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Technical Report

Extrinsic thoracic spinal cord compression related to supine position: from diagnosis to the creation of a spinal protection shield

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Abstract

BACKGROUND: Rapidly progressing extrinsic spinal cord compression syndromes are rare, especially when the compression is associated with the supine position.

PURPOSE: This work presents a case of extrinsic thoracic spinal cord compression related to the supine position and describes our approach from diagnosis to the technical therapeutic creation of a spinal protection shield.

STUDY DESIGN: One case of a patient suffering from extrinsic spinal cord compression syndrome is reported.

PATIENT SAMPLE: We report the case of a Coptic priest patient who, as a result of Pott disease sequelae, underwent several decompressive and stabilizing surgeries for major kyphoscoliosis. Consequently, he developed extrinsic thoracic spinal cord compression caused by the supine position.

OUTCOME MEASURES: After each instrumentation device removal, we noticed progressive severe paraparesis when the patient was supine. Imaging assessment confirmed spinal dynamic and intermittent compressions triggered by the supine position, which was facilitated by the exposure and vulnerability of the thoracic spine cord.

METHODS: We implanted a tailored titanium mesh spinal protection shield and a trapezius flap for spine coverage. This work presents the diagnostic aspects as well as several surgical technique options.

RESULTS: At the 6-year follow-up, the patient's neurologic conditions were significantly improved. We report neurologic improvements, no sphincter disorder, persistent spasticity, and lower limbs weakness not affecting full ambulation.

CONCLUSIONS: To our knowledge, no other case of spinal protection shield in compressions caused by the supine position have been studied. The surgical and technical management therefore remains innovative. © 2015 Elsevier Inc. All rights reserved.

Keywords: Cord compression; Pott disease; Spinal cord shield; Supine position; Extrinsic compression; Surgical management

Introduction

Acute or rapidly progressing spinal cord compression syndromes related to posture are rare. They can be difficult to diagnose especially when neurologic deficits only show up while the patient is in the supine position. The case discussed here was observed in a patient presenting major thoracic post Pott disease kyphoscoliosis sequelae. The patient had had biologically confirmed pulmonary tuberculosis, which was revealed by extrapulmonary symptoms and complications caused by thoracic Pott disease. Iterative surgery on the vulnerable and deformed spine led to extrinsic cord compression on the T5–T7 apex of a kyphoscoliosis. The diagnosis was challenging, as was the uncommon treatment, which consisted of the implantation of a spinal cord protection shield associated with a musculocutaneous flap.

To our knowledge no similar cases have been reported. Additionally, it seemed important to report the therapeutic efficacy of this custom-made spinal cord protection shield.

Clinical and technical presentation

A 27-year-old Coptic priest first consulted the author for a spinal disorder caused by T5–T7 thoracic deformation

FDA device/drug status: Not applicable.

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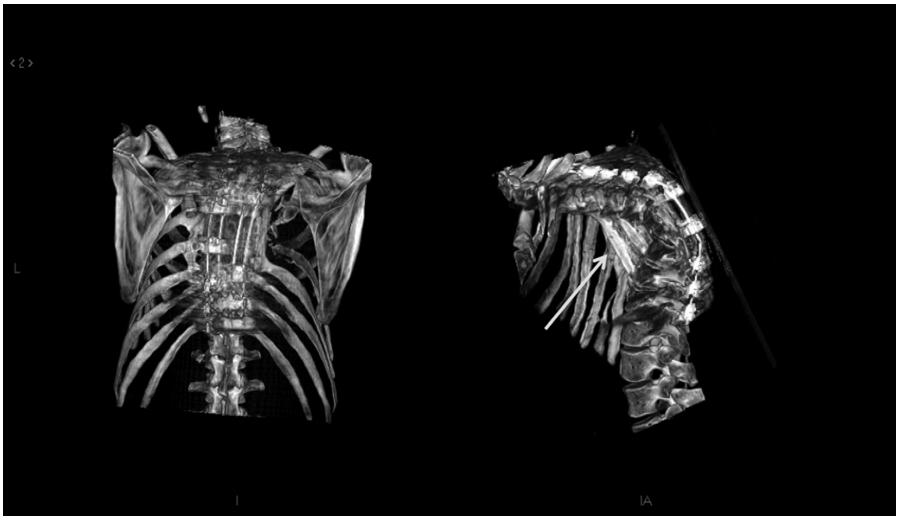

Fig. 1. 3-D thoracolumbar spine CT scan (Left), posterolateral instrumented kyphoscoliosis (Right), and anterior strut graft (white arrow).

