Jurnal Pengabdian Ilmu Kesehatan
Volume 6, Nomor 1, Maret 2026
E-ISSN: 2827-9204; P-ISSN: 2827-9212; Hal. 397-412
DOI: https://doi.org/10.55606/jpikes.v6i1.7065
Tersedia: https://journalshub.org/index.php/JPIKes

Vancomycin Induced Thrombocytopenia in Sepsis Patient

Prisilia Rianni Kaunang[1*], Eka Yudha Lantang[2], Waldemar Siahaan[3]
[1]Jurusan Anestesiologi dan Terapi Intensif, Fakultas Kedokteran, Universitas Sam Ratulangi, Indonesia
[2-3]Prof. Dr. R. D. Kandou Rumah Sakit Umum Manado, Indonesia
Email: riannikaunang@gmail.com[1], ekalantang@gmail.com[2], waldemarsiahaan@yahoo.com[3]*

**Author Correspondence: riannikaunang@gmail.com*

Riwayat Artikel:
Naskah Masuk: 19 Februari 2026;
Revisi: 13 Maret 2026;
Diterima: 28 Maret 2026;
Terbit: 31 Maret 2026

***Keywords**: Critically Ill Patients; Drug-Induced Thrombocytopenia; Sepsis; Vancomycin; Vancomycin-Induced Thrombocytopenia.*

***Abstract:** Vancomycin is a widely used glycopeptide antibiotic for severe Gram-positive infections, yet it may cause rare but serious adverse effects such as vancomycin-induced thrombocytopenia (VIT), which is often under-recognized in critically ill patients. This study aims to describe a case of suspected VIT in a septic patient and to highlight the diagnostic challenges and clinical implications in intensive care settings. A case report method was applied, involving detailed clinical observation, laboratory monitoring, and evaluation of temporal relationships between drug exposure and hematologic changes. The patient, a 70-year-old male with multiple comorbidities and severe sepsis, developed a sudden and profound decline in platelet count following approximately one week of vancomycin therapy. Despite the presence of other potential causes of thrombocytopenia, including sepsis and disseminated intravascular coagulation, the rapid onset and severity of platelet reduction, along with its temporal association with vancomycin administration, strongly suggested an immune-mediated mechanism. The clinical course was further complicated by multiorgan failure and multidrug-resistant infections, ultimately leading to mortality. This case underscores the importance of early recognition of drug-induced thrombocytopenia, careful evaluation of differential diagnoses, and timely discontinuation of the offending agent. Increased awareness of VIT among clinicians is essential to reduce morbidity and improve outcomes in critically ill patients.*

1. INTRODUCTION

Vancomycin is a tricyclic glycopeptide antibiotic widely used in the management of severe Gram-positive infections, particularly those caused by methicillin-resistant *Staphylococcus aureus* (MRSA) and coagulase-negative *Staphylococcus* species. It is also considered an important alternative in patients with hypersensitivity to beta-lactam antibiotics. Despite its clinical utility, vancomycin is associated with a spectrum of adverse effects, most commonly nephrotoxicity, infusion-related reactions, and hypersensitivity. Hematologic complications are less frequently reported but include neutropenia, agranulocytosis, pancytopenia, and thrombocytopenia.

Vancomycin-induced thrombocytopenia (VIT) is a rare but potentially life-threatening adverse drug reaction that remains under-recognized in clinical practice. First described in 1985, VIT is thought to be primarily immune-mediated, involving drug-dependent antibodies that bind to platelet glycoproteins in the presence of vancomycin, leading to platelet

destruction.[3,4] Non-immune mechanisms, such as direct platelet toxicity and activation, have also been proposed. The clinical significance of VIT lies in its potential to cause severe thrombocytopenia and bleeding complications, particularly in critically ill patients.

Recent real-world evidence suggests that the incidence of VIT may be higher than previously expected. A retrospective study involving 1,269 patients reported an incidence of 3.3%, with nearly half of affected patients experiencing severe (grade 3–4) thrombocytopenia. The onset of thrombocytopenia typically occurs after several days of therapy, with a median onset of approximately 9 days following vancomycin initiation, and platelet recovery generally observed after discontinuation of the drug. Several risk factors have been identified, including higher severity of illness (qSOFA score ≥2), underlying renal disease, prolonged duration of vancomycin therapy (≥8 days), lower baseline platelet count, and elevated blood urea nitrogen levels.

Importantly, thrombocytopenia is also a common hematologic abnormality in sepsis, often resulting from complex mechanisms such as disseminated intravascular coagulation, bone marrow suppression, and increased platelet consumption. This overlap creates a diagnostic challenge in distinguishing sepsis-related thrombocytopenia from drug-induced causes such as VIT.[6,7] Failure to recognize VIT may lead to continued exposure to the offending agent, thereby increasing the risk of bleeding, morbidity, and mortality. Given the widespread use of vancomycin in septic patients and the potential severity of VIT, early recognition and prompt discontinuation of the drug are essential. However, due to its rarity and nonspecific presentation, VIT is often overlooked or misattributed to other causes. Therefore, case reports remain valuable in highlighting this condition, improving clinician awareness, and aiding in early diagnosis.

In this report, we present a case of vancomycin-induced thrombocytopenia in a septic patient managed in the intensive care unit, highlighting the diagnostic challenges, clinical course, as well as critical care and hemodynamic management considerations associated with this condition.

2. CASE REPORT

Patient Identity and Clinical Course

The patient, Mr. FP, is a 70-year-old male with a body weight of 69 kg and a height of 170 cm. He was admitted to the ICU on April 4, 2025, at 08:48 and underwent a total hospitalization period of 33 days. The patient subsequently died on October 7, 2025, at 19:03.

