

***Myroides odoratus* polymicrobial cellulitis complicated by sepsis and acute kidney injury in an adolescent with focal segmental glomerulosclerosis: a case report**

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Case Report

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Abstract

Background

Myroides odoratus is a rare, non-fermenting gram-negative bacillus with intrinsic broad-spectrum antimicrobial resistance mediated by chromosome-encoded metallo- β -lactamases, rendering most empirical regimens ineffective. Although more than 100 human infections have been documented, *Myroides* cellulitis in the setting of nephrotic syndrome has not been previously reported.

Case presentation

A 17-year-old boy with newly diagnosed focal segmental glomerulosclerosis, receiving prednisolone, presented one month into therapy with bullous cellulitis of both lower limbs complicated by sepsis and rapidly progressive acute kidney injury. Initial empirical therapy with amoxicillin-clavulanate and clindamycin failed, and escalation to piperacillin-tazobactam and linezolid was undertaken at 48 hours. Pus culture results, available on Day 4, yielded *Klebsiella pneumoniae* and *Myroides odoratus*, the latter susceptible only to minocycline. Targeted therapy with intravenous minocycline and ceftazidime achieved defervescence by Day 10, with renal recovery and sterile repeat cultures by Day 21.

Conclusions

This case highlights the need for broad empirical gram-negative coverage and early culture-directed therapy in immunocompromised patients with refractory cellulitis. Minocycline remains the most reliably active agent against *Myroides* across geographical regions.

Background

Myroides species (formerly *Flavobacterium odoratum*) are environmental, non-fermentative, yellow-pigmented, aerobic, non-motile, gram-negative bacilli classified within the family Flavobacteriaceae. Two clinically relevant species, *M. odoratus* and *M. odoratimimus*, are recognised alongside several environmental species. They are ubiquitous in soil, freshwater, marine water and sewage, but are not part of the normal human microflora [1, 2].

Myroides species exhibit intrinsic resistance to most β -lactams (including carbapenems), aminoglycosides, fluoroquinolones and trimethoprim-sulfamethoxazole. Resistance mechanisms include chromosome-encoded metallo- β -lactamases (TUS-1 and MUS-1) and biofilm formation [3, 4]. More than 100 infections have been documented since 1996 [2], with skin and soft-tissue infections (approximately 43%) being the most common presentation [4].

We report, to our knowledge, the first case of *Myroides odoratus* cellulitis in a patient with nephrotic syndrome, occurring as a polymicrobial soft-tissue infection complicated by sepsis and acute kidney

injury.

Case presentation

A 17-year-old boy with no significant prior medical history presented with a two-month history of progressive generalised oedema, abdominal distension and frothy urine. Examination revealed a blood pressure of 144/90 mmHg, pitting oedema, periorbital puffiness and ascites. Laboratory evaluation demonstrated nephrotic-range proteinuria (24-hour urine protein 8.3 g/day), severe hypoalbuminaemia (1.9 g/dL) and hyperlipidaemia (cholesterol 336 mg/dL; triglycerides 474 mg/dL), with preserved renal function (creatinine 0.6 mg/dL) and normal hepatic function (AST 25 IU/L, ALT 34 IU/L, total bilirubin 0.7 mg/dL). Serological screening for hepatitis B, hepatitis C, HIV and autoimmune markers was negative. Renal biopsy revealed features consistent with primary focal segmental glomerulosclerosis (FSGS). The patient was started on oral prednisolone (1 mg/kg/day) together with diuretics and supportive management.

One month after initiating prednisolone, the patient returned with fever, progressive bilateral lower-limb swelling, excoriations, multiple fluid-filled bullous lesions and purulent discharge (Fig. 1); pus culture was obtained from the discharging lesions. Given the appearance of severe cellulitis in an immunocompromised host, empirical intravenous amoxicillin-clavulanate (1.2 g every 8 hours) plus oral clindamycin (300 mg every 8 hours) were initiated, targeting the gram-positive organisms and anaerobes commonly implicated in cellulitis.

