Open Access Journal

www.ijirms.in

Sepsis Induced Acute Kidney Injury: Clinical Profile, Risk Factors and Outcomes in Tertiary Referral Centre

Sunil R^{*1}, Mohammad fakruddin², R Sandeep³, Amogh C⁴, Nutan kumar DM⁵

Department of Nephrology, KIMS Hospital and Research Centre

Abstract:

Sepsis increases the risk of development of acute kidney injury (AKI) and is associated with significant patient morbidity and mortality. Sepsis induced AKI is characterized by a distinct pathophysiology and therefore requires a different approach and management. A prospective study was conducted to look for pattern of sepsis induced AKI in South Indian population. A total of 234 patients were enrolled in the study .Patient's detailed history, clinical examination and laboratory investigations were noted and source of sepsis was evaluated, patients were followed up till discharge or death. During follow up development of complications or requirement of renal replacement therapy was noted. Sepsis associated AKI was associated with significant morbidity and mortality.

Keywords: Acute Kidney Injury, