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Case Report

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Long-head biceps tendon rupture as a complication of brucellar tenosynovitis: a rare manifestation of multifocal osteoarticular brucellosis

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ABSTRACT

Brucellosis is a major zoonotic infection in endemic regions and can cause focal osteoarticular complications. Spinal involvement and peripheral septic arthritis are recognised manifestations; however, glenohumeral septic arthritis complicated by long-head biceps (LHB) infective tenosynovitis and tendon rupture is exceedingly rare. A 69-year-old immunocompetent man presented with severe low back pain and right shoulder pain without fever. Initial imaging suggested degenerative lumbar disease and the patient was treated conservatively. One week later, he returned with fever, night sweats, anorexia, worsening bilateral sciatica, and markedly restricted shoulder motion. Inflammatory markers were significantly elevated (CRP 121 mg/l; ESR 74 mm/h), and aerobic blood culture grew Brucella spp., with Brucella serology titre of 1:320. He subsequently reported raw camel milk consumption one month prior. MRI demonstrated L5-S1 spondylodiscitis, right glenohumeral joint effusion with synovial thickening, loculated fluid within the LHB tendon sheath, and complete proximal-third LHB tendon rupture. Lumbosacral MRI confirmed spondylodiscitis without epidural abscess or neurological compromise. Spine management was conservative. Arthroscopic shoulder debridement confirmed infective synovitis, yellowish intra-articular fluid, and complete LHB rupture. The patient received intravenous gentamicin, ceftriaxone, oral doxycycline, and rifampicin. At two months, shoulder pain had resolved, range of motion was almost full, back pain had improved, and CRP had normalised with no documented recurrence. This case represents a rare multifocal presentation of osteoarticular brucellosis and highlights the importance of early microbiological investigation in endemic regions when systemic symptoms coexist with severe musculoskeletal pain. Infective tenosynovitis should be considered a potential contributor to tendon rupture in patients with confirmed brucellosis. Multidisciplinary management is essential.

Keywords: Brucellosis, Brucella melitensis, Spondylodiscitis, Septic arthritis, Shoulder, Long-head biceps, Tenosynovitis, Tendon rupture, Camel milk

INTRODUCTION

Brucellosis is one of the most important zoonotic infections worldwide and remains a major public health problem in endemic regions, including the Middle East, Mediterranean basin, Latin America, and parts of Asia and Africa. It is caused by intracellular gram-negative coccobacilli of the genus Brucella, with B. melitensis, B. abortus, B. suis, and B. canis being the main species associated with human disease. Among these, B. melitensis is generally considered the most virulent and clinically significant species in humans.[1-3] Human

infection usually occurs through direct contact with infected animals or ingestion of unpasteurized animal products. Raw milk and dairy products from goats, sheep, cattle, and camels remain important transmission routes in endemic communities. In Oman and the wider Middle East, B. melitensis is an important cause of human brucellosis, and raw camel milk consumption remains a relevant cultural exposure, particularly in rural communities.[4-6] Osteoarticular involvement is the most common focal complication of brucellosis. The spine and sacroiliac joints are most frequently affected, whereas peripheral septic arthritis, bursitis, and tenosynovitis are less common. Shoulder involvement is unusual, and tendon-sheath infection complicated by tendon rupture is exceedingly rare.[7-10] We report a rare case of multifocal osteoarticular brucellosis presenting with simultaneous L5-S1 spondylodiscitis and right glenohumeral septic arthritis complicated by long-head biceps tenosynovitis and proximal long-head biceps tendon rupture. The case emphasizes diagnostic pitfalls, imaging findings, and the need for multidisciplinary management in endemic settings.

CASE REPORT

A 69-year-old man with no significant known comorbidities presented with severe low back pain and right shoulder pain. There was no history of trauma. On initial assessment, he was clinically stable, afebrile, and not septic.