Laboratory investigations at admission showed leukocytosis (white blood cell count 16760/ μ L). Procalcitonin and lactate were 0.3 ng/mL and 1.6 mmol/L, respectively. Urine and blood cultures were negative. Chest radiography demonstrated bilateral pleural effusions. Doppler ultrasonography of both lower limbs, the renal vessels and the inferior vena cava showed no evidence of venous thrombosis. Renal ultrasonography demonstrated raised cortical echogenicity bilaterally, consistent with the underlying FSGS, with no hydronephrosis or calculi.

No clinical improvement occurred over the next 48 hours. As the patient continued to worsen and remained refractory to initial therapy, biomarkers were obtained and confirmed sepsis: procalcitonin 12 ng/mL and lactate 3 mmol/L (Table 1).

Table 1
Laboratory investigations

Laboratory parameter	First admission (baseline)	Second admission (Day 0)	Second admission (Day 2, sepsis)	At discharge
Serum creatinine (mg/dL)	0.6	1.1	8.0 (peak)	0.8
Blood urea nitrogen (mg/dL)	29	53	140 (peak)	18
White blood cells (/ μ L)	8500	16760	20000	5000
Procalcitonin (ng/mL)	0.1	0.3	12	0.1
Lactate (mmol/L)	1.2	1.6	3	0.8
Serum albumin (g/dL)	1.9	1.4	-	1.8
24-hour urine protein (g/day)	8.3	6.67	-	5.6
Haemoglobin (g/dL)	15.0	16.7	16.6	15.1

The patient developed rapidly progressive acute kidney injury: serum creatinine peaked at 8.0 mg/dL and blood urea nitrogen at 140 mg/dL within approximately 48–72 hours of admission. Given the failure of initial therapy in an immunocompromised host with confirmed sepsis, amoxicillin-clavulanate and clindamycin were discontinued and empirical escalation to intravenous piperacillin-tazobactam (4.5 g every 6 hours) plus intravenous linezolid (600 mg every 12 hours) was initiated, in accordance with Infectious Diseases Society of America recommendations for severe skin and soft-tissue infections in immunocompromised patients [5]; vancomycin was avoided given the rapidly evolving renal impairment. Prednisolone was reduced from 1 mg/kg/day to 0.5 mg/kg/day; complete cessation was avoided given the active nephrotic state and the risk of relative adrenal insufficiency with abrupt withdrawal after one month of high-dose corticosteroid therapy.

The temporal relationship between sepsis onset and subsequent renal recovery following targeted therapy supported septic acute tubular necrosis as the primary aetiology, although concurrent FSGS progression and diuretic-related haemoconcentration (rising haemoglobin 15.0 to 16.7 g/dL) were also considered.

Haemodialysis was initiated for worsening uraemia, metabolic acidosis and fluid overload in the context of oliguria; six sessions of intermittent haemodialysis were administered over the course of hospitalisation. Despite improvement in renal parameters with haemodialysis, inflammatory markers remained persistently elevated (procalcitonin 10.5 ng/mL).

Pus culture, collected on Day 0, became available on Day 4 and yielded two organisms: *Myroides odoratus* (Fig. 2) and *Klebsiella pneumoniae*. The *Myroides* isolate was identified by VITEK 2 (bioMérieux, Marcy-l'Étoile, France) and matrix-assisted laser desorption/ionisation time-of-flight mass spectrometry (MALDI-TOF), and confirmed phenotypically: yellow-pigmented, convex, non-haemolytic colonies with a characteristic fruity odour on blood agar after 24–48 hours at 37°C, with no growth on MacConkey agar. Broth microdilution and disc diffusion, interpreted using Clinical and Laboratory Standards Institute non-fermenter breakpoints, revealed extensive drug resistance with susceptibility limited to minocycline (Table 2). The co-isolated *Klebsiella pneumoniae* was susceptible to third- and fourth-generation cephalosporins, meropenem, aminoglycosides and trimethoprim-sulfamethoxazole, but resistant to amoxicillin-clavulanate, piperacillin-tazobactam and ciprofloxacin (Table 3).

