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Neurologic Decline following Acute Vertebral Collapse in a Patient with Cervical Osteomyelitis

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Abstract

A 61 year-old male with 5 weeks of severe neck pain was diagnosed with cervical osteomyelitis. His past medical history was significant for end stage renal disease which required peritoneal dialysis and chronic recurrent respiratory infections. Baseline neurologic examination, gait, and muscle testing were all normal. Two weeks after the initial diagnosis, the patient developed severe atrophy of the hands, weakness involving multiple myotomes, and severe gait ataxia. Diagnostic imaging showed collapse of the C5 vertebral body with retropulsion into the central canal causing impingement on the spinal cord. Rapid diagnosis and treatment of osteomyelitis is pivotal in successful clinical outcomes.

Keywords

Cervical spine; vertebral infection; ataxia; spinal instability; neck pain

Introduction

Osteomyelitis is a rare condition accounting for only 2-4% of infectious bone disease, and typically occurs in the fifth/sixth decade of life with men more frequently afflicted than women [1,2]. Only 3-10% of vertebral osteomyelitis cases present in the cervical spine [3]. Symptoms typically present as neck pain and paravertebral muscle spasm. Hematogenous seeding is the most common cause of cervical osteomyelitis and frequently stems from intravenous drug use or skin ulcerations, as well as infections of the genitourinary tract, respiratory system, or oral cavity [3]. Patients with alcohol dependence, diabetes mellitus, chronic renal failure, malignancy, and prolonged corticosteroid use have an elevated risk of developing osteomyelitis [4]. While surgical intervention is the treatment of choice in patients that develop spinal instability [5], it may not always be an option due to the significant comorbidities in this patient population. This case describes a patient with cervical osteomyelitis who was treated conservatively that endured vertebral collapse and subsequent neurologic decline.

Case Presentation

A 61 year-old afebrile male presented to the emergency department with a 5 week history of severe neck pain and paravertebral muscle spasm. The patient had a past medical history of end-stage renal disease treated with peritoneal dialysis, congestive heart failure, and coronary artery disease with two previous myocardial infarctions. Cyclobenzaprine hydrochloride and hydrocodone bitartrate with acetaminophen prescribed at a previous physician encounter were unsuccessful in managing his neck symptoms.

Physical examination exhibited negative findings for ankle clonus and Hoffmann and Babinski testing. Deep tendon reflex testing was negative for hyperreflexia. Ambulation was independent without assistive device and there were no signs of ataxia. Strength testing identified mild weakness of the right hand, but was otherwise unremarkable.

Erythrocyte sedimentation rate, C-reactive protein levels, and neutrophils were elevated while white blood cell count was within normal limits. Radiographs were significant for destructive changes at the C5 vertebral body suggestive of osteomyelitis (Figure 1). Magnetic resonance imaging (MRI) also revealed findings that were representative of osteomyelitis (Figure 2) with blood cultures revealing *Staphylococcus aureus*. The patient was prescribed intravenous antibiotics, issued a Philadelphia collar, and referred to a spinal surgeon. The spinal surgeon determined the patient was at too great a medical risk for surgical intervention.

Three days after the discharge for this initial hospital admission, the patient was admitted to the hospital again for care following an episode of syncope. He denied chest pain, shortness of breath, and palpitations, however, he did report a feeling of impending doom. The patient was found to have an ST elevated myocardial infarction that required emergent placement of 2 stents. Repeat cervical radiographs of the cervical spine were ordered one week after the initial diagnosis, revealing vertebral collapse of the C5 vertebral body (Figure 3). However, no neurologic decline was found at that time.

Fourteen days after initial diagnosis, however, the patient developed severe ataxia requiring a front wheeled walker and moderate assistance from a physical therapist. A neurologic exam showed no hyperreflexia of deep tendon reflexes and negative findings for ankle clonus, Hoffmann's, and Babinski testing. However, the patient had developed bilateral weakness of the biceps brachii muscle as well as severe atrophy of the intrinsic musculature of the hands. Asterixis secondary to concomitant hepatic encephalopathy prevented more thorough examination of hand or wrist strength.

A cervical computed tomography scan and a neurology consult were ordered by the attending physician. The computed tomography scan revealed destruction of the C5 vertebral body with retropulsion of the destroyed vertebral body into the central canal causing compression of the spinal cord (Figure 4); MRI was not possible due to recent cardiac stent placement. The patient was referred to a spinal surgeon; however, surgery was deferred due to the patient's significant medical comorbidities.

One month after initiating antibiotic therapy, the patient's erythrocyte sedimentation rate dropped by 36% and his C-reactive protein level dropped 90%, indicating improvement of the osteomyelitis [6]. However, patient succumbed 6 weeks after initial diagnosis due to complications from a fourth myocardial infarction.

