Case Series

Brucellosis with hepatobiliary and pulmonary involvement: A case series of similar clinical patterns from a resource-limited setting

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Abstract

Brucellosis is a zoonotic disease with diverse and often misleading clinical presentations, including hepatobiliary and, less commonly, pulmonary involvement. In this case series, we discuss three patients presenting with complex hepatobiliary infections and pulmonary symptoms, initially misdiagnosed and treated as acute abdomen. Each patient exhibited a history of contact with cattle, abdominal pain, jaundice and systemic symptoms and required prolonged antibiotic courses due to persistent infection with diagnostic dilemma and slow clinical improvement. Diagnosis was a challenge due to negative conventional cultures and serology, necessitating empirical treatment based on clinical suspicion. In endemic regions like Sri Lanka, the lack of advanced diagnostics and the overlapping symptoms of brucellosis with other diseases mandate the need for increased clinical awareness and clinical pattern recognition, especially in resource-limited settings. These cases illustrate the importance of considering brucellosis in patients with prolonged fever, hepatobiliary symptoms and atypical pulmonary involvement. Early recognition of the clinical imprint of the disease can improve outcomes and potentially avoid unnecessary surgical interventions. Prevention measures are necessary to eliminate the spread.

Keywords*: Brucellosis, Zoonoses, Hepatobiliary Diseases, Lung Diseases, Sri Lanka*

Introduction

Brucellosis is a zoonotic infection caused by the *Brucella* species, primarily affecting livestock such as cattle, goats, and sheep, and can be transmitted to humans through direct contact with infected animals or consumption of contaminated animal products. The disease is endemic in many parts of the world, including the Mediterranean, the Middle East, and South Asia. Brucellosis in humans can lead to nonspecific symptoms such as fever, malaise and arthralgia, which can make

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diagnosis challenging. Chronic cases may cause more severe complications including arthritis, endocarditis, and neurological disorders.[1,2]

This case series presents three patients with complex hepatobiliary infections with pulmonary involvement, each characterised by close contact with cattle, who presented with right-sided abdominal pain, jaundice, and right sided lung involvement. They also had systemic inflammatory symptoms including prolonged fever, and each presented to the surgical unit with acute abdomen.

Despite diverse backgrounds and initial diagnoses, all 3 patients required prolonged courses of multiple broad-spectrum antibiotics due to persistent infection and slow clinical improvement. Notably, in each case, conventional cultures and serology remained negative, complicating the diagnostic pathway and requiring ongoing empirical treatment.

Recognising the clinical imprint of the disease and doing early cultures may help to diagnose the condition and is of paramount importance in a resource poor setting like Sri Lanka where sophisticated investigations are not available most of the time.

Only a few cases of human brucellosis have been reported in Sri Lanka. This case series addresses concomitant hepatobiliary and pulmonary involvement of brucellosis for the first time in Sri Lanka.

Case reports

Case 1

A 54-year-old previously healthy male farmer who lives close to a goat farm (and occasionally drinks raw milk), presented to the surgical ward with a 10-day history of continuous dull right-sided abdominal pain, pale stools, intermittent fever, jaundice and worsening dyspnoea. He had initially been treated at a base hospital with intravenous (IV) meropenem and metronidazole for suspected biliary sepsis based on ultrasound (imaging findings of dilated gallbladder with wall oedema and mild ascites). Despite one week of treatment, his symptoms did not improve.

Upon admission to the surgical ward, he was managed as a case of biliary sepsis with acute-on-chronic cholecystitis. Due to worsening clinical parameters including tachycardia, tachypnea and persistent fever, he was referred to the medical ward for further evaluation. On examination, he had right-sided lower lobe crepitations and pleural effusion, jaundice without pallor, and mild hepatomegaly with a positive Murphy's sign.

A comprehensive blood workup (Tables 1A and 1B) and imaging (Figure 1) was initiated and key differentials considered, included pyogenic subdiaphragmatic abscess, hepatobiliary sepsis with cholangitis, granulomatous infections, brucellosis with complications and parasitic infections.

The patient was treated for nearly three weeks with IV meropenem, metronidazole and vancomycin, showing gradual clinical and biochemical improvement. Brucella antibody titres were done around the 3rd week of illness and results (SAT titre 1/320) received 1 week later. He

was discharged with a 10-day course of doxycycline in the 4th week of illness as brucellosis was considered the most likely cause. Six weeks after completion of treatment at follow up he had full clinical recovery with normalising inflammatory markers, and normal ultrasound abdomen and chest Xray. His case was managed with a multidisciplinary team approach with timely inputs and follow up.