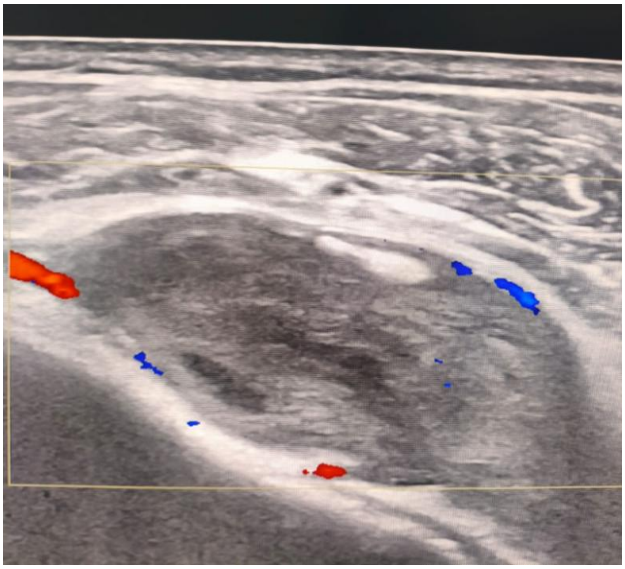

Figure 1: Doppler ultrasound of the right shoulder demonstrating a lobulated hypoechoic collection along the long-head biceps tendon sheath with peripheral vascularity, consistent with infective tenosynovitis.

Physical examination showed marked tenderness over the lower lumbar spine and painful but initially full range of motion of the right shoulder. No motor or sensory neurological deficit was detected. Plain radiographs showed lumbar spondylosis, and lumbar CT demonstrated advanced spondylodegenerative changes with reduced L5-S1 disc height. He was treated conservatively with physiotherapy and analgesics.

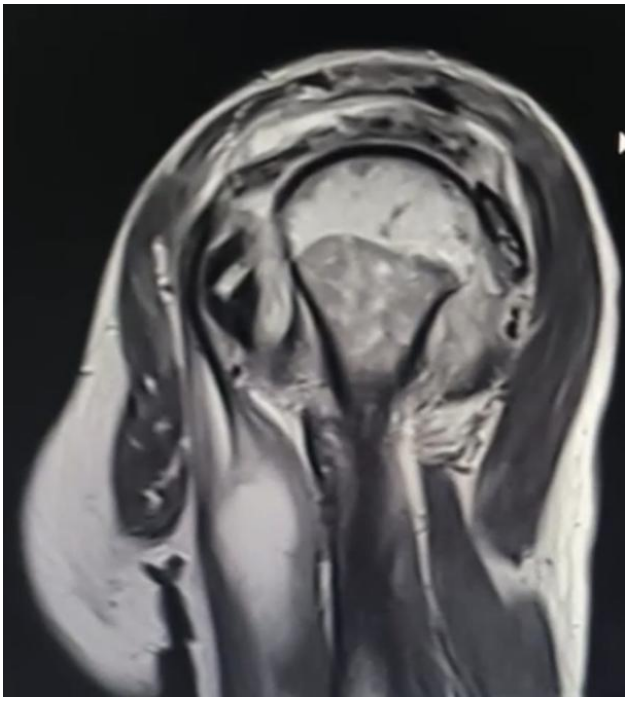

Figure 2: MRI of the right shoulder demonstrating glenohumeral joint effusion with synovial thickening and subchondral marrow signal abnormality of the humeral head, in keeping with septic arthritis.