following a Pott disease diagnosis at the age of 13. Progressive paraparesis and neurologic deterioration were observed during the preceding months. Computed tomography scans and plain X-ray investigation demonstrated a 95° major Pott kyphotic deformity with an apex at T5–T7. Corrective surgery (1987) was performed through a wide posterolateral T5–T8 laminoarthrectomy. The posterior wedge of the deformity (posterior wall of T6–T7) was cut down, allowing anterior spinal translocation. An additional posterolateral fusion associated with T2–T10 transpedicular plate instrumentation (Roy Camille device) was performed, without the intention to correct the global kyphotic deformity, but to stabilize it. Postoperative evolution was uneventful, and the patient recovered all his neural functions. After 8 years following the operation (late 1994), we removed the device due to instrumentation-related pain with scapular irradiation resurgence. During this surgery, a complete evaluation of the posterolateral bone graft was performed and fusion was assessed. Unfortunately, in the postoperative period (6 months), a neurologic deterioration occurred, which surprisingly included intermittent paraparesis. This paraparesis was quickly identified as a position-related phenomenon. The supine and standing positions were responsible for the emergence of this paraparesis. Plain and dynamic apex radiographs performed on recovery from Pott disease showed a small T5–T7 range of motion (6° ROM), while a computed tomography scan and magnetic resonance imaging (MRI) performed in the supine position surprisingly revealed a spontaneous apex deformity reduction of 20°. The delaminated spinal cord demonstrated signs of severe compression in front of the T5–T6 mobile kyphoscoliotic apex. The saccoradiculography corroborated intermittent epidural positions related to extrinsic compression. Following the removal of the device, the recovered flexibility of this kyphotic spine induced recurrent compression at the deformity apex level. Simultaneously, the large posterolateral laminoarthrectomy directly exposed the cord to compression when supine. In 1995, a revision surgery with anterior strut grafts was performed to definitively stabilize the kyphosis associated with a new posterior instrumentation (Fig. 1 Left and Right). Two additional cross-links were added to avoid cord exposure and to prevent the cord from dorsal soft tissue compression in the supine position. Fusion was also achieved by adding an autologous bone graft. After this procedure, the patient recovered full ambulation after rehabilitation but experienced lasting weakness in the lower limbs and persistent spasticity. Despite these sequelae, he was able to return to his priest position in France and Egypt. The patient experienced no further specific problems until 2005. The undernourished patient presented a local infection with fistula on the scar tissue. Following treatment of the operating area sepsis and complete posterior instrumentation removal over a 3-year time period, he started to develop a new postoperative progressing spinal cord compression syndrome. It was noted that scar area palpation or the supine position triggered neurologic deficits, whereas the lateral decubitus position reduced them. The supine position was responsible for the cord compression, which explained the intermittent neurologic symptoms. Imaging assessment confirmed thoracic spinal cord compression in the vulnerable area at the level of the large laminoarthectomy (Fig. 2A–F). An area of large, thin, and damaged scar tissue was directly covering the underlying cord. By this time, the kyphoscoliosis apex had completely fused and healed (Fig. 3 Left, Middle, Right). Finally, in February 2008, we decided to implant a spinal cord protection shield by the iterative posterior approach, along with a wide excision of the wound scar. A spinal cord protection shield was achieved preoperatively by shaping a cylindrical titanium mesh device (Fig. 4A). The "shield" was designed to fit the size of the exposed thoracic vertebral spine 6 cm×3 cm (Fig. 4B). The protective shield was fixed with short cortical bone screws into the posterolateral bony fusion mass (Fig. 4C). The protective shield fully overhung the

