World Journal of Case Reports and Clinical Images

ISSN: 2835-1568; CODEN: USA

DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433

CASE REPORT

Spontaneous Renal Recovery After Prolonged Oliguria in an Elderly Septic Patient Managed Without Renal Replacement Therapy: A Case Report

Dr. Arthi Gauthaman[1*], Dr. Jaganathan Selvanayagam[1], Dr. Rahul SM[2]

[1]Dr. Arthigauthaman MBBS, MD, Adult Intensive Care Unit, Dr Mehtas Multi-Speciality Hospital, Chennai, Tamil Nadu, India, Email: arthi85med@gmail.com; ORCID ID: 0000-0001-5544-9604.*

[1]Dr. Jaganathan Selvanayagam MBBS, MD, EDIC, Adult Intensive Care Unit, Dr Mehtas Multi-Speciality Hospital, Chennai 600034, TamilNadu, India, Email: jaganmmc@gmail.com.

[2]Dr. Rahul S M MBBS, FCCM, Adult Intensive Care Unit Dr. Mehtas Multi-Speciality Hospitals, Chennai 600034, TamilNadu, India, Email: drrahulsm23@gmail.com; ORCID ID: 0009-0008-5974-2291.

Received Date:
10 - August - 2026
Revised Date:
15 - August - 2026
Accepted Date:
18 - August – 2026
Published Date:
20 - August - 2026

Corresponding Author:
Dr. Arthigauthaman MBBS, MD, Adult Intensive care unit, Dr Mehtas multi-speciality Hospital, Chennai, Tamil Nadu, India, Email: arthi85med@gmail.com; ORCID ID: 0000-0001-5544-9604.

Citation:
Dr. Arthi Gauthaman, Dr. Jaganathan Selvanayagam, Dr. Rahul S M (2026) Spontaneous Renal Recovery After Prolonged Oliguria in an Elderly Septic Patient Managed Without Renal Replacement Therapy: A Case Report. World J Case Rep Clin Imag. 2026 August; 5(2), 1-7.

Abstract:

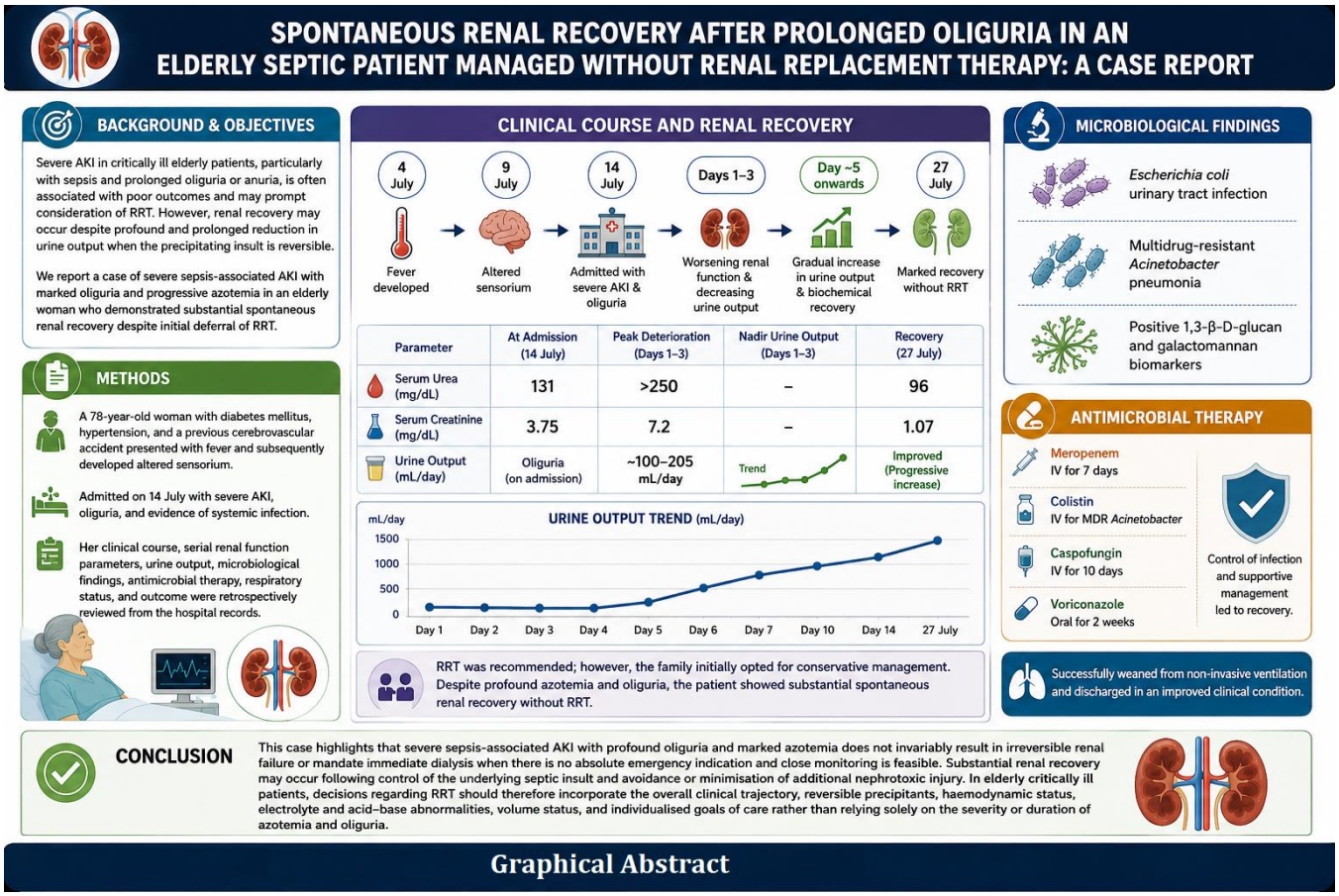
Background and Objectives: Severe acute kidney injury (AKI) in critically ill elderly patients, particularly when associated with sepsis and prolonged oliguria or anuria, is often associated with poor outcomes and may prompt consideration of renal replacement therapy (RRT). However, renal recovery may occur despite profound and prolonged reduction in urine output when the precipitating insult is reversible. We report a case of severe sepsis-associated AKI with marked oliguria and progressive azotemia in an elderly woman who demonstrated substantial spontaneous renal recovery despite initial deferral of RRT. ***Methods:*** A 78-year-old woman with diabetes mellitus, hypertension, and a previous cerebrovascular accident presented with fever and subsequently developed altered sensorium. She was admitted with severe AKI, oliguria, and evidence of systemic infection. Her clinical course, serial renal function parameters, urine output, microbiological findings, antimicrobial therapy, respiratory status, and outcome were retrospectively reviewed from the hospital records. ***Results:*** The patient developed fever on 4 July and altered sensorium on 9 July and was admitted on 14 July with severe AKI, with blood urea nitrogen equivalent to a urea level of 131 mg/dL and serum creatinine of 3.75 mg/dL, accompanied by oliguria. Renal function deteriorated further over the subsequent 3 days, with urea exceeding 250 mg/dL, creatinine rising to 7.2 mg/dL, and urine output declining to approximately 100–205 mL/day. RRT was recommended; however, the family initially opted for conservative management. The subsequent clinical course was notable for gradual spontaneous improvement in urine output from approximately day 5, followed by progressive biochemical recovery. By 27 July, serum urea and creatinine had decreased to 96 mg/dL and 1.07 mg/dL, respectively, without RRT. Microbiological evaluation demonstrated *Escherichia coli* urinary tract infection and multidrug-resistant *Acinetobacter* pneumonia. Positive 1,3-β-D-glucan and galactomannan biomarkers prompted antifungal therapy. Antimicrobial treatment included meropenem for 7 days, followed by intravenous colistin for multidrug-resistant Acinetobacter, intravenous caspofungin for 10 days, and oral voriconazole for 2 weeks. With control of the infectious process and supportive management, the patient was successfully weaned from non-invasive ventilation and discharged in an improved clinical condition. ***Conclusion:*** This case highlights that severe sepsis-associated AKI with profound oliguria and marked azotemia does not invariably result in irreversible renal failure or mandate immediate dialysis when there is no absolute emergency indication and close monitoring is feasible. Substantial renal recovery may occur following control of the underlying septic insult and avoidance or minimisation of additional nephrotoxic injury. In elderly critically ill patients, decisions regarding RRT should therefore incorporate the overall clinical trajectory, reversible precipitants, haemodynamic status, electrolyte and acid–base abnormalities, volume status, and individualised goals of care rather than relying solely on the severity or duration of azotemia and oliguria.

Keywords: Acute Kidney Injury, Sepsis-associated Acute Kidney Injury, Oliguria, Renal Recovery, Renal Replacement Therapy, Elderly, Sepsis, Conservative Management, Nephrotoxicity, Critical Care.