Table 1A: Basic blood investigations

Investigation	Summary of results							
Haematology & inflammatory markers		**Normal Range**	**Day 1**	**Day 5**	**Day 10**	**Day 17**	**Day 24**	**Before Discharge**
	WBC	4.0–11.0 × 10^9/)	22.07 (Neutrophils 78%)	21		23.2		7.1
	Haemoglobin	(13.0–17.0 g/dL for males)	12.1	12.0		11.9		11.9
	Platelets	(150–400 × 10^9/L)	154	160		185		195
	CRP	(<5mg/L)	309	211	294	195	65	36
Liver Function Tests		**Normal Ranges**	**Day 1**		**Day 10**		**Before Discharge**	
	AST	(10–40 IU/L)	175		160		55	
	ALT	(7–56 IU/L)	74.8		80		48	
	Total bilirubin	(3–17 µmol/L)	164.68		135		51	
	Direct bilirubin	(<3.4 µmol/L)	123.8		111		31	
	Alkaline phosphatase	(44–147 IU/L)	722		580		160	
	Gamma-GT	(8–61 IU/L)	1203		880		107	
	Total protein & Albumin	(6.0–8.3 g/dL) & (3.5–5.0 g/dL)	6.5 & 3.2		6.2 & 3.1		6.1 & 3.3	
	PT/INR	0.8-1.2	1.59		1.3		0.9	
Renal Function & Electrolytes	**S. Creatinine**	62–106 µmol/L	67					
	Serum Electrolytes (Na+, K+, Ca2+, PO4)		Normal ranges noted					
	Blood Urea	10–50 mg/dL	27					

AST: aspartate amino-transferase; ALT: Alanine amino-transferase; Gamma-GT: Gamma glutamyl-transferase; PT/INR: Prothrombin time/International Normalized Ratio; CRP: C-Reactive Protein; Intravenous (IV)

Table 1B: Radiological and microbiological investigations

Investigation	Summary of results
CXR	Right-sided pleural effusion with consolidation
CECT Abdomen (Triple phase):	Infected right subdiaphragmatic collection, mild to moderate ascites, single gallbladder calculus without signs of acute cholecystitis, mild right-sided pleural effusion, and calcified granulomas in the spleen.
Pleural Fluid Analysis and cytology	Protein: 2.9, Albumin: 1.5, and LDH: 148, WBC: 4 (100% polymorphs), RBC: 1760. In cytology, predominantly lymphocytes (80%), with a benign inflammatory smear. ADA: 30.6.
Microbiology & Serology	Negative for TB (Mantoux, sputum AFB, GeneXpert) and other infectious agents (Mycoplasma, Rickettsia, Leptospirosis, melioidois). Brucella Serum Agglutination Test (SAT) Antibody titer elevated at 1:320. Multiple cultures (blood, urine, pleural fluid, subdiaphragmatic collection, sputum, ascitic fluid) were negative.