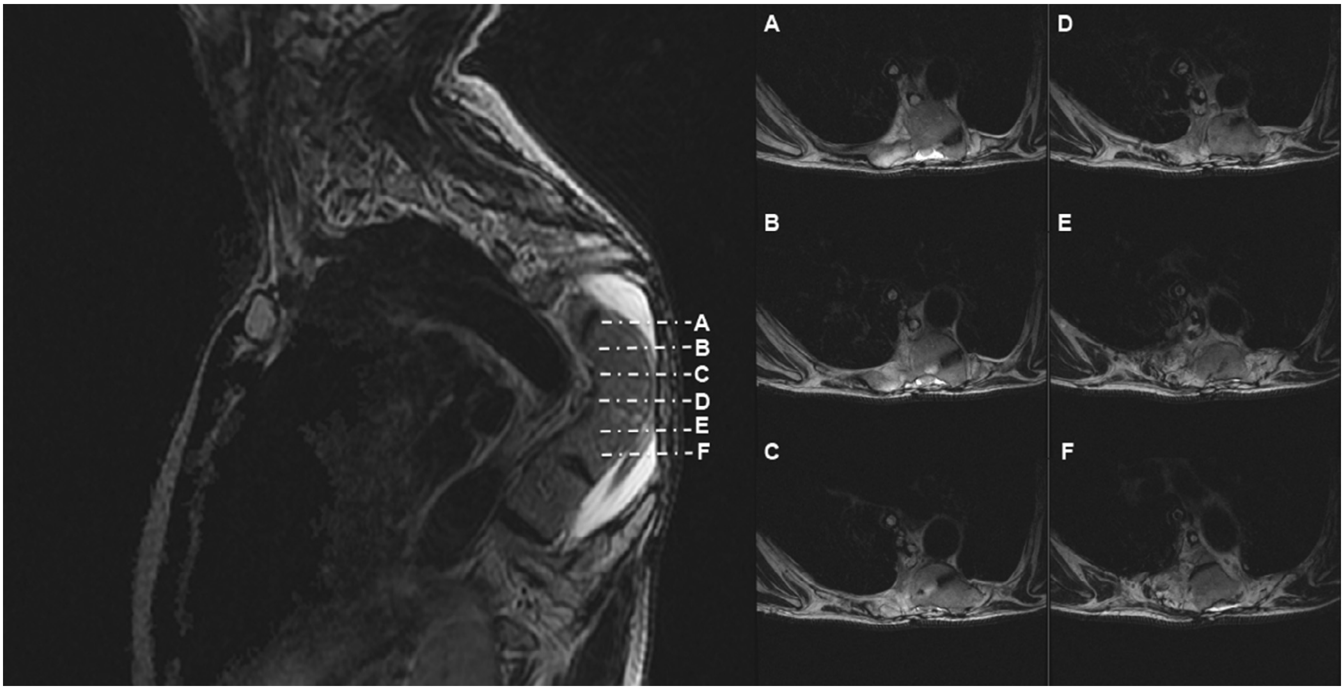

Fig. 2. MRI T2-weighted thoracic spine: sagittal level related to axial views (A–F) revealing extrinsic compression, spinal cord standing directly below soft tissues was stable and merged after 5 years.

delaminated cord at the T5–T8 laminoarthrectomy level. Soft tissue coverage after extensive scar resection was performed by means of a trapezius flap ending the procedure (Fig. 4D). Within 1 year, the patient's neurologic condition had significantly improved. We report neurologic improvement, no sphincter disorder, persistent spasticity, and lower limbs weakness not affecting full ambulation. The patient experienced no delayed healing or sepsis. The spinal shield remains intact at the last follow-up (2014) (Fig. 5C–F) and esthetic effect was "noticeable" (Fig. 5A and B).