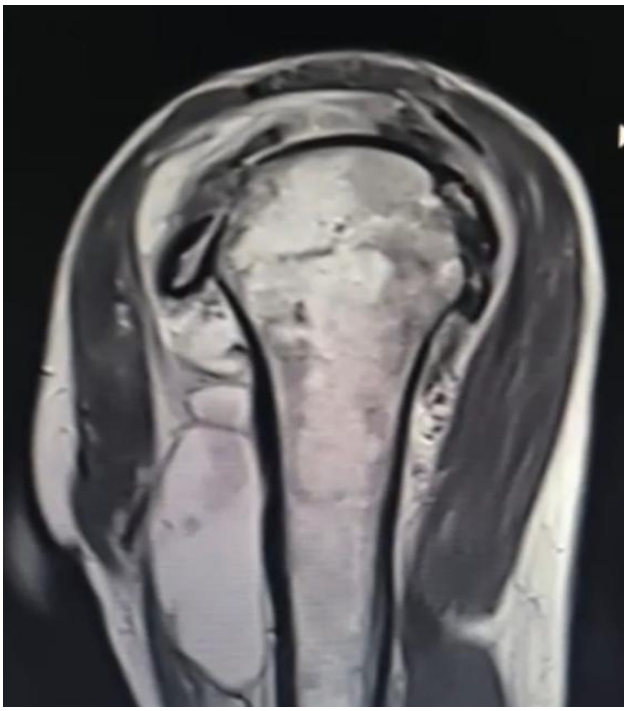

Figure 3: MRI of the right shoulder demonstrating complete proximal-third rupture of the long-head biceps tendon with surrounding fluid signal within the tendon sheath and glenohumeral joint effusion.

One week later, he returned with anorexia, fever, night sweats, severe constant pain unresponsive to treatment, restricted right shoulder motion, and newly developed bilateral sciatica. Neurological examination showed no motor or sensory deficit; however, the right ankle reflex was absent, and straight-leg raise testing was positive bilaterally at 20°. There were no bowel or urinary symptoms. Laboratory investigations showed mildly impaired renal function, ESR of 74 mm/h, and CRP of 121 mg/l.

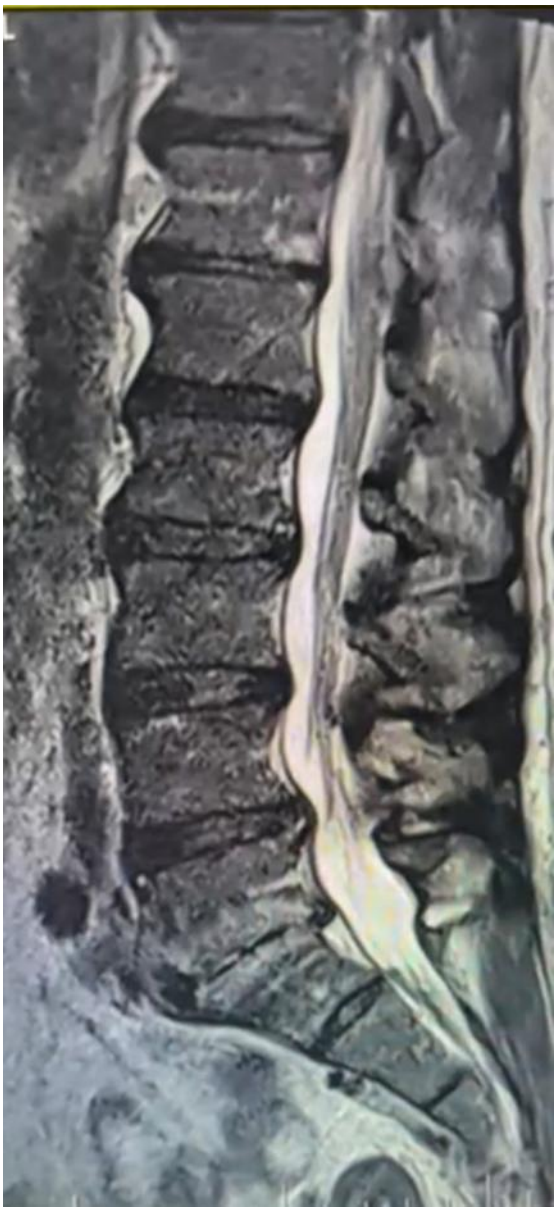

Figure 4: MRI of the lumbosacral spine demonstrating L5-S1 spondylodiscitis with disc-space fluid signal, adjacent end-plate marrow oedema, anterior paravertebral inflammatory fluid signal and severe disc-height loss.

