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Clinical Challenge

When the blood culture points one way, and the pattern points another: A diagnostic journey through sepsis, cholestasis, and a hidden abscess

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Introduction

The journal's Clinical Challenge section is designed to aid daily clinical practice by sharing real patient experiences. Each case is detailed with a step-by-step explanation, highlighting the rationale for investigative and management decisions and emphasising rational, cost-effective care in resource-limited healthcare environments.

In this issue, we present a case of Mr T, a 43-year-old hotel worker with poorly controlled diabetes mellitus who presented with fever followed by jaundice and lower urinary tract symptoms. Initially, the blood culture identified *Pseudomonas aeruginosa*, a familiar pathogen. However, the antimicrobial susceptibility pattern revealed a paradox that could not be explained by this bacterium. This case illustrates the diagnostic value of reading the full susceptibility report rather than the organism's name alone, the importance of recognising sepsis-induced cholestasis as a distinct clinical entity, and the need to pursue a focus of infection even when standard imaging and urine studies are reassuring

Case presentation: The initial encounter

Mr T, a 43-year-old married man employed in the housekeeping department of a hotel and residing in Bandaragama, a town in the Kalutara District of the Western Province of Sri Lanka, was admitted to our medical ward with a ten-day history of fever. The fever was intermittent and low-grade, accompanied by chills. Two days into the illness, he noticed dark-coloured urine and yellowish discolouration of his eyes, prompting him to seek medical attention.

The jaundice was painless, and there were no pale stools, pruritus, or biliary colic to suggest mechanical biliary obstruction. He reported dysuria and increased urinary frequency throughout the illness, although his urine output remained normal. There was no haematuria or symptoms of bladder outflow obstruction. He denied any respiratory, cardiovascular, or neurological symptoms. There were no joint pain, skin rashes, red or painful eyes, night sweats, or bleeding manifestations. He had no vomiting, altered bowel habits, or gastrointestinal upset. There was no significant weight loss.

His past medical history was significant for type 2 diabetes mellitus of five years' duration, which had been poorly controlled. He was maintained on metformin and gliclazide and was not taking any other medications, including herbal or alternative remedies. He had no previous surgical procedures and no known drug allergies. He was an occasional smoker and consumed alcohol infrequently, and he denied use of other substances. He kept pet fish at home and had no recent travel history, either locally or internationally. He worked in hotel housekeeping, with potential exposure to soil and environmental surfaces. His family history was unremarkable, with no known hepatic diseases or conditions requiring recurrent blood transfusions.

On physical examination, Mr T was febrile. He appeared unwell, with pallor and mild jaundice. Tattoos were noted on both arms. There was no cervical lymphadenopathy or ankle oedema. Cardiovascular examination revealed

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sinus tachycardia at 100 beats per minute and a blood pressure of 120/70 mmHg. Heart sounds were dual, with no added sounds or murmurs. Respiratory examination was unremarkable. Abdominal examination revealed a soft, non-tender abdomen. There was no organomegaly, no palpable gallbladder, no renal angle tenderness, and no ascites. Neurological examination was entirely normal. Digital rectal examination revealed a normal-sized, non-tender prostate without nodularity or induration.

Clinical pause: Your initial assessment

Before reading further, take a moment to reflect on this presentation. What is your working diagnosis for the fever and jaundice in this patient with diabetes? What are your top three differential diagnoses for the overall clinical picture? Given the combination of fever, jaundice, and lower urinary tract symptoms in a patient with poorly controlled diabetes mellitus in Sri Lanka, what would your initial investigation strategy be, and which specific findings would guide your next steps?

Diagnostic reasoning: The clinical debate

The clinical team was immediately struck by the combination of systemic sepsis and painless jaundice in a middle-aged diabetic male. The main question was not which antibiotic to start, but what was causing the jaundice. The answer to this would determine whether the patient needed immediate biliary intervention, further tests, or only the best available treatment for the underlying sepsis.