Initial ICU Admission and History

The patient was referred from Sentra Medika Hospital with a chief complaint of sudden decreased consciousness that had persisted for approximately 10 days. A prior CT scan performed on April 21, 2025, demonstrated a hyperdense lesion in the left basal region, accompanied by obstructive hydrocephalus. The patient underwent external ventricular drainage (EVD) placement on the same date. Postoperatively, the patient's level of consciousness worsened, necessitating intubation. The patient denied symptoms such as seizures, headaches, unilateral weakness, hearing or visual disturbances, fever, and chronic cough. His past medical history included obstructive hydrocephalus managed with EVD on April 21, 2025. He also had a history of hypertension for approximately 15 years but had poor medication adherence, previously taking amlodipine before being switched to candesartan 16 mg once daily. Additionally, the patient had a history of dyslipidemia for about 5 years, treated with Lipitor (atorvastatin) 10 mg once daily. Neurosurgical interventions included VP shunt placement on April 9, 2025 (right side), revision on June 9, 2025 (right side), and subsequent VP shunt placement on the left side on September 21, 2025.

ICU Procedures and Airway Management

The patient underwent multiple airway interventions during ICU care. Initial intubation with an endotracheal tube (ETT) size 7.5 was performed on April 4, 2025, at 08:50. He was extubated later the same day at 12:05. A central venous catheter (CVC) was inserted in the right jugular vein at Sentra Medika Hospital. Reintubation using ETT size 7.5 was performed on September 5, 2025, at 11:00. Another CVC was placed in the right subclavian vein on September 28, 2025, at 02:30. Due to prolonged airway needs, a tracheostomy was performed on September 13, 2025, at 10:15.

Initial Assessment

On initial ICU assessment using the ABCDE approach, the airway was clear. Breathing evaluation showed a respiratory rate of 14 breaths per minute with oxygen saturation of 98% on a simple mask. Circulation assessment revealed blood pressure of 148/88 mmHg, mean arterial pressure (MAP) of 108 mmHg, and heart rate of 86 beats per minute. Disability assessment indicated decreased consciousness with GCS under sedation (dexmedetomidine 0.2 mcg/kg/hour), approximately E3M5Vt, and anisocoria (unequal pupils). Exposure findings showed warm extremities with capillary refill time less than 2 seconds.

Physical Examination and Supporting Findings

General condition was severely ill. Airway was patent, with vesicular breath sounds and no adventitious sounds. The abdomen was soft with decreased bowel sounds, and there was no

edema in the extremities. Neurologically, the patient had decreased consciousness under sedation, with GCS E3M5Vt. Hemodynamically, the patient remained stable without signs of shock. Fluid management included administration of Ringer's lactate at 400 mL per hour (approximately 0.19 mL/kg/hour input), with urine output monitored. Renal function showed an estimated creatinine clearance of 75 mL/min.

On September 4, 2025, at the time of ICU admission, continuous bedside monitoring showed that the patient was hemodynamically stable but critically ill. The monitor displayed a respiratory rate of 14 breaths per minute, oxygen saturation of 99%, heart rate of 68 beats per minute, and blood pressure of 146/86 mmHg with a mean arterial pressure of approximately 109 mmHg. These findings indicate relatively stable vital signs under intensive monitoring, with adequate oxygenation and controlled circulation.

Laboratory evaluation prior to ICU admission (August 31, 2025) revealed leukocytosis with a white blood cell count of 18,770/µL, suggesting an ongoing inflammatory or infectious process. Red blood cell count was decreased at 3.95 million/µL, with hemoglobin of 12.1 g/dL and hematocrit of 34.6%, indicating mild anemia. Platelet count was within normal limits at 337,000/µL. Coagulation profile showed PT 13.9 seconds, INR 1.01, and APTT 27.4 seconds, all within normal range. Renal function tests demonstrated elevated urea (34 mg/dL) and reduced estimated glomerular filtration rate (eGFR) of 75 mL/min/1.73 m², indicating mild renal impairment. Electrolytes showed sodium 132 mmol/L, suggesting mild hyponatremia. Liver enzymes were mildly elevated, with SGOT/SGPT at 49/80 U/L. Serologic testing for HIV, HBsAg, and anti-HCV was non-reactive.

Electrocardiography performed on September 1, 2025, showed sinus rhythm with a heart rate of 93 beats per minute, normal electrical axis, and evidence of left ventricular hypertrophy with strain pattern.

Laboratory results on September 4, 2025, demonstrated persistent leukocytosis (11.16 $\times 10^3$/µL) with marked neutrophilia (95.2%) and lymphopenia (3.9%), consistent with acute stress or infection. Hemoglobin remained slightly decreased at 12.2 g/dL, and hematocrit was 34.8%. Platelet count increased to 489,000/µL. Renal function showed urea of 48 mg/dL and creatinine of 1.0 mg/dL, with eGFR remaining at 75 mL/min/1.73 m². Inflammatory markers were elevated, with C-reactive protein (CRP) at 12 mg/L and D-dimer markedly increased at 5.1 µg/mL. Liver function tests revealed mild elevation of ALT (58 U/L), while AST remained within normal limits. Albumin level was slightly decreased at 3.40 g/dL. Blood glucose was within normal range at 121 mg/dL. Electrolyte levels were within normal limits, with sodium 135 mmol/L, potassium 4.4 mmol/L, and chloride 97 mmol/L. Arterial blood gas analysis on

the second day of ICU care demonstrated a mixed acid–base disorder. The pH was 7.507, indicating alkalemia. Partial pressure of carbon dioxide ($PaCO_2$) was significantly reduced at 17.5 mmHg, consistent with respiratory alkalosis. Bicarbonate (HCO_3^-) was decreased at 13.8 mmol/L, with a base excess of −9 mmol/L, indicating underlying metabolic acidosis. Oxygenation was adequate, with PaO_2 of 85 mmHg and oxygen saturation of 98%. Lactate level was within normal limits at 1.5 mmol/L.