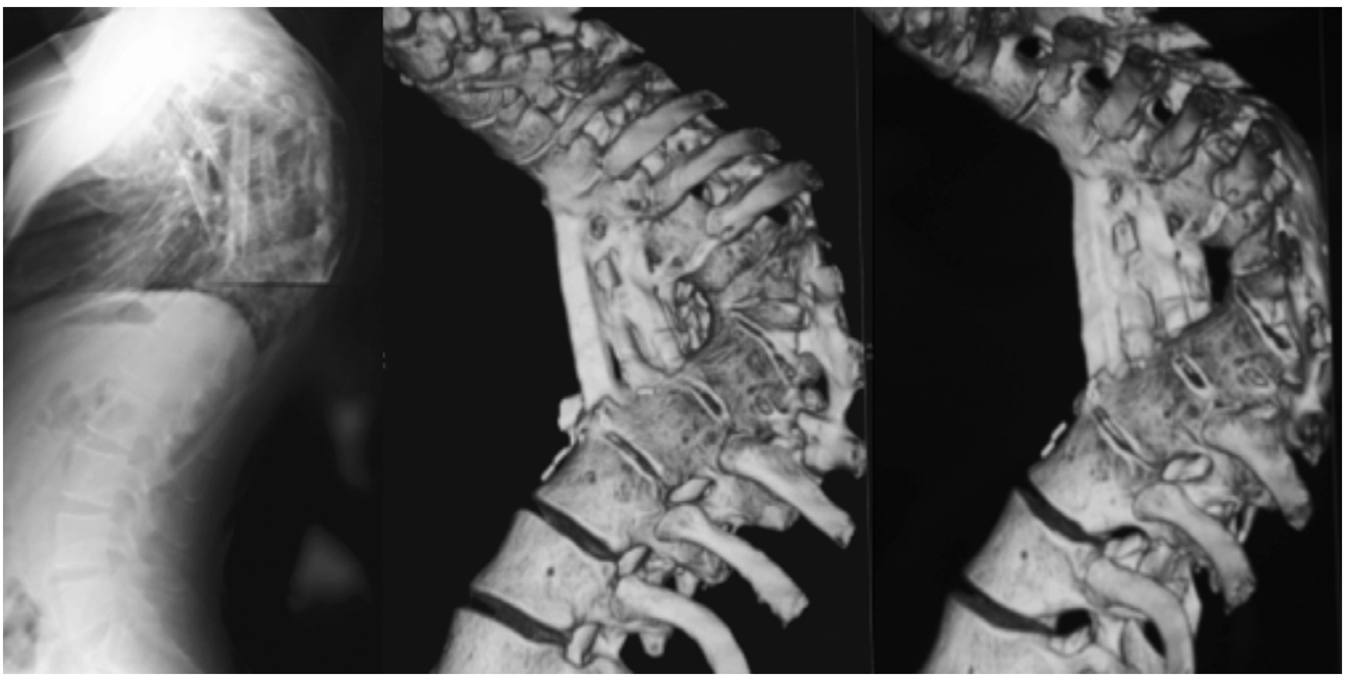

Fig. 3. Profile X-ray (Left) and 3-D thoracic spine CT scan reconstructions (Middle and Right) confirmed that the kyphoscoliosis apex was completely merged.

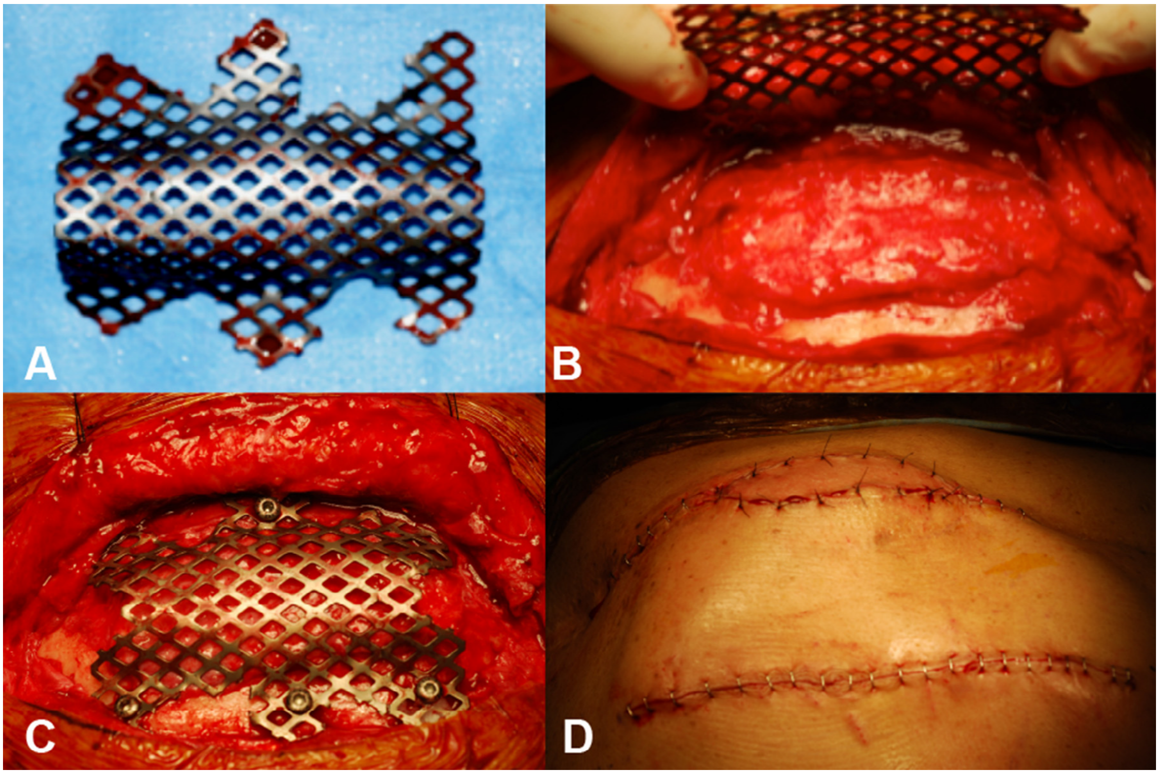

Fig. 4. Preoperative views of the spinal cord (B, C) protected by the tailored cylindrical titanium mesh device (A), and the spine coverage by a trapezius flap (D).

