International Journal of Preventive, Curative & Community Medicine
Volume 12, Issue 1 - 2026, Pg. No. 1-4
Peer Reviewed & Open Access Journal

Case Study

Dual Infection Of Brucellosis And Abdominal Tuberculosis: An Unusual Cause Of Febrile Jaundice

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DOI: https://doi.org/10.24321/2454.325X.202605

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How to cite this article:
Chakravartty D, Dhania S, Tiwari P K, Khushboo, "Dual Infection of Brucellosis and Abdominal Tuberculosis: An Unusual Cause of Febrile Jaundice Int J Preven Curat Comm Med. 2026;12(1):1-4.

Date of Submission: 2025-07-17
Date of Acceptance: 2025-08-25

A B S T R A C T

Introduction: Brucellosis and tuberculosis (TB) are important endemic infections in developing countries. Both conditions can present with overlapping systemic and abdominal features, making differentiation challenging. Clinically significant hepatic involvement with jaundice is rare in brucellosis, while abdominal TB often mimics other chronic infections. Co-infection of brucellosis and TB in a single patient is exceedingly rare and may lead to delayed or inappropriate treatment.

Case Presentation: We report a 22-year-old male with type 2 diabetes mellitus who presented with high-grade fever for 15 days, jaundice for 7 days, right upper abdominal pain, vomiting, and watery diarrhea. Examination revealed icterus and hepatomegaly with right hypochondrial tenderness. Investigations showed thrombocytopenia (platelets 60,000/µl), elevated bilirubin (5.4 mg/dl), transaminitis (SGOT 383 IU/L, SGPT 266 IU/L), and elevated ALP and GGT. Brucella IgM serology was positive, while viral hepatitis, leptospirosis, and scrub typhus serologies were negative. Imaging demonstrated hepatosplenomegaly, bowel wall thickening, enlarged mesenteric lymph nodes, and ascites, highly suggestive of abdominal TB.

Conclusion: This case emphasizes the diagnostic dilemma posed by overlapping features of brucellosis and TB. In endemic areas, physicians must consider co-infection in atypical presentations such as febrile jaundice. Rational therapy is essential to prevent mismanagement and reduce the risk of drug-resistant tuberculosis.

Keywords: Brucellosis, Abdominal Tuberculosis, Chronic Infections, Hypochondrial Tenderness, Leptospirosis

Introduction

Brucellosis is a zoonotic infection caused by Brucella species, often acquired through consumption of unpasteurized dairy products or exposure to infected animals. Its clinical spectrum is wide-ranging—patients commonly experience undulant fevers, weight loss, hepatosplenomegaly, and nonspecific gastrointestinal symptoms—but overt jaundice is quite rare, largely subclinical in most cases. In fact, clinically significant hepatic involvement in brucellosis occurs in only 1–3% of cases, with frank jaundice being notably uncommon[1,2]

Abdominal or extrapulmonary tuberculosis (TB), particularly involving the gastrointestinal tract or lymph nodes within the abdomen, also presents a diagnostic challenge. Its manifestations—such as fever, weight loss, abdominal pain, lymphadenopathy, and mesenteric changes—are often indistinguishable from other chronic infections.

Importantly, both brucellosis and TB share considerable overlap in clinical presentations and laboratory findings, including fever, hepatosplenomegaly, lymphadenopathy, and elevated inflammatory markers.[3,4] Nevertheless, their co-infection in a single patient remains exceedingly rare. Few case reports exist of this unusual dual involvement, and the potential for misdiagnosis or delayed treatment is significant.[5]

We report the case of a 22-year-old diabetic male presenting with high-grade fever, jaundice, and right upper quadrant tenderness, ultimately diagnosed with simultaneous brucellosis and abdominal tuberculosis based on serological and radiological data. This case underscores the diagnostic complexity and importance of considering atypical co-infections in patients with febrile jaundice, particularly in regions where both diseases are endemic.

