



Letters to the Editor

Community-acquired Carbapenem-resistant *Escherichia coli* Bacteremia Associated with Transient Aplastic Crisis

Keywords: Community-acquired; Carbapenem-resistant *Escherichia coli*; Bacteremia; Aplastic crisis.

Published: January 01, 2026

Received: November 16, 2025

Accepted: December 05, 2025

Citation: Chen Y., Zhang J., Kong W. Community-acquired carbapenem-resistant *Escherichia Coli* bacteremia associated with transient aplastic crisis. *Mediterr J Hematol Infect Dis* 2026, 18(1): e2026005, DOI: <http://dx.doi.org/10.4084/MJHD.2026.005>

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by-nc/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

To the editor.

The World Health Organization (WHO) has classified carbapenem-resistant *Enterobacterales* (CRE) as pathogens of critical priority, underscoring their significant global impact on clinical care and public health. These organisms pose major challenges related to transmission, treatment, and prevention.¹ Epidemiological studies indicate that community-acquired or community-onset CRE infections are rare, with reported rates generally low and varying considerably across regions.²

We describe a rare case of community-acquired bloodstream infection caused by carbapenem-resistant *Escherichia coli* (CRECO), which led to an aplastic crisis.

A 75-year-old woman, weighing 40 kg and with no prior medical history, was admitted on September 10, 2025, after experiencing one week of fever, chills, rigors, nausea, and vomiting. On admission, her vital signs were: temperature 38.2 °C, pulse 59 beats/min, respiratory rate 20 breaths/min, blood pressure 100/55 mmHg, and SpO₂ 96%. Physical examination revealed petechiae, ecchymoses, pallor, and an anemic appearance. Laboratory tests showed severe pancytopenia: white blood cell count $0.25 \times 10^9/L$, red blood cell count $2.5 \times 10^{12}/L$, hemoglobin 76.0 g/L, platelet count $9.0 \times 10^9/L$, absolute neutrophil count $0.02 \times 10^9/L$, lymphocyte count $0.21 \times 10^9/L$, and reticulocyte count $0.0028 \times 10^{12}/L$. C-reactive protein was 276.4 mg/L, and procalcitonin was 0.754 ng/mL. Blood culture (anaerobic bottle) yielded Gram-negative bacilli. Empirical therapy with imipenem/cilastatin was started, along with intravenous fluconazole for antifungal prophylaxis, oral trimethoprim-sulfamethoxazole for *Pneumocystis jirovecii* prophylaxis, and oral acyclovir for herpes zoster prevention. Platelet transfusion was also administered. Bone marrow cytology showed markedly reduced cellularity with relative lymphocytosis. A bone marrow biopsy performed on September 11, 2025, revealed

extensive fatty replacement, with hematopoietic tissue comprising about 20% and adipose tissue about 80% of the marrow. Both granulocytic and erythroid precursors were markedly decreased, consistent with very severe aplastic anemia. Further diagnostic evaluation was performed to investigate the cause of the severe pancytopenia and suspected aplastic crisis. In routine practice, the diagnostic workup of aplastic crisis includes viral studies such as parvovirus B19 PCR and serologies; however, these assays were not available at our institution. Other clinically relevant viral markers, including Epstein–Barr virus, cytomegalovirus, and hepatitis viruses, were assessed according to local laboratory capability and showed no evidence of acute infection. Bone marrow cytology and biopsy demonstrated markedly reduced cellularity, supporting the diagnosis of transient infection-related marrow suppression rather than primary aplastic anemia, particularly given the subsequent hematologic recovery following targeted antimicrobial therapy. Laboratory results are summarized in **Table 1**, and the temperature trend is shown in **Figure 1**.

On September 13, 2025, antimicrobial susceptibility testing identified carbapenem-resistant *E. coli* producing NDM metallo-lactamase (**Table 2**). In accordance with treatment guidelines,³ therapy was changed to polymyxin B plus amikacin. The patient's temperature normalized, hematologic indices gradually improved, and follow-up bone marrow examination showed recovery of hematopoiesis. (**Table 1, Figure 1**)

Infections are defined as community-acquired when they are made within 48 hours of admission in the absence of healthcare-associated risk factors, such as prior hospitalization, surgery, dialysis, residence in a long-term care facility, or use of invasive devices within the previous 12 months. This patient met all criteria for community-acquired infection, as blood cultures obtained on the first hospital day confirmed the presence of carbapenem-resistant *E. coli*.

Table 1. The laboratory findings of this patient.