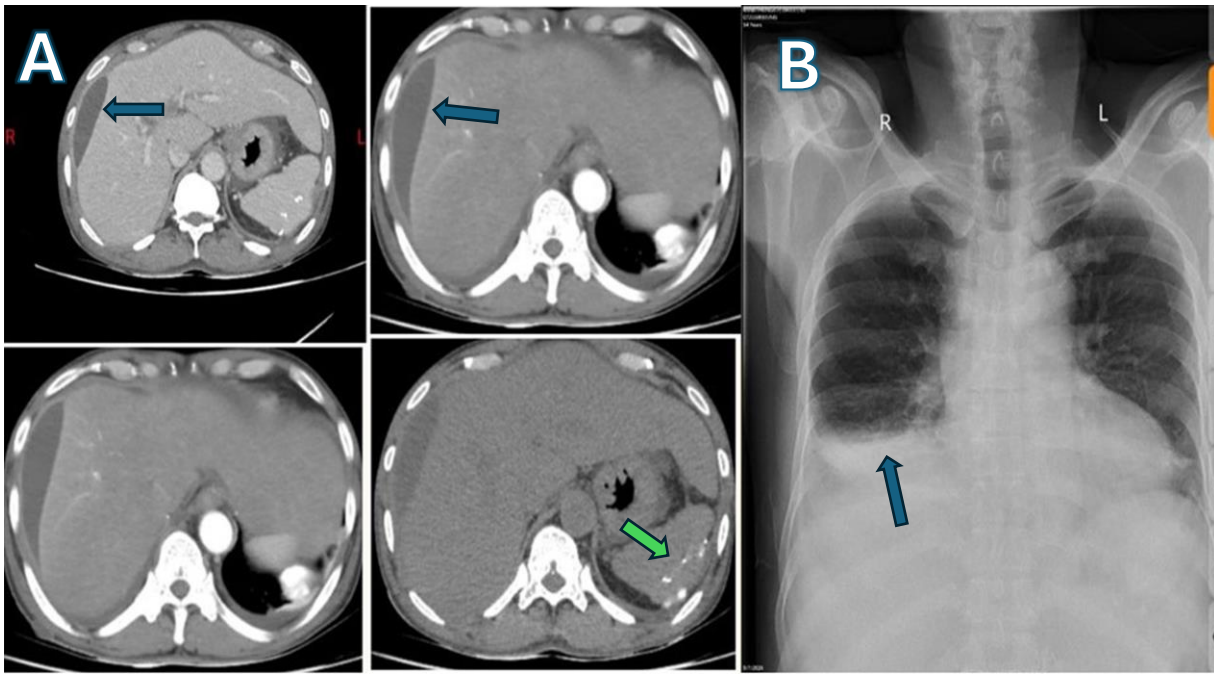

Figure 1:
A: CECT Abdomen: Right subdiaphragmatic fluid collection (blue arrows) and calcified granulomas in spleen (Green arrow)
B: Chest Xray: Right lower zone consolidation and pleural effusion (blue arrow)

The timeline of Case 1 is given below.

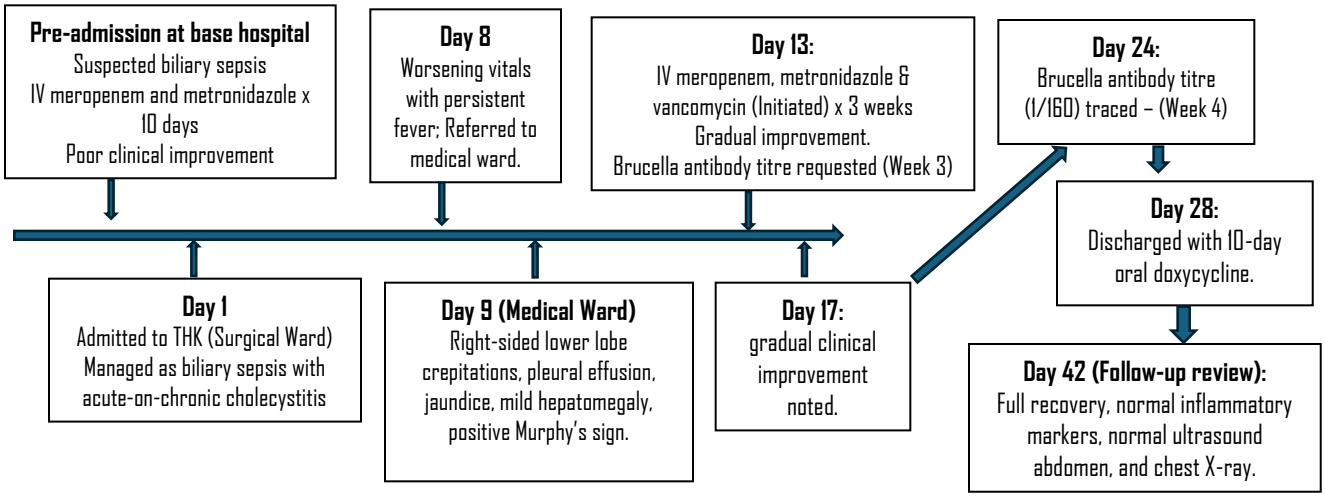

Figure 2: Case 1: Timeline of patient's progress

Case 2

A 64-year-old farmer with a history of diabetes and dyslipidemia who encounters his cows in his backyard daily and tastes raw milk occasionally while milking the cows, presented to the surgical ward with a 10-day history of intermittent right-sided abdominal pain, dark urine and pale stools, intermittent fever with chills and rigors, jaundice, constipation and several episodes of vomiting. He had initially been treated with IV ceftriaxone and metronidazole for suspected biliary sepsis based on ultrasound findings of multiple gallstones, gallbladder wall edema and mild ascites. After one week, his symptoms persisted, with static inflammatory markers, worsening tachypnea, and persistent fever, prompting a referral to the medical ward for further evaluation.

