

Meropenem-Induced Thrombocytosis: A Case Report

Review began 10/10/2026
Review ended 10/25/2026
Published 10/28/2026

© Copyright 2026
Guella et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118731

Adnane Guella¹, Ehab Kabk², Rabab Z. Muhammad², Rasha M. Elagamal², Thamer E. Samha², Aiat Ghadban²

1. Nephrology, University Hospital Sharjah, Sharjah, ARE 2. Internal Medicine, University Hospital Sharjah, Sharjah, ARE

Corresponding author: Adnane Guella, guella@gmail.com

Abstract

Thrombocytosis is a rarely reported adverse event associated with meropenem. A 64-year-old woman received meropenem for bilateral multifocal bacterial nephritis. Her urine culture grew multidrug-resistant extended-spectrum β -lactamase (ESBL)-producing *Escherichia coli*. On day 7 of meropenem therapy, thrombocytosis developed despite substantial clinical improvement and declining inflammatory markers. Meropenem was subsequently discontinued and replaced with ceftazidime-avibactam. The platelet count peaked after meropenem withdrawal before progressively declining and normalizing approximately 14 days after the last meropenem administration. A Naranjo score of five indicated a probable adverse drug reaction, although delayed infection-related reactive thrombocytosis could not be conclusively excluded. This case highlights the importance of monitoring platelet trends and considering medication-related causes when thrombocytosis develops during antimicrobial treatment.

Categories: Hematology, Internal Medicine, Infectious Disease

Keywords: acute focal bacterial nephritis, adverse drug reaction, drug-induced thrombocytosis, meropenem, meropenem-induced thrombocytosis

Introduction

Thrombocytosis, defined as a platelet count exceeding $450 \times 10^9/L$, is most commonly reactive in origin, accounting for approximately 88-97% of cases in adults [1]. Common causes include infection, inflammation, iron deficiency anemia, malignancy, surgery, and drug reactions [1,2]. Drug-induced thrombocytosis is relatively uncommon and may be difficult to distinguish from other causes of reactive thrombocytosis, particularly in patients being treated for active infections.

Carbapenems are broad-spectrum β -lactam antibiotics frequently used to treat severe infections caused by extended-spectrum β -lactamase (ESBL)-producing *Enterobacterales* [3]. Although generally well tolerated, carbapenems have been associated with hematological adverse reactions, including thrombocytopenia and, less frequently, thrombocytosis [4]. Meropenem-associated thrombocytosis has been described in published case reports, but its true incidence remains uncertain [4].

Distinguishing drug-associated thrombocytosis from infection-related reactive thrombocytosis remains challenging because both may occur during antimicrobial therapy, and platelet elevations may persist despite clinical improvement. We report a case of suspected meropenem-associated thrombocytosis in a 64-year-old woman treated for acute focal bacterial nephritis (AFBN). The case highlights the diagnostic challenge posed by increasing platelet counts despite improving inflammatory markers, followed by platelet normalization after meropenem discontinuation.

Case Presentation

A 64-year-old woman was admitted with a three-day history of high-grade fever, dysuria, nausea, headache, and abdominal pain radiating to the back. Her medical history was significant for hypertension, treated with amlodipine 5 mg daily. She had no previous history of thrombocytosis, hematological disorders, malignancy, or iron deficiency anemia. On examination, she appeared pale and was febrile, with a normal heart rate and blood pressure. Bilateral costovertebral angle tenderness was present. Admission vital signs and laboratory findings are summarized in Table 1. Laboratory investigations revealed leukocytosis and elevated inflammatory markers, while the platelet count was within normal limits ($295 \times 10^9/L$). Urinalysis was positive for nitrites, and renal function and serum electrolytes were normal. Contrast-enhanced computed tomography (CT) demonstrated bilateral multifocal bacterial nephritis (Figure 1). Empirical intravenous ceftriaxone (2 g once daily) was initiated.