Three categories of differential diagnosis were considered. The first was infective hepatitis, either viral (hepatitis A, B, or E) or bacterial, including leptospirosis, typhoid fever, and septicaemia with hepatic involvement. Leptospirosis warranted consideration given the patient's hotel housekeeping role, water exposure, and fever with jaundice.

The second and most important category was sepsis-induced cholestasis, a common but overlooked cause of jaundice in hospitalised febrile patients, accounting for 20% of cases. Systemic infection impairs bile flow through cytokine-mediated downregulation of hepato-canalicular transporters, primarily the bile salt export pump and multidrug resistance-associated protein. The main mediators are tumour necrosis factor-alpha, interleukin-1, and interleukin-6. The condition presents with conjugated hyperbilirubinemia, elevated ALP and GGT disproportionate to transaminases, and an R-factor below 2. Sepsis-induced cholestasis resolves with treatment of the infection without permanent liver damage and is distinguished from mechanical obstruction by the absence of biliary dilatation on imaging.

The third category was mechanical biliary obstruction with superimposed infection, which required exclusion. Untreated cholangitis is associated with high mortality and may present atypically in diabetic patients. The absence of biliary colic, pale stools, and pruritus argued against this diagnosis; however, imaging was essential. Lower urinary tract symptoms, such as dysuria and frequency, necessitated investigation for urosepsis and occult prostatic pathology.

Investigation strategy: Resource-conscious approach

Initial laboratory investigations revealed significant inflammatory and cholestatic abnormalities.

The full blood count showed marked leucocytosis with neutrophilia, suggestive of significant bacterial sepsis. Anaemia was normocytic, consistent with anaemia of chronic disease. Thrombocytosis was considered reactive in the setting of inflammation. Lactate dehydrogenase level was not consistent with significant haemolysis as a contributor to the jaundice.

Liver biochemistry showed a predominantly cholestatic pattern, with markedly elevated serum alkaline phosphatase and gamma-glutamyl transferase. Bilirubin was elevated in both conjugated and unconjugated fractions. The calculated R-factor was 0.7, consistent with cholestatic liver injury. Serum albumin was reduced, likely reflecting the acute inflammatory state, while INR remained within the normal range, indicating preserved hepatic synthetic function.

Renal function was preserved. Significant hyponatraemia was present, likely secondary to the septic state. Urinalysis and urine culture did not show evidence of urinary tract infection.

Ultrasound of the abdomen, performed on the same day, was normal, with no biliary duct dilatation, no hepatic or splenic enlargement, no focal lesions, and no free fluid. This single finding was critical, as it effectively excluded mechanical biliary obstruction and firmly directed our thinking towards sepsis-induced cholestasis as the mechanism of the jaundice. The chest radiograph was normal. A two-dimensional echocardiogram, performed given the persistent bacteraemia, did not identify any vegetations, making infective endocarditis unlikely.

Blood cultures were obtained before starting antibiotics, and this decision proved to be the single most important investigation in this case. The cultures grew an organism initially reported as a Pseudomonas species, sensitive to imipenem and ceftazidime and resistant to gentamicin, amikacin, netilmicin, and ciprofloxacin. Initially, it was considered a case of Pseudomonas aeruginosa sepsis, and the patient was given intravenous meropenem. However, on closer inspection, the sensitivity pattern altered the course of action.

The pivotal clue: reading the sensitivity pattern

The key was not in the organism's name but in its susceptibility and resistance pattern. In Sri Lanka, Pseudomonas aeruginosa is typically susceptible to aminoglycosides (gentamicin, amikacin, netilmicin) and resistant to co-amoxiclav.[1] This organism demonstrated the opposite antimicrobial susceptibility pattern: resistance to aminoglycosides and susceptibility to co-amoxiclav. Such a pattern is inconsistent with *Pseudomonas aeruginosa* but characteristic of *Burkholderia pseudomallei*, which causes melioidosis. Furthermore, Pseudomonas is sensitive to colistin, whereas *B. pseudomallei* is inherently resistant to colistin and polymyxin. This pattern — multiple aminoglycoside resistance, carbapenem and co-amoxiclav sensitivity — in a diabetic patient from an endemic region of Sri Lanka was the main diagnostic clue.

