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Case Report

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Neurologic Deficit During Halo-Gravity Traction in the Treatment of Severe Thoracic Kyphoscoliotic Spinal Deformity

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Correction of severe spinal deformity is a significant challenge for spinal surgeons. Although halo-gravity traction (HGT) has been shown to be well-tolerated and safe, we report here a case of neurologic decline during treatment. A 24-year-old male presents with severe thoracic kyphoscoliosis with $> 180^\circ$ of 3-dimensional deformity. Magnetic resonance imaging showed his thoracic spinal cord draped across his T7–9 apex. His neurologic exam showed lower extremity myelopathy. During week 7 at a goal traction weight of 18.1 kg, his distal lower extremity exam declined from 4+/5 to 2/5. His traction weight was lowered to 11.3 kg. He subsequently sustained a ground-level fall and became paraparetic with a motor exam of 1–2/5. He subsequently underwent a T1–L4 posterior spinal instrumentation and fusion with a T7–9 vertebral column resection. Postoperatively, he was noted to have a complete return to his baseline neurologic exam. At his 4-month postoperative visit, he was now full strength in his lower extremities with complete resolution of his myelopathy. We present here a case of neurologic decline in a patient with severe kyphoscoliosis who underwent HGT and discuss the management decisions associated with this challenging scenario.

Keywords: Spinal deformity, Halo-gravity traction, Neurologic deficit, Complications, Kyphoscoliosis

INTRODUCTION

Correction of severe and rigid spinal deformity represents a significant challenge for spinal surgeons. Several of the surgical goals include halting the progression of a painfully disabling deformity, providing cardiopulmonary stability, avoiding the long-term consequence of restrictive lung disease, and improving quality of life.¹⁻⁴ In patients with severe kyphoscoliosis, surgical correction is also aimed at reducing spinal cord tension across the apex that may be the cause of progressive neurologic impairments.¹

Halo-gravity traction has been successfully used to assist the multimodal management of severe complex spinal deformi-

ty.⁵⁻¹⁰ The gradual addition of traction weight provides for gradual lengthening of the spinal column and also allows time for clinical improvements in inpatient nutritional status and pulmonary function, and helps reduce the risks of neurologic compromise.¹¹⁻¹³ This nonsurgical partial correction of rigid and stiff curves while the patient is awake helps to mitigate the risks of these complications prior to definitive surgical correction.¹⁴

Although halo-gravity traction has been shown to be a safe and well-tolerated method of providing significant preoperative corrective forces,^{8,10} we report here a case of neurologic decline during halo-gravity traction in a 24-year-old patient with severe kyphoscoliosis and discuss the management decisions associated with this challenging scenario.

CASE REPORT

1. Presentation

A 24-year-old male is admitted to the hospital with a known history of neurofibromatosis and severe thoracic kyphoscoliosis. He was otherwise healthy and had no other significant past medical history or prior surgeries. Although he had undergone multiple evaluations by surgeons in his country of origin, his case was deemed too complicated and he therefore sought care at our hospital within the United States.

Plain X-rays demonstrated a severe thoracic kyphoscoliosis with $>180^\circ$ of 3-dimensional deformity (Fig. 1). Computed tomography (CT) with 3-dimensional reconstruction showed the bayoneted nature of the kyphosis in 2 separate coronal planes (Fig. 2). Magnetic resonance imaging (MRI) showed his thoracic spinal cord to be stretched across the apex of his deformity at T7–9 (Fig. 3). Although difficult to fully appreciate due to the severity of his deformity on orthogonal multiplanar MRI, there was also a dorsolateral soft tissue mass separate from the bony apex compressing the spinal cord. His baseline pulmonary function testing showed restrictive lung disease with a forced vital capacity of 38% (reference, 3.24; predicted, 1.24) and forced expiratory volume in one second of 38% (reference, 3.02; predicted, 0.95).

