Spinal Brucellosis

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KEYWORDS

• Spinal infection • Brucellosis • MR imaging

KEY POINTS

- Spinal involvement is common in human brucellosis.
- Osteoarticular disease and neurobrucellosis are the most common complications.
- Spinal brucellosis involves lumbar region in more than half of the cases.
- Preservation of vertebral architecture is typical.
- MR imaging is the currently the best imaging tool for diagnosis and follow-up in patients with spinal infections.

INTRODUCTION

Spinal brucellosis is a significant cause of morbidity and mortality, particularly in endemic areas. The diagnosis of spinal brucellosis is challenging but important to ensure proper treatment. Early diagnosis and treatment are crucial. Radiologic evaluations have gained importance in the diagnosis, evaluation, and treatment monitoring of all the spinal infections. Diagnosis can be made with imaging and isolation of the causative agent from blood, cerebrospinal fluid (CSF), or the lesion.

DEFINITION AND EPIDEMIOLOGY

Brucellosis (undulant fever, Malta fever) is a zoonotic disease that effects animals as the primary host (ie, camels, sheep, goats) and humans as the secondary host.[1,2] The infecting agent was first identified by Bruce in 1887 in a patient who died on the island of Malta. The disease is caused by small, nonmotile gram-negative facultative intracellular coccobacilli of the genus *Brucella*, including *Brucella melitensis*, *Brucella abortus*, *Brucella suis*, *Brucella canis*, and *Brucella ovis*, which are usually transmitted through the consumption of uncooked meat or unpasteurized dairy products.[1,2]

B. melitensis is the most common microorganism isolated in brucella spondylitis and neurobrucellosis, which is also endemic in certain parts of the world. The incidence of spinal involvement in brucellosis is 2% to 65%. Men are affected more frequently than women, which may be reflective of occupational risks such as those encountered in the stock industry, which mainly employs men.[3]

The type of skeletal involvement depends partly on the patient's age and the *Brucella* species involved. Although arthritis, bursitis, tenosynovitis, and sacroiliitis are more frequently observed in younger patients, the frequency of spondylodiscitis increases with age, and its diagnosis may be difficult because brucella spondylitis may resemble many diseases that affect the spine, such as tuberculosis, pyogenic osteomyelitis, intervertebral disc herniation, and malignancy. Brucella spondylitis presents in focal and diffuse forms, commonly in people aged 50 and 60 years of age in endemic areas.[4]

Spinal brucellosis suggests a spectrum of disease comprising infections of the numerous components of the spinal colon, including vertebral bodies (spondylitis), intervertebral discs (spondylodiscitis), facet joints (arthritis), ligaments, paraspinal soft tissues, epidural space (epidural

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phlegmon/abscesses), meninges, and subarachnoid space and the spinal cord itself (myelitis).[5,6]

The lumbar region was noted to be the site of involvement in more than half of the cases, which was considered to be the result of its rich blood supply and higher likelihood of endplate degeneration.[7] Thoracic vertebrae are involved in 19% of the cases.[8] Cervical vertebrae are rarely affected, but this involvement is more dangerous because of potentially life-threatening complications, such as paraplegia and tetraplegia, in 1% of cases.[9,10] Multilevel involvement of brucella spondylitis has been reported to occur in 6% to 36% of cases.[7,8,11,12]

The World Health Organization estimates the worldwide incidence of new brucellosis cases to be more than 500,000 per year.[13] Brucellosis is a common cause of vertebral osteomyelitis in geographic areas in which *B. melitensis* is endemic (ie, the Mediterranean basin, the Middle East, Latin America).[14]

MECHANISM

Brucellosis spreads hematogenously to tissues, and almost every organ can be affected. Microorganisms at a distant septic focus reach the spine by anterograde flow through the nutrient arterioles of the vertebral bodies or by retrograde flow through the paravertebral Batson venous plexus.[9,15]

Brucella spondylitis usually begins in the superior endplate because of its rich blood supply, and causes bone destruction even in the early stages. Occasionally the inferior endplate may also be involved. The infection spreads to the remainder of the vertebral body along the medullary spaces. With the inflammatory process, the process of bone healing begins almost simultaneously, and frequently spills over in the form of anterior osteophyte, like "parrot's beak." Initially discs are spared, as they are in tuberculosis spondylitis. In the later stage, brucella spondylitis extends to the adjacent vertebrae through the intervertebral disc space. The intervertebral disc is involved as a secondary process.[7,11]

Microorganisms may leak into the CSF with the help of an inflammatory vasculitic process, resulting in spondylitis and meningitis occurring simultaneously during the acute stage masking one's symptoms over other's.[5,16] Epidural masses may accompany the whole pathologic process, sometimes causing compression of a nerve root or spinal cord, mimicking a herniated intervertebral disc.[7,17] Facet involvement (6%–35%), intramedullary infections (1.2%–35.0%), and psoas abscess (1.2%–50.0%) are rare findings.[2,11]

Intramedullary brucella infection or abscess is rare.[18] The organism may act directly or indirectly through its endotoxins. The spinal cord or nerve root may be secondarily involved because of spondylitis, vasculitis, and arachnoiditis.[19]

Immune-mediated demyelination has been proposed to explain certain chronic forms of neurobrucellosis.[20] Impaired immune status is believed to be a risk factor for developing neurobrucellosis. Nervous system involvement in brucellosis might be from the persisting intracellular microorganisms or, perhaps, the infection triggering an immune mechanism, leading to neuropathology.[21] In an experimental animal model, the ganglioside-like molecules expressed on the surface of *B. melitensis* were found to induce antiganglioside membrane 1 ganglioside antibodies, resulting in flaccid limb weakness and ataxia-like symptoms.[22]

Infection triggers the immune-allergic mechanism, leading to myelopathy and/or a demyelinating state.[23] The occurrence of inflammatory central and peripheral demyelination as the pathologic manifestation of some types of neurobrucellosis, associated with inflammatory perivenular infiltration but not with histologically demonstrable organisms, strongly suggests that autoimmune mechanisms play a role in some types of neurobrucellosis.[24] This change in the spinal cord is virtually identical to that found in transverse myelitis or acute disseminated encephalomyelitis (ADEM), whereas the change that may be found in spinal roots may be identical to that found in acute inflammatory polyneuritis associated with Guillain-Barré syndrome. In these types of chronic neurobrucellosis cases, axons tend to be spared, although in severe cases, as in severe ADEM or Guillain-Barré syndrome, considerable axonal loss and associated peripheral neuroaxonal dissolution are seen.[24] It is also possible that the occurrence of a hyperergic immune response after latency from a bout of acute brucellosis is not caused by the slowed autoimmune response during a phase of chronic infection, but rather reinfection. Chronic inflammatory changes may be found in the perineurium in patients with neurobrucellosis, and adhesive arachnoiditis may develop in the subarachnoid space. Perineural inflammation may lead to dysfunction of peripheral nerves, with resulting radiculitis syndromes, especially if these inflammatory changes progress to the point of granuloma formation.[24] Recurrence of myelitis has also been reported.[25]