On examination, he had right-sided lower lobe crepitations and consolidation, jaundice without pallor, bilirubinuria, mild hepatomegaly, a negative Murphy's sign and tenderness in the right hypochondrium.

On workup (Tables 2A and 2B), elevated inflammatory markers, cholestasis and imaging findings were suggestive of biliary sepsis or complicated cholecystitis with a differential diagnosis of atypical pneumonia without current evidence of hepatitis or other specific infections. Empiric antibiotic therapy was adjusted to IV metronidazole, IV meropenem and a short course of clarithromycin after discontinuing ceftriaxone. After a 10-day antibiotic course, the patient experienced intermittent paroxysms of right hypochondrial pain and slowly improving jaundice for which the surgical team resumed his management again and performed laparoscopic cholecystectomy.

Postoperative findings showed a normal gallbladder with a few gallstones and bile-stained ascitic fluid, which was inconsistent with the clinical presentation. IV vancomycin was added for one-week post-surgery, and the patient discharged on a five-day course of cefuroxime. Much later after the surgery, brucella antibody titres were traced (SAT titre 1/320), with a retrospective diagnosis

of brucellosis. In the follow up review two weeks after discharge, the patient was asymptomatic and repeated inflammatory markers were normal.

Table 2A: Basic blood investigations

Investigations	Summary of Results			
Haematology & Inflammatory markers		Normal Ranges	Initial Value	On Discharge
	White Cell Count	(4.0–11.0 × 10^9/L)	14.37 (Neutrophil predominance)	6.5
	Hemoglobin	(13.0–17.0 g/dL for males)	12.6	12.5
	Platelets	(150–400 × 10^9/L)	210	267
	CRP	(<5mg/L)	340	61
Liver Function Tests	AST	(10–40 IU/L)	61.7	40
	ALT	(7–56 IU/L)	40.5	35
	Total bilirubin	(3–17 µmol/L)	136	27
	Direct bilirubin	(<3.4 µmol/L)	107.2	17
	Alkaline phosphatase	(44–147 IU/L)	255	160
	Gamma-GT	(8–61 IU/L)	367	65
	Total protein & Albumin	(6.0–8.3 g/dL) & (3.5–5.0 g/dL)	7.1 & 4.1	7.2 & 4.0
	PT/INR	0.8-1.2	1.2	1.0
Renal Function Electrolytes	S. Creatinine	62–106 µmol/L	98	100
	Serum Electrolytes (Na+, K+, Ca2+, PO4)	Normal Values noted		
	Blood Urea	10–50 mg/dL	18	
Urinalysis	Urinary bile: Positive, suggestive of cholestasis.			

AST: aspartate amino-transferase; ALT: Alanine amino-transferase;, Gamma-GT: Gamma glutamyl-transferase; PT/INR: Prothrombin time/International Normalized Ratio; CRP: C-Reactive Protein; Intravenous (IV)

Table 2B: Radiological and Microbiological Investigations

Investigations	Summary of Results
CXR	**Right-sided consolidation.**
CECT Abdomen (Triple phase):	Mild hepatomegaly, right-sided diaphragm elevation with associated consolidation, and mild bilateral pleural effusions. Findings of acute cholecystitis with a few gallbladder calculi, mild gallbladder wall edema, and moderate ascites.
Microbiology & Serology	Negative for Mycoplasma and Leptospirosis. Hepatitis viral panel: Negative. Blood and urine cultures: Negative. Brucella Serum Agglutination Test (SAT) antibody titers: I: 320

