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CASE REPORT

Early De-resuscitation Strategy using Diuretics Targeting Low Central Venous Pressure in the Management of Septic Shock with Acute Kidney Injury

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ABSTRACT

Septic shock remains a critical condition associated with high morbidity and mortality rates, particularly when complicated by Acute Kidney Injury (AKI). Traditional management strategies have predominantly emphasized fluid resuscitation to restore hemodynamic stability; however, the adverse effects of fluid overload are most pronounced in situations such as septic shock, which predisposes patients to acquired AKI. This case report presents an early de-resuscitation strategy using diuretics to target low central venous pressure (CVP) in a 58-year-old male with Urosepsis-associated AKI. The initial treatment included fluid loading and antibiotics, but his condition worsened, leading to ICU admission. A central venous catheter was placed, and a furosemide infusion was started due to positive fluid balance and high CVP. Over the next few days, the patient's condition improved significantly, with reduced ventilator and vasopressor support and improved renal function. The success of this strategy underscores the importance of re-evaluating fluid resuscitation practices and incorporating de-resuscitation as a key component of patient care.

Keywords: Acute kidney injury, de-resuscitation, central venous pressure, negative fluid balance, septic Shock.

INTRODUCTION

Septic shock remains a critical condition associated with high morbidity and mortality rates, particularly when complicated by Acute Kidney Injury (AKI). The coexistence of circulatory failure and renal dysfunction often reflects severe systemic inflammation and profound hemodynamic instability, both of which contribute to poor clinical outcomes and prolonged intensive care unit stays. In this setting, kidney injury not only serves as a marker of disease severity but also directly influences therapeutic decision-making and overall prognosis.

Traditional management strategies have predominantly emphasized fluid resuscitation to restore hemodynamic stability; however, the adverse effects of fluid overload maybe most pronounced in situations such as systemic sepsis which predispose to acquired AKI.[1] Although early fluid administration is essential to optimize preload and improve tissue perfusion, excessive or prolonged fluid loading may result in a persistently positive fluid balance with detrimental physiological consequences. This highlights the need for continuous reassessment of volume status during the course of septic shock management.

Patients with Sepsis-associated AKI are more likely to have fluid overload (FO), FO exacerbates organ edema which impairs organ functions and accelerates AKI progression due to decreased renal blood flow because of increased central venous pressure.[2] Elevated venous pressures may compromise renal perfusion gradients and worsen interstitial edema within the kidney, further aggravating renal dysfunction. As a result, strategies aimed at mitigating venous congestion have gained increasing clinical interest.

This paper presents an early de-resuscitation strategy utilizing diuretics to target low central venous pressure (CVP) in patients experiencing septic shock with concurrent AKI. By focusing on timely fluid removal after initial resuscitation, this approach seeks to balance hemodynamic support with prevention of fluid-related complications.

CASE

A 58-year-old male was brought to Emergency Department due to shortness of breath an hour before the admission. This symptom was accompanied by epigastric pain, vomiting, and diaphoresis. Three days prior, he had experienced fever and diarrhea. Examination revealed pain in the epigastric and right upper quadrant areas. The vital signs showed a mean arterial pressure of 65–70 mm Hg, accompanied by tachypnea, tachycardia, and fever. He had not urinated one day prior.

Laboratory assessments confirmed the diagnosis of Urosepsis-associated Acute Kidney Injury with elevated levels of urea (91.4 mg/dL), creatinine (4.57 mg/dL), C-Reactive Protein (254.7 mg/dL), and procalcitonin (65.4 ng/mL), along with significant bacterial infection in urinalysis. The electrocardiogram and chest X-ray were normal. Sepsis management protocols were initiated, including intravenous fluid loading therapy and antibiotic. The patient was then transferred to the ward for daily monitoring. Within 24 hours of monitoring, the patient became agitated and had difficulty breathing. An intensivist was consulted, and the patient was admitted to the Intensive Care Unit (ICU). He was in septic shock and respiratory distress, leading to intubation and the use of vasopressor. A central venous catheter was placed in the right subclavian vein, with a CVP of 13 mmHg. Subsequently, a furosemide infusion was commenced at a rate of 5 mg/hour. Prior to ICU admission, the cumulative fluid balance was positive, totaling 797cc. Additionally, the chest X-ray demonstrated acute lung edema.

