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Case Report Open Access

# Delayed Neurologic Deficit after Thoracolumbar Kyphosis Correction: One Case Report

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## Abstract

**Study design:** Case Report.

**Objective:** To present one case suffering from delayed neurologic deficit after kyphosis deformity correction and analyze its potential etiological factors, how to identify and treat it.

**Summary of background data:** Tardive neurologic deficits occur much less than acute ones after corrections of spinal deformities. Currently some risk factors, such as postoperative hypotension, deformity severity, and intraoperative haemorrhage are considered to be related with its occurrence and worsening though the true mechanisms are not clear. Extrinsic cord compression should be excluded by radiographic examinations before conservative treatments, such as improving blood perfusion, eliminating cord edema, suppressing local inflammation; promoting neurite outgrowth can be applied. If these don't lead to functional recovery of spinal cord, emergency operation returning the cord to its former kyphotic position can also be considered.

**Methods:** This 44 year-old female patient demonstrated thoracolumbar kyphosis of 136.9° combined with incompetence of spinal cord before operation, and underwent posterior vertebra column resection with the kyphotic correction of 68.8°. No neurologic dysfunction was found during operation.

**Results:** Neurologic deficit appeared on the sixth day after operation, showing motion disability and sensation weakening of both lower extremities. Imaging examinations failed to reveal mechanical compression, such as haematoma or displaced implant. After use of glucocorticoid, gamma globulin, monosialotetrahexosylganglioside, alprostadil and rehabilitation therapies, her myodynamia of both lower extremities recovered greatly, especially muscles extensor, however, her sensation didn't ameliorate.

**Conclusions:** Delayed-onset neurologic deficit is rarely seen. Detailed history collection and meticulous physical examination are strongly recommended. Early identification and correct treatments can avert incomplete spinal cord from permanent damage.

**Keywords:** Delayed neurologic complication; Spinal deformity; Kyphosis correction

## Case Report

### Case history

This 44 year-old female complained of "kyphosis for more than 40 years, worsening weakness and numbness of both lower extremities for two years". In recent months, she felt aggravating fatigue of lower limbs and gradually couldn't manage daily walking, as well as defecation and urination. Physical examination showed that below the umbilical plane sensation decreased; myodynamia of both lower extremities was Grade IV and muscular tension increased; abdominal reflex and perianal reflex disappeared; bilateral knee reflex and ankle reflex were hyperactive; Babinski sign and other pathologic signs were positive. Thoracolumbar plain film demonstrated her kyphotic Cobb angle was 136.9° and osseous fusion of four vertebras (T10, T11, T12 and L1) (Figure 1A), and preoperative computed tomography (CT) indicated osseous stenosis of kyphotic spinal canal (Figure 1B), while magnetic resonance imaging (MRI) didn't reveal tethered cord and other abnormity; pulmonary function test revealed moderately restrictive hypoventilation and cardiac function was also moderately restricted.

### Operation procedure

Under general anesthesia, this patient underwent posterior vertebra column resection. Screws were inserted into both pedicles of T8, T9, L2, L3 and then ribs connected to thoracic vertebraes were resected. From posterior route the whole osseous fusion composed of T10, T11, T12, L1 were removed completely until compression on spinal cord was resolved, and then titanium mesh filled with autogenous bone was placed between anterior column of T9 and L2. Kyphotic correction was accomplished through bilateral sequential rod compression. Finally, post-correction status was maintained through tightening screw-rod construct. Intraoperative Stagnara wake-up test didn't detect any neurologic deficit.

### Postoperation course

During five days following surgery, the patient showed no abnormality. The kyphotic angle decreased to 68.1° (Figure 1C). However, from the sixth day postoperation, she felt worsening fatigue and numbness of both lower extremities. According to her physical examination, sensation decreased further and myodynamia decreased to nearly Grade II, particularly musculus flexor, such as flexor pollicis longus and tibialis anterior. Emergent CT and MRI failed to demonstrate spinal cord compression by hematoma or slipped fixation implants (Figure 1D). Then this patient was immediately treated by use of methylprednisolone (MP), gamma globulin, monosialotetrahexosylganglioside (GM-1) and alprostadil. Ten days

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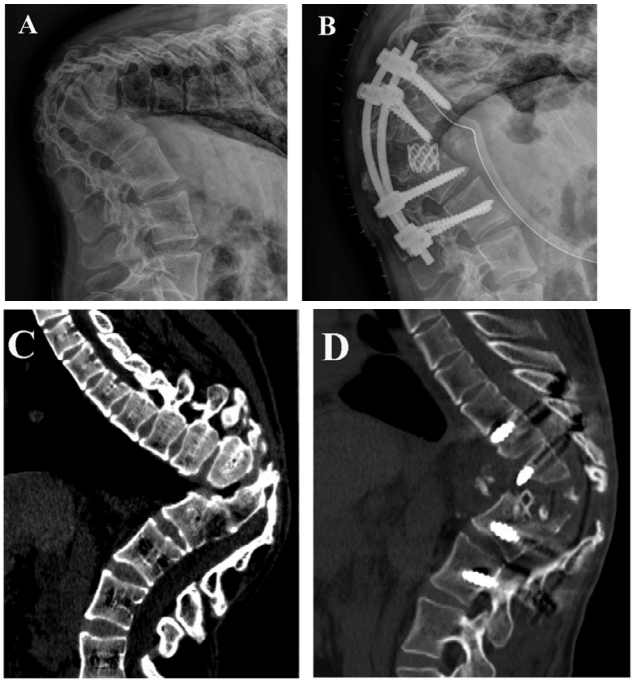


