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CASE REPORT

Marked Non-Infectious Elevation of Procalcitonin in a Hemodialysis Patient with Bladder Cancer: A Case Report

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Background: Here, we present a rare case of a sustained hemodialysis patient with bladder cancer who had the marked elevation of procalcitonin (PCT) in the absence of evidence for infection.

Case Presentation: A patient had significantly elevated PCT but without clinical manifestations of infection. Through evidence-based investigation and collaboration with physicians, clinical pharmacists successfully ruled out infection. Consequently, antibiotics were discontinued, and the patient improved and was discharged from the hospital.

Conclusion: The aim is to remind clinical pharmacists and clinicians to conduct comprehensive examination and judgment when encountering similar cases to reduce the occurrence of misdiagnosis in clinical practice.

Keywords: end-stage renal disease, procalcitonin, sepsis, C-reactive protein, pharmaceutical care

Background

As is well known, procalcitonin (PCT) is considered as an important biomarker for bacterial infection and sepsis. PCT has relatively higher sensitivity and specificity compared with other inflammatory biomarkers such as white blood cells count and neutrophil percentage, which can be used to determine the severity of infections, the use of antibiotics, the evaluation of effectiveness, and the cessation of antibiotics.[1,2] However, it can also yield false-positive results in some special conditions, such as renal dysfunction and heart failure. Here, we report a rare case of the markedly elevated PCT level in a patient with end-stage renal disease (ESRD) and bladder cancer, without the evidence of infection.

Case Presentation

A 67-year-old Chinese woman presented to our hospital for fatigue on November 14, 2024. The patient had a history of ESRD, bladder cancer, supraventricular tachycardia, and thrombosis of the lower extremities and internal jugular vein. On admission, physical examination revealed the patient suffered from fever, with a temperature of 38.5°C. The results of blood routine examination at the time of admission were as follows: red blood cells 1.78×10^{12}/L, hemoglobin 45 g/L, urea 20.72 mmol/L, creatinine 528.1 μmol/L, and brain natriuretic peptide (BNP) 619.91 pg/mL. The C-reactive protein (CRP) level was 39.72 mg/L. Other test results were unremarkable, including the nine-item respiratory pathogen panel detection, T-Spot. TB test, and other relevant tests. There were no significant changes in the strip shadow of the upper lobe of the left lung and the inflammation of the lower lobes of both lungs when compared to the computed tomography (CT) scan of the chest on October 31, 2023, except bilateral pleural effusion was recently discovered. The patient had unobvious respiratory symptoms, such as cough, sore throat, backache, or abdominal pain. Hence, no treatment was given regardless of the new pulmonary infection. Regular hemodialysis (three times per week) was performed. On November 17, 2024, the PCT level was 3.37 ng/mL, and the CRP increased to 42.38 mg/L. There were also no

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respiratory symptoms found. The doctor considered that the lung infection caused sepsis, and piperacillin-tazobactam was prescribed on November 18, 2024.

On November 25, 2024, the symptom of fever was well controlled, and the patient was present in good spirits. However, the CRP increased significantly to 82.88 mg/L, and the PCT slightly declined to 2.55 ng/mL. Considering that the patient had a central venous catheter, it was considered a catheter-related infection. Doxycycline capsules were added for anti-infection.

On November 28, 2024, the body temperature of the patient is 37.8°C, and the PCT increased significantly to 7.47 ng/mL. The chest CT scan was unchanged from the previous examination, and pneumonia was ruled out. The antibiotics were upgraded to meropenem for anti-infection treatment until December 2, 2024.

On December 3, 2024, the patient's body temperature was 38.8°C, and the PCT level increased to the highest point in admission monitoring, at 19.6 ng/mL. The hospital conducted a multidisciplinary consultation for the patient. After comprehensively analyzing the patient's vital signs (such as body temperature, respiration, pulse, and blood pressure) and laboratory tests (such as routine blood tests, inflammatory markers, and blood cultures), as well as listening to the opinions of relevant physicians and consulting literature, the treatment team considered the patient to be in a good state.

Twice blood cultures were performed, and the results were negative. The sputum smear showed that the dominant bacteria and acid-fast bacilli were negative, the (1,3)-β-D-glucan detection assay was negative, and only PCT increased. Therefore, we considered that the evidence of sepsis was always insufficient, the antibiotics were stopped, and supportive treatment was carried out.