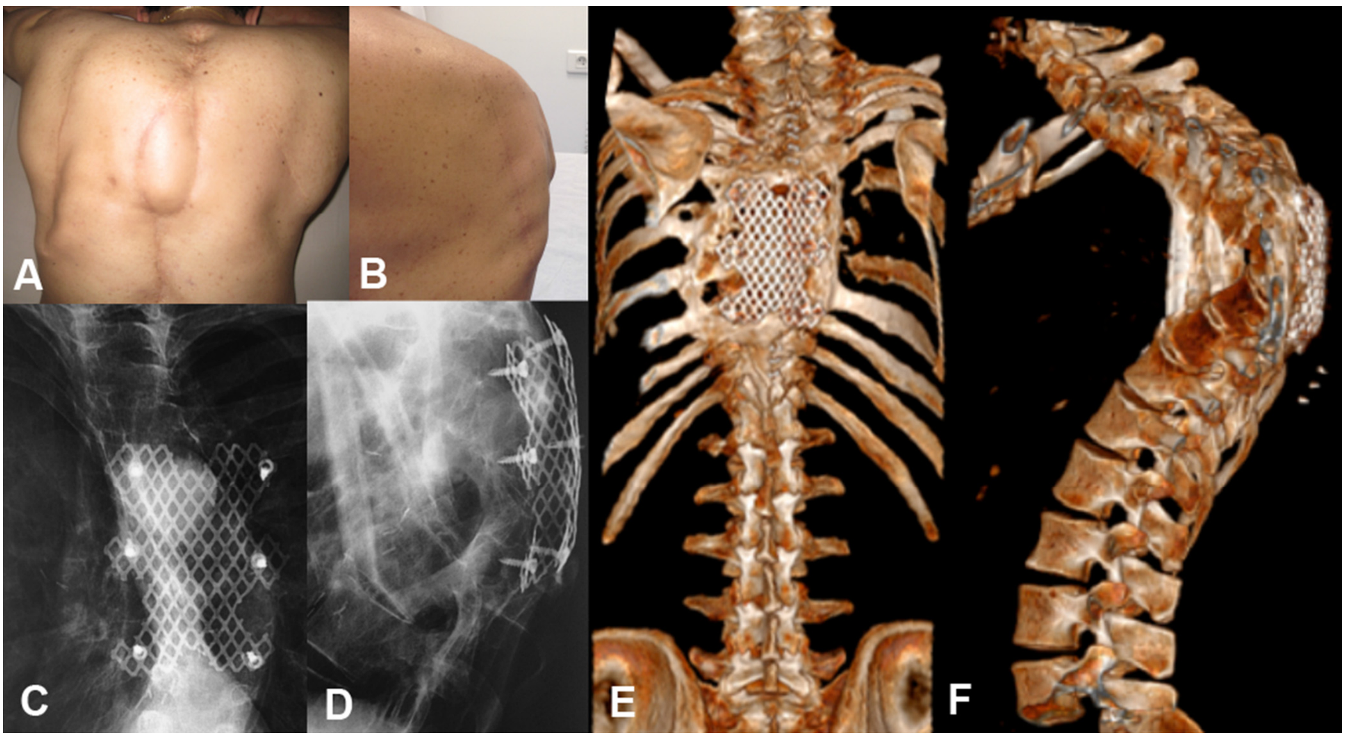

Fig. 5. Dorsal (A) and lateral (B) views of skin evolution. Spinal shield remaining intact at 5 years follow-up, based on plain X-ray in posterior-anterior (C) and lateral views (D), and on 3-D CT scan reconstructions (E and F).

Discussion

In the present study, we have described a rare case of dynamic spinal cord extrinsic compression. The supine position triggered this compression, which was induced by spinal cord vulnerability associated with multiple surgeries following Pott disease. The outward signs, the diagnostic approach, and the surgical strategy were unusual. The unique context of major thoracic deformations as a result of Pott disease sequelae was decisive in the onset of neurologic deficits. Decisive … but not sufficient! Spinal cord compression syndromes in Pott disease have been reported. According to the literature, neurologic deficits are progressive in the beginning, and their evolution is gradual. Spinal cord compression is most often associated with a tuberculosis abscess or kyphoscoliosis deformations [1–3].