On physical exam, he stood 4'8" tall and weighed 34.9 kg (body mass index, 17.3 kg/m^2). His truncal deformity was se-

vere and rigid on clinical inspection. His neurologic exam on evaluation showed full strength in his upper extremities, but a baseline 4+/5 strength in his tibialis anterior and extensor hallucis longus bilaterally. He showed hyperreflexia and several beats of clonus bilaterally and although he did ambulate independently, he did so with a spastic-type gait.

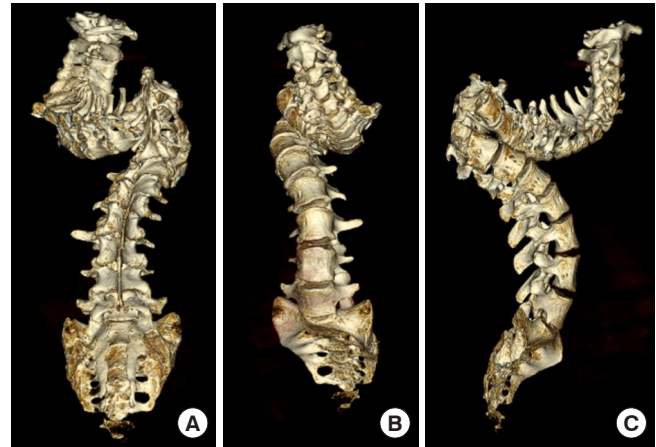


Fig. 2. Posterior (A), anterior (B), and lateral (C) views of a 3-dimensional reconstruction from computed tomography showing the bayoneted nature of the kyphosis in 2 separate coronal planes.

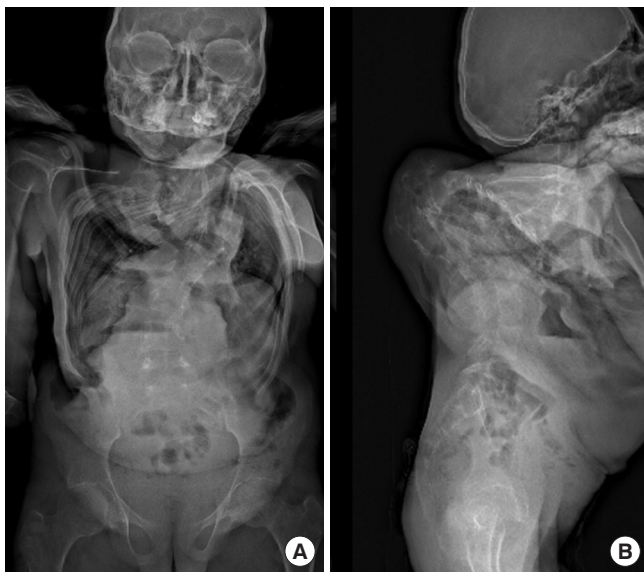


Fig. 1. Posterioranterior (A) and lateral (B) X-rays showing a severe thoracic kyphoscoliosis with $>180^\circ$ of 3-dimensional deformity.

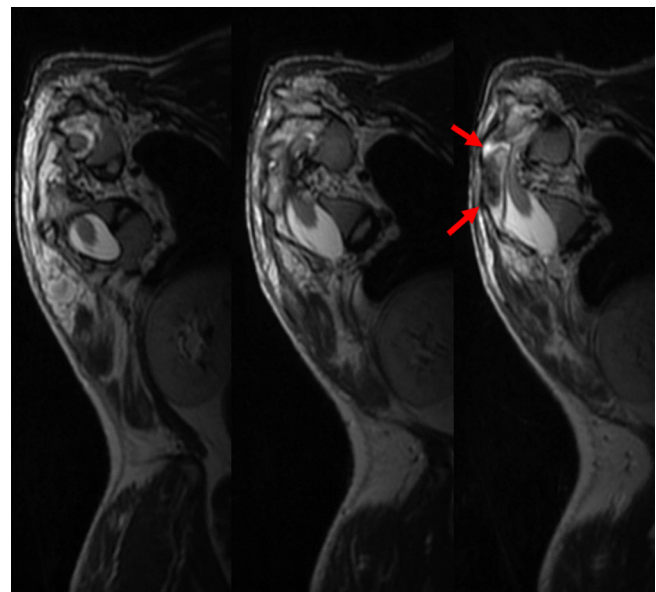


Fig. 3. Sequential parasagittal images from a T2-weighted magnetic resonance imaging sequence showing the draped spinal cord across the apex as well as the dorsolaterally positioned soft tissue mass causing compression posteriorly. Arrows denote the soft tissue mass.