On December 4, 2024, the CT scan of the abdomen revealed that the tumor had progressed rapidly. The counts of white blood cells and neutrophils were unremarkable. After that, the patient had no obvious discomfort, the PCT dropped gradually to 5.96 ng/mL, and CRP increased slightly (Figure 1). On December 7, 2024, the patient was discharged from the hospital. Follow-up after discharge showed that the patient had a normal body temperature with no signs of infection. However, the patient died of end-stage cancer more than a month later.

Discussion

It was well-known that PCT was considered as a biomarker to diagnose bacterial infection and sepsis. However, PCT levels may be elevated in certain conditions, such as renal dysfunction and heart failure, without strong evidence of bacterial infection. The aim of this case was to explore the causes for the marked increase of PCT in patients without sepsis or infection. Knowledge of this field is helpful for improving the value of PCT in special populations.

The Value of PCT in the Diagnosis of Sepsis and Special Populations

According to the 2016 International Guidelines for the Management of Sepsis and Septic Shock, sepsis could be diagnosed in the presence of suspected infection and organ failure.[3] The quick sequential organ failure score (qSOFA) was more commonly used, and qSOFA >2 suggested sepsis.[4] In this case, the patient had a qSOFA score of 0, and blood culture results were negative, which did not support the diagnosis of sepsis. However, studies reported that the patients with advanced cancer and ESRD may present with atypical clinical manifestations, and the qSOFA and blood culture had high specificity but low sensitivity for sepsis.[4] In addition, the 2021 International Guidelines for the Management of Sepsis and Septic Shock did not recommend using qSOFA scores alone to diagnose sepsis.[5] Nevertheless, PCT levels >2 ng/mL strongly suggested systemic infection, and >10 ng/mL indicated sepsis or septic shock.[6]

Therefore, we searched relevant literature and observed that elevated PCT levels may be found in patients with ESRD, advanced cancer, heart failure, tuberculosis, trauma, and other diseases. Several studies have reported that in hemodialytic individuals, with the PCT levels at the cut-off range of 0.75–1.66 ng/mL associated with identifying infection, the increased PCT levels are not significantly related to the severity of infection, and the increased PCT levels are affected by the dialysis method and the adequacy of dialysis.[7–9] At the same time, PCT was significantly affected by cancer type and cancer stage.[10–12] To summarize, as a biomarker for infection, PCT had poor specificity in identifying infection and sepsis in patients with ESRD and cancer, which may lead to a high rate of misdiagnosis. Consequently, PCT is controversial as the biomarker of infection in special populations.