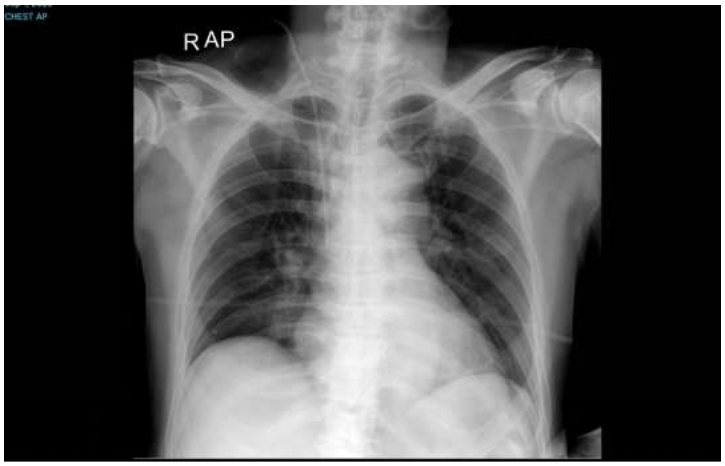

Figure 1. Patient chest radiograph on september 1st, 2025.

A chest X-ray performed at RS Kandou on September 1, 2025, showed no apparent abnormalities. There were no visible signs of active pulmonary pathology, and the cardiac and pulmonary structures appeared within normal limits on preliminary assessment.

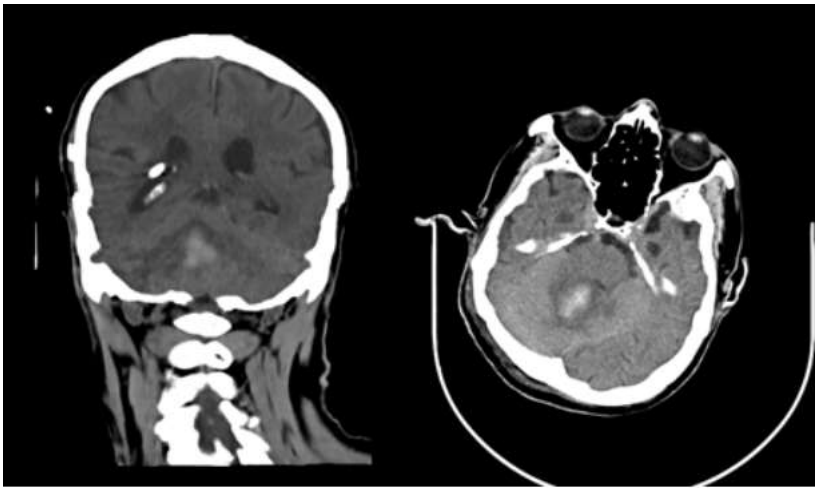

Figure 2. Head CT-Scan on september 1st, 2025.

A head CT scan performed on the same date, compared with the previous study from August 21, 2025, demonstrated several important findings. There was a hyperdense lesion located in the right cerebellar region and vermis, with an estimated volume of approximately 6.2 cc. This finding is consistent with an intracerebral hemorrhage (ICH) in the resorption phase, suggesting a subacute stage of bleeding. Additionally, there was evidence of dilatation

of both lateral ventricles and the third ventricle, although this appeared reduced compared to the prior CT scan, indicating improvement of obstructive hydrocephalus. An external ventricular drain (EVD) was visualized in the right lateral ventricle, consistent with prior neurosurgical intervention. Furthermore, multiple hypodense lesions were noted in the centrum semiovale, corona radiata, and periventricular regions, which are suggestive of chronic cerebral small vessel disease, classified as Fazekas grade 1, indicating mild microvascular ischemic changes.

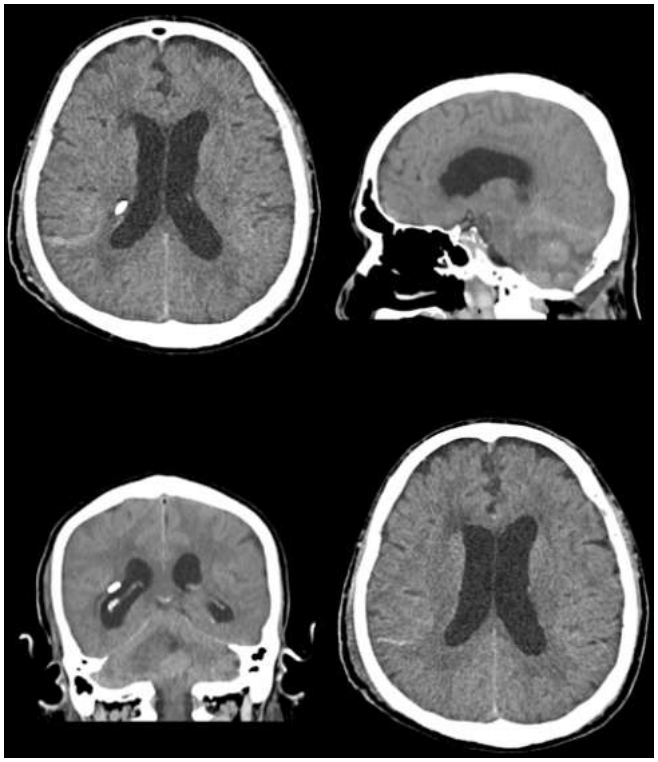

Figure 3. Follow-up CT scan on September 5, 2025.

During the ICU stay, serial laboratory findings showed progressive and dynamic abnormalities involving hematologic, renal, hepatic, inflammatory, and coagulation parameters, indicating worsening critical illness. Initially, on September 5, there was significant leukocytosis (25.54 $\times 10^3/\mu L$) that gradually declined to near-normal levels by mid-September. However, a second marked increase occurred toward the end of September, peaking at 43.87 $\times 10^3/\mu L$ on October 4, suggesting ongoing or recurrent severe infection or sepsis. This was accompanied by persistent neutrophilia (up to 95.8%) and relative lymphopenia, consistent with a septic response. Platelet counts were initially normal but later fluctuated and dropped sharply in late September, reaching 17,000/µL on October 3 before slight recovery, suggesting consumptive coagulopathy, sepsis-related thrombocytopenia, or bone marrow suppression. Hematologic parameters showed progressive anemia, with hemoglobin declining from 13.7 g/dL to approximately 7–9 g/dL.

