH was probably due to a complication of spinal compression fracture.

The EMG study showed prominent active denervation, increased polyphasic potentials, reduced recruitment and presence of involuntary motor unit potentials in the muscles of bilateral lower limbs, which indicated that both cauda equina and spinal cord lesion were present. The absence of bilateral H-reflex could be due to compromise of bilateral S1 roots in the cauda equina or a normal variation in the aged (H reflex was recorded bilaterally in 92% of healthy individuals aged 60-88 years).[24] Mild reduction of left common peroneal nerve CMAP amplitude and a normal sensory conduction study of bilateral sural nerves also suggested that the lesion was located proximal to the sensory ganglions, which was compatible with lesion of cauda equina and spinal cord.

The prognosis of neurologic deficits depends on the time interval between onset of symptoms and surgical decompression (in some cases, conservative treatment). It is important how long the patient recognizes the symptoms and visits the medical system for help, and how soon the physician recognizes the clinical signs of SEH and obtains the image study to confirm the diagnosis. The physician can minimize the surgical delay by early and accurate diagnosis, and therefore improve the outcome. Close observation after spinal trauma and education of patients and physicians about symptoms of SEH may contribute to reduce the incidence of delayed diagnosis of SEH and improve the clinical outcome.

CONCLUSION

Delayed SEH after a spinal compression fracture is a very rare but important cause of symptomatic spinal cord compression. MRI is the best choice for early diagnosis

of SEH. Decompressive surgery is necessary if there is presence of spinal cord or cauda equina compression. Proper education and close follow-up of the patient with spinal compression fracture enhances the chance of early diagnosis and management, which are important to improve the prognosis and avoid medico-legal issues.

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第十二節胸椎壓迫性骨折合併遲發性脊椎硬膜外血腫：病例報告

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脊椎壓迫性骨折後合併遲發性脊椎硬腦膜外血腫雖屬罕見，但卻是造成脊髓損傷的重要原因之一。本篇提出一外傷性第十二節胸椎壓迫性骨折合併遲發性脊椎硬腦膜外血腫，因而導致脊髓壓迫及馬尾症候群的個案。一位 74 歲男性因外傷性第十二節胸椎壓迫性骨折發生嚴重下背痛，一開始並無神經學的缺損；但在後續的兩個半月中逐漸出現雙下肢無力、感覺缺損，及排尿功能異常。磁振造影檢查顯示第十二節胸椎至第五節腰椎之硬腦膜外血腫。患者接受減壓手術清除血塊，並於術後接受復健治療，神經功能逐漸恢復。追蹤至受傷後一年半時，患者可持四腳拐獨立行走。提出本報告的目的，旨在提醒臨床醫師在處理外傷性脊椎壓迫性骨折時，需注意後續的神經學變化及其他併發症，早期診斷及治療，以確保患者有較好的預後及避免醫療糾紛的發生。（台灣復健醫誌 2011；39(4)：227 - 232）

關鍵詞：脊椎骨折(spinal fracture)，脊椎硬膜外血腫(spinal epidural hematoma)，壓迫性骨折(compression fracture)，脊髓損傷(spinal cord injury)，馬尾症候群(cauda equina syndrome)

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