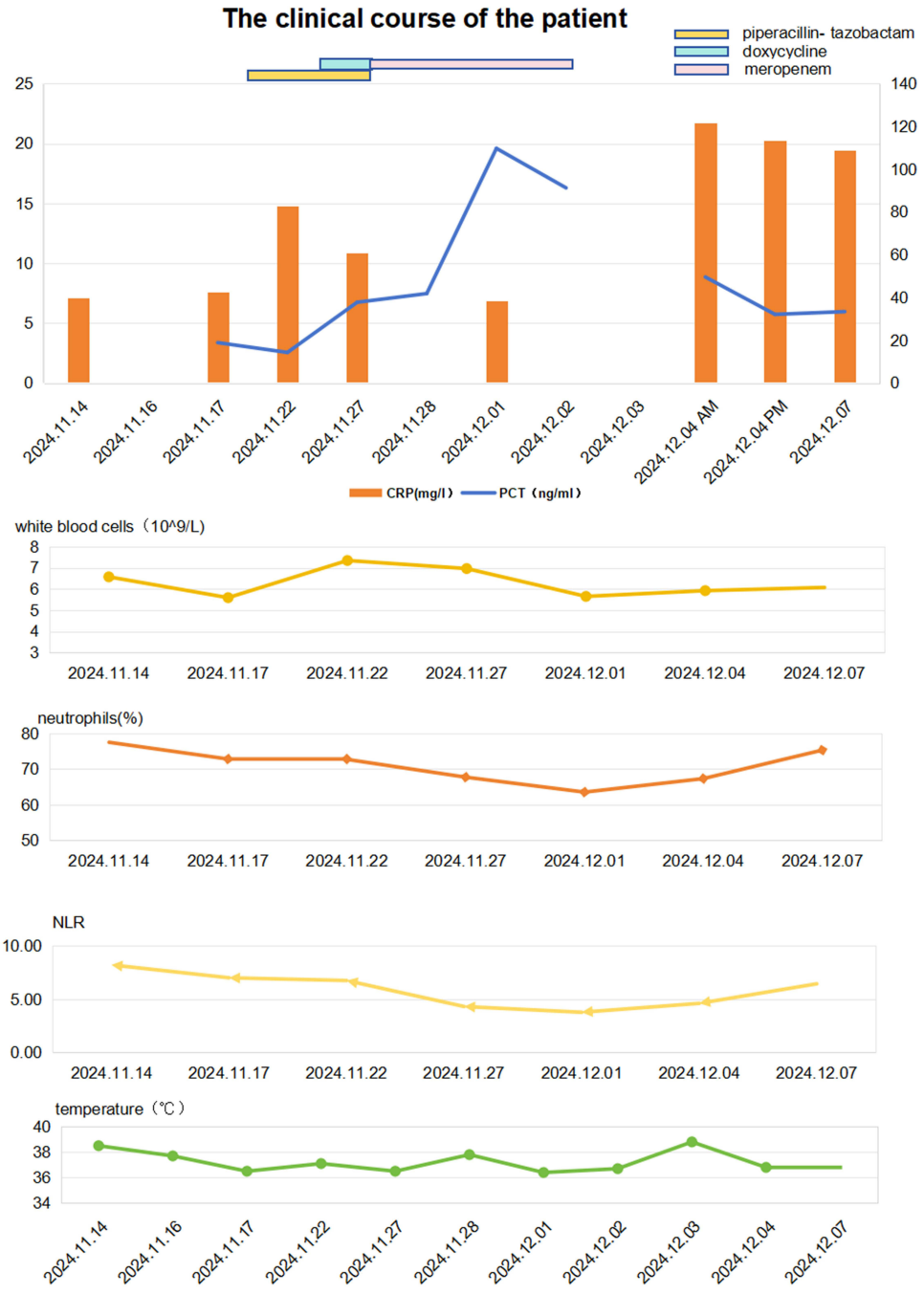

Figure 1 The clinical course of the present case. The X-axis shows the number of days after admission. The serum procalcitonin levels, C-reactive protein levels, white blood cells, neutrophils, NLR, and temperature variations were recorded. Multiple antimicrobials were given to the patient. Periods using piperacillin-tazobactam, doxycycline, and meropenem were marked by orange, blue, and pink lines, respectively.

The Value of Other Inflammatory Biomarkers in the Identification of Infection, Sepsis, and Prognosis

In addition to PCT, inflammatory biomarkers used to diagnose infection and sepsis include white blood cells, neutrophils, CRP, lactic acid, and other relevant indicators.

Chronic kidney disease was positively correlated with the neutrophil percentage and PCT, but it did not influence white blood cells and CRP.[13] However, the count of white blood cells was the earliest inflammatory marker used to distinguish between bacterial infection and viral infection. Studies have shown that when white blood cells or the

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percentage of neutrophils increased, it could indicate bacterial infection.[2] Furthermore, one of the diagnostic criteria for sepsis was white blood cells >12×10^9/L or <4×10^9/L, which had good sensitivity in the sepsis diagnosis.[2]

CRP level was not influenced by renal function or immunosuppression, while PCT was greatly affected by renal function.[14] Although CRP had poor specificity in sepsis diagnosis, 53 biomarkers were evaluated in the differentiation of sepsis and non-sepsis, and it was discovered that no biomarker or combined biomarker performed better than CRP.[15] Additionally, sepsis was indicated by a CRP of more than 87 mg/L and a daily increase of more than 41 mg/L or by a CRP of more than 50 mg/L and a daily increase rate of more than 25%.[15] Thus, the initial value and changes of CRP may better reflect the severity of infection and the occurrence of sepsis.

The neutrophil to lymphocyte ratio (NLR) may reflect changes in immunity and inflammation to some extent. The value of NLR in healthy individuals should be 1–5, which is positively correlated with the severity of inflammation.[6,16] Studies reported that a cut-off value of NLR at 5.52 was appropriate for determining pulmonary infection and sepsis in hemodialytic patients with ESRD, which had strong specificity and sensitivity.[17] Li et al[18] demonstrated that NLR was over 5.2 for patients with infection and 10–13 for patients with sepsis. And it was negatively correlated with treatment efficacy. On the third day of antibacterial drug treatment, the NLR value exceeding 15 indicated a higher mortality rate.[18]

Huang et al[19] reported that the monocyte distribution width (MDW) at a cut-off value of 20.3 was an appropriate predictor of sepsis, with high sensitivity and moderate specificity in the diagnosis of sepsis, and the value of MDW was positively correlated with prognosis. However, MDW is not included in the routine blood test at our hospital.