Right shoulder ultrasound demonstrated a lobulated heterogeneously hypoechoic collection with internal hyperechoic debris anterior to the humerus along the long-head biceps tendon sheath, extending intra- and extra-articularly. There was associated turbid septated fluid tracking along the tendon sheath, minimal turbid glenohumeral joint effusion, thick irregular capsular thickening, and irregularity of the supraspinatus footprint with altered fibrillar pattern. Because infection was suspected, blood cultures were obtained, and empirical amoxicillin-clavulanate was started (Figure 1).

Two days later, aerobic blood culture grew B. melitensis, and Brucella serology titre was 1:320. After further discussion, the patient reported drinking raw camel milk in a rural area one month before presentation.

Urgent MRI of the right shoulder and lumbosacral spine was performed. Shoulder MRI showed moderate glenohumeral joint effusion, synovial thickening, articular cartilage thinning, subchondral marrow signal abnormality of the humeral head, a severely thinned and frayed glenoid labrum, and an anterosuperior labral tear involving the biceps-labral complex remnant. Loculated septated fluid was seen within the long-head biceps tendon sheath, associated with complete proximal-third rupture of the long-head biceps tendon. Severe supraspinatus tendinopathy with a partial-thickness tear was also noted (Figures 2 and 3).

Lumbosacral MRI demonstrated abnormal fluid signal within the L5-S1 disc, adjacent end-plate marrow oedema, anterior paravertebral inflammatory fluid signal, severe L5-S1 disc-height loss, and posterior disc bulge causing moderate bilateral foraminal narrowing with nerve-root impingement (Figure 4). There was no epidural abscess, paraspinal abscess, psoas abscess, spinal canal compromise, or radiological spinal instability. These findings were consistent with L5-S1 spondylodiscitis in the context of confirmed systemic brucellosis.

Following discussion with the spine surgery team, conservative management was chosen for the spondylodiscitis because there was no objective neurological deficit, spinal instability, or abscess requiring drainage. Arthroscopic debridement of the right shoulder was subsequently performed. Intraoperatively, a collection was found over the long-head biceps tendon, and 12 ml of yellowish fluid was aspirated and sent for culture and sensitivity. Diagnostic arthroscopy demonstrated synovitis, mild arthritic changes of the glenoid and humeral head, a frayed labrum, and complete rupture of the long-head biceps tendon with capsular retraction.

The patient was treated with intravenous gentamicin 360 mg once daily and ceftriaxone 2 g once daily for seven days, in addition to oral rifampicin 300 mg twice daily and doxycycline 100 mg twice daily. The aspirated shoulder fluid culture showed no growth after two weeks. The postoperative period was uneventful, with significant improvement in shoulder pain. He was discharged one week after surgery with oral antibiotics, regular follow-up,

and physiotherapy. Completion of antimicrobial therapy was transferred to an external infectious disease team.

At two months after surgery, he was pain-free in the right shoulder, shoulder range of motion was almost full, back pain had improved, CRP had normalized, and no recurrence was documented.

DISCUSSION

Osteoarticular disease is the most frequent focal complication of brucellosis, with reported prevalence varying across studies depending on region, case definition, and diagnostic method. A systematic review found that patients with brucellosis have at least a 27% risk of osteoarticular involvement, while other reviews report broader ranges in endemic settings.[8,9] The present case illustrates a common diagnostic pitfall: the patient initially presented with low back pain and imaging evidence of degenerative lumbar disease, which delayed recognition of an infective process. This is particularly relevant in older patients, where spondylosis and disc-height loss may coexist with early infectious spondylodiscitis.[10]

Brucellar spondylodiscitis is one of the most serious skeletal manifestations of brucellosis and commonly affects the lumbar spine. It may mimic pyogenic or tuberculous spondylodiscitis clinically and radiologically. MRI is the imaging modality of choice because it can detect disc-space fluid, end-plate oedema, paravertebral inflammation, epidural extension, and abscess formation.[11-13] In this patient, MRI demonstrated L5-S1 disc fluid signal, adjacent end-plate marrow oedema, anterior paravertebral inflammatory fluid signal, and severe disc-height loss, supporting the diagnosis of brucellar spondylodiscitis in the context of positive blood culture, positive serology, and compatible exposure history.