Case Presentation

A 22-year-old male was admitted to the Department of Medicine with complaints of high-grade fever for 15 days, jaundice for 7 days, right upper abdominal pain for 7 days, one episode of vomiting 5 days prior to admission, and watery, non-bloody loose stools 6–7 times daily for 4 days. The fever was documented between 102–104 °F, continuous in nature. The patient also reported loss of appetite, subjective weight loss, nausea, and dryness of mouth.

He was a known case of type 2 diabetes mellitus for 7 years, managed with vildagliptin (50 mg twice daily), gliclazide (60 mg once daily), and metformin (500 mg twice daily). His last HbA1c was 6.5%. C-peptide was >1.5 ng/ml and anti-GAD antibody was negative, consistent with early T2DM. He was also a known asthmatic, using a budesonide/formoterol inhaler as needed. There was no past history of tuberculosis, other chronic illness, or previous surgery. Family history of tuberculosis was absent. Before admission, he had received two courses of oral antibiotics (cefuroxime-clavulanate and cefditoren pivoxil) without improvement.

On examination, he was icteric and had right hypochondrial tenderness. The liver was palpable 2 cm below the costal margin, soft in consistency. Other systemic examination was unremarkable.

Laboratory evaluation revealed hemoglobin 13.4 g/dl, total leukocyte count 6600/µl (neutrophils 72%, lymphocytes 25%, eosinophils 3%), and thrombocytopenia (platelets 60,000/µl). Blood urea was 47 mg/dl and serum creatinine 1.3 mg/dl. Liver function tests showed total bilirubin 5.4 mg/dl (direct 4.0, indirect 1.4), SGOT 383 IU/L, SGPT 266 IU/L, ALP 500 IU/L, and GGT 704 IU/L. Serum proteins were reduced (total protein 5.6 g/dl, albumin 3.08 g/dl, globulin 2.52 g/dl).

Serological investigations showed positive Brucella IgM (1.88), while IgG was negative. Scrub typhus IgM, leptospira IgM, hepatitis A and E IgM were all negative. Blood and urine cultures yielded no growth.

Ultrasonography of the abdomen revealed hepatomegaly with altered echotexture, diffuse bowel wall thickening, hypoechoic mesenteric lymph nodes (largest short-axis diameter 11 mm), minimal interbowel free fluid, and mild bilateral pleural effusion. Contrast-enhanced CT (CECT) of the abdomen demonstrated hepatomegaly (span 175 mm) with diffuse fatty changes, splenomegaly (span 139 mm), circumferential thickening of the caecum, ileocecal junction, and terminal ileum (maximum 14.5 mm), multiple enlarged mesenteric and retroperitoneal lymph nodes (largest right iliac node 35×20 mm), mild ascites, and bilateral basal consolidation with pleural effusion. These findings were highly suggestive of an infective etiology, most likely abdominal tuberculosis.

Although colonoscopy with biopsy was advised for confirmation, the patient refused invasive procedures. Based on clinical, serological, and radiological findings, a provisional diagnosis of dual infection: brucellosis with abdominal tuberculosis presenting as febrile jaundice was made.

He was initiated on modified antitubercular therapy consisting of intramuscular streptomycin (1 g daily), intravenous moxifloxacin (100 mg daily), and oral ethambutol (800 mg daily), along with intravenous doxycycline (100 mg twice daily). Supportive therapy included insulin infusion, antipyretics, analgesics, and intravenous fluids. The patient's fever subsided by day 8 of doxycycline and day 6 of modified ATT.

He was discharged afebrile and clinically stable after 36 hours of sustained apyrexia. On follow-up, doxycycline was continued for a total of 14 days. By day 25 of modified ATT, liver function tests showed marked improvement (total bilirubin 1.83 mg/dl, SGOT 76 IU/L, SGPT 37 IU/L, ALP 440 IU/L), and repeat Brucella IgM ELISA had become negative. Modified ATT was continued, with plans to add standard drugs as per National TB Elimination Programme (NTEP) guidelines.

Discussion

The diagnosis of brucellosis requires isolation of the organism in blood or bone marrow cultures.[6] or demonstration of serological evidence using the standard tube agglutination test or ELISA.[7,8] However, such techniques are frequently unavailable in many developing regions, resulting in underreporting of cases.[9] In our case, the diagnosis was made

ISSN: 2454-325X
DOI: https://doi.org/10.24321/2454.325X.202605

on the basis of positive Brucella IgM serology (1.88) along with compatible clinical features and exclusion of other infectious etiologies such as scrub typhus, leptospirosis, and viral hepatitis.