World Journal of Case Reports and Clinical Images

ISSN: 2835-1568; CODEN: USA

DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433



Graphical Abstract

1. Introduction:

Acute kidney injury (AKI) is a frequent and clinically important complication of critical illness and sepsis and is associated with increased morbidity, mortality, and healthcare utilisation. The multinational AKI-EPI study demonstrated the substantial burden of AKI among critically ill patients and highlighted its strong association with adverse outcomes [1]. In severe sepsis-associated AKI, renal dysfunction may result from a complex interaction of systemic inflammation, altered renal microcirculation, endothelial dysfunction, tubular injury, haemodynamic disturbances, and exposure to potentially nephrotoxic therapies.

When AKI progresses to severe oliguria with rapidly increasing azotaemia, Renal Replacement Therapy (RRT) may become necessary, particularly in the presence of refractory hyperkalaemia, severe metabolic acidosis, pulmonary oedema or other clinically significant uraemic complications. However, current recommendations emphasise that the decision to initiate RRT should not be based solely on an isolated serum creatinine or urea concentration; rather, the broader clinical context, trends in biochemical - parameters, urine output, fluid status and the presence of complications should be considered [2]. This approach has also been supported by the STARRT-AKI trial, in which an accelerated strategy for RRT initiation did not reduce 90-day mortality compared with a standard strategy in critically ill patients with severe AKI [3].

We report the case of a 78-year-old woman with diabetes mellitus, systemic hypertension and a previous cerebrovascular accident who developed severe sepsis-associated AKI characterised by profound oliguria, rapidly progressive azotaemia and a peak serum creatinine of 7.2 mg/dL. Although RRT was recommended during the period of severe renal dysfunction, it was not initiated because the family initially preferred conservative management. With intensive supportive care and treatment of the underlying infectious processes, urine output subsequently recovered, followed by a rapid and substantial improvement in renal function without RRT. This case illustrates the potential for renal recovery even after severe sepsis-associated AKI and emphasises the importance of serial clinical

World Journal of Case Reports and Clinical Images
ISSN: 2835-1568; CODEN: USA
DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433

assessment rather than prognostication based solely on the magnitude of biochemical derangement.

2. Case Report:

A 78-year-old woman with known diabetes mellitus, systemic hypertension and a previous cerebrovascular accident developed fever on 4 July, followed by altered sensorium from 9 July. She was admitted on 14 July with persistent, altered sensorium and markedly reduced urine output.

Initial investigations demonstrated significant renal dysfunction, with a serum urea concentration of 131 mg/dL and serum creatinine of 3.75 mg/dL. Computed Tomography (CT) of the abdomen demonstrated a simple hepatic cyst, with kidneys of normal size and no reported evidence of obstructive uropathy. CT of the chest demonstrated minimal infective changes. During the subsequent 3 days, renal function deteriorated rapidly, with serum urea increasing from 131 mg/dL on admission to 190 mg/dL on 15 July, 208 mg/dL on 16 July and 250 mg/dL on 17 July. Serum creatinine increased concurrently from 3.75 mg/dL to 5.1, 6.7 and 7.2 mg/dL, respectively.

Urine output was profoundly reduced, measuring approximately 100 mL on both 14 and 15 July and 205 mL on 16 July. Thus, although complete anuria was not documented, the patient developed severe oliguria approaching anuric levels. The clinical and biochemical trajectory was consistent with severe KDIGO stage 3 AKI in the setting of sepsis [2].

Given the severity of renal dysfunction and progressive oliguria, RRT was recommended. However, the family initially declined RRT and preferred conservative/palliative management. In view of the absence of documented severe hyperkalaemia, refractory acid–base disturbance, uncontrolled volume overload or another immediately life-threatening indication for emergent RRT, conservative management was continued with close monitoring of urine output, renal function, electrolytes, fluid balance and clinical status. This approach was consistent with the principle that RRT decisions should incorporate the overall clinical context rather than rely on isolated urea or creatinine thresholds [2].

Interestingly, renal recovery subsequently became evident. On 18 July, serum creatinine had begun to decline to 6.9 mg/dL despite persistent marked azotaemia. By 20 July, serum creatinine had fallen further to 3.74 mg/dL. This biochemical improvement was preceded by a striking increase in urine output, which reached approximately 2350 mL on day 5. The sequence of severe oliguria followed by brisk urine output and subsequent rapid decline in serum creatinine was compatible with transition from the oliguric phase of acute tubular injury to the recovery/diuretic phase.

