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Case report

Gibbus deformity: Lessons from incompletely treated osteomyelitis☆

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ABSTRACT

Gibbus deformity is defined by the collapse of one or more vertebral bodies, which results in kyphosis and is often the consequence of infection, metabolic, or congenital irregularities in the vertebrae. In this report we present a unique case of a young male with inadequately treated MRSA bacteremia complicated by lumbar osteomyelitis with progression to severe joint destruction and a marked Gibbus deformity.

Introduction

Gibbus deformity is defined by the collapse of one or more vertebral bodies, which results in kyphosis and is often the consequence of infection, metabolic, or congenital irregularities in the vertebrae. Anterior vertebral collapse results in progressive kyphosis and if left untreated, spinal cord compression and paraplegia [1]. While this condition is most common in young patients with spinal tuberculosis [2], other infectious etiologies exist, but are exceedingly rare. In this report we present a unique case of a young male with inadequately treated MRSA bacteremia complicated by lumbar osteomyelitis with progression to severe joint destruction and a marked Gibbus deformity.

Case presentation

A 45-year-old undomiciled male with a history of motor vehicle collision (2016), intravenous (IV) drug abuse complicated by Methicillin-Resistant Staphylococcus aureus (MRSA) bacteremia, recurrent thoracic/ lumbar discitis/osteomyelitis, and left hallux osteomyelitis status post amputation, presented to our facility with progressive acute on chronic back pain.

☆ Competing interests: The authors have no competing interests.

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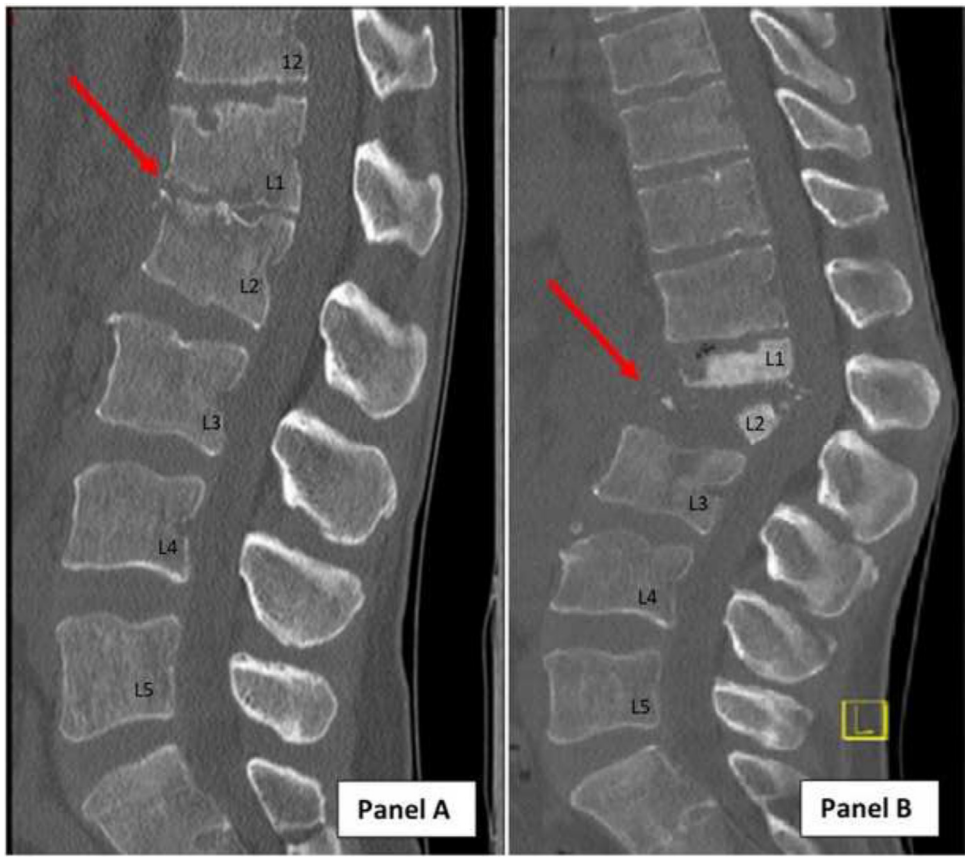

Fig. 1 – CT scan of the thoracolumbar spine. CT scan demonstrates progression of osteomyelitis and progressive Gibbous deformity of the spine in 3 mo after first presentation - August 2020 (Panel A) and 1 year after first presentation - March 2021 (Panel B).