Renal function deteriorated over time, with early elevated urea (82 mg/dL) and relatively preserved creatinine, followed by a significant rise in both parameters in late September, with urea peaking at 444 mg/dL and creatinine at 4.2 mg/dL, alongside a decline in eGFR to 13 mL/min/1.73m², consistent with severe acute kidney injury. Electrolyte evaluation showed mild hyponatremia (Na 128–134 mmol/L), relatively stable potassium levels, a tendency toward hypochloremia, and significant hypocalcemia toward the end (as low as 6.77 mg/dL). Magnesium levels fluctuated within or slightly above normal limits.

Liver function tests revealed marked transaminitis early in the course, with AST and ALT peaking at 488 U/L and 609 U/L, respectively, suggestive of acute hepatic injury, possibly due to hypoperfusion, sepsis, or drug effects. Although levels gradually decreased, they remained elevated. Hypoalbuminemia persisted (around 2.0–2.5 g/dL), indicating chronic inflammation, malnutrition, or hepatic dysfunction, while bilirubin showed only mild elevation.

Coagulation profiles demonstrated prolonged PT and elevated INR (up to 1.71), with intermittent APTT prolongation, consistent with coagulopathy, possibly due to sepsis-associated DIC or liver dysfunction. Inflammatory markers remained elevated, with CRP up to 48 mg/L and procalcitonin initially decreasing before rising sharply to 85.4 ng/mL, indicating severe bacterial infection. D-dimer levels were persistently high (>10 µg/mL), reflecting increased fibrinolysis and systemic inflammation. Blood glucose levels were generally controlled with occasional mild hyperglycemia.

Microbiological cultures from September 15, 2025, revealed multiple multidrug-resistant organisms. An aerobic culture identified ESBL-producing Klebsiella pneumoniae, sensitive only to amikacin and resistant to multiple antibiotics including ampicillin, ampicillin/sulbactam, cefazolin, ceftriaxone, and ciprofloxacin. Urine culture grew carbapenem-resistant Acinetobacter baumannii (CRAB), showing extensive resistance to nearly all tested antibiotics, with tigecycline demonstrating intermediate susceptibility. Similarly, CSF culture also identified CRAB, sensitive to amikacin but resistant to most other antibiotic classes, with tigecycline again showing intermediate susceptibility.

Table 1. The patient's laboratory results from September 5, 2025 to October 5, 2025.

Parameter	05-Sep	06-Sep	07-Sep	08-Sep	09-Sep	10-Sep	11-Sep	12-Sep	13-Sep	14-Sep	15-Sep	16-Sep	17-Sep	18-Sep	19-Sep	20-Sep
Leukosit (×10³/µL)	25.54	-	12.41	9.70	9.60	10.20	11.80	12.60	15.80	16.18	14.10	14.10	12.60	11.10	10.10	9.30
Trombosit	354000	-	168000	136000	160000	132000	126000	201000	155000	287000	298000	306000	249000	364000	303000	362000
Hb (g/dL)	13.7	-	10.7	9.7	10.0	10.7	11.0	10.1	10.2	10.4	9.3	9.4	8.0	7.5	7.2	7.4
Ht (%)	38.5	-	29.8	27.3	28.4	30.5	31.6	29.2	29.7	30.2	26.9	27.2	23.2	24.6	20.5	21.1
Eritrosit (×10⁶/µL)	4.45	-	3.49	3.07	3.20	3.39	3.54	3.29	3.32	3.41	3.01	3.03	-	2.43	2.30	2.34
Neutrofil (%)	-	-	88.3	-	-	-	-	-	-	-	87.5	85.3	-	-	-	-
Limfosit (%)	-	-	3.8	2.9	3.4	4.6	-	-	-	4.5	5.2	6.4	6.2	5.8	5.2	-
Monosit (%)	-	-	3.0	4.6	5.6	6.8	-	-	-	4.4	6.2	17.4	5.3	4.2	5.0	-
Ureum (mg/dL)	-	-	82	70	49	48	50	63	70	70	72	79	101	96	-	73
Kreatinin (mg/dL)	-	-	1.3	1.0	0.9	0.9	0.9	1.1	1.2	1.3	1.1	1.2	1.2	1.2	-	0.8
eGFR (mL/min/1.73m²)	-	-	55	75	86	86	86	67	60	55	67	60	60	60	-	90
Na (mmol/L)	-	-	-	138	134	134	132	130	128	131	134	134	133	132	132	133
K (mmol/L)	-	-	4.5	4.3	4.1	3.9	4.4	4.5	4.0	3.4	3.5	3.7	4.0	4.2	3.8	4.0
Cl (mmol/L)		-	97	97	98	93	91	93	89	85	90	92	93	92	94	92
Calcium		-	8.92	8.71	-	-	8.29	-	8.54	8.34	-	8.40	-	-	-	-
Magnesium	-	-	2.06	2.23	-	-	1.39	-	1.77	1.69	-	2.25	-	-	-	-
AST (U/L)	-	-	25	-	145	204	153	281	270	488	269	214	110	132	107	116
ALT (U/L)	-	-	31	-	200	223	274	358	425	609	390	386	230	213	176	171
Albumin (g/dL)	-	-	2.48	-	2.29	-	2.29	2.00	2.57	-	-	2.51	-	2.17	-	2.31
Bilirubin total	-	-	0.51	-	0.83	-	1.00	-	0.78	-	-	-	-	-	-	-
Bilirubin direct	-	-	0.35	-	0.69	-	0.73	-	0.25	-	-	-	-	-	-	-
PT (detik)	-	-	13.5/14.2	13.6/14.5	13.0/13.6	13.9/14.1	13.7/14.4	-	14.9/14.9	15.3/14.4	-	17.0/14.5	15.9/15.1	16.4/14.7	15.1/14.3	15.5/14.3
INR	-	-	0.98/1.03	1.00/1.05	0.93/0.98	1.01/1.02	1.00/1.05	-	1.09/1.09	1.13/1.02	-	1.27/1.06	1.18/1.10	1.22/1.07	1.11/1.03	1.14/1.03
APTT (detik)	-	-	27.3/30.5	41.5/31.0	27.8/31.0	34.2/30.5	28.9/30.3		29.8/29.8	28.8/31.4	-	35.1/30.5	49.9/31.2	38.5/32.3	27.3/31.4	29.3/30.1
GDS	-	-	105	-	124	174	145	-	93	126	-	-	106	104	127	-
CRP (mg/L)	12.0	-	48.0	-	48.0	-	24.0	-	-	-	48.00	-	-	-	-	-
Prokalsitonin (ng/mL)	-	-	-	-	3.45	1.85	1.09	-	-	-	0.302	-	0.254	-	-	-
D-dimer (µg/mL)	-	-	-	-	2.0	-	2.8	-	10.0	13.2	-	-	-	-	-	-