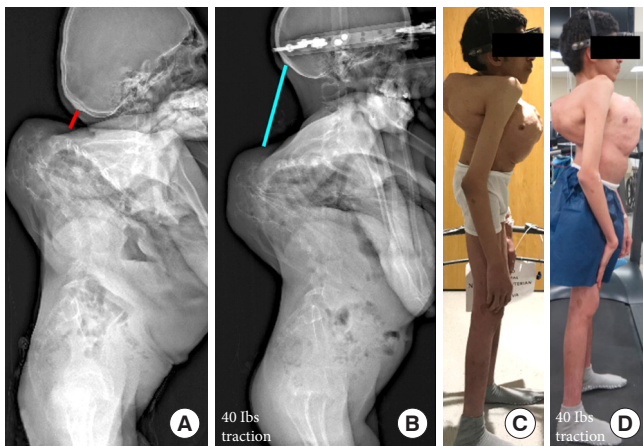


Fig. 4. Lateral X-rays showing pretraction (A) and halo-gravity traction at 18.1 kg (B) with partial correction of the kyphotic deformity. Lines denote the distance from the posterior inferior iliac spine to the trunci deformity for planning a surgical access corridor for instrumentation placement. Clinical pictures pretraction (C) and in halo-gravity traction at 18.1 kg (D) again showing partial correction of the kyphosis.

2. Traction Course

The initial plan for his spinal deformity correction was halo-gravity traction over the course of 12 weeks to allow time to reach a goal traction weight of 18.1 kg (52% body weight), and to allow for respiratory and nutritional support. Over the course of the first 6 weeks by increasing his traction weight by 1.3 kg per day, he reached his goal traction weight of 18.1 kg (Fig. 4). His weight also increased from 34.9 kg to 39.9 kg.

During week 7, he began developing bilateral tibialis anterior and extensor hallucis longus weakness, from his baseline of 4+/5 to 2/5. His traction was decreased from 15.9 kg (45% body weight) and then 11.3 kg (32% body weight) over the following days with improvement in his distal lower extremity exam to 3 and 4-/5. Subsequently, he sustained a ground-level fall after going to the restroom. His neurologic exam worsened immediately after the fall to 1-2/5 proximally and 1-3/5 distally.

He was immediately transferred to the intensive care unit (ICU) to drive his mean arterial pressure goals to > 90 mmHg. He was also started on high-dose dexamethasone. An MRI of his full spine did not show any appreciable worsening of his known cord signal change, and a CT scan did not show any fractures. His neurologic exam began slowly improving to 4-/5 proximally and 2-3/5 distally and he regained the ability to ambulate with a walker. Due to this tenuous clinical exam, his surgical date was moved from week 12 to week 8.

3. Surgical Technique

He was brought to the operating room and underwent a T1–L4 posterior spinal instrumentation and fusion with a T7–9 vertebral column resection. After exposure in the usual fashion, pedicle screw instrumentation was placed on the left-sided concavity from L4 to T12 below the apex, and T6 to T1 above the apex. On the right-sided convexity, pedicle screw instrumentation was placed from L4 to T11 below the apex, and T7 to T1 above the apex. Appropriate positioning was verified with an intraoperative CT scan and electromyography potential stimulation.