By 27 July, serum urea had decreased to 96 mg/dL and serum creatinine to 1.07 mg/dL, with sustained urine output. Serum potassium remained below 4 mEq/L throughout the documented period.

The infectious work-up identified *Escherichia coli* in the urine culture and multidrug-resistant *Acinetobacter* in sputum. In addition, serum 1,3-β-D-glucan and galactomannan were positive, raising concern for an invasive fungal process in the appropriate clinical context. These fungal biomarkers were interpreted alongside the patient's clinical condition and microbiological findings rather than as definitive evidence of invasive fungal disease in isolation [4,5]. The patient received meropenem for 7 days, followed by intravenous colistin for multidrug-resistant *Acinetobacter.* Antifungal therapy consisted of intravenous caspofungin for 10 days and oral voriconazole for 2 weeks. The antifungal strategy was undertaken in view of the clinical suspicion of invasive fungal infection and the positive fungal biomarkers, with therapeutic decisions guided by the overall clinical context [4,5].

During the hospitalisation, the patient also required non-invasive ventilatory support because of respiratory compromise. Her respiratory status subsequently improved, allowing successful weaning from non-invasive ventilation. Parallel improvement in her systemic clinical condition, urine output and renal biochemical parameters was observed following treatment of the documented infectious processes.

The patient was eventually discharged in an improved clinical condition with near-normal renal function and without requiring RRT during the hospitalisation.

Investigations: The principal investigations were as follows: *14 July:* serum urea 131 mg/dL, serum creatinine 3.75 mg/dL. *15 July*: serum urea 190 mg/dL, serum creatinine 5.1 mg/dL. *16 July:* serum urea 208 mg/dL, serum creatinine 6.7 mg/dL. *17 July:* serum urea 250 mg/dL, serum creatinine 7.2 mg/dL. *18 July:* serum urea >200 mg/dL, serum creatinine 6.9 mg/dL. *20 July:* serum urea >250 mg/dL, serum creatinine 3.74 mg/dL. *27 July:* serum urea 96 mg/dL, serum creatinine 1.07 mg/dL. Serum potassium remained below 4

World Journal of Case Reports and Clinical Images
ISSN: 2835-1568; CODEN: USA
DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433

mEq/L during the documented period. Urine output was approximately 100 mL on day 1, 100 mL on day 2, 205 mL on day 3 and 2350 mL on day 5. Urine culture grew *Escherichia coli,* while sputum culture yielded multidrug-resistant *Acinetobacter*. Serum 1,3-β-D-glucan and galactomannan were positive. CT abdomen demonstrated a simple hepatic cyst with normal-sized kidneys and no reported obstructive abnormality. CT chest demonstrated minimal infective changes.

Differential Diagnoses: The principal differential diagnoses considered were sepsis-associated acute tubular injury, acute interstitial nephritis, obstructive uropathy and acute-on-chronic kidney disease.

Sepsis-associated acute tubular injury was considered the most likely diagnosis because of the close temporal relationship between systemic infection and renal deterioration, the rapid rise in serum creatinine, profound oliguria, subsequent brisk diuretic recovery and rapid restoration of renal function. The absence of documented pre-existing chronic kidney disease and the presence of normal-sized kidneys on CT further supported substantial pre-existing renal structural reserve.

Acute interstitial nephritis was considered less likely in the absence of a characteristic clinical or laboratory profile and because the renal dysfunction resolved without specific therapy directed at interstitial nephritis.

Obstructive uropathy was considered unlikely because CT imaging demonstrated normal-sized kidneys without reported hydronephrosis or another obstructive lesion.

Acute-on-chronic kidney disease was also considered less likely because there was no documented history of established chronic kidney disease and renal function subsequently returned to near-normal levels.

Treatment: Management consisted of intensive conservative treatment of AKI, including meticulous fluid and haemodynamic management, avoidance of unnecessary nephrotoxic exposure, serial monitoring of renal function and electrolytes, and close assessment of urine output.

The underlying infectious processes were treated with meropenem for 7 days followed by intravenous colistin for multidrug-resistant Acinetobacter. Antifungal treatment consisted of intravenous caspofungin for 10 days and oral voriconazole for 2 weeks. The selection and duration of antimicrobial and antifungal therapy were determined according to the microbiological findings and clinical suspicion of invasive fungal infection [4,5].

Respiratory failure was managed with non-invasive ventilation, from which the patient was subsequently successfully weaned.

