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Adjacent Segment Disease 44 Years Following Posterior Spinal Fusion for Congenital Lumbar Kyphosis

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Abstract

Study Design: Case report.
Objective: To report the clinical and imaging findings of a patient with lumbar stenosis 44 years after posterior spinal fusion for congenital lumbar kyphosis.
Summary of Background Data: To our knowledge, there are no long-term follow-up reports after posterior spine fusion (PSF) for congenital kyphosis. Congenital kyphosis is an uncommon deformity with the potential to progress rapidly and result in deformity and neurologic deficits.
Methods: We report the patient's history, physical examination, imaging findings, and management in addition to providing a literature review.
Results: A 54-year-old-male who underwent T8–L3 PSF in 1972 because of congenital kyphosis presented 44 years after surgery with lower back pain, buttock, and bilateral posterior leg pain. On physical examination, no weakness was elicited and magnetic resonance imaging demonstrated L4–L5 lumbar stenosis. The patient was enrolled in physical therapy and responded well to medical/interventional management.
Conclusion: To our knowledge, this is the longest follow-up of surgical management of congenital lumbar kyphosis. Posterior fusion only halted the progression of the kyphosis with subsequent developed of adjacent segment disease distal to the fusion.
Level of Evidence: Level IV.

Keywords: Congenital kyphosis; Emivertebrae; Adjacent segment disease; Posterior spinal fusion; Spinal deformity

Congenital kyphosis is a rare deformity in which vertebral anomalies impair the longitudinal growth of vertebral elements in the sagittal plane with subsequent development of posterior angulation [1-10]. Surgery is indicated over nonsurgical approaches (ie, bracing) because spinal cord compression and neurologic deficits can occur as the kyphosis progresses [1,2]. The type of vertebral anomaly, size of deformity, and degree of spinal cord compression determine the operative approach [1-10]. We report a 44-year follow-up of congenital kyphosis of L1 managed with posterior spinal fusion (PSF) and subsequent development of adjacent segment disease at the distal part of the fusion.

Case Report

A 54-year-old man who underwent PSF for correction of congenital L1 kyphosis 44 years earlier presented to the office for low back pain and leg pain with ambulation. At the age of five months a chest radiograph was performed for an upper respiratory tract infection and incidentally revealed an L1 Type I (failure of anterior vertebral body formation) with a 50-degree kyphosis from T12 through L2. As the patient grew older, a gibbus deformity in his thoracolumbar junction was noted. Given the absence of neurologic symptoms, the patient was managed medically/interventionally while being closely monitored for the progression of kyphosis. In 1972, the patient presented with lower back pain when seated and a 10-degree limitation of lateral bend bilaterally. Radiographs revealed a kyphosis of 71 degrees from T12 to L2 and 14-degree scoliosis from T12 to L5 convex to the right. A myelogram revealed a myelographic block at T12 and L1. Despite the presence of a myelographic block, the patient did not demonstrate any neurologic deficits.

Author disclosures: none.

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Because of the progression of the kyphosis and the spinal cord compression, the patient underwent T8–L4 PSF with autogenous left iliac bone graft. Postoperatively, the patient was placed in a plaster cast. Ten months postoperatively, radiographs revealed a 10-degree decrease in kyphosis. In 1975, three years postoperation an intact fusion mass, a 54-degree kyphosis from T12 to L2 and a 73-degree lordosis from L2 to S1 were noted.

From 2013 to 2016 (44 years postoperation), the patient has been followed for axial back pain symptoms in addition to neurogenic claudication symptoms. He has no motor or sensory deficits. His radiographs demonstrate a 57-degree kyphosis from T12 to L2 (Fig. 1), and computed tomography demonstrates a solid fusion mass from T8 to L4 (Fig. 2). The patient's pelvic incidence (PI) was 73 degrees, with a kyphosis of 7 degrees from L1 to L5 and a PI lordosis mismatch of 80 degrees. Sagittal vertebral axis was positive 2.7 cm. Magnetic resonance imaging (MRI) demonstrated cord signal changes in the T12–L1 region because of the L1 hemivertebrae as well adjacent segment disease (L4–L5 stenosis) (Fig. 3). Subsequent MRI of the lumbar spine obtained 31 months later did not demonstrate significant worsening of the stenosis and degenerative at L4–L5 (Fig. 3C). The patient continues to be managed medically/interventionally with physical therapy, nonsteroidal anti-inflammatory drugs, antiepileptics, as well as