Both brucellosis and abdominal tuberculosis share overlapping clinical features such as prolonged fever, abdominal pain, weight loss, hepatosplenomegaly, and lymphadenopathy.[3] In our patient, febrile jaundice, hepatomegaly, bowel wall thickening, mesenteric lymphadenopathy, and splenomegaly were initially suggestive of abdominal tuberculosis on radiological imaging. This diagnostic overlap increases the risk of misdiagnosis, with patients of brucellosis potentially being treated as tuberculosis and vice versa.[9] Several antitubercular agents, including rifampicin and streptomycin, also possess excellent activity against Brucella species.[10] This therapeutic overlap has important implications. On one hand, undetected brucellosis may improve with standard antitubercular therapy, while on the other, patients with undiagnosed tuberculosis who are treated as brucellosis may receive inadequate doses or incomplete therapy, predisposing them to the development of drug-resistant TB strains.

In our patient, modified ATT (streptomycin, moxifloxacin, ethambutol) was initiated along with doxycycline, targeting both infections simultaneously. The patient responded with resolution of fever by day 8 of doxycycline and day 6 of ATT, with significant improvement in liver function by day 25. This clinical response highlights the benefit of a regimen with dual activity in suspected co-infections.

The emergence of multidrug-resistant (MDR) tuberculosis poses a major global threat, with an estimated 440,000 new cases annually, nearly half of which occur in India and China.[11] The economic burden of MDR-TB is substantial, requiring prolonged and expensive therapy with second-line agents.[12] The principal driver of MDR strains is selective antibiotic pressure caused by inadequate or inappropriate therapy.[13–15]

In endemic areas where tuberculosis and brucellosis co-exist, empirical treatment of undifferentiated febrile illnesses with rifampicin or streptomycin can inadvertently contribute to resistance. Our case underlines this concern, since the patient had already received two prior empirical antibiotic courses without improvement before admission. Physicians must therefore exercise caution in prescribing overlapping regimens without definitive diagnosis.

The diagnostic dilemma posed by tuberculosis is not unique to brucellosis. Several studies have reported similar overlap between tuberculosis and melioidosis.[16] or histoplasmosis.[17–19] However, unlike these infections, brucellosis treatment itself relies heavily on rifampicin and streptomycin—drugs that are also first-line agents for TB.[10] The WHO-recommended oral regimen for brucellosis consists of doxycycline 200 mg and rifampicin 600 mg daily for at least 6 weeks, while the alternate oral–parenteral regimen uses doxycycline combined with parenteral streptomycin for 14–21 days.[20,21] In regions with high TB prevalence, indiscriminate use of these regimens without ruling out concomitant tuberculosis can have disastrous implications for resistance control.

Our patient illustrates the degree of overlap between brucellosis and abdominal tuberculosis in terms of presentation and laboratory features. He presented with high-grade fever, jaundice, hepatosplenomegaly, bowel wall thickening, mesenteric lymphadenopathy, and thrombocytopenia, mimicking abdominal tuberculosis. Serological evidence of brucellosis alongside radiological findings consistent with tuberculosis established the diagnosis of dual infection. Clinical improvement with a modified ATT regimen combined with doxycycline highlights the importance of considering atypical co-infections in endemic areas.

Conclusion

This case underscores the diagnostic challenge of differentiating brucellosis from abdominal tuberculosis due to overlapping clinical and laboratory features. Our patient presented with febrile jaundice and abdominal findings, ultimately diagnosed with dual infection based on serology and imaging, and improved with combined modified antitubercular therapy and doxycycline. In endemic regions, clinicians should consider co-infection in atypical presentations and avoid empirical use of overlapping drugs without confirmation, to ensure appropriate management and reduce the risk of drug resistance.

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ISSN: 2454-325X
DOI: https://doi.org/10.24321/2454.325X.202605