The intermittent neurologic deficits observed were multifactorial: major kyphoscoliosis, a small local instability, and significant spine cord exposure. In our case, the supine position triggered the compression. This compression was quite surprising because of its extrinsic and intermittent aspects related to the supine position. Indeed, it was difficult to assess the relationship between the onset of the neurologic deficits and the direct cord compression, just under the patient's skin. This case was undocumented, as were the mechanical explanations.

Usually in major kyphosis Scheuermann disease [4] and Pott disease sequelae [5], the physiopathologic spinal cord compression mechanism involves the anterior column and the ventral spinal thoracic cord [4,6]. Care is generally successful and consists of deformation corrections, posterior or circumferential spinal cord decompression, and instrumented fusion. Up until now, a similar strategy of a posterior spinal shield concept resulting in spinal cord protection has never been reported. In the present case, the physiopathologic mechanism was very specific. We supposed that when the patient was in the supine position, the spinal cord was flattened and compressed between the kyphotic apex and the dorsal soft tissue, thus acting as a block. The extended posterolateral bony resection removing the lateral part of the vertebral canal was responsible for the cord exposure to compression at the apex level. Diagnostic tools were limited, as were the mechanical considerations. Some authors proposed to perform a dynamic MRI when the compression mechanism was not revealed by a simple MRI [7]. Based on our experience, only MRI and myelography in the supine position successfully revealed spinal cord compression. Plain or dynamic radiographs, as well as weight-bearing myelography, were not conducive at this last stage of the condition's progression. Could Dynamic MRI have been useful? We assumed that in atypical cases, alternative imaging investigations, such as a dynamic spinal electrophysiological investigation [5], could be used. Such investigations, performed in lateral decubitus or in flexion or extension posture compared with the supine position, would have probably helped us to point out the spinal cord compression mechanism and its trigger. This can be helpful in detecting the causative factors. Knowing that the spinal cord compression was linked to cord vulnerability in the supine position, a common surgical choice would have been inadequate [8]. A posterior spinal cord shield was probably the only available option. We therefore performed the most adapted procedure, which was shielding the posterior spine cord aspect with a custom-made shaped mesh titanium cylinder. Skin coverage was similarly challenging in this post-septic situation. A trapezius flap was the only solution to address this additional difficulty.

Alternatively, we could have implanted two cross-links, at least, to protect the spinal cord [9]. We believed that a single cross-link would have recreated a posterior block that could have pressed and crushed the thoracic spinal cord in the supine position. We reported similar spinal shielding in a recent experimental rat study [10]. Two cohorts of rats, both having undergone a wide laminectomy, were compared. One group experienced extrinsic compression in the absence of spinal cord shield, whereas the other group had a mesh titanium spinal cord shield. Macroscopic and electrophysiological spine assessments showed significantly higher cord integrity rates in the shield group compared with the control group, thus demonstrating the potential usefulness and effectiveness of the spinal cord shield.

Chung et al. [11] reported three columns reconstruction after total en bloc spondylectomy by a posterior approach in eight patients treated for vertebral tumoral disease. More than just creating a posterior shield, seven patients underwent posterior column reconstruction with a posterior mesh titanium shield used as an autologous bone graft support. The remaining patients underwent device removal due to septic complications. According to the authors, no neurologic deterioration followed the procedures. Total spondylectomy, for example, may be a procedure which could require spinal cord shield implantation.

Biomechanical failure or complications related to the spinal cord shield are unknown because of the limited number of clinical trials and experience with these preoperative custom-made devices.

We have no information regarding skin coverage complications following such spinal cord shield procedures. However, in the present case, the patient's body mass index was less than 16 kg/m^2 and had challenging sepsis, deteriorated general health, and skin conditions. A musculocutaneous trapezius flap, which healed, was performed. Limited studies found appropriate applications, to this musculocutaneous flap, in thoracic spine coverage, as well as thoracolumbar spine, chest, and face coverage [12–15]. In our case, we found that this procedure was reliable and reproducible in terms of healing.

Conclusions

The present case confirms that spinal cord compression related to the supine position exists. Imaging investigations may reproduce the condition's trigger and reveal the spinal cord compression mechanism. Spinal cord shielding and instrumented stabilization should therefore be performed. The iterative dorsal approach may require trapezius flap coverage planning. A prompt recovery and favorable long-term outcomes can be expected following such a procedure.

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