Historically, he was first diagnosed with MRSA bacteremia with concurrent L2-L4 lumbar discitis in 2016, which was incompletely treated due to medication non-adherence and an against medical advice (AMA) discharge. He subsequently presented to our facility 11 months prior to the current presentation where he was diagnosed with MRSA bacteremia and left hallux osteomyelitis. He was managed with Ceftaroline, given mild Vancomycin resistance, and incision & drainage and amputation, but subsequently left the hospital AMA with a course of oral Trimethoprim-Sulfamethoxazole. Two months after this AMA discharge, he represented in the context of ongoing back pain, was placed on Ceftaroline once again, and was discharged on long-term Ceftaroline therapy to a skilled nursing facility from which he ultimately eloped. One month later, he presented in the outpatient setting with worsening back pain and was found to have a new thoracic spinal deformity and was recommended for emergency evaluation which he declined since his symptoms were stable. Three months following this visit, he experienced acute worsening of his back pain and presented to the emergency department for evaluation.

Upon presentation, the patient reported usual health until one week prior, at which time he experienced disabling back pain complicated by an inability to ambulate. He denied new onset weakness, paranesthesia, saddle anesthesia, bowel, or bladder incontinence, fever, or chills. He furthermore denied recent trauma or ongoing IV substance use. He was hemodynamically stable. Physical examination was remarkable for a protruding, bony deformity localized to the T11-T12 spinal area with associated spinal and paraspinal tenderness extending to the lower lumbar region, without evidence of crepitus or overlying erythema. Cardiovascular examination remained unremarkable without evidence of murmurs or peripheral stigmata of endocarditis. Neurological examination was unremarkable except for mild left lower extremity weakness and difficulty ambulating secondary to pain.

Diagnostic workup was relatively unremarkable except for a mild normocytic anemia, CRP of 4.5 mg/L (n: 0-3) and ESR of 62 mm/h (n: 0-15). Computed Tomography (Fig. 1) and Magnetic Resonance Imaging demonstrated evidence of osteomyelitis of L1-3, with vertebral collapse and a severe kyphotic deformity at L1-2, in addition to bilateral psoas abscesses. He was placed on Ceftaroline and ultimately transferred to a tertiary care facility where he underwent anterior vertebral corpectomies L1-L2, anterior spinal fusion T12-L3 with prosthetic vertebral replacement cage, local autogenous bone graft, and cancellous and local rib allograft.

Discussion

Gibbus' etymological origin comes from the Latin 'gibbus' meaning 'hump' [3]. Vertebral osteomyelitis remains rare, with an incidence of 2.2/100,000 annually [3], with most cases secondary to tuberculosis. Vertebral osteomyelitis is usually a sequala of bloodstream or soft tissue infection [4].

Cases of Gibbus deformity generally present with back pain, limiting mobility and causing weakness. It distorts the spinal canal anatomy leading to severe kyphosis, myelopathy, and even paraplegia [5]. The patient presented in this report demonstrated evidence of progressive vertebral destruction in the setting of multiple courses of incompletely treated vertebral osteomyelitis of spanning six years, with underlying IV drug use as an ongoing risk factor. This case serves to highlight the importance of completing antibiotic therapy and longitudinal follow up to prevent long-term complications.

Patient consent

Full attempts were made to contact patients and/or family members. Unfortunately, in the context of ongoing intravenous drug use and domicile status the patient remains MIA. Sufficient patient information has been removed and this patient cannot be identified.

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