Tabel 2. (cont) The patient's laboratory results from September 5, 2025 to October 5, 2025.

Parameter	21-Sep	22-Sep	23-Sep	24-Sep	25-Sep	26-Sep	27-Sep	28-Sep	29-Sep	30-Sep	01-Oct	02-Oct	03-Oct	04-Oct	05-Oct
Leukosit (×10³/µL)	8.77	8.70	8.30	10.40	-	9.9	-	30.21	22.50	15.30	14.97	17.70	37.60	43.87	27.89
Trombosit	254000	422000	416000	455000	-	497000	-	194000	186000	158000	106000	50000	17000	35000	16000
Hb (g/dL)	8.2	8.5	8.1	8.5	-	8.4	-	7.4	8.2	9.0	9.2	9.4	9.0	8.6	8.1
Ht (%)	24.1	24.3	22.3	25.8	-	23.5	-	-	23.7	26.7	25.7	27.5	26.8	24.1	23.2
Eritrosit (×10⁶/µL)	2.69	2.75	2.52	2.71	-	2.67	-	2.42	2.73	3.03	3.03	3.17	3.08	2.92	2.77
Neutrofil (%)	-	-	-	85.8	-	-	-	-	-	-	-	-	-	-	95.8
Limfosit (%)	7.2	-	-	8.2	-	-	-	5.7	-	4.0	4.2	4.6	-	-	2.4
Monosit (%)	6.5	-	-	4.1	-	-	-	2.9	-	3.9	6.3	3.7	-	-	1.7
Ureum (mg/dL)	64	62	-	-	-	44	54	112	158	219	265	307	365	444	349
Kreatinin (mg/dL)	0.7	0.7	-	-	-	0.5	0.6	1.6	2.3	-	2.9	3.2	3.5	4.2	3.3
eGFR (mL/min/1.73m²)	95	95	-	-	-	109	107	43	28	-	21	18	17	13	18
Na (mmol/L)	133	132	132	-	-	130	130	131	131	130	133	134	135	135	134
K (mmol/L)	4.2	4.4	4.5	-	-	4.0	3.9	3.7	3.8	4.2	4.0	4.0	4.1	4.1	4.1
Cl (mmol/L)	87	90	91	-	-	83	83	84	84	84	84	86	85	86	87
Calcium	-	-	8.9	-	-	8.5	-	-	-	-	-	7.09	-	6.77	7.21
Magnesium	-	-	1.98	-	-	1.69	-	-	-	-	2.05	2.16	-	2.63	2.48
AST (U/L)	116	92	94	-	-	149	163	82	98	138	38	25	25	29	26
ALT (U/L)	158	145	144	-	-	172	242	148	140	215	131	85	55	40	37
Albumin (g/dL)	-	-	2.49	-	-	2.5	2.50		2.30	2.36	-	2.28	1.93	2.17	1.98
Bilirubin total	0.76	-	-	-	-	-	1.25	-	-	-	0.64	0.86	-	-	-
Bilirubin direct	0.42	-	-	-	-	-	0.77	-	-	-	0.39	0.57	-	-	-
PT (detik)	16.1/14.1	-	-	14.5/14.2	-	14.0/14.6	-	-	13.1/14.1	-	14.2/14.2	19.2/14.0	20.8/14.4	22.1/14.1	-
INR	1.20/1.03	-	-	1.06/1.02	-	60.7/1.07	-	-	0.94/1.01	-	1.28/1.03	1.45/1.00	1.60/1.04	1.71/1.03	-
APTT (detik)	28.4/30.8	-	-	27.4/31.5	-	24.6/31.3	-	-	26.9/33.2	-	31.7/31.3	35.8/31.4	37.8/32.3	39.7/32.3	-
GDS	136	-	150	-	-	148	175	-	134	133	132	184	-	-	96
CRP (mg/L)	-	-	-	-	-	48.00	-	-	48.00	-	-	24.00	-	-	48.00
Prokalsitonin (ng/mL)	-	-	-	-	-	0.239	-	-	85.4	-	13.9	19.3	-	-	-
D-dimer (µg/mL)	-	-	8.0	6.9	-	7.0	-	-	7.8	-	6.8	5.2	-	-	6.6