Discussion

Prompt clinical diagnosis of cervical osteomyelitis with laboratory and imaging confirmation is paramount in preventing catastrophic neurologic injury [7]. Because of the insidious onset of symptoms, frequent lack of fever, and absence of leukocytosis [6,8], diagnosis can be elusive and is typically delayed until 2 to 4 months of disease progression [9]. Erythrocyte sedimentation rate and C-reactive protein levels have been shown to be highly sensitive in excluding osteomyelitis [6]. Since the bone and disc involvement associated with osteomyelitis may be radiographically occult, [1] especially in the early

stages of the disease, magnetic resonance imaging is recommended since it is the most sensitive and specific imaging study for diagnosing spinal infections [10].

The standard of care for patients with cervical osteomyelitis treated nonsurgically is intravenous antibiotics followed by oral antibiotics and spinal immobilization [11]. Surgical intervention is typically reserved to patients with clear spinal instability, neurologic compromise, intractable pain, or poor response to antibiotic treatment [5]. Surgical intervention, especially for patients who are medically compromised, may pose significant risk in prolonging the course of the infection or introducing new pathogens into the region [12]. Medically compromised patients have shown to have an increased risk for respiratory complications, postoperative infections, symptomatic hematomas, longer length of hospital stay, and increased likelihood of discharge to a skilled nursing facility [13]. Studies have shown as high as an 11% mortality rate within 7 months of surgical intervention for patient with spinal infection [5].

The patient described in this case report had a past medical history of end-stage renal disease treated with peritoneal dialysis. There was a possibility that cervical osteomyelitis in this patient may have resulted from a catheter infection for the peritoneal dialysis. However, at no point did this patient have any signs of a catheter infection from the peritoneal dialysis, so the likelihood that the osteomyelitis in this patient may have resulted from a catheter infection is likely low.

When surgical intervention is elected for patients with spinal infection, early intervention either before or shortly after neurologic decline is paramount to post-operative outcomes. Previous studies have identified that surgical intervention delayed as little as 24 hours after onset of severe neurologic impairment may lead to minimal neurologic recovery [14]. In addition, patients undergoing cervical fusion after developing neurologic compromise have shown a 10-fold increase in death-rate, a 5-fold increase in infection-rate, and a 4-fold increase in developing pneumonia [15].

Figures

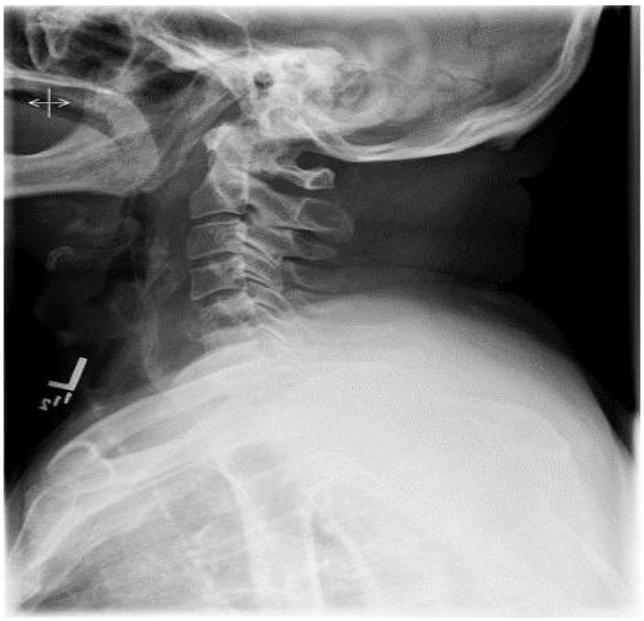

Figure 1: Lateral radiograph of the cervical spine demonstrating loss of height of the C5 vertebral body by approximately 50% with narrowing of the C5-C6 disc space and destruction of the inferior endplate of C5.

