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Case Report

Sepsis-Associated Acute Kidney Injury in Medical Intensive Care Unit (ICU). A Case Report

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Abstract

Sepsis-associated acute kidney injury (SA-AKI), considered a common impediment of the critically sick patients and is linked with undesirable illness. Avoidance of SA-AKI can be achieved by prompt and plenty fluid restoration especially with crystalloid solutions. Meta-analysis revealed three basic phenomena contributing to SA-AKI that are microvascular dysfunction, inflammation, and metabolic reprogramming.

Keywords: prevention, sepsis-associated acute kidney injury (SA-AKI), medical intensive care unit (M-ICU), Septic AKI (SAKI).

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Introduction

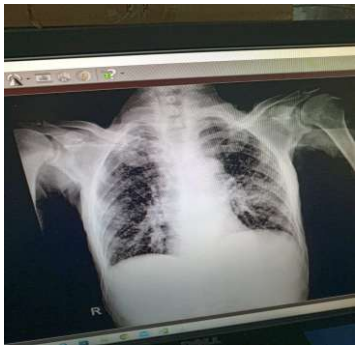
Sepsis is described by an uncontrolled host reaction against pathogens causing lethal organ dysfunction, generally causing acute kidney injury (AKI). Sepsis contributes to about 45 to 70 percent of all AKI cases amongst the critically sick population.[1] Sepsis-associated AKI (SA-AKI) indicates a poorer prognostic outcome and accompanying long-lasting intensive care unit (ICU) pauses, greater death rates, with long-standing frailty and condensed life quality in all group of ages.[2]

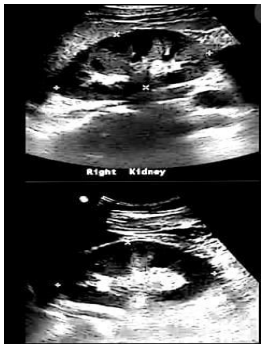
In cooperation sepsis and acute kidney injury (AKI) are illnesses of chief alarm in critical patients. Drastic sepsis is frequently complicated by AKI. The inclusive occurrence of septic AKI (SAKI) amongst all intensive care unit (ICU) population ranks in between 15 and 20 percent.[1] A widespread calculation of 750 million admissions in the hospital of United States era 1979-2000 established that sepsis increased from 82 to 240 per 100,000 people, an annual growth rate and mortality rates were 8.7% and 17.9% respectively.[3]

The pathophysiology of S-AKI is multifactorial, including gastroenteritis, surgeries, congestive cardiac failure, and fluid loss leading to shock. Consequently, it is inviting to point AKI to ischemia and universal hemodynamic variations.[4] The QSOFA score ranges from 0-3 with one point assigned for each of the following clinical signs: a). systolic blood pressure <100 mmHg b). Respiratory rate > 22/minutes, c) altered mental status from baseline. A score >2 showed more severity with increased ICU length of stay and death ratio.

Case report: A 70 year old man previously neither hypertensive nor diabetic presented to emergency department of Khyber teaching hospital Peshawar with high grade fever (recorded spike 100F), shortness of breath with palpitations having productive cough with rusty sputum. In previous weeks he has had severe gastroenteritis which was improved now by fluid resuscitation therapy in private hospital somewhere. For one day patient was retained in medical assessment and decision unit (MADU), then shifted to medical Intensive care unit (M-ICU) for better care. The general physical examination revealed cachectic person, moribund look, anemic, sarcopenia, grade 2 clubbing with mild pedal edema. There was no jaundice, lymphadenopathy or thyromegaly. The vitals showed blood pressure of 100/70 mmHg, pulse rate of 130beats/minute but irregular, oxygen saturation of 88% on oxygen support with a non-rebreathable mask. The systemic examination revealed bilateral scattered coarse crackles all over the chest with bronchial breathing and a respiratory rate of 28 breaths/min. The patient was commenced on crystalloid fluids 3L/day along with broad spectrum antibiotic

coverage for gram negative coverage. The patient was catheterized and nasogastric tube passed. A shot of Lasix (furosemide) 40mg was given alongside a fluid challenge but no improvement in blood pressure was seen at 30 minutes after a flush of 0.9% normal saline at 30ml/kg was given. Despite fluid challenge the patient was in oliguric phase with output of 200ml/24hours. In view of oliguria, patient had an altered mental status GCS 10/15, hyperkalemia, increasing urea and creatinine (no disproportion). The patient was commenced on renal replacement therapy. Three sessions of hemodialysis done. After this management, the patient's urine output improved and gradually so did his GCS. All the baseline investigations, arterial blood gases (ABGs) were done and have been tabulated as shown below.





