

Serial Polymicrobial Wound Infection Including Multidrug-Resistant *Acinetobacter baumannii* in a Patient with Budd-Chiari Syndrome: A Case Report

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ABSTRACT

Budd-Chiari syndrome is an uncommon disorder of hepatic venous outflow obstruction that leads to portal hypertension, ascites, hypoalbuminaemia and impaired tissue repair. Such patients are predisposed to chronic, poorly healing wounds and to recurrent infection. We report a 40-year-old woman with established Budd-Chiari syndrome and intrahepatic inferior vena caval obstruction, previously treated with balloon angioplasty and maintained on oral anticoagulation, who presented with a recurrent, non-healing right lower limb ulcer and cellulitis. Over a five-week admission, serial wound cultures yielded three different gram-negative organisms in sequence: *Klebsiella pneumoniae*, multidrug-resistant *Acinetobacter baumannii*, and *Enterobacter cloacae*. Antimicrobial therapy was adjusted at each step according to susceptibility, comprising empirical piperacillin-tazobactam and ciprofloxacin, followed by meropenem, tigecycline and oral minocycline. Care also addressed the systemic contributors to poor healing through albumin infusion, correction of anaemia, diuresis, therapeutic ascitic tapping and nutritional support. The ulcer showed healthy granulation with minimal discharge at discharge, although complete epithelialisation was not documented. This case illustrates the potential value of repeated culture, susceptibility-guided antimicrobial adjustment and coordinated multidisciplinary care in the recurrent, polymicrobial wound infections that complicate chronic liver disease.

Keywords: *Acinetobacter baumannii*, Budd-Chiari syndrome, Cellulitis, Multidrug resistance, Polymicrobial infection, Wound infection.

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INTRODUCTION

Budd-Chiari syndrome is an uncommon vascular disorder in which obstruction of hepatic venous outflow gives rise to hepatomegaly, ascites, portal hypertension and the chronic consequences of venous congestion (Hitawala *et al.*, 2025; Northup *et al.*, 2021). The oedema, hypoalbuminaemia and impaired immunity that accompany advanced liver disease undermine skin integrity and delay wound healing, leaving such patients vulnerable to recurrent soft tissue infection (Stevens *et al.*, 2014; Wiedermann, 2021).

Chronic wounds in this setting are frequently colonised and infected by more than one organism, and the responsible

pathogens may change over time as antibiotic pressure and repeated hospital exposure select for resistant flora. Among these, Multidrug-Resistant (MDR) *Acinetobacter baumannii* is of particular concern; carbapenem-resistant strains are ranked among the critical-priority antimicrobial-resistant pathogens by the World Health Organization (Sati *et al.*, 2025). MDR is conventionally defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories (Ahuatzin-Flores *et al.*, 2024). We describe a patient with Budd-Chiari syndrome in whom a recurrent lower-limb ulcer yielded three different organisms on serial culture, and in whom therapy was adjusted at each step according to susceptibility.

CASE PRESENTATION

A 40-year-old woman presented to the surgical department with swelling of the right lower limb of approximately two months' duration, an ulcer over the right leg, pain and two brief episodes of fever before admission. She had been apparently well until two months earlier, when insidious, gradually progressive swelling with throbbing pain developed. She had been admitted previously



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at the same institution for limb swelling and ulceration that had healed, and now re-presented at follow-up with a recurrent ulcer - a history that establishes the chronic, relapsing nature of the wound.

She was a known case of Budd-Chiari syndrome with intrahepatic Inferior Vena Caval (IVC) obstruction; complete (100%) IVC obstruction had been documented and balloon angioplasty had been performed on 20 May 2025. She was maintained on oral anticoagulation with acenocoumarol 4 mg once daily under cardiology supervision, and had a history of moderate ascites. She was not a known diabetic or hypertensive and had no history of tuberculosis, asthma or significant family illness.