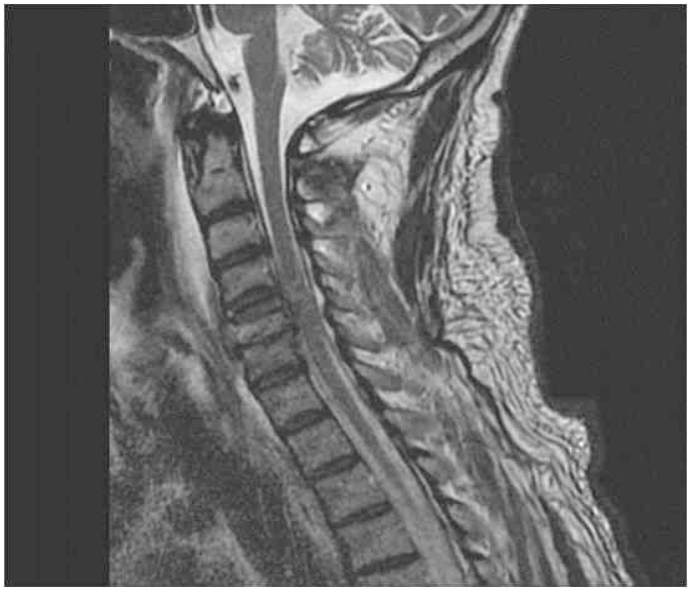

Figure 2: Sagittal T2-weighted magnetic resonance image demonstrating diminished height of the C5 vertebral body, increased signal within the C5-C6 disc space, and retrolisthesis of C5 upon C6 with mild cervical cord compression at this level without abnormal cord signal.

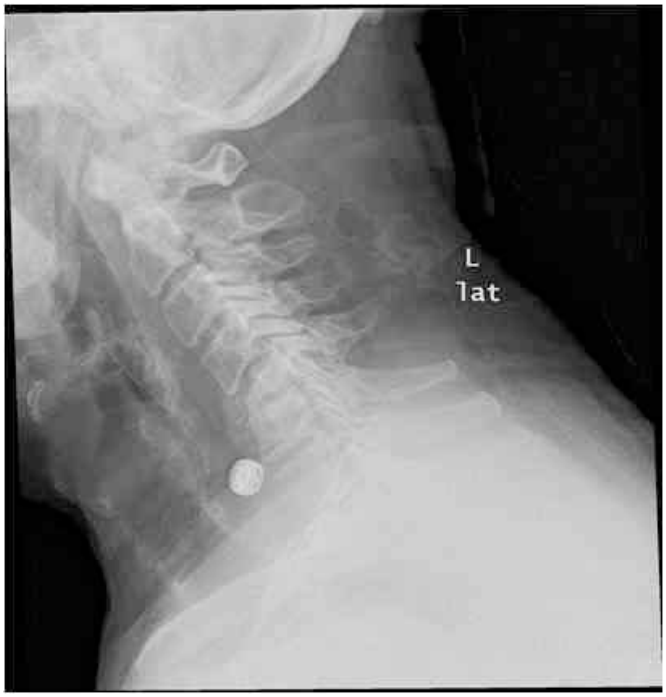

Figure 3: Lateral radiograph of the cervical spine demonstrating collapse of the C5 vertebral body with retropulsion, focal kyphosis, and narrowing of the central canal.

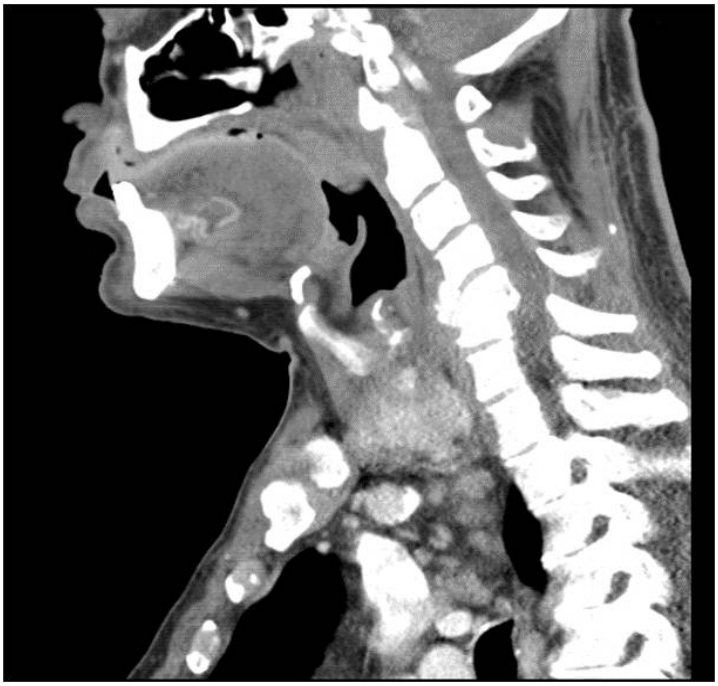

Figure 4: Sagittal computed tomography scan revealing destruction of the C5 vertebral body with retropulsion of the posterior aspect of the destroyed vertebral body, loss of disc height and kyphosis. This caused severe central canal narrowing with compression on the spinal cord.

Conclusion

Frequent neurologic examination in patients with cervical osteomyelitis is pivotal to early detection of neurologic decline, thereby facilitating expedited surgical intervention and maximizing surgical outcomes.

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