RRT was recommended because of the severity and trajectory of AKI but was not initiated after the family initially declined the intervention. Importantly, management remained under close clinical and biochemical surveillance, with readiness to initiate RRT if conventional indications developed. KDIGO recommendations emphasise emergent RRT in the presence of life-threatening disturbances of fluid, electrolyte or acid–base balance and recommend that broader clinical circumstances and trends be considered rather than isolated urea or creatinine values [2].

Outcome and follow-up: The patient's renal function improved progressively following the recovery of urine output. By 27 July, serum urea had decreased to 96 mg/dL and serum creatinine to 1.07 mg/dL, with good urine output. Her respiratory and neurological status also improved, allowing discontinuation of non-invasive ventilatory support. She was discharged in a clinically stable condition with near-normal renal function and without having required RRT during the hospitalisation.

3. Discussion:

This case demonstrates an important and clinically challenging feature of severe sepsis-associated AKI: marked biochemical renal dysfunction and profound oliguria do not invariably imply irreversible renal failure or inevitable dialysis dependence.

AKI is common among critically ill patients and is particularly frequent in those with severe sepsis and multiple co-morbidities [1]. Sepsis-associated AKI is mechanistically complex and cannot be explained solely by reduced renal perfusion. Inflammatory activation, endothelial dysfunction, altered renal microcirculation, tubular epithelial injury and cellular metabolic adaptation may all contribute to the development of renal dysfunction. Consequently, substantial functional impairment may occur despite preservation of renal structural architecture.

In the present case, the trajectory of renal recovery was particularly striking. Serum creatinine increased from 3.75 mg/dL on admission to a peak of 7.2 mg/dL within

World Journal of Case Reports and Clinical Images

ISSN: 2835-1568; CODEN: USA

DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433

3 days, while urine output remained severely depressed. Subsequently, urine output increased abruptly to approximately 2350 mL on day 5, followed by a rapid decline in serum creatinine to 3.74 mg/dL by 20 July and 1.07 mg/dL by 27 July. This temporal sequence strongly suggests transition from a severe oliguric phase of acute tubular injury to a recovery phase characterised by restoration of tubular function and brisk diuresis.

The absence of hyperkalaemia was notable. Serum potassium remained below 4 mEq/L despite severe azotaemia. In addition, no refractory acid–base disturbance or uncontrolled volume overload requiring emergent extracorporeal support was documented. This distinction is important because the severity of azotaemia alone should not be equated with an absolute indication for RRT. KDIGO recommends initiation of RRT emergently when life-threatening abnormalities of fluid, electrolyte or acid–base balance occur and advises clinicians to consider the overall clinical context and trends rather than isolated serum urea or creatinine concentrations [2].

The STARRT-AKI trial provides important contemporary evidence regarding this issue. In more than 3000 critically ill patients with severe AKI, an accelerated strategy of RRT initiation did not reduce 90-day mortality compared with a standard strategy. Furthermore, among survivors, RRT dependence at 90 days was more frequent in the accelerated-initiation group [3]. These findings do not support withholding RRT when conventional indications are present; rather, they reinforce the principle that immediate RRT is not necessarily beneficial for every patient with severe biochemical AKI in the absence of urgent complications.

The present case should therefore not be interpreted as evidence that RRT can routinely be deferred in patients with severe AKI. Instead, it illustrates the importance of individualised assessment and repeated reassessment. In this patient, the preservation of renal size, absence of documented pre-existing CKD, maintained potassium control and subsequent rapid restoration of urine output were clinical features compatible with significant renal recovery potential.

Control of the underlying infectious process was another important component of the patient's recovery. The patient had microbiologically documented *E. coli* urinary infection and multidrug-resistant Acinetobacter isolated from respiratory secretions, together with positive serum fungal biomarkers that raised concern for an invasive fungal process. Effective treatment of the infectious burden and optimisation of systemic physiological conditions likely reduced the ongoing septic insult to the kidneys.

The interpretation of the fungal biomarkers warrants particular caution. Serum 1,3-β-D-glucan and galactomannan can provide supportive evidence in patients at risk of invasive fungal disease, but neither test should be regarded as definitive proof of invasive infection in isolation [4,5]. Their significance must be interpreted in conjunction with host factors, clinical presentation, imaging and microbiological findings. In this case, antifungal treatment was undertaken because of the overall clinical suspicion rather than solely on the basis of biomarker positivity.

The case also raises an important issue concerning antimicrobial-associated renal injury. Colistin is itself potentially nephrotoxic, making the subsequent improvement in renal function despite its administration clinically noteworthy. The recovery nevertheless cannot be attributed to any individual antimicrobial agent, and the temporal association between infection control and renal recovery does not establish causality.