**Figure 1: (A)** Preoperative thoracolumbar lateral radiograph **(B)** correction of kyphosis from 136.9° to 68.1° after operation. **(C)** Preoperative thoracolumbar CT showing stenosis of kyphotic spinal canal. (**D)** sagittal CT obtained after neurologic dysfunction without demonstrating extrinsic cord compression, such as hematoma or implants malposition.

later, her motion dysfunction started to ameliorate. When discharged, her myodynamia of musculus extensor in both lower limbs improved to Grade IV, and that of hallex flexor was also Grade IV, while myodynamia of remaining flexor were Grade II; she still can't manage defecation and urination, and her sensation of bilateral lower limbs showed no improvement. She was followed up for two years with the continuing improvement of myodynamia, which returned to Grade V finally, and her defecation and urination function were restored to normality through rehabilitation therapy. However, her sensation revealed no improvement, even during the final follow-up.

## Disscussion

Neurologic deficits following spinal osteotomy are thought to be associated with cord ischemia or compression because of multiple reasons, such as subluxation, residual dorsal impingement or dural buckling [1-3]. Among these cases, delayed-onset neurologic deficit is rare and its etiologies are not very clear [4]. Postoperative hypotension has been proved as one direct reason of delayed cord dysfunction due to lowering tissue perfusion [5]. A great degree of kyphosis correction is also associated with late postoperative neurologic complication, for this may induce foraminal stenosis, enhance posterior drift or distraction of spinal cord [6,7]. Previous studies have indicated that patients with sagittal deformities, especially hyperkyphosis are at higher risks of neurologic deficits [8,9]. Besides, Patients who show incomplete cardiac output and pulmonary ventilation preoperation may suffer from more incompetence of blood oxygenation and cardiac output because of discomfort after operation, leading to higher risk for ischemic injury of spinal cord. Also failure to make the necessary anesthetic adjustments for patients and intraoperative mechanical distraction may also induce potential ischemic injury of spinal cord and its dysfunction [10,11]. Prolonged hypotension and excessive haemorrhage during operation may be other risk factors for delayed-onset neurologic compromise for lowering oxygen transportation capacity [12]. It is also demonstrated that patients with incompetence of spinal cord or intraspinal abnormalities are more vulnerable to etiological factors, finally resulting in late-onset neurological lesion postoperation [10]. For this patient demonstrating hyperkyphosis, preoperative cord dysfunction, cardiopulmonary comorbidities and massive intraoperative blood loss, it is hypothesized that these may be risk factors for her delayed-onset neurologic compromise due to possible alteration of blood perfusion to spinal cord and its intolerance to the new straightened position [4]. While her perioperative blood pressure and other vital signs were maintained well; her kyphosis correction rate was just 50.3%; no mechanical distraction or other injury was observed during surgery, so all these facts may be not considered as risk factors. In short, deformity patients with risk factors mentioned above should be approached with caution in order to decrease the possibility of delayed neurologic complications.

All of these risk factors may alter spinal cord blood supply, and finally causing apoptosis and necrosis of neurons [13,14]. Cord dysfunction occurs and even worsens though postoperative immediate neurologic function improves. Symptoms may exacerbate progressively from motion and sensation dysfunction of lower extremities to quadriplegia. Up to now, no medication can completely reverse physiopathologic procedures of spinal cord ischemia. Increasing cord blood supply is essential, for only this can ameliorate aerobic metabolism effectively. Glucocorticoid, especially MP is most widely used. Its pharmacological effects include suppressing lipid peroxidation and inflammatory factors, improving blood perfusion and decreasing intracellular calcium concentration. Among all these effects, suppressing oxidative stress is regarded as most important. Compared with other kinds of glucocorticoid, MP holds some advantages in this aspect [15]. GM-1 is also proved to be effective in the recovery of neurologic function by augmenting neurite outgrowth, as well as inducing regeneration and sprouting of neurons [16,17]. Besides these therapies that we have used, some other medications may have their own therapeutic effects in the future clinical practices. For instance, Vitamin C and E are strong antioxidant, which can provide best protection against cord ischemia [18-20]. A novel N-methyl-D-aspartate (NMDA) antagonist, Gacyclidine, owns its capacity for reducing the extent of an ischemic lesion and may promise its future use [21,22]. If medical managements don't reveal significant improvement of delayed neurologic compromise, surgery that return spinal cord to its former kyphotic position by releasing part of the correction may be considered [4,23]. However, great cautions should be given to the whole surgical procedures lest the cord suffer from secondary injury.

## Conclusion

Delayed spinal cord ischemia is rare. Once highly suspected, emergency imaging examinations should be performed to exclude the possibility of mechanical obstructions which compress spinal cord. Conservative treatments or surgery should be employed as soon as possible in order to minimize or ideally eliminate neurologic dysfunction.

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