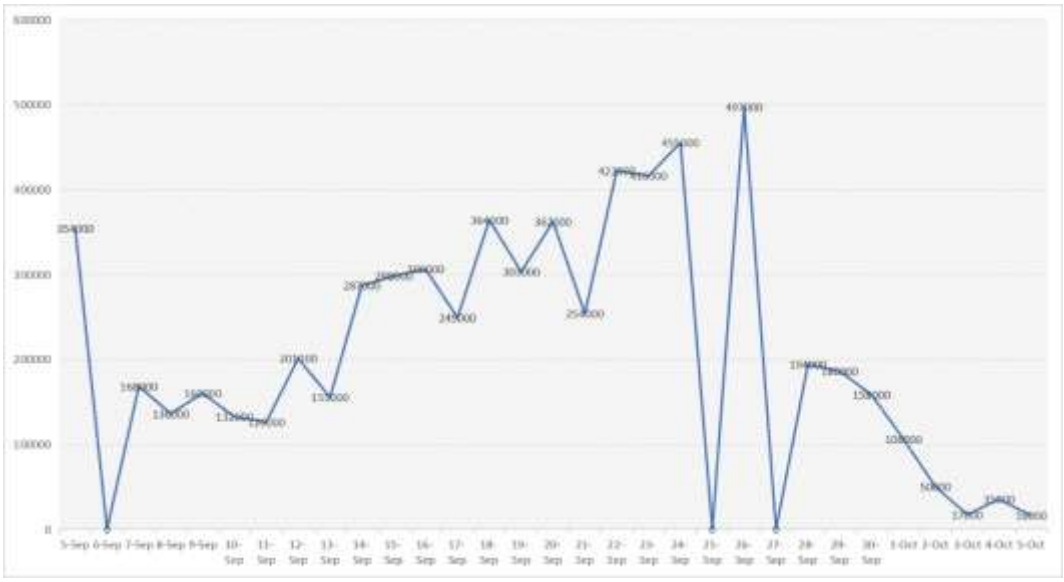

Figure 4. Trend of platelet values from September 5, 2025 to October 5, 2025.

Diagnosis and Management

The patient's clinical severity scores indicate a marked deterioration in condition over time. The SOFA score increased from 4 points to 16 points, reflecting a progression from an estimated mortality of less than 33% to more than 95%. Similarly, the APACHE score rose from 12 to 31 points, with the predicted postoperative mortality increasing from approximately 7% to 73%. The NUTRIC score also worsened from 3 to 8, indicating a transition from low nutritional risk, with an estimated 28-day mortality of around 20%, to a high-risk category with mortality reaching approximately 58%. These findings collectively suggest severe critical illness with a very high risk of mortality.

Clinically, the patient is experiencing respiratory failure requiring ventilatory support via a tracheostomy cannula. The patient is also in septic shock, with sepsis likely originating from a urosepsis source, although central nervous system infection remains a differential diagnosis. Hematologically, the patient presents with severe thrombocytopenia and is suspected of having disseminated intravascular coagulation (DIC). The patient also has a history of intracerebral hemorrhage (ICH) with sequelae, has undergone bilateral ventriculoperitoneal (VP) shunt placement, and is currently experiencing acute-on-chronic kidney disease requiring hemodialysis.

Regarding antimicrobial therapy, the patient had a history of prior antibiotic use, including meropenem 1 gram every 8 hours intravenously and levofloxacin 750 mg once daily intravenously, both initiated 12 days prior and continued from the referring hospital. Current antibiotic therapy includes vancomycin 1 gram every 24 hours intravenously, initiated on September 27, 2025 (day 10), and tigecycline 100 mg every 12 hours intravenously, started on

September 29, 2025 (day 8). Antifungal therapy consists of fluconazole 200 mg intravenously once daily, adjusted for renal function and currently on day 4.

Additional supportive therapies include intravenous fluids with Ahep 500 mL per day and Renxamin 200 mL per day. Symptomatic and adjunctive medications include ibuprofen 200 mg every 8 hours intravenously as needed, lansoprazole 30 mg daily intravenously for stress ulcer prophylaxis, diphenhydramine, metoclopramide, sucralfate, calcium carbonate, bromocriptine, N-acetylcysteine, and various vitamin supplements such as vitamin C, vitamin B complex, zinc, and folic acid. Antihypertensive medications (nifedipine and candesartan) are currently withheld. The patient is also receiving atorvastatin and levetiracetam administered rectally.

Based on the FAST HUGS IN BED approach, the patient's daily caloric requirement is estimated at 1,725 kcal. Nutritional support is provided through enteral nutrition via nasogastric tube (NGT) at 5 feeds of 200 mL (totaling 1,000 kcal), supplemented with parenteral nutrition (Renxamin and Ahep). Analgesia includes ibuprofen and paracetamol. The patient is post-operative, with the head of the bed elevated to 15–30 degrees. Gastrointestinal prophylaxis is maintained with lansoprazole, and blood glucose is monitored every 8 hours. Skin care includes the use of moisturizers and artificial tears.

The patient is supported with multiple invasive devices, including a central venous catheter (inserted September 28), nasogastric tube (September 30), urinary catheter (September 26), and tracheostomy cannula (September 13). Enteral feeding is ongoing with a gastric residual volume of 50 mL over the last 4 hours. Passive mobilization is performed regularly, and the room temperature is maintained at 26°C. The family has been educated regarding the patient's worsening condition.

The management plan includes ongoing evaluation of antibiotic therapy, implementation of ventilator and central line care bundles, and proper tracheostomy care. Follow-up of the central venous catheter tip culture is planned. In view of severe thrombocytopenia, platelet transfusion is planned, with follow-up on the availability of thrombocyte concentrate or apheresis platelets. Laboratory parameters will also be reassessed following hemodialysis.

Follow-up and Monitoring

On Monday, October 6, 2025, the bedside monitor recorded a heart rate of 99 beats per minute, oxygen saturation of 100%, respiratory rate of 34 breaths per minute, and blood pressure of 130/70mmHg. On Tuesday, October 7, 2025, the monitor showed a heart rate of 105 beats per minute, oxygen saturation of 100%, respiratory rate of 25 breaths per minute, and blood pressure of 116/57 mmHg. On October 7, 2025, at 15:42, the patient's mean arterial

pressure (MAP) was 58 mmHg, and norepinephrine was increased to 0.5 mcg/kgBW/min. At 15:50, blood glucose was 93 mg/dL. At 16:00, MAP decreased to 51 mmHg, and norepinephrine was titrated up to 0.6 mcg/kgBW/min. At 16:18, MAP improved to 67 mmHg, and norepinephrine was increased to 0.7 mcg/kgBW/min. At 16:44, MAP dropped again to 56 mmHg, and norepinephrine was further titrated up to 1 mcg/kgBW/min. At 17:32, MAP fell critically to 16 mmHg, prompting escalation of norepinephrine to 1.2 mcg/kgBW/min. At 17:50, blood glucose was found to be 43 mg/dL, and a bolus of 75 mL of 40% dextrose (D40%) was administered. At 18:05, repeat blood glucose measurement showed improvement to 116 mg/dL. At 18:58, the heart rate decreased to 60 beats per minute. At 19:00, the monitor showed ventricular tachycardia without a palpable pulse. The family requested that no cardiopulmonary resuscitation be performed. At 19:03, electrocardiography confirmed true asystole, and the patient was pronounced deceased in the presence of the family and nursing staff.