The timeline of Case 2 is given below

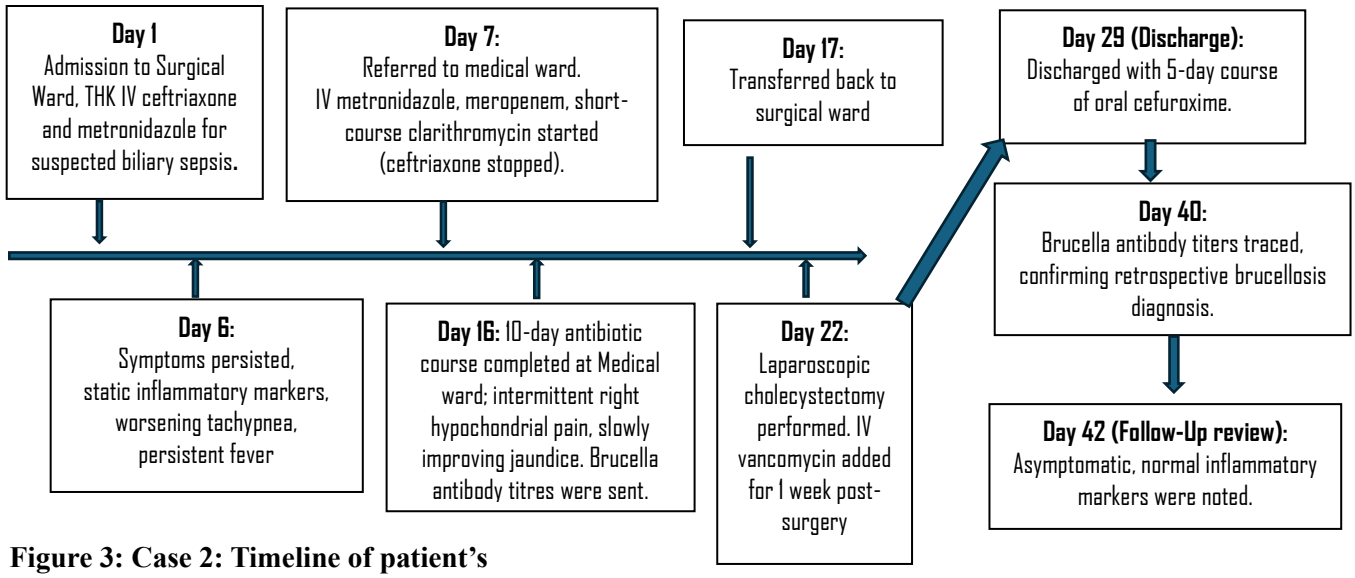

Figure 3: Case 2: Timeline of patient's

Case 3

A 35-year-old otherwise healthy South African forest monk living in the Mahiyangana forest ranges presented to the surgical ward with a 5-day history of continuous dull right-sided abdominal pain and fever with chills and rigors. He feeds animals and cattle every day but denied consumption of raw milk in his monastery. He had initially been treated for suspected cholangitis based on an ultrasound showing gallstones, mildly dilated common bile ducts and mild ascites. On the third day after admission, he developed right-sided pleuritic chest pain and was referred to our medical team for further evaluation. On examination, he had right-sided lower lobe crepitations, a tinge of jaundice without pallor, mild hepatomegaly, and a positive Murphy's sign.

A thorough workup (Tables 3A and 3B) was initiated and resulted in the differentials of hepatobiliary sepsis with cholangitis or an atypical infection. The patient received IV meropenem and metronidazole for nearly a week, showing gradual clinical improvement. However, with

ongoing pain and minimal improvement, he underwent a CECT scan which revealed a forming hepatic abscess and was referred for a gastrointestinal surgical opinion (Figure 4). IV gentamicin was added, which resolved his fever. He was then transitioned to oral cefixime and on the advice of the microbiology team, discharged on a two-week course of ciprofloxacin. Initial brucella antibody titres done at 3rd week (traced after the discharge) of illness was elevated at 1:160. Follow-up at the ward after two weeks of discharge was uneventful with full clinical improvement. The patient was asked to be reviewed with repeat antibody titres but unfortunately defaulted.