Date	WBC (10 ⁹ /L)	ANC (10 ⁹ /L)	RBC (10 ¹² /L)	Hg (g/L)	Ret (10 ¹² /L)	PLT (10 ⁹ /L)	CRP (mg/L)
2025/9/10	0.25	0.02	2.5	76	0.0028	9	276.4
2025/9/13	0.32	0.02	2.18	67	0.005	36	199.3
2025/9/16	1.84	1.00	2.15	67	0.006	43	154.9
2025/9/19	3.29	2.16	2.49	77	0.020	28	108.8
2025/9/22	3.61	2.58	2.28	73	0.037	37	94.3
2025/9/25	3.72	2.41	2.18	68	0.048	78	116.4
2025/9/29	3.01	2.06	2.13	68	0.034	112	64.5

Abbreviation: WBC, white blood cell; ANC, absolute neutrophil count; RBC, red blood cell; Hb, hemoglobin; Ret, reticulocyte; PLT, platelet; CRP, C-reactive protein.

Table 2. The antimicrobial susceptibility testing for *E. coli* based on CLSI M100

Antibiotic	MIC (ug/ml)	D-D (mm)	Results
Ertapenem	>=8.0	-	Resistant
Amikacin	<=2.0	-	Susceptible
Ceftazidime	>=64.0	-	Resistant
Ciprofloxacin	>=4.0	-	Resistant
Ampicillin	>=32.0	-	Resistant
Piperacillin/Tazobactam	64.0	-	Resistant
Tobramycin	>=16.0	-	Resistant
Cefepime	8.0	-	Resistant
Ceftriaxone	>=64.0	-	Resistant
Gentamicin	>=16.0	-	Resistant
Imipenem	>=16.0	-	Resistant
Levofloxacin	>=8.0	-	Resistant
Cefotetan	>=64.0	-	Resistant
Ampicillin / Sulbactam	>=32.0	-	Resistant
Aztreonam	<=1.0	-	Susceptible
Trimethoprim-Sulfamethoxazole	>=320.0	-	Resistant
Amoxicillin /Clavulanic Acid	-	7	Resistant
Meropenem	-	15	Resistant
Cefuroxime	-	6	Resistant
Cefazolin	>=64.0	-	Resistant
Colistin	<=2	-	Intermediate

Abbreviation: MIC, Minimal Inhibitory Concentration; D-D, disk diffusion method.

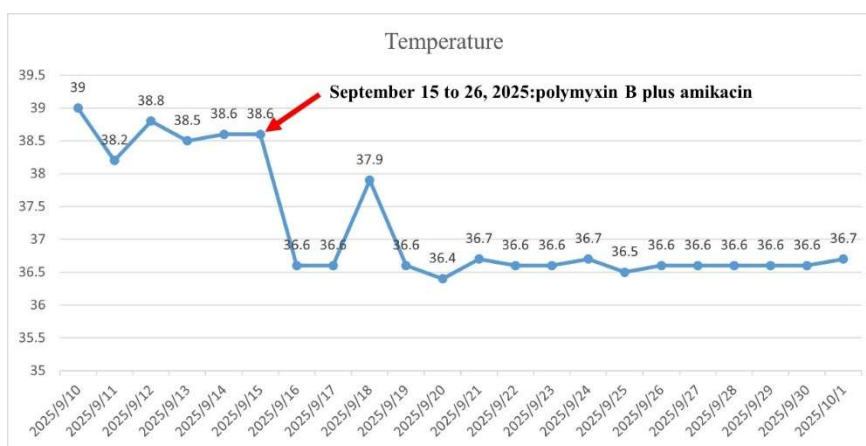


Figure 1. The temperature trend.

Epidemiologic data on community-acquired CRE bloodstream infections are limited because such cases are rare. Reported rates of community-associated or community-onset CRE infections range from 0.04% to 29.5%.² Potential sources include environmental reservoirs and animal-to-human transmission, which can lead to colonization. Previous studies suggest that community-onset CRE bacteremia may have a better prognosis than hospital-acquired infection.⁵

In this case, the infection also triggered an aplastic crisis. Transient bone-marrow suppression during severe bacterial sepsis is thought to result mainly from overwhelming systemic inflammation. High levels of cytokines such as TNF- α and IFN- γ can directly inhibit hematopoietic stem and progenitor cells, induce apoptosis, and impair erythroid and myeloid proliferation. In addition, bacterial components such as lipopolysaccharide can activate Toll-like receptor pathways within the marrow niche, further suppressing hematopoiesis.⁶⁻⁹ These effects are functional and reversible, and marrow recovery usually occurs after infection control measures are implemented. Published reports of bacterial sepsis presenting as aplastic-appearing marrow are uncommon, and most involve hospital-acquired infections in critically ill or immunocompromised patients.^{10,11} Unlike those

previous cases, our patient developed profound but reversible marrow hypoplasia following community-acquired CRE bacteremia, highlighting that even community infections can cause transient bone-marrow failure when systemic inflammation is severe. This case reinforces the importance of early pathogen identification and targeted therapy to promptly reverse marrow suppression and prevent progression to irreversible marrow failure.