On examination, she was moderately built, afebrile, with pallor and lower-limb oedema; blood pressure was 110/70 mmHg, pulse 80/min and oxygen saturation 98% on room air. There was oedema of the right lower limb extending to the knee, with a healing ulcer of about 5 × 4 cm over the anterior aspect of the shin and a smaller ulcer of about 1 × 2 cm just above it. The ulcers had sloping edges, an irregular outline and a red, granulating floor that bled on touch, with serous discharge, surrounding hyperpigmentation and a base extending to underlying muscle. The abdomen was distended with an everted umbilicus, caput medusae, a positive fluid thrill and shifting dullness, consistent with gross ascites; hernial orifices were intact. Cardiovascular, respiratory and neurological examinations were unremarkable. A summary of the clinical findings is presented in Table 1.

Laboratory investigations showed anaemia (haemoglobin 8.0 g/dL), hypoalbuminaemia (albumin 2.9 g/dL), conjugated hyperbilirubinaemia (total bilirubin 4.2 mg/dL, direct 2.4 mg/dL), mildly raised alkaline phosphatase, a markedly elevated erythrocyte sedimentation rate (105 mm/h), mild hyponatraemia and preserved renal function. The peripheral smear showed a microcytic hypochromic anaemia. The laboratory findings are shown in Table 2. Abdominal ultrasonography demonstrated hepatomegaly with coarse, altered echotexture, splenomegaly, gross ascites, reactive gallbladder wall oedema and partial recanalisation of the inferior vena cava (Figure 1).

Direct Gram staining of the wound pus showed a few pus cells with gram-positive cocci, and Ziehl-Neelsen staining was negative for acid-fast bacilli; however, aerobic culture consistently grew gram-negative organisms. This discordance between the direct smear and culture is in keeping with a polymicrobial, chronically colonised wound, in which the organism seen on smear need not be the one recovered on culture. Because of the non-healing course, the wound was cultured on more than one occasion, and the isolates and their susceptibility changed over time (Table 3). The first culture grew *Klebsiella pneumoniae*, sensitive to ciprofloxacin and meropenem. A subsequent culture grew multidrug-resistant *Acinetobacter baumannii*, reported sensitive to tigecycline, amikacin and minocycline; the

organism was designated MDR by the laboratory, consistent with non-susceptibility across multiple antimicrobial categories. A later culture grew *Enterobacter cloacae*, sensitive to meropenem and ciprofloxacin.

Antimicrobial therapy was adjusted in line with these evolving results. Empirical broad-spectrum cover was begun with intravenous piperacillin-tazobactam (1.5 g twice daily for five days) and intravenous ciprofloxacin (400 mg twice daily for five days), and intravenous meropenem (1 g three times daily for twelve days) was used to cover the meropenem-sensitive isolates. When multidrug-resistant *Acinetobacter baumannii* was identified, treatment was directed to intravenous tigecycline (50 mg twice daily for six days), followed by oral minocycline (100 mg twice daily for fifteen days) as a susceptibility-guided oral agent to complete therapy. The therapeutic framework is summarised in Table 4.

Attention was directed equally to the systemic factors impairing healing. Anaemia was corrected with serial blood transfusions; hypoalbuminaemia was addressed with two infusions of 20% albumin together with high-protein nutritional supplementation; ascites was managed with a combination diuretic (furosemide with spironolactone) and a therapeutic ascitic tap; ursodeoxycholic acid and acenocoumarol were continued for the underlying liver disease and its thrombotic basis. The wound was managed conservatively with regular dressings and sitz baths.

Over the course of the admission the wound improved steadily. The floor granulated healthily and serous discharge diminished, and the patient remained vitally stable. At discharge, the ulcer showed healthy granulation tissue with minimal serous discharge,

Table 1: Summary of clinical findings.