Table 3A : Basic blood investigations

Investigations	Summary of Results (Case 3)			
Haematology & Inflammatory Markers		**Normal Ranges**	**Initial Value**	**On Discharge**
	White Cell Count	(4.0–11.0 × 10^9/L)	17.2 (With Neutrophil Predominance)	5.4
	Haemoglobin	(13.0–17.0 g/dL for males)	13.2	13.0
	Platelets	(150–400 × 10^9/L)	37	167
	CRP	(<5mg/L)	327	47
Liver Function Tests				
	AST	(10–40 IU/L)	44.8	40
	ALT	(7–56 IU/L)	66.2	57
	Total bilirubin	(3–17 µmol/L)	38.8	21
	Direct bilirubin	(<3.4 µmol/L)	27.8	5.6
	Alkaline phosphatase	(44–147 IU/L)	164	122
	Gamma-GT	(8–61 IU/L)	145	65
	Total protein Albumin	(6.0–8.3 g/dL) (3.5–5.0 g/dL)	6.9 & 4.0	7.0 & 4.1
	PT/INR	0.8-1.2	1.1	1.0
Renal Function Electrolytes	**S. Creatinine**	62–106 µmol/L	102	94
	Serum Electrolytes (Na+, K+, Ca2+, PO4)		Normal range	
	Blood Urea	10–50 mg/dL	31	33

AST: aspartate amino-transferase; ALT: Alanine amino-transferase; Gamma-GT: Gamma glutamyl-transferase; PT/INR: Prothrombin time/International Normalized Ratio; CRP: C-Reactive Protein; Intravenous (IV)

Table 3B: Radiological and microbiological investigations

Investigations	Summary of Results (Case 3)
CXR	Few atelectatic shadows on the right lower lobe.
CECT Abdomen (Triple phase):	Mild hepatomegaly with early hepatic abscess formation, calculous cholecystitis, mild atelectasis in the right lower lung lobe, and prominent porta hepatis lymph nodes.
Microbiology & Serology	Negative for Mycoplasma, Rickettsial, and leptospirosis. Brucella Serum Agglutination Test (SAT) antibody titers elevated at 1:160. Blood, urine, and sputum cultures: Repeatedly negative.

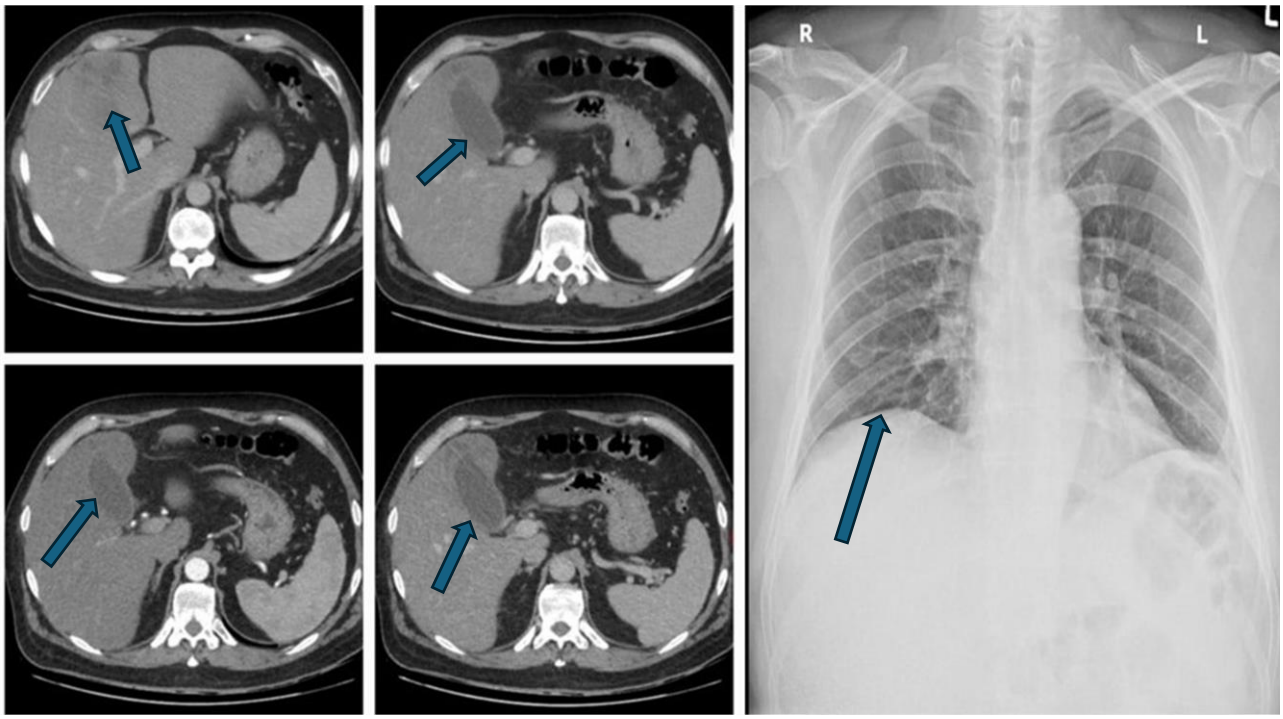

Figure 4

A: CECT Abdomen: Indicates early hepatic abscess formation (arrow)

B: Chest x-ray: Indicates few right lower zone atelectatic shadows (arrow)

The time line of Case 3 is given below.