Thrombocytopenia was a common complication of sepsis, and persistent thrombocytopenia was an independent risk factor for mortality in patients with sepsis. Hua et al[20] demonstrated that the platelet count at a cut-off value of 84×10^9/L was an appropriate predictor of the severity and mortality of sepsis in ICU patients, with a sensitivity of 55.6% and a specificity of 91.8%, respectively. Additionally, the meta-analysis further reported that platelet distribution width (PDW) and mean platelet volume (MPV) were significantly elevated in sepsis. The specificity of PDW for sepsis reached 90.7% at a cut-off of 26%, and MPV reached 85.1% at a cut-off of 13 fL. However, they had poorer sensitivity.[21]

Lactic acid is a metabolic indicator that reflects the perfusion state of the body. Studies have shown that a lactic acid level >2 mmol/l has been used as an indicator for the diagnosis of septic shock.[22] The patient's blood gas analysis showed a lactic acid level of 0.6 mmol/L on December 2, indicated without the clinical manifestations of septic shock.

In this case, we comprehensively analyzed other sensitive inflammatory biomarkers to help in judging the presence of infection and the severity of sepsis. All these biomarkers were within the normal range, except for CRP and NRL (Figure 1). Although CRP and NRL were slightly elevated, this finding alone was insufficient to diagnose sepsis. Additionally, Luo et al[23] also reported a similar case of the markedly elevated PCT level in a patient without the evidence of infection. Based on comprehensive judgment, sepsis could be reasonably ruled out, or the prognosis of sepsis (if present) would be relatively good. Accordingly, after discussion with the physicians, antibiotics were stopped. Finally, thanks to individualized pharmaceutical care from clinical pharmacists and the combined efforts of clinicians, the patient was successfully discharged.

Analysis of the Reasons for the Elevation of PCT in the Absence of Evidence for Infection

Of note, this patient was also diagnosed with malignancy, renal dysfunction, and heart failure, without severe infection or sepsis, and these conditions might have been major factors influencing PCT levels.

Patients who had renal failure pre-dialysis had significantly higher PCT levels compared with healthy individuals, and PCT was also affected by dialysis type. High-flux hemodialysis could lead to a significant decrease in PCT, and low-flux hemodialysis did not decrease significantly in PCT.[3] A similar study further found that the PCT level decreased significantly in patients with ESRD after hemodialysis, considering the PCT at a cut-off value of 1.5 ng/mL was an appropriate predictor.[7] Meanwhile, Luo et al[23] also reported that in a patient with ESRD and tuberculosis, PCT levels fluctuated between 2.9 ng/mL and 94.9 ng/mL in the absence of infection. In this case, renal failure and insufficient dialysis might be the main factors for the elevated PCT levels.

Studies had found that PCT was higher in patients with malignancies than in healthy individuals. This elevation was attributed to inflammatory factors and treatment-related complications, and PCT levels increased with the severity of clinical staging.[9] Furthermore, the baseline PCT level of patients with cancer was the same as the groups with local infections. For cancer patients, a PCT cut-off value > 0.5 ng/mL had 63% specificity for diagnosing sepsis,[24] while a cut-off of 2 ng/mL only had 75% specificity for the same diagnosis.[25] The patient was in the late stage of malignancy, which may have been one of the causes of the high baseline level of PCT.

Schüttrumpf et al[26] observed that monoclonal antibody therapy has been reported to cause massive tumor lysis and cytokine release in cancer patients, with PCT concentrations up to 85.0 ng/mL. The patient in the present case had a history of treatment with tislelizumab and toripalimab before admission. Therefore, monoclonal antibody therapy may be one of the factors contributing to the elevated baseline PCT level.

Myocardial injury may indirectly activate the PCT pathway. Studies have reported that PCT levels increased with the severity of heart failure. The recommended cut-off values of PCT for diagnosing infection in patients with NYHA class II, III, and IV congestive heart failure were 0.086, 0.192, and 0.657 ng/mL, respectively.[11,27] Picariello et al[28] reported that PCT level was markedly elevated in patients with cardiogenic shock who had no sign of infections. Brain natriuretic peptide (BNP) was an important indicator for assessing cardiac injury and was positively correlated with the severity of the myocardial injury. During hospitalization, the patient had a high BNP concentration and large fluctuations, and myocardial injury may have a certain impact on the gradual increase of PCT.