Parameter	Finding
Age and sex	40-year-old woman
Presenting complaints	Recurrent right lower limb swelling and ulceration, pain; brief pre-admission fever
Major comorbidity	Budd-Chiari syndrome with intrahepatic IVC obstruction (post balloon angioplasty); on acenocoumarol
Local findings	Two granulating ulcers (approx. 5 × 4 cm and 1 × 2 cm) on anterior right shin; sloping edges, serous discharge, surrounding hyperpigmentation; limb oedema to knee
Abdominal findings	Distension, caput medusae, fluid thrill, shifting dullness (gross ascites)
Microbiology	Serial wound cultures: <i>Klebsiella pneumoniae</i> , MDR <i>Acinetobacter baumannii</i> , <i>Enterobacter cloacae</i>
Outcome	Granulating, healing wound with minimal discharge at discharge

IVC: inferior vena cava; MDR: multidrug-resistant.

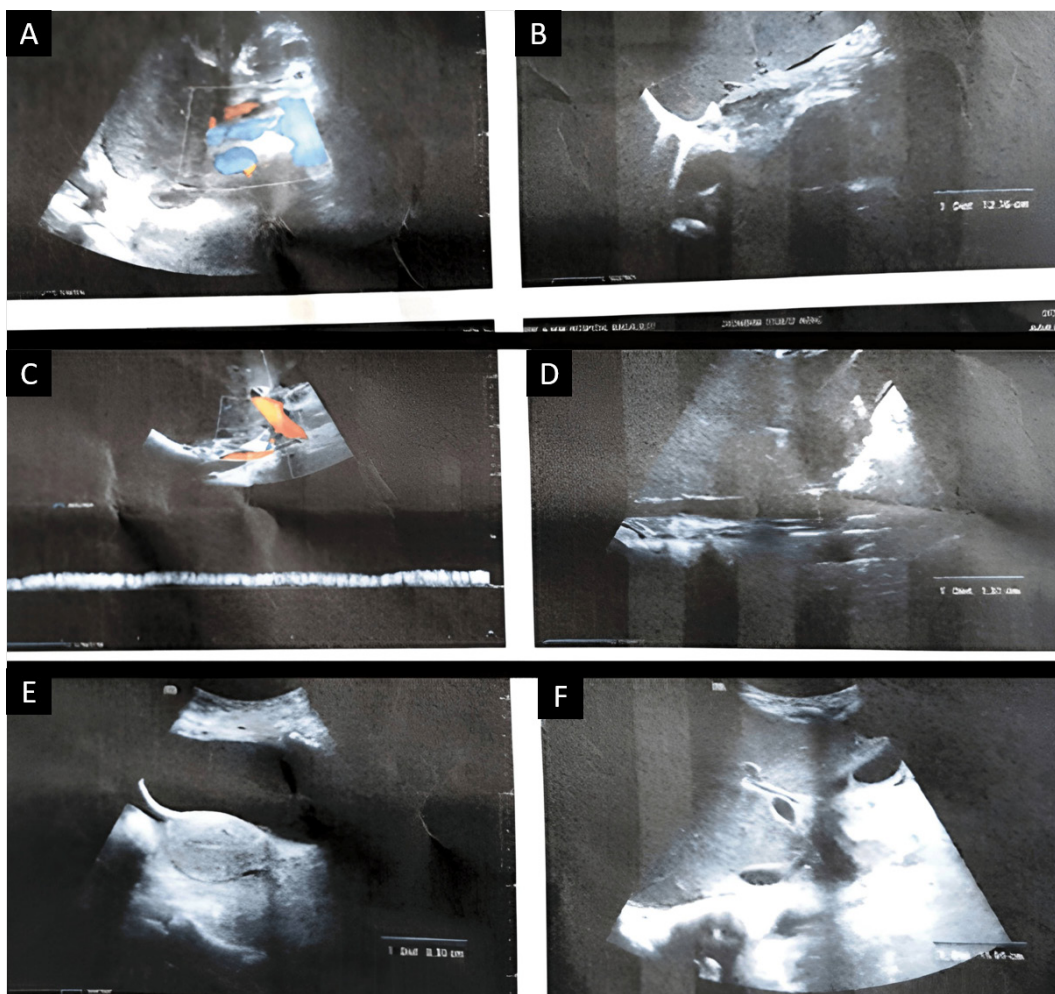


Figure 1: Abdominal ultrasonography panels (A-F) showing hepatomegaly with coarse, altered hepatic echotexture, splenomegaly, gross ascites, and colour Doppler assessment of hepatic venous and inferior vena caval flow, consistent with Budd-Chiari syndrome and portal hypertension.