Table 1
Haematological and serological investigations.

Investigation	Result	Reference
White blood cell count	20.05 × 10^9/L	4.00-10.00
Neutrophils	13.82 × 10^9/L (68.9%)	2.00-7.00
Haemoglobin	7.9 g/dL	11.0-16.0
Mean corpuscular volume (MCV)	82.6 fL	84.4
Mean corpuscular haemoglobin (MCH)	27.0 pg	28.5
Platelet count	450 × 10^9/L	150-400
C-reactive protein (CRP)	118.1 mg/L	<5
Lactate dehydrogenase (LDH)	133 U/L	170-283

Table 2
Liver function tests.

Investigation	Result	Reference
Alanine aminotransferase (ALT)	75.4 U/L	0.00-50.00
Aspartate aminotransferase (AST)	129.7 U/L	0.00-50.00
Alkaline phosphatase (ALP)	369 U/L	59–164
Gamma-glutamyl transferase (GGT)	163 U/L	0–55
Total bilirubin	128.63 µmol/L	5.00-21.00
Conjugated bilirubin	67.5 µmol/L	0.00-3.40
Unconjugated bilirubin	61.13 µmol/L	
Serum albumin	25.7 g/L	35.00-52.00
International normalized ratio (INR)	1.09	

Table 3
Renal profile and urine investigations.

Investigation	Result	Reference
Serum creatinine	66.8 µmol/L	74.00-110.00
Estimated glomerular filtration rate (eGFR)	112 mL/min/1.73m^2	
Serum sodium	124.7 mmol/L	136.00-146.00
Serum potassium	4.4 mmol/L	3.5-5.1
Urine full report	Negative for protein, pus cells, red cells, and casts	
Urine culture	No growth	

Systematic exclusion process

We systematically excluded other serious diagnoses. Given the clinical picture and Sri Lanka's tuberculosis burden, a Mantoux test was negative. Contrast-enhanced CT of the chest, abdomen, and pelvis showed no abscesses, lymphadenopathy, or focal disease. However, a normal CT may miss small or early abscesses, particularly in the prostate. The absence of pyuria and a negative urine culture argued against a simple urinary tract infection but did not exclude prostatic involvement, as prostatic abscesses in melioidosis can present with sterile urine in up to 7% of cases.[2]

The susceptibility profile strongly indicated *B. pseudomallei*, prompting urgent serological testing. The indirect haemagglutination antibody titre for melioidosis was >1:10,240, a level of great significance in the Sri Lankan endemic setting, effectively confirming the diagnosis. Blood cultures were then re-evaluated, confirming *B. pseudomallei* as the causative agent. With the diagnosis of melioidosis established, the team realised that the entire clinical presentation — including sepsis, cholestasis, and urinary symptoms — could be attributed to this single pathogen.

Advanced imaging: The hidden focus

The patient was discharged after 2 weeks of intravenous meropenem, with apparent clinical improvement, and commenced on oral co-trimoxazole for the eradication phase. However, he returned three days after discharge with recurrence of fever and dysuria. On examination, there were no new clinical findings. Repeat urine full report and culture

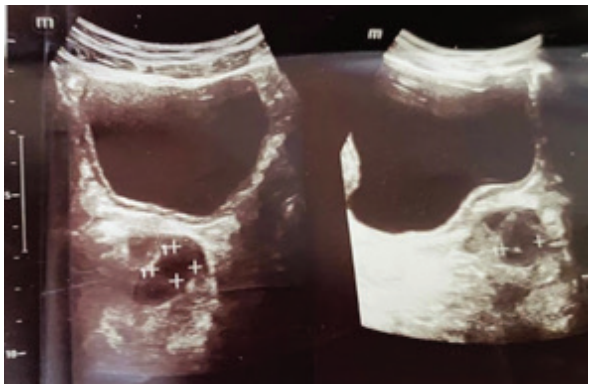

Figure 1. TRUS scan showing bilateral prostate abscesses. A left-sided abscess measuring 1.4 × 0.9 × 1.2 cm^3 and a right-sided abscess measuring 0.5 × 0.6 × 0.7 cm^3.

again showed no significant abnormality and no bacterial growth. The CRP had risen to 250 mg/L, suggesting active and ongoing inflammation. The previous CT had shown no abscess, and the repeat chest radiograph was clear.

