

Technical Report

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

L. Ajavon, MD, A. Amelot, MD, PhD*, C. Conso, MD, L. Balabaud, MD, C. Mazel, MD, PhD

Department of Orthopaedics Surgery, Institut Mutualiste Montsouris, 42 Boulevard Jourdan, 75014 Paris, France

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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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* Corresponding author. Orthopedy Department, Institut Mutualiste Montsouris, Jourdan Boulevard, 42, 75014 Paris, France. Tel.: +33 1 56 61 63 63; fax: +33 1 45 80 60 41.

E-mail address: aymmed@hotmail.fr (A. Amelot)

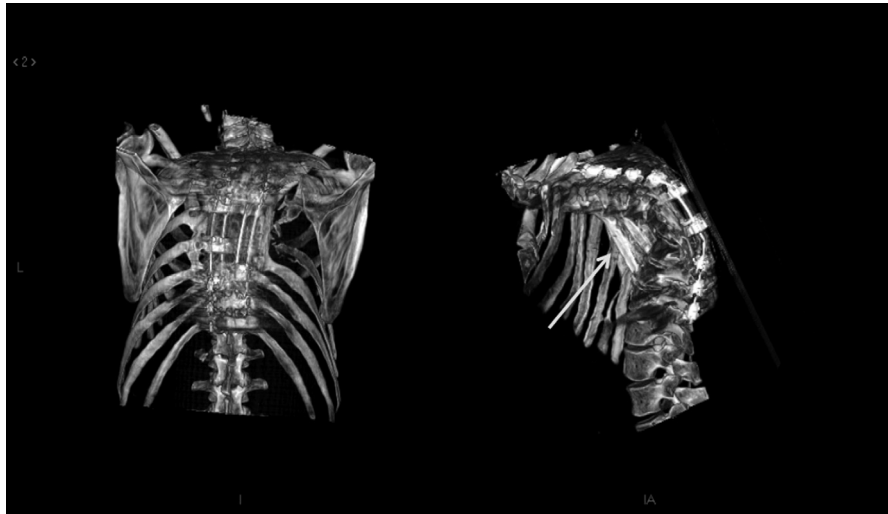


Fig. 1. 3-D thoracolumbar spine CT scan (Left), posterolateral instrumented kyphoscoliosis (Right), and anterior strut graft (white arrow).

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 laminectomy. 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 saccoradiography 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 laminectomy 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 laminectomy (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

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