Table 2: Laboratory investigations with reference ranges.

Investigation	Patient value	Reference range
Haemoglobin	8.0 g/dL	11.5-16.5 g/dL
Total leukocyte count	9,000 cells/cumm	4,000-11,000 cells/cumm
Platelet count	1,52,000/cumm	1,50,000-4,00,000 /cumm
Peripheral smear	Microcytic hypochromic anaemia	-
Serum sodium	133 mEq/L	135-153 mEq/L
Blood urea	17 mg/dL	15-39 mg/dL
Serum creatinine	0.6 mg/dL	0.6-1.3 mg/dL
Total bilirubin	4.2 mg/dL	0.2-1.0 mg/dL
Direct bilirubin	2.4 mg/dL	0.1-0.4 mg/dL
Serum albumin	2.9 g/dL	3.5-5.0 g/dL
Alkaline phosphatase	145.8 U/L	35-140 U/L
Erythrocyte sedimentation rate	105 mm/h	15-20 mm/h

although complete epithelialisation was not documented. She was discharged on a high-protein diet, oral antibiotics, continued hepatic and anticoagulant therapy, regular dressings and sitz baths, with advice for follow-up in the surgery, gastroenterology and cardiology clinics.

PATIENT PERSPECTIVE

The patient expressed relief at the steady improvement in her wound and the easing of her symptoms during the admission. She understood that her underlying liver condition was long-standing and that it required continued anticoagulation and regular follow-up, and she consented to the publication of her case.

DISCUSSION

This case is instructive because a common problem - a lower-limb ulcer with cellulitis - arose in a patient whose chronic liver disease had made healing precarious, and because the wound proved to be infected not by one organism but by a succession of them. Budd-Chiari syndrome produces hepatic venous congestion and portal hypertension, and with them ascites, oedema,

hypoalbuminaemia and nutritional compromise (Hitawala *et al.*, 2025; Khan *et al.*, 2019). Each of these undermines the skin and slows repair, and hypoalbuminaemia in particular is a recognised marker of poor physiological reserve and of an increased risk of infective complications (Soeters *et al.*, 2019; Wiedermann, 2021). The patient's prolonged limb oedema and her earlier, recurrent ulceration reflect this vulnerability.

The most striking feature of the case was the evolving microbiology. Serial cultures of the same wound grew *Klebsiella pneumoniae*, then multidrug-resistant *Acinetobacter baumannii*, and then *Enterobacter cloacae* (Table 3). Such shifting, polymicrobial flora is characteristic of chronic wounds subjected to repeated antibiotic exposure and hospital contact, and it carries a clear practical lesson: a single culture may not capture the responsible pathogen, and re-culturing a wound that fails to heal is often necessary. *A. baumannii* is notorious for environmental persistence and for accumulating resistance across antimicrobial classes (Ahuatzin-Flores *et al.*, 2024; Mea *et al.*, 2021), and its appearance here, designated MDR by the laboratory, is consistent with the global concern reflected in the WHO priority pathogen list (Sati *et al.*, 2025).

Table 3: Serial wound cultures and susceptibility.

Date	Organism isolated	Sensitive to
24/04/2026	<i>Klebsiella pneumoniae</i>	Ciprofloxacin, meropenem
13/05/2026	<i>Acinetobacter baumannii</i> (MDR)	Tigecycline, amikacin, minocycline
29/05/2026	<i>Enterobacter cloacae</i>	Meropenem, ciprofloxacin

MDR: Multidrug-resistant. The *Acinetobacter* isolate was designated multidrug-resistant by the reporting laboratory, consistent with non-susceptibility to agents across three or more antimicrobial categories.