Conservative management is appropriate in brucellar spondylodiscitis when there is no progressive neurological deficit, spinal instability, or abscess requiring drainage.[11] In this case, the spine team opted for non-operative management because there was no motor or sensory deficit, no epidural or paraspinal abscess, no significant spinal canal compromise, and no radiological instability. The patient's back pain improved with antimicrobial therapy and follow-up care.

Peripheral septic arthritis is a recognized but less common manifestation of brucellosis compared with axial skeletal disease. Large joints such as the knee, hip, and ankle are more frequently involved, while glenohumeral involvement is uncommon.[14-16] Shoulder brucellosis may be misdiagnosed as degenerative rotator cuff disease, bursitis, adhesive capsulitis, or non-specific inflammatory arthritis, particularly in elderly patients with coexisting degenerative shoulder pathology. In this case, the shoulder findings were clinically significant because the patient had severe pain, restricted motion, ultrasound evidence of septated fluid, MRI evidence of glenohumeral effusion and synovial thickening, tendon-sheath fluid around the long-head biceps tendon, and operative confirmation of yellowish fluid and synovitis. Although shoulder fluid culture was negative, the diagnosis of presumed brucellar glenohumeral septic arthritis and long-head biceps infective tenosynovitis was supported by positive blood culture, positive Brucella serology, raw camel milk exposure, compatible imaging, intraoperative findings, and clinical response to brucellosis-directed therapy.

Tenosynovitis is a rare manifestation of brucellosis. Published reports mainly describe tendon-sheath infection in the hand and wrist, with very limited literature describing larger tendon sheaths or tendon rupture.[14-18] The most distinctive feature in this case is involvement of the long-head biceps tendon sheath with complete proximal-third tendon rupture, directly confirmed arthroscopically.

The relationship between infection and tendon rupture warrants cautious interpretation. In a 69-year-old patient, proximal long-head biceps rupture may occur due to chronic degenerative tendinopathy, commonly associated with rotator cuff disease, labral degeneration, and age-related tendon attrition. However, Brucella infection likely contributed through infective synovitis within the tendon sheath, inflammatory degradation of tendon collagen, increased local pressure, and mechanical attrition within the inflamed bicipital groove.[18] Because histopathological analysis of the tendon and synovium was not performed, direct causation cannot be proven. The most accurate interpretation is that Brucella infection likely contributed to proximal long-head biceps tendon rupture through infective tenosynovitis superimposed on pre-existing degenerative shoulder disease.

The absence of histopathological analysis of the resected synovium and long-head biceps tendon stump represents the most significant limitation of this case. Tissue-level confirmation of Brucella invasion of the tendon sheath was not obtained, as synovial and tendon biopsy were not part of the routine arthroscopic protocol at our institution. Consequently, a definitive causal link between Brucella infective tenosynovitis and tendon rupture cannot be established with certainty, and the contribution of pre-existing degenerative tendinopathy well documented on preoperative MRI cannot be excluded. However, the diagnosis of brucellar glenohumeral septic arthritis and long-head biceps infective tenosynovitis rests on the convergence of six independent criteria: positive aerobic blood culture for Brucella spp.; Brucella serology titre of 1:320, consistent with active systemic infection; epidemiologically relevant exposure through raw camel milk; MRI evidence of glenohumeral joint effusion, synovial thickening, and loculated long-head biceps tendon sheath fluid; intraoperative confirmation of synovitis and turbid intra-articular fluid on arthroscopy; and favourable clinical and biochemical response to brucellosis-directed antimicrobial therapy. This integrative diagnostic approach is consistent with established