How to cite this article

Guella A, Kabk E, Muhammad R Z, et al. (October 28, 2026) Meropenem-Induced Thrombocytosis: A Case Report. Cureus 18(10): e118731. DOI 10.7759/cureus.118731

Parameter	Result	Reference range	Unit
Temperature	38.0	36.0-37.5	°C
Heart rate	75	60-100	beats/min
Blood pressure	115/68	90/60-120/80	mmHg
WBC	18.8	4.0-10.0	×10 ⁹ /L
Platelet count	295	150-410	×10 ⁹ /L
CRP	318	0-8	mg/L
Procalcitonin	0.6	0.01-0.15	ng/mL
Creatinine	74	49-90	μmol/L
Sodium	138	136-145	mmol/L
Potassium	4.1	3.5-5.1	mmol/L
Urine nitrite	Positive	Negative	-

TABLE 1: Admission vital signs and laboratory findings.

CRP, C-reactive protein; WBC, white blood cell count

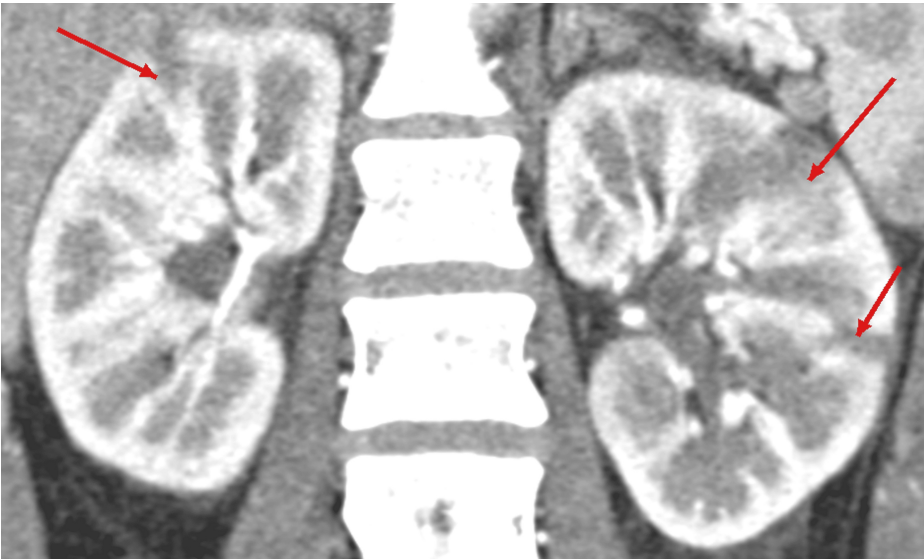


FIGURE 1: Coronal contrast-enhanced computed tomography (CT) image demonstrating bilateral multifocal areas of decreased renal parenchymal enhancement (red arrows), consistent with acute focal bacterial nephritis (AFBN).

On hospital day 3, ceftriaxone was replaced with intravenous meropenem (1 g every eight hours) because of inadequate clinical improvement. Urine culture identified multidrug-resistant ESBL-producing *Escherichia coli* susceptible to meropenem, supporting the antibiotic change. Blood cultures remained negative.

Serial laboratory findings during antimicrobial treatment are presented in Table 2 and illustrated in Figure 2. On hospital day 9, corresponding to day 7 of meropenem therapy, thrombocytosis was observed, with a platelet count of 740 × 10⁹/L. This occurred despite substantial clinical improvement and a progressive decline in inflammatory markers, particularly C-reactive protein (CRP), which had decreased from 318 mg/L at admission to 51 mg/L.

Laboratory parameter	Reference range	D1	D3	D5	D9	D11	D12	D13	D14	D15	D17	D18	D25
CRP (mg/L)	0-8	318	293	235	51	21	-	10	-	6	-	-	3
WBC ($\times 10^9/L$)	4.0-10.0	18.8	16.4	14.0	9.5	9.5	-	9.1	-	7.0	-	-	7.9
PLT ($\times 10^9/L$)	150-410	295	326	386	740	848	-	856	791	705	662	513	358
Antibiotic	-	CTX	MP (D1)	MP (D3)	MP (D7)	MP (D9)	CA (D1)	CA	CA	CA	CA	CA	CA

TABLE 2: Serial laboratory findings and antibiotic therapy during hospitalization, demonstrating thrombocytosis during meropenem treatment despite improvement in inflammatory markers.