Two days later, test results revealed elevated urea and creatinine levels, and the patient continued to experience oliguria. The furosemide infusion was increased to 10 mg/hour. From days 4 to 6, the patient's condition improved, particularly following the initiation of renal replacement therapy. He became alert, allowing for reduced ventilator support and lower doses of norepinephrine and furosemide. The CVP dropped from 13 to 4 mmHg, and creatinine levels improved to 2.74 mg/dL, resulting in a cumulative fluid balance of negative 4581 cc. By the seventh day, the patient was extubated

Table 1. Various parameters assessed as predictors of an effective de-resuscitation strategy in managing septic shock

Day of Hospitalization	Cumulative Balance (cc)	CVP level (mm Hg)	Furosemide Dose (mg/hour)	Creatinine Level (mg/dL)	Norepinephrine Dose (mcg/kg/min)
1	+ 250	-	-	4,57	-
2	+ 797	13	5	-	0,8
3	- 896	10	10	-	0,5
4	- 3032	4	5	-	0,25
5	- 4636	4	5	7,58	0,05
6	- 4581	4	2	2,74	0,01
7	- 4710	1	2	1,77	-
8	- 6831	1	-	-	-

with creatinine levels at 1.77 mg/dL, leading to the discontinuation of vasopressor support and a CVP of 1 mmHg. The cumulative fluid balance reached negative 6831 cc before transfer to the ward. (Table 1.)

During a week of management in the ICU, we effectively resolved the septic shock by the administration of furosemide and closed monitoring of critical parameters, including cumulative fluid balance, CVP, and creatinine levels. This positive outcome was sustained by a reduction in both vasopressor and ventilator support. The patient was hospitalized for 12 days and had a followup appointment three weeks later. He remained stable, returned to his routine, and the creatinine levels were significantly reduced to 1.3 mg/dL.

DISCUSSION

Sepsis is a severe condition characterized by a dysregulated host response to infection, leading to life-threatening organ dysfunction that commonly associated with hypotension due to a variable combination of peripheral vasodilatation, myocardial depression and fluid extravasation. If severe, this can progress to septic shock, characterised by tissue hypoperfusion and harmful cellular and metabolic consequences.[3,4] Severe sepsis is a major concern in critically ill patients and is often complicated AKI.[5] Sepsisassociated AKI (SA-AKI) carries a graver prognosis compared to sepsis or AKI alone, resulting in longer hospital stays, higher mortality rates, increased disability, and reduced quality of life in both adult and pediatric populations.[3] SA-AKI is best defined as the occurrence of AKI within 7 days of sepsis onset (diagnosed according to Kidney Disease Improving Global Outcome criteria and Sepsis 3 criteria, respectively). The KDIGO criteria, based on specific increases in serum creatinine (SCr) levels and reduction in urine output.[3,6] Upon initial admission to the hospital, this patient exhibited signs consistent with SA-AKI, classified as KDIGO stage 3. He possessed no prior history of renal dysfunction; however, he reported that his most recent micturition occurred 12 hours preceding his admission.

