Fatal Invasive Skin and Soft Tissue Infection (SSTI) Caused by Multidrug-Resistant *Klebsiella pneumoniae* in a Neutropenic Patient

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Background

Klebsiella pneumoniae is a ubiquitous Gram-negative opportunistic pathogen, but severe, invasive Skin and Soft Tissue Infections (SSTIs) remain rare.

The intersection of **profound neutropenia** (post-chemotherapy) and multidrug resistance (MDR)creates a highly aggressive, rapidly progressive, and frequently fatal clinical trajectory.

The Resource Barrier: Managing Carbapenem-Resistant *K. pneumoniae* (CRKP) in resource-constrained settings highlights a critical gap between global therapeutic guidelines and local availability.

Case report

Patient: 51-year-old male.

Underlying Comorbidities: Diabetes Mellitus and B-cell Acute Lymphoblastic Leukaemia (B-ALL).

Admission Status: Admitted to the Medical Intensive Care Unit (MICU) on post-chemotherapy Day 10 presenting with respiratory distress, neutropenic sepsis, and left lower limb cellulitis.

Day 1–2: Admission & Initial Escalation: Empirical Therapy Failure.

Patient initially managed for left lower limb cellulitis with Piperacillin-Tazobactam. Due to clinical decline and a soaring CRP (410 mg/L), therapy was escalated to Meropenem, Amikacin, and Teicoplanin. On Day 2, blood cultures flagged positive for MDR *Klebsiella pneumoniae.*

Day 3–4: Targeted Shifts: Profound Neutropenia Discovered.

VITEK system confirmed *K. pneumoniae* sensitive only to Colistin and Tigecycline. White Blood Cell (WBC) count dropped to 50/μL (Absolute Neutrophil Count [ANC]: 0). Tigecycline was initiated; Colistin was omitted due to acute local unavailability.

Day 5–9: Deepening Sepsis: Systemic Deterioration.

WBC bottomed out at 10/μL. Persistent bacteremia prompted the addition of Colistin once procured. By Day 8, Procalcitonin skyrocketed to 70.4 ng/mL, and abdominal ultrasound revealed new-onset typhlitis (bowel wall edema).

Day 10–11: Terminal Phase: Source Control Barrier.

Left lower limb SSTI visibly progressed with signs of microvascular failure. Repeated blood cultures remained stubbornly positive for the same strain. Profound, persistent thrombocytopenia (10-11 x10^3/μL) completely barred surgical debridement or tissue biopsy. The patient succumbed to septic shock on Day 11.

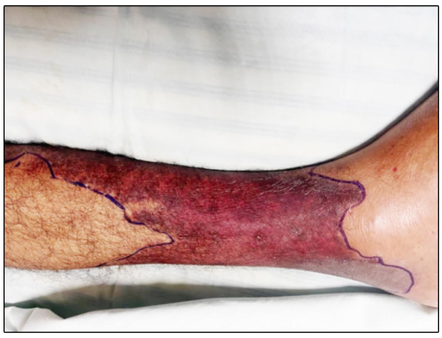

Figure 1. Clinical presentation of lower limb cellulitis on admission (Day 01). Severe, poorly demarcated erythema and localized soft tissue oedema.

Lab Trends Analysis

A compact table maps out the profound hematological crash alongside rampant inflammatory markers, demonstrating why the immune system was entirely weaponless.

Table 1. Timeline of Clinical & Diagnostic Investigations During MICU Admission

Physiological Domain & Parameter (Ref. Range)	Day 1	Day 3	Day 5	Day 8	Day 10
Hematological Profile					
Total Leukocyte Count (4.0–11.0×103/μL)	0.33	0.05	0.01	0.06	0.08
Absolute Neutrophil Count (1.5–7.5×103/μL)	0.03	0	0	0	0
Platelet Count (150–400×103/ μL)	11	32	51	10	10
Biomarkers of Inflammation					
C-Reactive Protein (<5.0 mg/L)	410	499	366.9	375	-
Procalcitonin (<0.5 ng/mL)	-	-	-	70.4	-
Organ Function & Coagulation					
Serum Creatinine (60–110 μmol/L)	108	-	81.3	72	-
International Normalized Ratio (INR)	2.13	1.52	1.34	-	-
Cardiac Troponin I (<40 ng/L)	232	-	-	-	-
Microbiological Surveillance					
Blood Culture	-	*K.pneumoniae*	*K.pneumoniae*	-	*K.pneumoniae*
Antimicrobial Susceptibility (ABST)	-	Col/Tig-S	Col/Tig-S	-	Col/Tig-S
Urinalysis & Culture	Normal /Negative	Negative	-	-	-
Diagnostic Imaging Timeline					
Chest Radiography (CXR)	Unremarkable	-	-	-	-
Transthoracic Echocardiogram (TTE)	Mild LVD (EF 50%)	-	-	-	-
Lower Limb Duplex Ultrasonography	-	-	Negative for DVT	-	-
Abdominal Ultrasonography (US)	Normal peristalsis	-	Normal peristalsis	Confirmed Typhlitis	-

Discussion

Pathogenicity: MDR lineages leverage mobile genetic elements carrying beta-lactamases (ESBLs, AmpC, Carbapenemases) alongside aggressive virulence factors (capsular polysaccharides, siderophores) that accelerate deep tissue destruction.

Critical Clinical Barrier: Persistent, severe thrombocytopenia created a Catch-22. Surgical debridement (source control) was critically needed to halt the necrotic SSTI, but the profound bleeding risk rendered surgery impossible.

The Resource Paradox: While international paradigms advocate for novel, targeted agents for CRKP, frontline clinicians in developing healthcare systems face a dual diagnostic and therapeutic barrier: the unavailability of phenotypic tests (Carba NP) alongside a lack of advanced molecular testing (PCR and sequencing) and specialized pharmaceuticals."

Novel Therapeutic Arsenal for CRKP

Ceftazidime-Avibactam / Aztreonam- Avibactam: Ideal if phenotypic testing identifies OXA- 48 or Metallo-β-lactamases (NDM).
Meropenem-Vaborbactam: Highly effective against KPC-producing strains.
Cefiderocol / Eravacycline: Vital backup options when traditional polymyxins (Colistin) fail or are unavailable

Conclusion

Early Recognition Is Key: In hematological malignancy patients, atypical presentations of Gram-negative infections can mimic classic Gram-positive cellulitis but progress far more violently.

The Source Control Dilemma: Hematological failure (thrombocytopenia/neutropenia) completely strips away our two strongest defenses: surgical debridement and innate immunity.

Global Health Call to Action: There is an urgent, systemic need to improve access to novel reserve antibiotics and rapid molecular resistance testing in lower-to-middle-income countries (LMICs).

References

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