Temporary rods were then placed bilaterally across the apex, and a multirib thoracoplasty was performed with the removal of 2–3 cm of ribs 5–10 on the left and ribs 6–10 on the right. A T7–9 laminectomy was then completed with subsequent identification and full exposure of the dural sac. Due to the rotatory component of the deformity at the apex, the spinal cord was draped anteriorly-posteriorly on top of the T7–9 pedicles, which were subsequently drilled out to the pedicle-body junction for a nearly complete decompression from the left side concavity. The right-side of the spinal cord was decompressed first through resection of the dorsolaterally compressive neurofibroma that was originating from the right-sided T8 nerve root. Resection of the tumor opened the exposure to vertebral bodies and allowed for the completion of the vertebral column resection at T7–9 from the right-sided convexity.

With the spinal cord completely and circumferentially decompressed, the temporary rods were exchanged with permanent rods, and the deformity was corrected through a compressive cantilever technique. After the correction, the dural sac was noted to be soft and no longer under tension, and spinal cord monitoring data improved. The now mostly closed T7–9 corpectomy defect was decorticated and packed anteriorly with autogenous bone. Posteriorly, a structural allograft rib spanning from T6–10 was laid over the laminectomy defect and sutured in place. A third accessory rod was placed. Bone grafting and a multilayer closure then proceeded in the usual fashion.

4. Postoperative Course

Postoperatively on wake-up, he was noted to have a complete return to his baseline neurologic exam of 5/5 proximally and 4+/5 distally. He was monitored initially in the ICU and then transferred to the floor with physical and occupational therapy training. Postoperative standing films and clinical pictures were taken prior to his transfer to an acute rehabilitation center (Fig. 5). His total hospital stay was 11 weeks and 4 days with the inclusion of halo-gravity traction, with 19 of those days being postop-

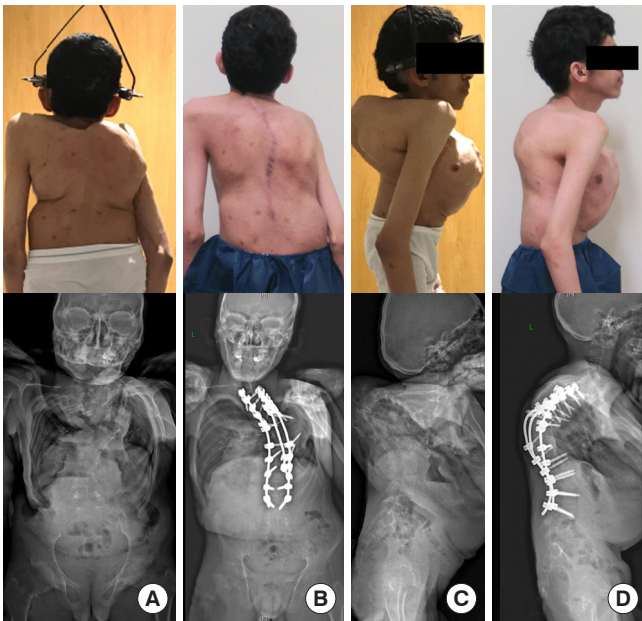


Fig. 5. Preoperative (A, C) and postoperative (B, D) clinical pictures and posterior-anterior/lateral X-rays showing correction of the patient's severe kyphoscoliosis.

erative recovery. There were no other major complications and the majority of his postoperative stay was due to the logistics of securing acute rehabilitation for an international patient.

At his 5-month postoperative follow-up visit, he was now full strength in his lower extremities with complete resolution of his hyperreflexia and clonus. Height measurements showed that he had gained 5" in height (preop, 4'8"; postop, 5'1") and 2.7 kg in weight (preop, 34.9 kg; postop, 37.6 kg). Surveillance X-rays showed his instrumentation to be in good position.

A patient consent was obtained for the surgical procedure and for publishing of the material.