frameworks for focal brucellosis, in which tissue culture sensitivity from joint fluid is reported as low as 4.5%, and histopathological confirmation is frequently absent in published case reports of brucellar tenosynovitis and septic arthritis. Furthermore, the mechanism of tendon rupture in this context is biologically plausible: infective tenosynovitis within the bicipital groove likely promoted mechanical friction and increased local compartment pressure, while infection-driven pro-inflammatory cytokines including interleukin-1β, tumour necrosis factor-α, and matrix metalloproteinases accelerated degradation of type I tendon collagen. This inflammatory collagenolytic mechanism, superimposed on a substrate of pre-existing degenerative tendinopathy, represents the most probable explanation for the complete proximal rupture observed. This "two-hit" model degenerative attrition compounded by infective collagenolysis best reconciles the clinical, imaging, and operative findings in this case.[19]

The management strategy was clinically reasonable and individualized. The spinal disease was treated conservatively because there was no neurological deficit, instability, or drainable abscess. By contrast, the shoulder had loculated fluid, synovitis, severe pain, restricted motion, and functional limitation, making arthroscopic washout and debridement appropriate. Arthroscopic management of septic shoulder arthritis allows lavage, drainage, debridement, microbiological sampling, and assessment of intra-articular pathology while limiting surgical morbidity.[20]

The negative shoulder aspirate culture is an important diagnostic limitation but does not exclude brucellar septic arthritis. Brucella is a fastidious organism, and culture yield may be limited, particularly after antibiotics have already been administered. In this patient, empirical antibiotics were started before shoulder aspiration, which likely reduced culture sensitivity. Culture-negative focal infection is a recognized challenge in brucellosis, and diagnosis often depends on integrating exposure history, serology, blood culture, imaging, operative findings, and treatment response rather than relying solely on isolation from the affected site.[7]

The antibiotic regimen for brucellosis typically uses a doxycycline and rifampicin backbone, often supplemented by an aminoglycoside such as streptomycin or gentamicin in complicated disease.[21,22] Osteoarticular brucellosis and spondylodiscitis generally require prolonged treatment to reduce relapse risk. In this case, gentamicin and ceftriaxone were used during the inpatient period, while doxycycline and rifampicin were continued orally after discharge. Ceftriaxone was used as empirical adjunctive coverage for suspected septic arthritis while awaiting microbiological clarification, rather than as a primary anti-brucellosis agent. The total duration of therapy was completed under the care of an external infectious disease team. At two months, the patient showed clinical and biochemical improvement, with resolved shoulder pain, almost full shoulder range of motion, improved back pain, normalized CRP, and no recurrence.

This case has several limitations. First, the shoulder fluid culture was negative, most likely because antibiotics had already been given before aspiration. Second, histopathological assessment of the tendon and synovium was not performed, so direct proof of Brucella invasion of the tendon sheath or tendon tissue was not available. Third, antimicrobial treatment completion occurred under an external infectious disease team, limiting detailed reporting of total treatment duration. Despite these limitations, the combination of positive blood culture, positive serology, raw camel milk exposure, multifocal MRI findings, operative evidence of shoulder infection, and favourable response to brucellosis-directed treatment supports the final diagnosis.

CONCLUSION

This case demonstrates a rare and clinically important presentation of multifocal osteoarticular brucellosis involving simultaneous L5-S1 spondylodiscitis, presumed culture-negative glenohumeral septic arthritis, long-head biceps infective tenosynovitis, and proximal long-head biceps tendon rupture after raw camel milk exposure. It emphasizes the need to consider brucellosis in endemic regions when severe back or shoulder pain is associated with fever, night sweats, elevated inflammatory markers, and raw milk exposure. MRI is essential for defining multifocal disease, and management should be individualized through multidisciplinary discussion. The contribution of infection to biceps tendon rupture is biologically plausible but cannot be proven without tissue histopathology.

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