Antimicrobial management illustrates the value of allowing culture, rather than habit, to direct therapy. Broad-spectrum agents were begun empirically, but treatment was revised at each step as new organisms and susceptibilities emerged, with meropenem covering the meropenem-sensitive isolates and tigecycline followed by oral minocycline directed against the *Acinetobacter*. This sequence, though pragmatic, embodies the principles of antimicrobial stewardship: repeated sampling, prompt de-escalation or escalation as results dictate, and completion of therapy with an appropriate oral agent (Stevens *et al.*, 2014). Equally important, the systemic contributors to poor healing were treated in parallel; correcting albumin and haemoglobin, relieving ascites and supporting nutrition were not adjuncts but central components of care.

The report has limitations. It describes a single patient, and while serial culture results were documented in the clinical record, the complete written susceptibility report was available for one isolate only; the full case record is available from the corresponding author on request. Histopathology and cross-sectional venous imaging were not performed during this admission, and long-term follow-up was limited. Some empirical agents were later found to be inactive against particular isolates, reflecting the reality of treating before culture results return. Notwithstanding these constraints, the case offers a practical illustration of managing recurrent, polymicrobial wound infection in a patient with complex hepatovascular disease.

CONCLUSION

A recurrent lower-limb ulcer complicated by serial polymicrobial infection, including multidrug-resistant *Acinetobacter baumannii*, in a patient with Budd-Chiari syndrome represents a demanding clinical problem. This case demonstrates the potential value of repeated wound culture, susceptibility-guided antimicrobial

Table 4: Therapeutic management framework.

Problem	Intervention	Rationale
Wound infection (empirical)	IV piperacillin-tazobactam 1.5 g BD (5 d); IV ciprofloxacin 400 mg BD (5 d)	Broad cover before culture results are available
Meropenem-sensitive isolates	IV meropenem 1 g TDS (12 d)	Directed cover for <i>Klebsiella</i> and <i>Enterobacter</i>
MDR <i>A. baumannii</i>	IV tigecycline 50 mg BD (6 d), then oral minocycline 100 mg BD (15 d)	Susceptibility-directed therapy and oral step-down
Anaemia	Serial blood transfusions	Improves oxygen delivery and healing
Hypoalbuminaemia	20% albumin × 2; high-protein diet	Supports oncotic balance and tissue repair
Ascites	Furosemide + spironolactone; therapeutic ascitic tap	Reduces fluid burden
Liver disease/BCS	Ursodeoxycholic acid; acenocoumarol	Hepatic support and anticoagulation for IVC obstruction
Local wound	Regular dressings; sitz baths	Reduces bacterial burden, supports granulation

BCS: Budd-Chiari syndrome; BD: Twice daily; IV: Intravenous; IVC: Inferior vena cava; MDR: Multidrug-resistant; TDS: Three times daily.

adjustment, correction of the systemic factors that impair healing, and coordinated multidisciplinary care. Clinicians caring for patients with chronic hepatovascular disease should remain alert to the likelihood of recurrent and evolving wound infection, and to the importance of addressing local infection and systemic risk factors together.

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ABBREVIATIONS

BCS: Budd-Chiari syndrome; **BD:** Twice Daily; **IV:** Intravenous; **IVC:** Inferior Vena Cava; **MDR:** Multidrug-Resistant; **TDS:** Three Times Daily.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS CONTRIBUTION

S.M. and R.S.H.: Contributed to the conception and design of the report and drafted the manuscript. **B.V.G.:** Supervised patient management and the surgical assessment. **A.J.:** Performed and

interpreted the microbiological investigations. **B.V.G. and A.J.:** Critically reviewed the manuscript for important intellectual content. All authors read and approved the final version and agreed to be accountable for all aspects of the work.

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