This condition occurred as a consequence of several antecedent causes, including septic shock, with the source of infection likely originating from urosepsis, with a differential diagnosis of central nervous system (CNS) infection. The clinical course was further complicated by multidrug-resistant (MDR) infection and disseminated intravascular coagulation (DIC), as well as acute-on-chronic kidney disease (CKD). These acute and subacute conditions developed on the background of underlying chronic neurological disorders, namely obstructive hydrocephalus and intraventricular hemorrhage (IVH).

3. DISCUSSION

The diagnosis of drug-induced immune thrombocytopenia (DITP) is challenging due to the numerous potential causes of acquired thrombocytopenia in critically ill ICU patients. In patients with severe infections, distinguishing DITP from other etiologies is particularly important. Initially, primary hematologic and autoimmune disorders were excluded based on clinical history and laboratory findings. Subsequently, other conditions such as thrombotic microangiopathy (TMA), hepatic failure, and disseminated intravascular coagulation (DIC) were ruled out through evaluation of renal function, liver function, and coagulation parameters. Attention was then directed toward infection and medication-related factors. Although severe infection was initially considered a major contributor, it showed signs of improvement, while the platelet count continued to decline. Vancomycin-induced thrombocytopenia is a rare immune-mediated adverse reaction characterized by the formation of drug-dependent antibodies that bind to platelet glycoproteins in the presence of vancomycin, leading to accelerated platelet destruction. Typically, thrombocytopenia develops after 5–10 days of

exposure and may be severe, with platelet counts often dropping below 20,000/µL.[8] In this case, the patient received vancomycin starting on September 27, 2025, and subsequently developed a sharp decline in platelet count, reaching a nadir of 17,000/µL on October 3, which is consistent with the typical time course and severity described in VIT.

Vancomycin, a glycopeptide antibiotic widely used to treat Gram-positive bacterial infections, has been associated with a rare but important hematologic adverse effect known as VIT. The primary mechanism is thought to be immune-mediated, involving the formation of drug-dependent antibodies that bind to platelet glycoproteins in the presence of vancomycin, leading to accelerated peripheral platelet destruction. This is supported by the detection of vancomycin-specific antibodies in a significant number of reported cases. Clinically, VIT typically develops after several days of therapy, with a mean time to platelet nadir of approximately 8 days. However, in patients with prior exposure, thrombocytopenia may occur more rapidly due to a secondary immune response. The severity of thrombocytopenia can vary widely, ranging from mild reductions to severe cases (<10,000/µL), and may be accompanied by bleeding manifestations.[9]

In critically ill patients, thrombocytopenia is multifactorial, with sepsis being one of the most common causes due to mechanisms such as disseminated intravascular coagulation (DIC), endothelial dysfunction, and increased platelet consumption. However, distinguishing sepsis-associated thrombocytopenia from drug-induced causes is crucial.[8,10] In this patient, thrombocytopenia developed abruptly after a period of relatively stable platelet counts, coinciding temporally with vancomycin administration, suggesting a possible drug-related etiology superimposed on sepsis-related mechanisms.

Sepsis-induced thrombocytopenia is usually gradual and associated with other signs of systemic coagulopathy, whereas VIT often presents with a sudden and profound decrease in platelet count. In this case, the patient initially had normal to elevated platelet levels, followed by a rapid decline in late September. Although the patient also exhibited laboratory features suggestive of DIC, including elevated D-dimer and prolonged coagulation parameters, the magnitude and rapidity of platelet decline raise suspicion for an additional immune-mediated mechanism such as VIT.

Renal dysfunction is a recognized risk factor for vancomycin toxicity, as impaired clearance may lead to higher plasma concentrations and prolonged exposure.[14] The patient developed acute-on-chronic kidney disease with significantly elevated urea and creatinine levels, which may have contributed to increased vancomycin exposure and a higher risk of adverse hematologic effects, including thrombocytopenia.

Critically ill patients with multiple comorbidities and exposure to broad-spectrum antibiotics are at increased risk for multidrug-resistant (MDR) infections, necessitating the use of agents such as vancomycin.[1,8] In this case, the presence of ESBL-producing Klebsiella pneumoniae and carbapenem-resistant Acinetobacter baumannii complicated antimicrobial management and prolonged antibiotic exposure, which may have increased the likelihood of adverse drug reactions, including VIT.

The diagnosis of VIT is primarily clinical, based on temporal association, exclusion of other causes, and improvement after discontinuation of the offending agent,[8] relying on the temporal relationship between vancomycin administration and platelet decline, as well as recovery following drug discontinuation. Tools such as the Naranjo Adverse Drug Reaction Probability Scale may aid in assessing causality. The cornerstone of management is immediate discontinuation of vancomycin, which typically results in platelet recovery within 5–7 days. In severe cases with active bleeding, platelet transfusion may be considered, although its effectiveness can be limited in the presence of circulating antibodies. Additional therapies, including intravenous immunoglobulin (IVIG), corticosteroids, rituximab, or plasma exchange, are generally reserved for refractory cases or those with life-threatening bleeding. Early recognition of VIT is essential to prevent serious complications, particularly in patients receiving prolonged or repeated courses of vancomycin.[9] However, in this case, ongoing severe sepsis, DIC, and multiorgan failure made it challenging to definitively confirm VIT. Despite this, the temporal relationship between vancomycin initiation and the onset of severe thrombocytopenia strongly supports the diagnosis.