Table 2
Antimicrobial susceptibility of the *Myroides odoratus* isolate

Antimicrobial agent	Broth microdilution	Disc diffusion
Ampicillin	Resistant	Resistant
Amoxicillin-clavulanate	Resistant	Resistant
Ceftazidime	Resistant	Resistant
Meropenem	Resistant	Resistant
Imipenem	Resistant	Resistant
Ciprofloxacin	Resistant	Resistant
Trimethoprim-sulfamethoxazole	Resistant	Resistant
Gentamicin	Resistant	Resistant
Minocycline	Susceptible	Susceptible
Doxycycline	Intermediate	Intermediate
Tetracycline	Intermediate	Intermediate

Testing was performed using Clinical and Laboratory Standards Institute non-fermenter breakpoints. The profile is extensively drug-resistant, with susceptibility limited to minocycline.

Table 3
Antimicrobial susceptibility of the *Klebsiella pneumoniae* isolate

Antimicrobial agent	Broth microdilution	Disc diffusion
Ampicillin	Resistant	Resistant
Amoxicillin-clavulanate	Resistant	Resistant
Piperacillin-tazobactam	Resistant	Resistant
Ceftriaxone	Susceptible	Susceptible
Ceftazidime	Susceptible	Susceptible
Cefepime	Susceptible	Susceptible
Meropenem	Susceptible	Susceptible
Gentamicin	Susceptible	Susceptible
Amikacin	Susceptible	Susceptible
Ciprofloxacin	Resistant	Resistant
Trimethoprim-sulfamethoxazole	Susceptible	Susceptible

Based on susceptibility results, the regimen was changed to targeted therapy: linezolid and piperacillin-tazobactam were discontinued, and intravenous minocycline (200 mg loading dose followed by 100 mg every 12 hours) was initiated on Day 4 for *Myroides odoratus*, together with intravenous ceftazidime (2 g every 8 hours) for the co-isolated *Klebsiella pneumoniae*. Although some references recommend dose reduction in renal impairment, standard dosing was maintained given the severity of infection, the predominantly hepatic metabolism of the drug, its negligible removal by haemodialysis [6] and the patient's normal baseline hepatic function. Antimicrobial therapy was continued for 21 days given the severity of the presentation.

The patient achieved progressive improvement: defervescence by Day 10; normalisation of leukocyte count and serum creatinine (0.8 mg/dL) by Day 21; and complete healing of skin lesions by discharge. Repeat pus culture at discharge was sterile. The full 21-day antimicrobial course was administered without interruption or dose modification, and no adverse drug events, haemodialysis-related complications or other unanticipated events occurred during the admission. Twenty-four-hour urine protein remained elevated at 5.6 g/day, reflecting ongoing nephrotic-range proteinuria from FSGS. Tacrolimus was introduced as a steroid-sparing agent following confirmed microbiological clearance and renal recovery [7]; it was deliberately deferred until infection was completely controlled, given the additional immunosuppressive effect of calcineurin inhibition. A detailed clinical timeline is provided in Additional file 1.

Discussion and conclusions

This case represents, to our knowledge, the first reported case of *Myroides odoratus* cellulitis with polymicrobial involvement in a patient with nephrotic syndrome (FSGS) receiving corticosteroids, complicated by sepsis and acute kidney injury. A systematic search of PubMed and Embase (inception to January 2026) identified no prior reports of *Myroides* cellulitis in nephrotic syndrome. The case illustrates several clinically important themes: the vulnerability of patients with nephrotic syndrome receiving corticosteroids to opportunistic polymicrobial soft-tissue infection; the diagnostic and therapeutic challenges posed by *Myroides odoratus* as a co-pathogen; and the importance of early empirical escalation in immunocompromised hosts with sepsis.