Other factors such as cirrhosis, chronic obstructive pulmonary disease, anastomotic fistula, and trauma could also elevate the baseline level of PCT.[2,27] However, the patient had no history of other diseases, so other diseases were not considered as the cause of elevated PCT.

Conclusion

The diagnosis and treatment of infection and sepsis have long been major challenges in clinical work. Although PCT is widely used as a biomarker for bacterial infection in clinical settings, misdiagnosis and inappropriate treatment still occur. In conclusion, we reported a rare case of marked elevation of PCT in a sustained hemodialysis patient with cancer and heart failure, who was initially misdiagnosed with sepsis. It is hoped that this case will help clinical pharmacists and clinicians to reduce the incidence of misdiagnosis in clinical practice.

Abbreviations

PCT, procalcitonin; ESRD, end-stage renal disease; BNP, brain natriuretic peptide; CRP, C-reactive protein; NLR, neutrophil to lymphocyte ratio; MDW, monocyte distribution width; PDW, platelet distribution width; MPV, mean platelet volume.

Data Sharing Statement

The data are available from the corresponding author upon reasonable request.

Ethics Approval and Consent for Publication

The patient provided written informed consent to allow the case details and any accompanying images to be published. No specific ethics committee approval was required for this study.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Schneider HG, Thanh Lam Q. Procalcitonin for the clinical laboratory: a review. *Pathology*. 2007;39(4):383–390. doi:10.1080/00313020701444564
2. Castelli GP, Pognani C, Cita M, et al. Procalcitonin, C-reactive protein, white blood cells and SOFA score in ICU: diagnosis and monitoring of sepsis. *Minerva Anestesiologica*. 2006;72(1–2):69. doi:10.1097/01.ta.0000199389.16214.eb
3. Lee WS, Kang DW, Back JH, et al. Cutoff value of serum procalcitonin as a diagnostic biomarker of infection in end-stage renal disease patients. *Kor J Intern Med*. 2015;30(2):198. doi:10.3904/kjim.2015.30.2.198
4. Rhodes A, Evans LE, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. *Intensive Care Med*. 2017;43(3):304–377. doi:10.1007/s00134-017-4683-6
5. Evans L, Rhodes A, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for the management of sepsis and septic shock 2021. *Crit Care Med*. 2021;49(11):1974–1982. doi:10.1097/CCM.0000000000005357
6. Méndez Hernández R, Ramasco Rueda F. Biomarkers as prognostic predictors and therapeutic guide in critically ill patients: clinical evidence. *J Personalized Med*. 2023;13(2):333. doi:10.3390/jpm13020333
7. Dahaba AA, Rehak PH, List WF. Procalcitonin and C-reactive protein plasma concentrations in nonseptic uremic patients undergoing hemodialysis. *Intensive Care Med*. 2003;29(4):579–583. doi:10.1007/s00134-003-1664-8
8. El Haddad H, Chaftari AM, Hachem R, et al. Biomarkers of sepsis and bloodstream infections: the role of procalcitonin and proadrenomedullin with emphasis in patients with cancer. *Clin Infectious Dis*. 2018;67(6):971–977. doi:10.1093/cid/ciy331
9. Chaftari AM, Hachem R, Reitzel R, et al. Role of procalcitonin and interleukin-6 in predicting cancer, and its progression independent of infection. *PLoS One*. 2015;10(7):e0130999. doi:10.1371/journal.pone.0130999
10. Lee YC, Yeh HT, Lu SW, et al. Diagnostic accuracy of procalcitonin in adult non-neutropenic cancer patients with suspected infection: a systematic review and meta-analysis. *BMC Infect Dis*. 2024;24(1):278. doi:10.1186/s12879-024-09174-7
11. Niemirowicz K, Durnaś B, Wątek M, et al. Utility of blood procalcitonin concentration in the management of cancer patients with infections. *Onco Targets Ther*. 2016:469–475. 10.2147/OTT.S95600.
12. Sedef AM, Kose F, Mertsoylu H, et al. Procalcitonin as a biomarker for infection-related mortality in cancer patients. *Current Opinion Support Palliative Care*. 2015;9(2):168–173. doi:10.1097/SPC.0000000000000142