Management of VIT involves immediate discontinuation of vancomycin, supportive care, and platelet transfusion if clinically indicated. In septic patients, this decision is complex due to the need for continued broad-spectrum antimicrobial coverage.[15-17] In this patient, vancomycin was part of the regimen for severe infection, highlighting the clinical dilemma between treating life-threatening infection and preventing drug-induced complications. This case illustrates the significant impact of combined sepsis, MDR infection, coagulopathy, and possible VIT on patient outcomes. The patient's clinical course was marked by progressive deterioration, culminating in septic shock, refractory hypotension, and death. This underscores the importance of early recognition of drug-induced thrombocytopenia, especially in critically ill patients, to minimize additional morbidity and improve clinical outcomes.

4. CONCLUSION

In conclusion, this case highlights the complexity of diagnosing VIT in critically ill septic patients, where multiple overlapping mechanisms contribute to thrombocytopenia. Despite the presence of sepsis, disseminated intravascular coagulation, and multiorgan failure, the temporal association between vancomycin administration and the abrupt, severe decline in platelet count strongly suggests a drug-induced immune mechanism. The coexistence of renal impairment and prolonged antibiotic exposure may have further increased the risk of VIT. This case underscores the importance of maintaining a high index of suspicion for drug-induced thrombocytopenia in ICU settings, particularly when platelet counts decline disproportionately to the clinical course of infection. Early recognition and prompt discontinuation of the offending agent are essential to prevent further complications, although management remains challenging in patients requiring broad-spectrum antimicrobial therapy.

REFERENCE

Al-Harbi, D., Alturaiki, A., Alshngeetee, A., Aldabas, H., AlBreacan, L., Aljohani, R., Alshahrani, E. H., Althemery, A., & Esba, L. C. A. (2022). Linezolid vs vancomycin in induced thrombocytopenia. Infectious Diseases and Therapy, 11(4), 1649–1660. https://doi.org/10.1007/s40121-022-00663-3

Alqeeq, B. F., Ayyad, M., Albandak, M., Almadhoun, W. J., Kullab, M., Ghabayen, A. W., & Al-Tawil, M. (2025). The clinical significance of thrombocytopenia in sepsis and septic shock: A systematic review and meta-analysis. BMC Anesthesiology, 25, 327. https://doi.org/10.1186/s12871-025-03188-7

Cox, D. (2023). Sepsis—It is all about the platelets. Frontiers in Immunology, 14, 1210219. https://doi.org/10.3389/fimmu.2023.1210219

Guleng, S. R., Wu, R. H., & Guo, X. B. (2021). Vancomycin-induced thrombocytopenia in endocarditis: A case report and review of literature. World Journal of Clinical Cases, 9(7), 1696–1704. https://doi.org/10.12998/wjcc.v9.i7.1696

He, N., Su, S., Ye, Z., Du, G., He, B., Li, D., et al. (2020). Evidence-based guideline for therapeutic drug monitoring of vancomycin: 2020 update. Clinical Infectious Diseases, 71(Suppl. 4), S363–S371. https://doi.org/10.1093/cid/ciaa1536

Hunzeker, Z., Chu, V., & Agrawal, P. (2026). Platelets disappear overnight: A case report of vancomycin-induced immune thrombocytopenia. Annals of Internal Medicine Clinical Cases, 5(3). https://doi.org/10.7326/aimcc.2025.0666

Ju, G., Zhang, Y., Ye, C., et al. (2024). Comparative effectiveness and safety of six antibiotics in treating MRSA infections: A network meta-analysis. International Journal of Infectious Diseases, 107109. https://doi.org/10.1016/j.ijid.2024.107109

Kan, W. C., Chen, Y. C., Wu, V. C., & Shiao, C. C. (2022). Vancomycin-associated acute kidney injury: A narrative review from pathophysiology to clinical application. International Journal of Molecular Sciences, 23(4), 2052. https://doi.org/10.3390/ijms23042052

Li, X. X., Wang, G. R., Li, C., He, N., Yao, P., Cheng, Y. C., Wang, C. H., Ge, Q. G., Shi, L. W., & Zhao, R. S. (2022). Severe thrombocytopenia caused by vancomycin in the intensive care unit: A case report. Frontiers in Medicine, 9, 829267. https://doi.org/10.3389/fmed.2022.829267

Mohammadi, M., Jahangard-Rafsanjani, Z., Sarayani, A., Hadjibabaei, M., & Taghizadeh-Ghehi, M. (2017). Vancomycin-induced thrombocytopenia: A narrative review. Drug Safety, 40, 49–59. https://doi.org/10.1007/s40264-016-0469-y

Péju, E., Fouqué, G., Charpentier, J., et al. (2023). Clinical significance of thrombocytopenia in patients with septic shock: An observational retrospective study. Journal of Critical Care, 76, 154293. https://doi.org/10.1016/j.jcrc.2023.154293

Savchenko, I., Birg, T., Sharipov, O., & Davydova, Y. (2021). Severe vancomycin-induced thrombocytopenia in a patient with meningitis. BMJ Case Reports, 14(8), e244209. https://doi.org/10.1136/bcr-2021-244209

Schupp, T., Weidner, K., Rusnak, J., et al. (2023). Diagnostic and prognostic role of platelets in patients with sepsis and septic shock. Platelets, 34, 2131753. https://doi.org/10.1080/09537104.2022.2131753

Yang, H., Zhang, Y., Feng, X., & An, Z. (2023). Bleeding complications in vancomycin-induced thrombocytopenia: A real-world postmarketing pharmacovigilance analysis. Clinical Therapeutics, 45(9), 868–872. https://doi.org/10.1016/j.clinthera.2023.06.022

Zhu, Y., Huang, L., Zhang, J., Liang, L., & Jin, P. (2025). Incidence and risk factors of vancomycin-induced thrombocytopenia: A six-year real-world study. BMC Infectious Diseases, 25, 7. https://doi.org/10.1186/s12879-024-10393-1

Zhu, Y., Zhao, F., & Jin, P. (2023). Clinical manifestations and risk factors of tigecycline-associated thrombocytopenia. Infection and Drug Resistance, 16, 6225–6235. https://doi.org/10.2147/IDR.S426259