At this point, the team faced a critical decision. The clinical question was whether an undrained focus of infection was sustaining the bacteraemia and preventing cure. Given melioidosis's well-documented predilection for prostatic abscess formation in diabetic males,[2] and given that the initial DRE had been normal and the CT had not shown a prostatic abnormality, a transrectal ultrasound (TRUS) of the prostate was requested. TRUS is considered the most sensitive and first-line imaging modality for detecting prostatic abscesses, with reported sensitivities approaching 80%.[3] It should be considered the investigation of choice when prostatic melioidosis is suspected, particularly when the standard workup is non-contributory.

The transrectal ultrasound identified bilateral prostatic abscesses (figure 1). This single finding explained the persistent symptoms, the elevated CRP, and the early relapse after discharge. The genitourinary surgery team was consulted. Given the small size of both abscesses and the patient's haemodynamic stability, the decision was made to manage without surgical drainage and to extend the intensive phase of antimicrobial therapy.

The diagnostic picture was now complete. The blood culture grew *B. pseudomallei*, the melioidosis antibody titre was greater than 1:10,240, and transrectal ultrasound confirmed bilateral prostatic abscesses. The organism's characteristic susceptibility pattern — resistance to gentamicin, amikacin, netilmicin, and ciprofloxacin, with sensitivity to ceftazidime, carbapenems, and co-amoxiclav — was consistent with the known intrinsic resistance profile of *B. pseudomallei*. The cholestatic jaundice, confirmed by an R-factor of 0.7, a cholestatic biochemical profile, and normal biliary ducts on imaging, represented sepsis-induced cholestasis secondary to the systemic *B. pseudomallei* infection. The lower urinary tract symptoms, negative urine studies, and occult bilateral prostatic abscesses on CT completed the clinical triad.

Final diagnosis: Melioidosis (*B. pseudomallei* bacteraemia) presenting with sepsis-induced cholestasis and bilateral prostatic abscesses

Mr T exhibited a typical high-risk profile for melioidosis, characterised by poorly managed diabetes mellitus, working in an environment with potential exposure to melioidosis, and experiencing systemic sepsis without an immediately identifiable source. He met all the diagnostic criteria for melioidosis: a positive blood culture for *B. pseudomallei*, a confirmatory antibody titre greater than 1:10,240, and a sensitivity pattern consistent with the organism's known antibiotic resistance. The cholestasis and prostatic abscesses were direct complications of the systemic *B. pseudomallei* infection.

Treatment and outcomes

After confirming melioidosis and identifying prostatic abscesses, the treatment strategy was revised following input from the microbiology and genitourinary surgery teams. Given bilateral prostatic abscesses indicating deep-seated infection, the intensive phase was extended to four weeks of intravenous meropenem. This extension from the usual two-week phase aligns with international guidelines recommending prolonged intensive therapy for melioidosis with deep-seated infections.[4,5,6]

Following completion of the intensive phase, the eradication phase was initiated with oral co-amoxiclav and doxycycline for a planned duration of 3 months. Eradication therapy is mandatory in all cases of melioidosis; clinical improvement alone is not a surrogate for cure, and early discontinuation carries a relapse risk of 10 to 25%.[5,6] The patient and his family were counselled about the need to complete the full course of eradication therapy even if he felt clinically well.

A transrectal ultrasound examination was arranged after 4 weeks, during the intensive phase, to monitor abscess size. This demonstrated progressive reduction in abscess dimensions bilaterally, confirming an adequate clinical response to the extended antibiotic course without the need for surgical drainage. Blood glucose was optimised with insulin therapy during admission. By the end of the intensive phase, the fever had resolved, CRP had normalised, and liver function tests had returned to near-normal levels, indicating resolution of sepsis-induced cholestasis. A monitoring plan was established with six-weekly clinical reviews during eradication, inflammatory markers, and a final TRUS to confirm resolution.