CA, ceftazidime-avibactam; CRP, C-reactive protein; CTX, ceftriaxone; D, day; MP, meropenem; PLT, platelet count; WBC, white blood cell count; -, not measured

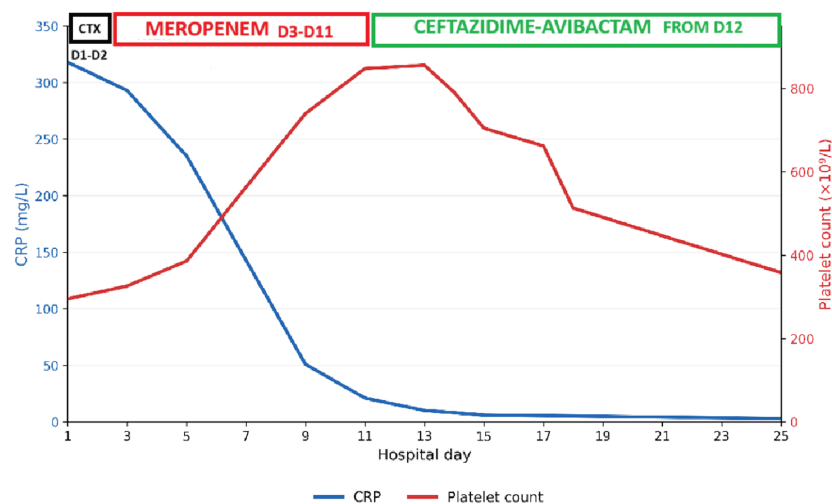


FIGURE 2: Changes in platelet count and CRP levels during hospitalization. Platelet count increased during meropenem therapy despite declining CRP levels. The periods of meropenem administration and its subsequent replacement with ceftazidime-avibactam are indicated.

CRP: C-reactive protein; CTX: ceftriaxone

The platelet count continued to increase, reaching $848 \times 10^9/L$ on hospital day 11 (meropenem treatment day 9), while CRP had decreased further to 21 mg/L. These laboratory results became available and were reviewed on hospital day 12, prompting discontinuation of meropenem before administration of the scheduled 10th-day doses. Intravenous ceftazidime-avibactam (2.5 g every eight hours) was initiated to continue antimicrobial therapy for AFBN.

On hospital day 13, following meropenem discontinuation, the platelet count reached a peak of $856 \times 10^9/L$ before progressively declining. The platelet count subsequently normalized to $358 \times 10^9/L$ on hospital day 25, approximately 14 days after the last meropenem administration. This platelet trend following withdrawal raised suspicion of a drug-associated reaction, although infection-related reactive thrombocytosis remained a possible alternative explanation.

During hospitalization, the patient received subcutaneous enoxaparin 4,000 IU once daily for venous thromboembolism prophylaxis. No other concomitant medications were reported apart from her regular

amlodipine. No thrombotic or bleeding complications were observed, and no antiplatelet therapy was administered.

The patient completed the prescribed intravenous antimicrobial treatment and was discharged in excellent clinical condition. The prolonged hospital stay was primarily related to the need for intravenous therapy, as outpatient parenteral antibiotic administration was not feasible because of poor venous access, together with the complexity of the multidrug-resistant infection and bilateral multifocal renal involvement.

Discussion

Despite the widespread use of carbapenems, carbapenem-associated thrombocytosis remains sparsely reported. A recent systematic review of carbapenem-associated platelet abnormalities, with a literature search extending through June 30, 2025, identified 42 cases. Among these, thrombocytosis was reported in 20 patients, including 12 cases associated with meropenem [4]. Meropenem was the most frequently implicated carbapenem among all reported platelet abnormalities (64.3%), followed by imipenem (26.2%). However, these percentages represent the distribution of published cases rather than the incidence of platelet abnormalities among patients receiving these antibiotics.