The management of sepsis has seen limited changes in the past two decades, primarily through the introduction of bundles prescribing specific interventions within defined timeframes.[7] On the other hand, even though the sepsis protocol was implemented in this case, the patient's clinical condition was worsening, which led to septic shock and required mechanical ventilation support along with continuous monitoring in the ICU. In the early stage of fluid resuscitation, with no urine output present, the patient faced fluid accumulation (FA) resulting in a positive fluid balance within the first 24 hours after admission. In critical care settings, intravenous fluid therapy is crucial for managing patients with inadequate tissue perfusion, aiming to improve renal perfusion and prevent AKI. Despite its benefits, excessive fluid administration can harm patients by causing FA and interstitial

edema due to capillary leakage.[8] Renal edema due to fluid overload occurs when excessive fluid administration leads to interstitial edema, which can impair renal function. This edema obstructs capillary blood flow and lymphatic drainage, resulting in decreased renal blood flow and glomerular filtration rate (GFR). The kidney, being an encapsulated organ, is particularly susceptible to increased intracapsular pressure from fluid congestion, which can further exacerbate AKI and prolong recovery. Thus, careful fluid management is crucial in critically ill patients.[2]

Likewise, hemodynamic monitoring plays a key role in the management of septic shock. Legrand et al. found that high CVP values within 24 h of admission to the ICU were closely related to the occurrence of AKI in patients with septic shock.[9] Fluid removal was carefully carried out using furosemide, aimed at creating a negative fluid balance that can ultimately reduce organ congestion and decrease CVP levels, respectively. Along with diuretics, ultrafiltration was used as an extra treatment method. This approach not only helps eliminate excess fluid but also effectively removes pro-inflammatory cytokines from the blood.[10]

The graphic (Figure 1.) below illustrates a linear correlation among three key variables: cumulative fluid balance, CVP levels, and kidney function. As the cumulative balance becomes increasingly negative, there is a notable decline in CVP levels from 13 mmHg to 1 mmHg. This decrease in CVP is also accompanied by a reduction in creatinine levels, indicating an improvement in kidney function and urine output. As a result of this improvement, it becomes feasible to discontinue the administration of furosemide. Upon closer examination, the underlying cause of SA-AKI in this case can be attributed to kidney edema and congestion, which result from elevated CVP levels. Current literature has shifted the understanding of CVP; it is no longer viewed as a reliable preload marker but is increasingly recognized as a surrogate indicator of fluid overload. In this case, the more negative cumulative balance demonstrates an inverse relationship with CVP, suggesting a reduction in interstitial edema due to the leakage syndrome in septic shock.

The term de-resuscitation was coined in 2014 and defined as active fluid removal in patients with fluid overload using drugs and/or ultrafiltration (UF). Measures to remove excess fluid can be pharmacologic and non-pharmacologic (net UF), combined with fluid restriction. Diuretics are usually tried first, either as monotherapy or in combination.[11] The role for using diuretics in AKI includes prevention of tubular obstruction, reduction in medullary oxygen consumption and increase in renal blood flow as well as reducing fluid overload and venous congestion.[12] De-resuscitation should be stopped when the goal is met (i.e. benefit has been achieved) or when safety concerns arise. This goal can be fluid-related (neutral balance), physiologic (e.g. CVP) or clinical (improved oxygenation, extubation).[11]

The patient has successfully achieved the de-resuscitation objective, evidenced by a favorable clinical outcome marked by a negative cumulative fluid balance. This positive development is closely correlated with a notable reduction in key indicators such as creatinine levels and CVP values. Furthermore, there has been a significant decrease in the necessity for both ventilatory support and vasopressor medications, which collectively contribute to a lower 30-day mortality rate. Throughout this process, the patient has maintained robust renal function, with other critical biomarkers remaining within normal ranges. This comprehensive improvement underscores the effectiveness of the de-resuscitation strategy and its impact on enhancing the overall health status of the patient.

CONCLUSION

This case study highlights the potential benefits of an early de-resuscitation strategy in the management of septic shock complicated by acute kidney injury. The innovative approach of utilizing diuretics to achieve a negative fluid balance, alongside meticulous monitoring of central venous pressure and renal function, demonstrated significant improvements in

the patient's clinical status. The successful resolution of septic shock, marked by enhanced renal function and decreased reliance on vasopressors and ventilatory support, underscores the importance of re-evaluating traditional fluid resuscitation strategies. As the medical community continues to confront the challenges posed by septic shock and associated complications, the findings from this case advocate for a paradigm shift towards fluid stewardship, emphasizing de-resuscitation as a critical component of patient care.