Key learning points

This case highlights the importance of reviewing the entire antimicrobial susceptibility report rather than relying solely on the organism's name in the laboratory report. In Sri Lanka, *P. aeruginosa* typically remains aminoglycoside-sensitive, and any Gram-negative bacillus reported as aminoglycoside-resistant yet sensitive to co-amoxiclav and carbapenems should immediately raise suspicion of *B. pseudomallei*, particularly in a diabetic patient from an endemic area. This resistance pattern is not a laboratory anomaly; it is the organism's defining feature. Failing to recognise the susceptibility paradox and treating this patient as having straightforward Pseudomonas sepsis would have resulted in under-treatment, an overlooked diagnosis, and a significant risk of recurrence with potentially fatal consequences.

This case also illustrates the pathophysiology and clinical recognition of sepsis-induced cholestasis. This is a functional, rather than mechanical, cause of jaundice, resulting from cytokine-mediated downregulation of hepatocanalicular bile transporters.[7] The clinical triad of active infection or sepsis, jaundice developing during rather than before sepsis, and a cholestatic biochemical profile without biliary obstruction on imaging should prompt the clinician to treat the underlying infection rather than pursue invasive biliary procedures. In this patient, the R-factor of 0.7, higher levels of ALP and GGT relative to the transaminases, and a normal biliary ultrasound were the main diagnostic indicators. Typically, bilirubin levels return to normal within 2 to 4 weeks after sepsis is effectively treated, provided there is no permanent liver damage.[7,8]

The hidden prostatic abscess was the most important learning point in the second phase of this case. Negative urine studies, including sterile urine microscopy and negative culture, do not exclude prostatic involvement in melioidosis. Published evidence indicates that up to 7% of prostatic abscesses in melioidosis can be microbiologically silent, with neither pyuria nor a positive urine culture.[2] Computed tomography, though useful for large abscesses, can miss small early prostatic collections that are readily identified by transrectal ultrasound.[2,3] In any male patient with melioidosis who has genitourinary symptoms, persistent fever despite appropriate antibiotics, or an elevated CRP after treatment, TRUS should be performed early. The threshold for TRUS should be particularly low in diabetic males, in whom prostatic abscess formation is relatively common.

Finally, this case underscored the critical importance of completing the full two-phase treatment protocol for melioidosis. The distinction between clinical improvement and microbiological cure is essential. Patients with melioidosis may appear and feel much better during the intensive phase while harbouring residual infection in deep-seated foci. The eradication phase typically involves three to six months of oral therapy with co-trimoxazole, co-amoxiclav, or doxycycline. Shortening eradication therapy carries a relapse risk of 10 to 25%,[5,6] and relapses can occur months to years after the apparent initial cure.

Conclusion: Lessons for clinical practice

This case exemplifies the importance of interpreting laboratory results in their clinical context and never accepting a microbiological report at face value without interrogating the full susceptibility pattern. Melioidosis is a disease of the immunocompromised host, and in Sri Lanka, the diabetic patient with fever of uncertain aetiology must always be considered in the differential diagnosis. Recognising sepsis-induced cholestasis as a distinct and reversible cause of jaundice prevents unnecessary invasive biliary interventions. The low threshold for TRUS in diabetic males with melioidosis and genitourinary symptoms is a lesson that can genuinely be lifesaving. Adhering to the entire two-phase treatment plan, even when the patient starts to feel better, is not just good practice; it is crucial to ensuring a cure rather than a relapse. Mr T's diagnosis of melioidosis not only resolved his clinical issues but also provided significant prognostic reassurance: with an accurate diagnosis, appropriate antibiotics, and the right treatment duration, melioidosis can be cured.

Author declaration

Conflicts of interest

The authors declare no conflicts of interest.

Patient consent

Written informed consent was obtained from the patient for publication of this clinical practice and accompanying images.

Data availability

Data supporting this clinical practice are available from the corresponding author upon reasonable request.

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