13. Sun Y, Jiang L, Shao X. Predictive value of procalcitonin for diagnosis of infections in patients with chronic kidney disease: a comparison with traditional inflammatory markers C-reactive protein, white blood cell count, and neutrophil percentage. *Int Urol Nephrol*. 2017;49(12):2205–2216. doi:10.1007/s11255-017-1710-z
14. Póvoa P, Coelho L, Dal-Pizzol F, et al. How to use biomarkers of infection or sepsis at the bedside: guide to clinicians. *Intensive Care Med*. 2023;49(2):142–153. doi:10.1007/s00134-022-06956-y
15. Parlato M, Philippart F, Rouquette A, et al. Circulating biomarkers may be unable to detect infection at the early phase of sepsis in ICU patients: the captain prospective multicenter cohort study. *Intensive Care Med*. 2018;44:1061–1070. doi:10.1007/s00134-018-5228-3
16. Widjaja H, Rusmawatiningtyas D, Makrufardi F, et al. Neutrophil lymphocyte ratio as predictor of mortality in pediatric patients with bacterial meningitis: a retrospective cohort study. *Annals Med Surg*. 2022:73. 10.1016/j.amsu.2021.103191.
17. Li LL, Yang YQ, Qiu M, et al. The clinical significance of neutrophil-lymphocyte ratio in patients treated with hemodialysis complicated with lung infection. *Medicine*. 2021;100(29):e26591. doi:10.1097/MD.0000000000026591
18. Sari R, Karakurt Z, Ay M, et al. Neutrophil to lymphocyte ratio as a predictor of treatment response and mortality in septic shock patients in the intensive care unit. *Turk J Med Sci*. 2019;49(5):1336–1349. doi:10.3906/sag-1901-105
19. Huang YH, Chen CJ, Shao SC, et al. Comparison of the diagnostic accuracies of monocyte distribution width, procalcitonin, and C-Reactive protein for sepsis: a systematic review and meta-analysis. *Crit Care Med*. 2023;51(5):e106–e114. doi:10.1097/CCM.0000000000005820
20. Hua Y, Wang R, Yang J, et al. Platelet count predicts mortality in patients with sepsis: a retrospective observational study. *Medicine*. 2023;102(38):e35335. doi:10.1097/MD.0000000000035335
21. Gupta A, Kumar S, Acharya S, et al. Utility of platelet indices as prognostic markers of sepsis: a medical intensive care unit-based cross-sectional study at a rural setup. *Cureus*. 2024;16(2). doi:10.7759/cureus.54490
22. Shankar-Hari M, Phillips GS, Levy ML, et al. Developing a new definition and assessing new clinical criteria for septic shock: for the third international consensus definitions for sepsis and septic shock (Sepsis-3). *JAMA*. 2016;315(8):775. doi:10.1001/jama.2016.0289
23. Luo P, Long Y, Ma L, et al. The sustained and marked elevation of serum procalcitonin in a hemodialysis patient with tuberculous lymphadenitis, but without the evidence of sepsis: a case report. *Infection Drug Resistance*. 2022;Volume 15:5161–5166. doi:10.2147/IDR.S378894
24. Bele N, Darmon M, Coquet I, et al. Diagnostic accuracy of procalcitonin in critically Ill immunocompromised patients. *BMC Infect Dis*. 2011;11(1):224. doi:10.1186/1471-2334-11-224
25. Mauro MV, Cavalcanti P, Perugini D, et al. Diagnostic utility of LightCycler SeptiFast and procalcitonin assays in the diagnosis of bloodstream infection in immunocompromised patients. *Diagn Microbiol Infectious Dis*. 2012;73(4):308–311. doi:10.1016/j.diagmicrobio.2012.04.006
26. Schüttrumpf S, Binder L, Hagemann T, et al. Utility of procalcitonin concentration in the evaluation of patients with malignant diseases and elevated C-Reactive protein plasma concentrations. *Clin Infectious Dis*. 2006;43(4):468–473. doi:10.1086/505394
27. Choi JJ, McCarthy MW. Novel applications for serum procalcitonin testing in clinical practice. *Expert Rev Mol Diagn*. 2018;18(1):27–34. doi:10.1080/14737159.2018.1407244
28. Picariello C, Lazzeri C, Chiostri M, et al. Kinetic of procalcitonin in patients with cardiogenic shock following acute myocardial infarction: preliminary data. *HSR Proc Intensive Care Cardiovasc Anesth*. 2010;2(3):201–207. doi:10.3109/17482941.2010.498920