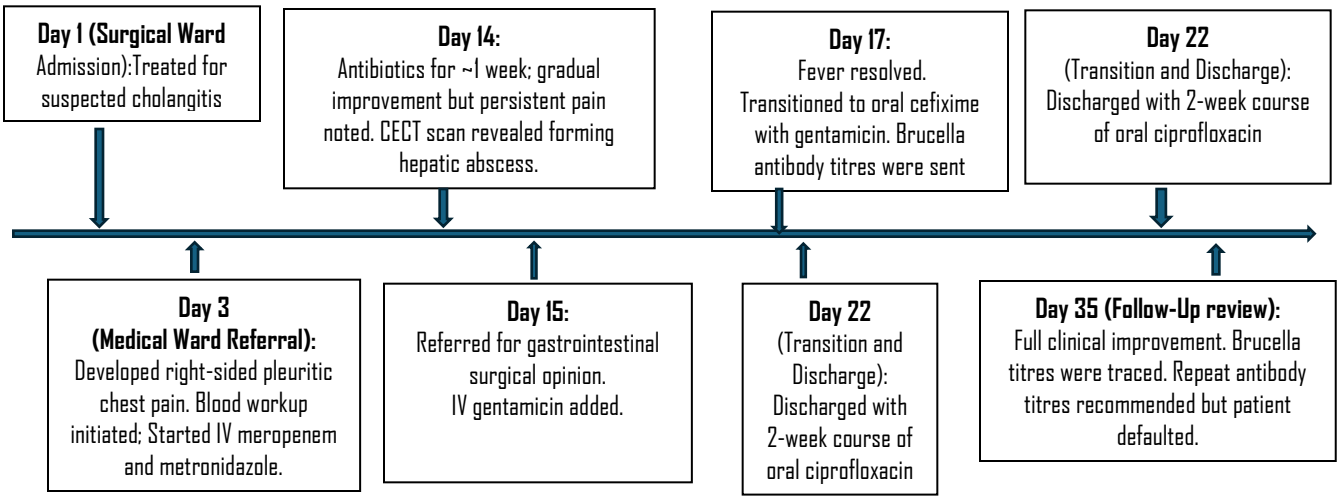

Figure 5: Case 3: Timeline of patient's progress

Discussion

Brucellosis is a zoonotic disease with diverse clinical manifestations, often leading to misdiagnosis. The disease can present in acute, sub-acute, or chronic forms, with most cases being acute. Laboratory findings often include anaemia, leukopaenia, thrombocytopaenia and elevated liver enzymes. Diagnosis relies on clinical presentation and laboratory tests, with serological tests-serum agglutination tests (SAT), immunofluorescence assays (IFA), and ELISA being preferred due to their simplicity and reliability. PCR techniques offer high sensitivity and specificity but may be too expensive for routine use in resource poor settings.[3] In each of our cases, SAT was done as it was the only available test in the state sector (and therefore without cost to the patient). However, as the investigation was requested late in the illness (around the 3rd week of illness), it is possible the antibody titre was in the declining phase even though it was still above the expected range. Though the definitive serologic diagnostic cut off titre has not been established locally, universally many authors consider a SAT titer of ≥1:160 to be indicative of active brucellosis. However, active brucellosis cannot be excluded in patients with SAT titers lower than 1:160.[5,6] Surveillance data of antibody titres in the cattle exposed population in Sri Lanka would be useful in establishing diagnostic titres.