REFERENCES

1. Hayashi Y, Shimazui T, Tomita K, Shimada T, Miura R E, Nakada TA. Associations between fluid overload and outcomes in critically ill patients with acute kidney injury: a retrospective observational study. Sci Rep. 2023;13(1): 1–8. Doi: https://doi.org/10.1038/s41598023-44778-0
2. Prowle JR, Echeverri JE, Ligabo EV, Ronco C, Bellomo R. Fluid balance and acute kidney injury. Nat Rev Nephrol. 2010;6(2):107–15. Doi: https://doi.org/10.1038/NRNEPH.2009.213
3. Zarbock A, Nadim MK, Pickkers P, Gomez H, Bell S, Joannidis M, et al. Sepsis-associated acute a kidney injury: consensus report of the 28th acute disease quality initiative workgroup. Nat Rev Nephrol. 2023; 19(6): 401–17. Doi: https://doi.org/10.1038/s41581-023-00683-3
4. Macdonald SPJ, Keijzers G, McD Taylor D, Kinnear F, Arendts G, Fatovich DM, et al. Restricted fluid resuscitation in suspected sepsis associated hypotension (REFRESH): a pilot randomised controlled trial. Int Care Med. 2018; 44(12): 2070–078.Doi: https://doi.org/10.1007/S00134-018-5433-0
5. Minqiang Huang, Wei Han, Han M, Kuang L. Variability of CVP and Fluid Overload are Associated with Sepsis-associated Acute Kidney Injury: a case-control study. 2019, 1–12. Doi: https://doi.org/10.21203/rs.2.15438/v1
6. Fan Y, Chen L, Jiang S, Huang Y, Leng Y, Gao C. Timely renal replacement therapy linked to better outcome in patients with sepsis-associated acute kidney injury. J Int Med. 2022;2(3): 173–82. Doi: https://doi.org/10.1016/j.jointm.2022.03.004
7. Moschopoulos CD, Dimopoulou D, Dimopoulou A, Dimopoulou K, Protopapas K, Zavras N, et al. New Insights into the fluid management in patients with septic Shock. Medicina. 2023; 59(6): 1–20. Doi: https://doi.org/10.3390/medicina59061047
8. Hofer DM, Ruzzante L, Waskowski J, Messmer AS, Pfortmueller CA. Influence of fluid accumulation on major adverse kidney events in critically ill patients - an observational cohort study. Ann Intensive Care. 2024; 14(1): 1–1052. Doi; https://doi.org/10.1186/s13613-024-01281-7
9. Xiao W, Liu W, Zhang J, Huang L, Liu Y, Hu J, et al. Early persistent exposure to high CVP is associated with increased mortality and AKI in septic shock: A retrospective study. Am J Emerg Med. 2023; 74, 146–51. Doi: https://doi.org/10.1016/j.ajem.2023.09.043
10. AS E, Lestari MI, George YW. Restrictive fluid and fluid removal approach in diabetic ketoacidosis with septic AKI: A case report. Crit Care Shock. 2021; 24, 125–29. Doi: https://criticalcareshock.com/2021/06/3445/
11. Malbrain MLNG, Martin G, Ostermann M. Everything you need to know about deresuscitation. Int Care Med. 2022; 48(12), 1781–786. Doi: https://doi.org/10.1007/s00134-02206761-7
12. Joannidis M, Druml W, Forni LG, Groeneveld ABJ, Honore PM, Hoste E, et al. Prevention of acute kidney injury and protection of renal function in the intensive care unit: update 2017 : Expert opinion of the Working Group on Prevention, AKI section, European Society of Intensive Care Medicine. Intensive Care Med. 2017; 43(6): 730–49. Doi: https://doi.org/10.1007/S00134-017-4832-Y