The mechanism underlying meropenem-associated thrombocytosis remains uncertain. One possible explanation involves the interleukin-6 (IL-6)-thrombopoietin pathway, in which IL-6 stimulates thrombopoietin production, subsequently increasing megakaryocyte activity and platelet production [5]. However, this mechanism has not been established specifically for meropenem. Infection is also a major cause of reactive thrombocytosis, as demonstrated by Rose et al. in their epidemiological study [6]. Consequently, the development of thrombocytosis during meropenem therapy presents a diagnostic challenge, as platelet elevation may be related to the underlying infection or to a drug-associated reaction.

In our patient, thrombocytosis developed on day 7 of meropenem therapy despite substantial clinical improvement and a progressive decline in inflammatory markers, particularly CRP, which remained above the normal range. The possibility of infection-related reactive thrombocytosis was considered. However, the continued increase in platelet count despite clinical improvement, the absence of other identified pre-existing causes, and progressive platelet normalization following meropenem discontinuation supported a drug-associated reaction.

Nevertheless, delayed infection-related reactive thrombocytosis could not be conclusively excluded, as platelet elevations may persist during recovery from acute infections despite declining inflammatory markers. The positive dechallenge further supported a temporal association with meropenem. Accordingly, Question 5 of the Naranjo algorithm was scored as unknown (0). The resulting total score was 5, indicating a probable adverse drug reaction (Table 3) [7].

No.	assessment question	Answer	Score
1	Are there previous conclusive reports of this reaction?	Yes	+1
2	Did the adverse event appear after administration of the suspected drug?	Yes	+2
3	Did the reaction improve after discontinuation of the drug?	Yes	+1
4	Did the reaction reappear after readministration?	Not assessed	0
5	Are there alternative causes that could independently explain the reaction?	Do not know*	0
6	Did the reaction reappear after placebo administration?	Not assessed	0
7	Was the drug detected in toxic concentrations?	Not assessed	0
8	Was a dose-response relationship demonstrated?	Not assessed	0
9	Was a similar reaction documented during previous exposure?	Not documented	0
10	Was the adverse event confirmed by objective evidence?	Yes	+1
Total		Probable	5

TABLE 3: Naranjo adverse drug reaction probability assessment for suspected meropenem-associated thrombocytosis.

*Infection-related reactive thrombocytosis could not be conclusively excluded as an alternative explanation. Therefore, Question 5 was scored as unknown (0).

The timing of thrombocytosis in our patient was consistent with previously published observations. Pan et al. reported a median time to platelet abnormality of three days (range: 0.125-12 days), with 79.5% of cases occurring within the first week of carbapenem therapy. Additionally, 54.8% of patients were older than 60 years [4]. In our patient, meropenem was administered for nine days, and the platelet count reached its peak of 856 × 10⁹/L on hospital day 13, approximately two days after the last meropenem administration. This timing was consistent with the reported interval to peak platelet count in meropenem-associated thrombocytosis (4-18 days) [4]. The platelet count subsequently normalized to 358 × 10⁹/L on hospital day 25, approximately 14 days after the last meropenem administration, within the recovery intervals reported in previous literature (1-26 days and 4-18 days, respectively) [4,8]. These observations support consideration of platelet count monitoring during meropenem treatment, particularly within the first week, although available evidence does not establish a specific monitoring schedule.

Secondary thrombocytosis is generally transient, and management primarily involves addressing the underlying cause. Antiplatelet therapy is not routinely indicated because the risk of thrombosis associated with reactive thrombocytosis is generally low, although individual risk factors and clinical circumstances should be considered [9]. In our patient, the platelet count peaked at 856 × 10⁹/L without observed thrombotic or bleeding complications. She received subcutaneous enoxaparin 4,000 IU daily for venous thromboembolism prophylaxis during hospitalization. No antiplatelet therapy was initiated because thrombocytosis was considered secondary, and there was no documented thrombosis or other specific indication for antiplatelet treatment. Nevertheless, her overall thrombotic risk required consideration independently of the platelet count, particularly in the context of hospitalization and acute infection.