Patients with nephrotic syndrome carry a well-established predisposition to serious bacterial infections, including cellulitis, peritonitis and sepsis [8]. This susceptibility is multifactorial: urinary losses of immunoglobulin G and complement factors impair opsonisation; oedematous tissues provide a protein-rich medium with diluted local defences; and corticosteroids further suppress T-cell function and neutrophil chemotaxis. In hospitalised children with nephrotic syndrome, major infections occur in approximately 44%, with cellulitis accounting for up to 14% [8]. Our patient embodied this convergence: active nephrotic syndrome with severe hypoalbuminaemia (1.9 g/dL), extensive lower-limb oedema, and full-dose prednisolone for one month prior to presentation.

Since reclassification of the genus in 1996, only approximately 100 cases of human *Myroides* infection have been documented, the majority in immunocompromised hosts, particularly those with diabetes mellitus, hepatic cirrhosis, chronic obstructive pulmonary disease or prolonged corticosteroid therapy [2, 9]. The clinical spectrum ranges from urinary tract and soft-tissue infections to bacteraemia and, rarely, fatal pericardial effusion [10] and necrotising fasciitis [2, 11]. The predominant presentations reported in the literature are cellulitis and bacteraemia [2].

Our case adds to this limited but growing body of literature and is notable for several reasons. First, the co-isolation of *M. odoratus* with *Klebsiella pneumoniae* from a severe soft-tissue infection in a nephrotic adolescent receiving corticosteroids represents a host profile not previously emphasised in the *Myroides* literature. It is also noteworthy that the co-isolated *Klebsiella pneumoniae* was resistant to both amoxicillin-clavulanate and piperacillin-tazobactam (Table 3), indicating that neither the *Myroides* nor the *Klebsiella* component was adequately covered until targeted therapy with minocycline and ceftazidime was initiated on Day 4. The co-isolate exhibited piperacillin-tazobactam resistance with preserved cephalosporin susceptibility, an increasingly recognised phenotype attributed to hyperproduction of narrow-spectrum chromosomal β -lactamases (for example SHV-1) that overwhelm β -lactamase inhibitor activity without affecting cephalosporins [12, 13]. This reinforces the central message that early pus culture and susceptibility-directed therapy are indispensable in polymicrobial soft-tissue infections in immunocompromised hosts. Second, the extensive drug resistance profile of the *Myroides* isolate, with susceptibility confined to minocycline alone, is consistent with a pattern increasingly recognised in recent case series, in which minocycline and moxifloxacin have shown the highest rates of activity against *Myroides* isolates [14]. Although a 2025 cohort reported that many Western isolates retained susceptibility to meropenem and ciprofloxacin [4], minocycline is the only agent with near-universal

activity across geographical regions and disparate resistance patterns. Our isolate matched the extensively drug-resistant pattern commonly described in South Asian and European settings, being susceptible only to minocycline [9, 14].

A critical consideration in polymicrobial wound infection is distinguishing true pathogens from colonisers. Several observations support the genuine pathogenicity of *M. odoratus* in this case. The organism was isolated from purulent wound discharge rather than a superficial swab of intact skin, indicating tissue-level infection. Furthermore, pus cultures were repeated on two separate occasions and both yielded the same organisms, reinforcing the significance of the isolates as true pathogens rather than incidental colonisers. More importantly, the clinical timeline supports a causal role: the patient deteriorated progressively on empirical therapy that covered gram-positive, gram-negative and anaerobic organisms but lacked anti-*Myroides* activity, and clinical improvement commenced only after the introduction of minocycline on Day 4. Although ceftazidime was initiated simultaneously, making it impossible to attribute recovery to either agent alone, this does not diminish the clinical rationale: without minocycline the *Myroides* component would have remained entirely untreated, a clinically unacceptable gap in the management of severe polymicrobial sepsis. Selected *Myroides* soft-tissue infections from the literature, including outcomes and resistance patterns, are summarised in Table 4.