Hepatobiliary involvement in brucellosis is not uncommon, with 10-20% of patients experiencing abnormal liver function tests. Presentations can range from mild to moderate enzyme elevations in chronic granulomatous hepatitis to rare cases of acute hepatitis and even rarely cholestasis. Liver granulomas, often indistinguishable from sarcoidosis, are a characteristic feature. Histologically, these granulomas may show central necrosis, portal and peripheral infiltration, and Kupffer cell hyperplasia.[4,7,8] Brucellosis can also manifest as intrahepatic cholestasis and may involve the bone marrow. Brucellosis can cause acute cholecystitis as a rare hepatobiliary manifestation.[7]

Diagnosis is typically based on blood cultures, serum agglutination titres, and imaging findings. In some cases, hepatobiliary brucellosis can present as a refractory infection, emphasising the

importance of considering this diagnosis in endemic regions.[6] Appropriate antibiotic treatment usually leads to clinical and biochemical improvement.

Pulmonary involvement in brucellosis is rare but may be more common than currently estimated. [8] In our cases, pulmonary involvement might have been due to direct involvement of lungs due to intermittent bacteraemia or due to reactive inflammatory lung involvement. Symptoms typically include fever, cough, and chest pain.[9] Pulmonary brucellosis may be associated with other systemic manifestations, including osteoarticular complications, liver enzyme elevation, and thrombocytopaenia.[10] In endemic regions, brucellosis should be considered in patients with prolonged fever, arthritis, and pulmonary symptoms, even in the absence of significant exposure, as it can mimic other conditions like systemic juvenile idiopathic arthritis.[8] Radiological findings can vary, including lobar consolidation, pleural effusion, and nodular lesions.[10] Diagnosis is confirmed through blood cultures or serology with sputum cultures being rarely positive. Traditional methods for isolation of *Brucella* spp. can be challenging. This is because the bacteria often cause mild ongoing infection in the blood (low-grade bacteraemia) requiring longer incubation times. Additionally, the bacteria may hide inside macrophages, leading to intermittent bacteraemia with initial clearing from the bloodstream. Another issue is that patients may have already taken antibiotics before testing, which can further complicate culture-based detection.[4]

To increase the detection rate; authors recommend taking blood for culture prior to starting antibiotics, inclusion of brucellosis in the differentials in the request form, performing serology in the earlier phase of illness, repeating cultures if clinical suspicion of brucellosis, collecting tissue samples also in sterile blood culture bottles and increasing awareness among clinicians.[13,14]

Treatment usually involves combination antibiotic therapy with good prognosis and rapid improvement.[10,11] The choice and duration of antibiotic therapy for brucellosis depends on the presence of focal disease and patient factors with doxycycline plus aminoglycosides or rifampicin for 6 weeks being the most common regimen.[~~10,13~~ 12,15] In the Sri Lankan context, treatment options for uncomplicated infections include doxycycline 100 mg twice daily for 45 days, combined with streptomycin 1 g daily for 15 days. An alternative regimen involves doxycycline for 45 days, paired with rifampicin for the same duration. While gentamicin may be used as a substitute for streptomycin, there is currently no direct study comparing these two regimens. The optimal treatment for pregnant women, neonates, and children under 8years remains uncertain. For children, treatment options may include co-trimoxazole in combination with an aminoglycoside (streptomycin or gentamicin) or rifampicin. [3] When complicated cases arise, special consideration of total duration of antibiotics, and types of antibiotics should be decided necessarily by experts (microbiologists) rather than physicians.

Prevention strategy of human brucellosis recommended by the World Health Organization (WHO) mainly consists of creating public awareness, improving hygienic practices in livestock farms and slaughterhouses, promoting the consumption of pasteurised milk, consumption of cooked meat, adhering to hygienic practices during laboratory handling of the organism and proper handling of companion animals. The recommended prevention and control methods of brucellosis in animals include culling of infected animals, vaccination and restriction of the movement of serologically positive animals.[~~3,4~~ 5,6]

Conclusion

Brucellosis can rarely present with acute cholecystitis or hepatic abscess or hepatobiliary sepsis along with pulmonary involvement. As demonstrated in this series, patients could present initially to surgical wards with typical acute abdomen. Increased awareness with a high index of suspicion is needed for early diagnosis as an early accurate diagnosis would help avoid unnecessary surgical interventions.

Declaration

Conflict of interest: The authors declare no conflicts of interest..
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Manuscript Drafting: Dr Risly NMM, Dr Sahana JF,
Critical Review and Final Approval: Dr Athauda N, Dr Jayasinghe IK, Dr Abeywardane M.

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