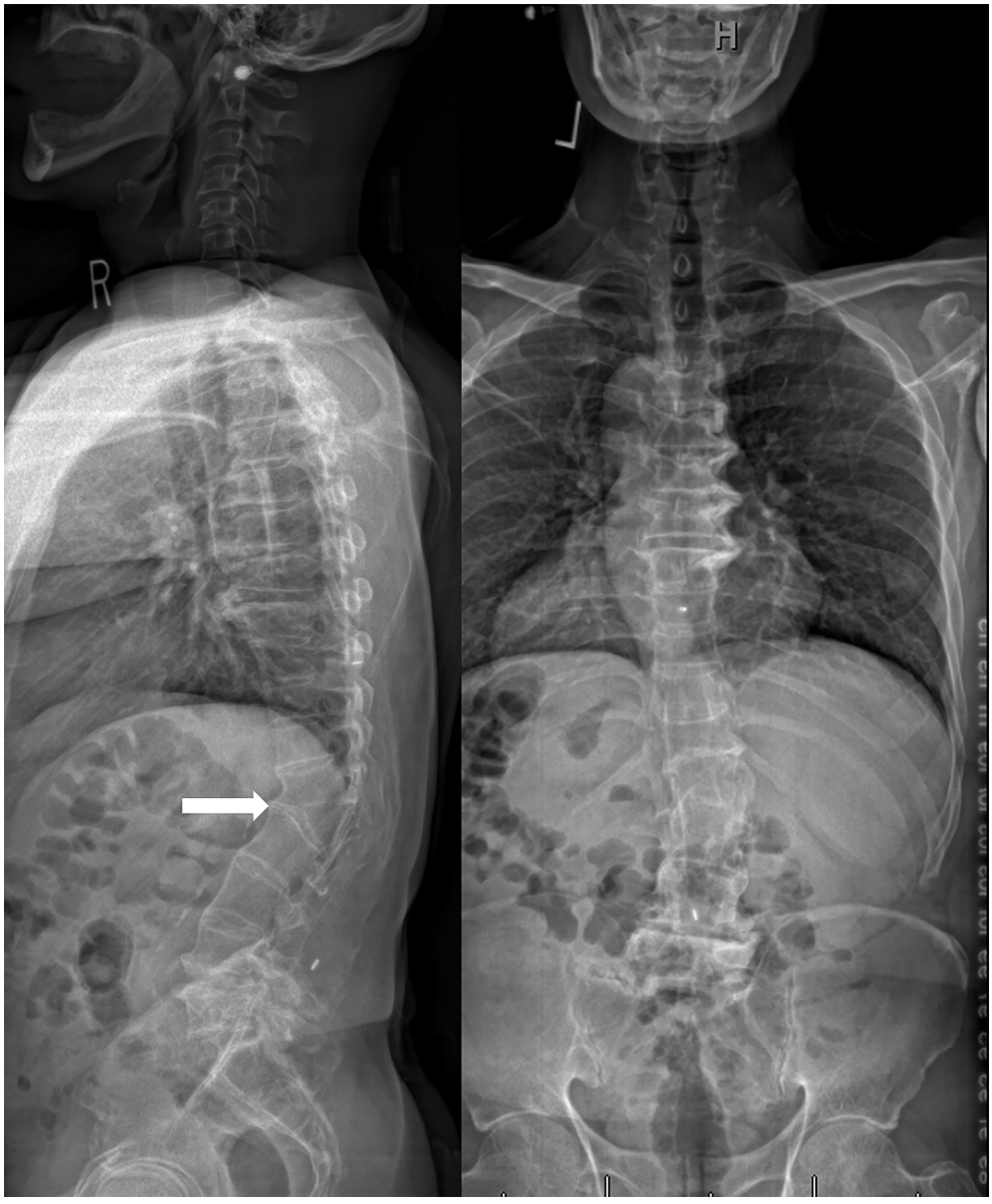

Fig. 1. Anteroposterior (AP) and lateral scoliosis radiographs demonstrating the L1 hemivertebrae (arrow) and posterior spinal fusion. A mild scoliosis is noted on the AP view.