Table 4
Selected *Myroides* soft-tissue infections reported in the literature

Case / reference	Patient	Presentation	Resistance	Outcome
Green et al. (2001) [15]	Apparently immunocompetent man	Cellulitis, bacteraemia	MDR (fluoroquinolone susceptible)	Resolved with fluoroquinolone
Beathard et al. (2021) [16]	88-year-old male with type 2 diabetes, heart failure and CKD	Cellulitis, bacteraemia	MDR (susceptible to meropenem and ciprofloxacin)	Resolved with meropenem followed by ciprofloxacin
Lovering et al. (2024) [17]	Elderly male with cirrhosis	Cellulitis, bacteraemia	XDR (levofloxacin susceptible)	Resolved with levofloxacin
Juusola et al. (2025) [18]	80-year-old male with chronic kidney failure, heart failure and chronic leg ulcers	Infected chronic leg ulcers, bacteraemia	XDR; levofloxacin susceptible at high dose	Recovered with meropenem plus levofloxacin after susceptibility testing
O'Neal et al. (2022) [14]	Critically ill, immunocompetent (2 cases)	VAP (case 1); wound infection, bacteraemia (case 2)	XDR	Case 1: resolved with minocycline; case 2: fatal despite minocycline + cefiderocol
Crum-Cianflone et al. (2014) [11]	Liver disease and open wounds	Necrotising fasciitis	XDR	Fatal despite antibiotics
Current case	Adolescent with FSGS, nephrotic syndrome	Cellulitis, sepsis, AKI	XDR, minocycline-only	Resolved; complete recovery

AKI acute kidney injury, CKD chronic kidney disease, FSGS focal segmental glomerulosclerosis, MDR multidrug-resistant, VAP ventilator-associated pneumonia, XDR extensively drug-resistant

This case has inherent limitations. As a single case report, the findings are not generalisable. Species identification relied on VITEK 2, MALDI-TOF and phenotypic features; 16S rRNA sequencing was unavailable, although MALDI-TOF provides species-level identification with high reliability. In addition, the simultaneous introduction of minocycline and ceftazidime precludes definitive attribution of clinical improvement to either agent individually.

In conclusion, this case highlights the importance of maintaining a high index of suspicion for extensively drug-resistant organisms in immunocompromised patients with nephrotic syndrome who present with severe soft-tissue infection. *Myroides odoratus*, though rare, is an emerging pathogen with intrinsic resistance to most standard antimicrobials. Early culture acquisition, timely identification by

MALDI-TOF or VITEK 2, and prompt susceptibility-guided de-escalation are essential; minocycline remains the most reliably active agent across geographical regions.

Abbreviations

AKI

Acute kidney injury

ALT

Alanine aminotransferase

AST

Aspartate aminotransferase

CKD

Chronic kidney disease

CLSI

Clinical and Laboratory Standards Institute

FSGS

Focal segmental glomerulosclerosis

HIV

Human immunodeficiency virus

IDSA

Infectious Diseases Society of America

IV

Intravenous

KDIGO

Kidney Disease:Improving Global Outcomes

MALDI-TOF

Matrix-assisted laser desorption/ionisation time-of-flight mass spectrometry

MDR

Multidrug-resistant

VAP

Ventilator-associated pneumonia

XDR

Extensively drug-resistant

16S rRNA

16S ribosomal ribonucleic acid

Declarations

Ethics approval and consent to participate

Institutional ethics committee approval was not required for this single-patient retrospective case report in accordance with the policy of the Institutional Ethics Committee, Maulana Azad Medical College, New Delhi, India. Written informed consent to participate was obtained from the patient's legal guardian (father), and written assent was obtained from the patient.

Consent for publication

Written informed consent for publication of this case report and any accompanying images was obtained from the patient's legal guardian (father). The patient also provided written assent.

Competing interests

The authors declare that they have no competing interests.

Authors' information

Not applicable.

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Author Contribution

SK and SML acquired the clinical and microbiological data and were major contributors in writing the original draft. SA reviewed and edited the manuscript. SG conceptualised and supervised the work and reviewed and edited the manuscript. SK and SML contributed equally to this work. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Figures



Figure 1

Bilateral lower-limb cellulitis with tense bullous lesions and purulent discharge at second presentation.

- [SupplementaryTable.docx](#)