The clinical relevance of this case lies in the challenge of distinguishing suspected meropenem-associated thrombocytosis from a delayed reactive response to infection. Although the temporal association, positive dechallenge, and Naranjo assessment support a probable drug-associated reaction, definitive causality cannot be established from a single case. These findings emphasize the importance of interpreting platelet trends in conjunction with the clinical course, inflammatory markers, and medication exposure.

Conclusions

We report a case of suspected meropenem-associated thrombocytosis in a 64-year-old woman treated for bilateral multifocal bacterial nephritis. The development of thrombocytosis despite clinical improvement and declining inflammatory markers, followed by platelet normalization after meropenem discontinuation, supports a probable drug-associated reaction, as indicated by a Naranjo score of five. However, delayed infection-related reactive thrombocytosis cannot be conclusively excluded. This case highlights the importance of monitoring platelet trends during antimicrobial therapy and considering drug-related causes when unexplained thrombocytosis develops.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Adnane Guella

Drafting of the manuscript: Adnane Guella, Rabab Z. Muhammad, Thamer E. Samha, Aiat Ghadban, Ehab Kabk, Rasha M. Elagamal

Acquisition, analysis, or interpretation of data: Rabab Z. Muhammad, Thamer E. Samha, Aiat Ghadban, Ehab Kabk, Rasha M. Elagamal

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Bleeker JS, Hogan WJ: Thrombocytosis: diagnostic evaluation, thrombotic risk stratification, and risk-based management strategies. *Thrombosis*. 2011, 2011:536062. [10.1155/2011/536062](https://doi.org/10.1155/2011/536062)
2. Vo QT, Thompson DF: A review and assessment of drug-induced thrombocytosis. *Ann Pharmacother*. 2019, 53:523-36. [10.1177/1060028018819450](https://doi.org/10.1177/1060028018819450)
3. Zhanel GG, Wiebe R, Dilay L, et al.: Comparative review of the carbapenems. *Drugs*. 2007, 67:1027-52. [10.2165/00003495-200767070-00006](https://doi.org/10.2165/00003495-200767070-00006)
4. Pan J, Peng W, Ye C, et al.: Carbapenem-induced platelet abnormalities: a systematic review literature analysis of platelet abnormality caused by carbapenems. *Infect Drug Resist*. 2025, 18:6059-70. [10.2147/IDR.S555737](https://doi.org/10.2147/IDR.S555737)
5. Kaser A, Brandacher G, Steurer W, et al.: Interleukin-6 stimulates thrombopoiesis through thrombopoietin: role in inflammatory thrombocytosis. *Blood*. 2001, 98:2720-5. [10.1182/blood.v98.9.2720](https://doi.org/10.1182/blood.v98.9.2720)
6. Rose SR, Petersen NJ, Gardner TJ, Hamill RJ, Trautner BW: Etiology of thrombocytosis in a general medicine population: analysis of 801 cases with emphasis on infectious causes. *J Clin Med Res*. 2012, 4:415-23. [10.4021/jocmr1125w](https://doi.org/10.4021/jocmr1125w)
7. Naranjo CA, Busto U, Sellers EM, et al.: A method for estimating the probability of adverse drug reactions. *Clin Pharmacol Ther*. 1981, 30:239-45. [10.1038/clpt.1981.154](https://doi.org/10.1038/clpt.1981.154)
8. Chang X, Han L, Liu Y, et al.: Analysis of adverse drug reactions caused by carbapenems and review of related literature. *J Chin Pharm Sci*. 2023, 32:835.
9. Rokkam VR, Killeen RB, Kotagiri R: Secondary thrombocytosis. *StatPearls*. StatPearls Publishing, Treasure Island, FL; 2026.