Laboratory investigations

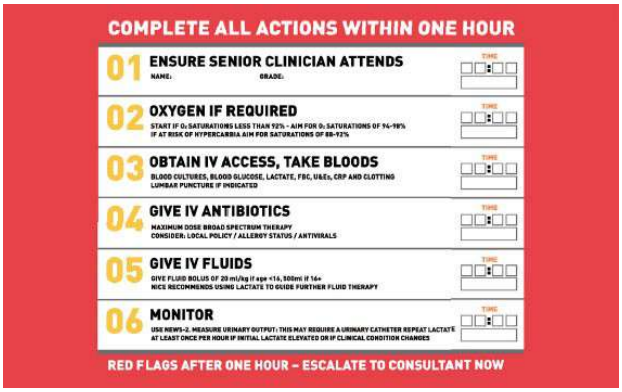
Investigation	Reference Range	21-12-2023	23-12-2023	26-12-2023
Leucocyte Count (10.e3/uL)	4-11	28	24	15
Hemoglobin (g/dl)	12-17	13	11	11
Platelet Count (10.e3/uL)	150-450	50	60	70
Lactate Dehydrogenase (LDH) (U/L)	91-180	5874	4540	3693
Serum Creatinine (mg/dL)	0.6-1.2	4	5	5.2
Blood Urea (mg/dL)	10-50	92	115	108
Alanine Transaminase(ALT)	10-50	56	60	49
Peripheral Smear (RBC Morphology) with Retic count (%)	-	Bicytopenia Anisocytosis ++ Hypochromic Cells ++ Atypical cells 15% Retic count 0.8%	-	-
Uric Acid (mg/dL)	3.4-7	5	-	5.6
Urine R/E	-	Protein- Trace Rest-Unremarkable	-	-
Sodium (mmol/L)	135-145	150	135	134
Potassium (mmol/L)	3.5-5.1	4	4.5	3.4
Serum Calcium (mg/dL)	8-10	8.5	7	7.8

Chest X-ray of Patient & Kidneys Ultrasound

Discussion

Criterion	Threshold	
	SIRS	qSOFA
Body temperature (°C)	<36 or >38	-
Heart rate (beats/min)	>90	-
While blood cell count ($10^3/\mu L$)	<4 or >12	-
Respiratory rate (breaths/min)	>20	≥22
Systolic blood pressure (mmHg)	-	≤100
Glasgow Coma Scale	-	≤13

SIRS: systemic inflammatory response syndrome, qSOFA: quick Sepsis-related Organ Failure Assessment.



Sepsis is a frequent fatal clinical disorder in the intensive care units, distressing 20–30 million individuals internationally per annum. Sepsis severity straight away causing multi-organ dysfunction is the principal root cause of death in ICU population. Acute kidney injury (AKI) is a traditional and severe hurdle to sepsis treatment with Sepsis associated acute kidney injury (SAKI) ratio being up to 47.5%.[5]

The identification of AKI is presently established on a rise in serum creatinine levels and/or a reduction in urine output. Concurrently, serum creatinine can be an indifferent sign of renal injury, and oliguria can be common in S-AKI. Conversely, in sepsis, oliguria seems to convey enlarged consequences, and even by 3 to 5 hours, a relationship concerning oliguria and AKI might be measurable.[6]

Even though sepsis devours well-known primary cause of AKI in the critically ill patients, Mehta et al established, 40% of critically sick patients progress sepsis afterwards AKI, signifying that AKI might perhaps

upsurge the sepsis hazards. Yet, both sepsis and AKI are clinical diagnoses, and it is typically challenging to describe the accurate interval of their initiation.[6]

In lieu of patients in the ICU, sepsis is present in 40-50% of patients with AKI in the ICU. A prospective cohort study comprising 1177 septic patients through 198 ICUs in 24 European nations testified a 51% prevalence of AKI with an ICU death rate of 41%. A retrospective study crosswise China containing 146,148 patients found AKI in 47.1% of sepsis cases. An auxiliary exploration of a multicenter randomized controlled trial (RCT) in septic shock comprising 1243 patients, AKI was existing at registration in the crisis sector in 50.4% and additional 18.7% established succeeding AKI within one week. 70% of patients with AKI were categorized as stage 2 or 3. Nonetheless, AKI is common even amongst patients without severe sepsis or shock; 34% of non-severe community-acquired pneumonia showed established AKI.[7]

Entirely intravenous fluids can add hostile renal and patient consequences by fluid excess and renal edema. In a potential observational learning, comprising 2526 ICU patients, every day and collective fluid equilibrium in first 3 days was greater in patients with AKI and fluid overload remain an autonomous threat for AKI and its severity.[8]

Conclusion

In conclusion, Avoidance of SAKI flinches with timely and sufficient fluid administration. Crystalloid fluids are chosen over colloid fluids but well-adjusted crystalloids do not seem grander over classic crystalloids for neutralizing SAKI. Lactate clearance rate, however, is well interrelated with SAKI-related mortality. Norepinephrine considered the vasopressor of high-quality for inhibiting SAKI. Prompt commencement of RRT is mandatory when fluid overload is extreme or stubborn to diuretic therapy. CRRT is progressively well-thought-out as primary cure in hemodynamically insecure SAKI.

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