DISCUSSION

Surgical treatment of severe spinal deformity remains a significant challenge despite advancements in modern instrumentation and the development of safer operative techniques. The use of halo-gravity traction serves as an adjunct to assist in the partial preoperative correction of highly rigid spinal deformities.⁵ This has been shown to increase pulmonary function, improve overall tolerance for a complex operative procedure, and has the potential to reduce the need of aggressive corrective measures such as vertebral column resections that are known to have higher complication rates.^{1,5,11}

Neurologic complications related to halo-gravity traction are

extremely rare and have been reported to be 1% in a recent systematic review and meta-analysis by Yang et al.⁵ Although overall traction-related complications were common at 22% (43 nonneurologic traction-related complications in 197 patients across 10 articles), they did find 3 patients (of 292 patients in 13 studies) who experienced neurologic deficits in halo-gravity traction. Janus et al.⁶ reported on a patient who developed hyperreflexia of the legs which disappeared after reducing traction weight. Sink et al.⁷ reported on a case of cervical distraction after halo-gravity traction that presented as numbness to the roof of the patient's mouth, and traction was discontinued. Lastly, Bouchoucha et al.⁹ reported on a patient who already had a pre-existing neurologic deficit who then developed spastic paraplegia while in halo-gravity traction.

A report of Bouchoucha et al.⁹ report mirrors our patient who initially presented with pre-existing neurologic deficits manifesting as hyperreflexia, clonus, spasticity, and mild weakness in his bilateral distal lower extremities. This would suggest that in some patients with preoperative neurologic deficit, the spinal cord has much less tolerance even for gradual correction with increasing weight in halo-gravity traction. The neurologic deficits are also evidence that the spinal cord is already compromised and showing objective signs of dysfunction via tension at the apex, as well as a reduction in the normal blood supply it would otherwise receive. Our patient also had the added compressive effects of a dorsolaterally positioned neurofibroma. Although his exam did improve after a reduction in his traction weight, it was likely the subsequent trivial trauma of a ground-level fall in the setting of an already impaired spinal cord that precipitated his sudden paraparesis.

The option of immediate and emergent surgical intervention was considered after our patient fell and developed a significant decline in neurologic function. However, because his surgical date had not been planned for several weeks, surgical adjuncts such as his custom cage and 3-dimensional spinal model for definitive surgical correction had not yet been processed. Due to the complex configuration of his anatomy, we knew that there would be few salvage options for good bony fixation for a high-stress correction after a completely destabilizing circumferential decompression with vertebral column resection. His improving neurologic exam on just conservative measures was reassuring that we did have some time to prepare and move up his definitive surgical correction. Certainly, however, had his neurologic exam not improved or declined further, he would have been brought emergently for surgical decompression for preservation of neurologic function which would have likely entailed a staged temporary construct that would have required a plan to return for his definitive con-

struct on a subsequent date. Because of the highly destabilizing nature of a circumferential decompression with only temporary fixation in the setting of a severe rigid deformity, this salvage plan would have been undertaken with a significant degree of caution.

It should be noted that while this report discusses the occurrence of neurologic deficits during halo-gravity traction, this event is suitably rare. With modern technical advances in surgical release and segmental instrumentation, the potential for intraoperative neurologic compromise with rapid intraoperative correction of deformity has increased.^{4,15,16} Koller et al.¹ reported on 10 patients with preoperative neurologic deficits of which 2 were not ambulatory and 9 of those patients had radiographic evidence on MRI of a sick spinal cord. While in halo-gravity traction, all patients experienced improvements in spasticity, hyperreflexia, subjective weakness, and dysesthesias; 2 of those patients had noted objective improvements in motor strength and gait. They concluded that halo-gravity traction improved progressive neurologic deficits especially in the setting of a sick spinal cord draped over the apex of a severe decompensating kyphoscoliosis deformity. Our personal experience with halo-gravity traction has also been similar in that it is an otherwise safe and usually a very well-tolerated method of preoperative correction.

We present here a case of neurologic deficit manifesting as worsening bilateral lower extremity weakness after the application of halo-gravity traction, subsequently complicated by an inpatient ground-level fall which rendered our patient paraparetic. He subsequently improved with conservative measures and his weakness and myelopathy completely resolved after expedited definitive surgical correction. Although halo-gravity traction is a safe technique for the nonsurgical partial correction of severe rigid deformity, a high index of suspicion for potential neurological complications needs to be maintained.

CONFLICT OF INTEREST

The authors have nothing to disclose.

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