This case has two notable limitations. First, molecular genotyping of the resistance determinants was not performed, preventing detailed characterization of the genetic context of NDM production and limiting epidemiologic interpretation. Second, parvovirus B19 molecular testing and serology were not available at our institution, limiting our ability to exclude B19-associated aplastic crisis definitively. However, the negative results from other viral evaluations and the rapid recovery of hematopoiesis after targeted antibacterial therapy support the interpretation of transient sepsis-related marrow suppression.

The study protocol was approved by the Medical Ethics Committee of the First People's Hospital of Zigong. The patient gave informed consent for the publication of this case report.

Yu Chen¹, Jin Zhang² and Wenqiang Kong³.

¹ Department of Clinical Laboratory, Zigong First People's Hospital, Zigong, 643000, Sichuan, China

² Department of Hematology, Zigong First People's Hospital, Zigong, 643000, Sichuan, China.

³ Department of Pharmacy, Zigong First People's Hospital, Zigong, 643000, Sichuan, China.

Competing interests: The authors declare no conflict of Interest.

Correspondence to: Wenqiang Kong. Department of Pharmacy, Zigong First People's Hospital, No. 42, Shangyihao Branch Road, Zigong, 643000, Sichuan, China. E-mail: wqkongpharmacist@outlook.com

References:

1. WHO bacterial priority pathogens list, 2024 (2024-05-17): Bacterial pathogens of public health importance to guide research, development and strategies to prevent and control antimicrobial resistance. <https://www.who.int/publications/i/item/9789240093461>
2. Kelly AM, Mathema B, Larson EL. Carbapenem-resistant Enterobacteriaceae in the community: a scoping review. *Int J Antimicrob Agents*. 2017 Aug;50(2):127-134. <https://doi.org/10.1016/j.ijantimicag.2017.03.012> PMID:28647532 PMCID:PMC5726257
3. Zeng, M., et al., Guidelines for the diagnosis, treatment, prevention and control of infections caused by carbapenem-resistant gram-negative bacilli. *Journal of microbiology, immunology, and infection = Wei mian yu gan ran za zhi* 2023, 56 (4), 653-671. <https://doi.org/10.1016/j.jmii.2023.01.017> PMID:36868960
4. Thaden, J. T., et al., Rising rates of carbapenem-resistant enterobacteriaceae in community hospitals: a mixed-methods review of epidemiology and microbiology practices in a network of community hospitals in the southeastern United States. *Infection control and hospital epidemiology* 2014, 35 (8), 978-83. <https://doi.org/10.1086/677157> PMID:25026612 PMCID:PMC4217156
5. Boutzoukas, A. E., et al., Increased mortality in hospital- compared to community-onset carbapenem-resistant enterobacterales infections. *The Journal of antimicrobial chemotherapy* 2024, 79 (11), 2916-2922. <https://doi.org/10.1093/jac/dkac306> PMID:39236214 PMCID:PMC11531819
6. Selleri, C., et al., Interferon-gamma and tumor necrosis factor-alpha suppress both early and late stages of hematopoiesis and induce programmed cell death. *Journal of cellular physiology* 1995, 165 (3), 538-46. <https://doi.org/10.1002/jcp.1041650312> PMID:7593233
7. Espinoza, J. L., et al., Microbe-Induced Inflammatory Signals Triggering Acquired Bone Marrow Failure Syndromes. *Frontiers in immunology* 2017, 8, 186. <https://doi.org/10.3389/fimmu.2017.00186>
8. Nagaraju, N., et al., Bone Marrow Changes in Septic Shock: A Comprehensive Review. *Cureus* 2023, 15 (7), e42517. <https://doi.org/10.7759/cureus.42517>
9. Davis, F. M., et al., Sepsis Induces Prolonged Epigenetic Modifications in Bone Marrow and Peripheral Macrophages Impairing Inflammation and Wound Healing. *Arteriosclerosis, thrombosis, and vascular biology* 2019, 39 (11), 2353-2366. <https://doi.org/10.1161/ATVBAHA.119.312754> PMID:31644352 PMCID:PMC6818743
10. Wang, H.-W., et al., Acquired Aplastic Anemia After Nosocomial Serratia marcescens Bone Marrow Infection in an Elderly Patient. *International Journal of Gerontology* 2011, 5 (4), 222-224. <https://doi.org/10.1016/j.ijge.2011.12.002>

11. Rajendran, A., et al., Severe systemic infection masking underlying childhood leukemia. *Indian J Hematol Blood Transfus* 2013, 29 (3), 167-70.
<https://doi.org/10.1007/s12288-012-0166-7>
PMid:24426366 PMCID:PMC3710556
12. Giordano, P., et al., An Epidemic of Parvovirus B19-Induced Aplastic Crises in Pediatric Patients with Hereditary Spherocytosis Following the COVID-19 Pandemic: A Single-Center Retrospective Study. *Children (Basel, Switzerland)* 2025, 12 (6).
<https://doi.org/10.3390/children12060772>
PMid:40564730 PMCID:PMC12191504