The family's initial refusal of RRT introduced an additional dimension to management. In severe AKI, clinicians must communicate that the trajectory of renal recovery can be difficult to predict, while simultaneously explaining the potentially life-threatening consequences of delaying extracorporeal support. Shared decision-making should include discussion of prognosis, anticipated benefits and burdens of RRT, the patient's previously expressed values, and the possibility of renal recovery. In the present case, continued conservative management was accompanied by close monitoring, with RRT remaining available should urgent indications have developed.

Limitations: This report has several limitations. First, it represents a single patient and therefore cannot establish that conservative management without RRT is appropriate for patients with comparable biochemical severity. Second, the precise contribution of antimicrobial therapy, antifungal therapy, haemodynamic optimisation and natural recovery to the improvement in renal function cannot be independently quantified. Third, renal histopathology was not available to definitively demonstrate acute tubular injury. Finally, long-term renal function and the patient's subsequent risk of chronic kidney disease were not assessed beyond the period of hospital discharge.

World Journal of Case Reports and Clinical Images

ISSN: 2835-1568; CODEN: USA

DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433

4. Conclusion:

Severe sepsis-associated AKI may occasionally demonstrate substantial and rapid renal recovery despite profound oliguria and marked azotaemia. This case illustrates that a peak serum creatinine of 7.2 mg/dL and serum urea of approximately 250 mg/dL do not, in isolation, establish irreversible renal failure or mandate immediate RRT in the absence of life-threatening complications. The subsequent recovery of urine output followed by near-complete restoration of renal function emphasises the importance of serial assessment of urine output, biochemical trends, electrolyte status, acid–base balance and fluid status when evaluating the need for RRT [2,3].

The case further highlights the importance of timely treatment of the underlying septic process and cautious prognostication in elderly patients with potentially reversible AKI. Conservative management should not be regarded as a substitute for RRT when established indications are present; rather, in carefully selected patients without urgent indications, closely monitored supportive management may allow sufficient time for intrinsic renal recovery. Individualised decision-making, shared goals of care and repeated reassessment remain central to the management of severe sepsis-associated AKI.

5. Declarations:

Conflict of Interest: Authors report no conflict of interest.

Financial Support and Sponsorship: Nil

Data Availability Statement: The data supporting the findings of this case report are contained within the article. Additional clinical information is not publicly available because of patient privacy and confidentiality considerations.

Acknowledgements: Special thanks to Dr. Jaganathan Selvanayagam Medical director, HOD of Adult Intensive Care Unit, for his kind permission to submit this article for publication, as well as his major contribution towards his guidance and evaluation and management of the patient during the ICU stay.

Authors' Contributions: All authors contributed to the conception and design of the case report, acquisition and interpretation of clinical data, and preparation of the manuscript. All authors critically reviewed the manuscript for important intellectual content, approved the final version, and agreed to be accountable for all aspects of the work.

Ethical Approval and Consent to Participate (Human Ethics): Formal ethical approval was not required for this case report in accordance with the institutional policy governing the reporting of individual clinical cases.

Consent (Declaration of Patient's Consent): Authors certify that they have obtained written informed consent for publication of the Patient's clinical information. They have been informed regarding the purpose of publication and the measures taken to maintain the patient's privacy and confidentiality.

6. References:

1. Hoste EAJ, Bagshaw SM, Bellomo R, Cely CM, Colman R, Cruz DN, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. Intensive Care Med. 2015;41(8):1411–1423. doi:10.1007/s00134-015-3934-7.
2. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl. 2012;2(1):1–138.
3. STARRT-AKI Investigators. Timing of initiation of renal-replacement therapy in acute kidney injury. N Engl J Med. 2020;383(3):240–251. doi:10.1056/NEJMoa2000741.
4. Pappas PG, Kauffman CA, Andes DR, Clancy CJ, Marr KA, Ostrosky-Zeichner L, et al. Clinical Practice Guideline for the Management of Candidiasis: 2016 Update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;62(4)–e50. doi:10.1093/cid/civ933.
5. Patterson TF, Thompson GR 3rd, Denning DW, Fishman JA, Hadley S, Herbrecht R, et al. Practice Guidelines for the Diagnosis and Management of Aspergillosis: 2016 Update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;63(4)–e60. doi:10.1093/cid/ciw326.

World Journal of Case Reports and Clinical Images

ISSN: 2835-1568; CODEN: USA

DOI: http://dx.doi.org/10.51521/WJCRCI.2026.e52.433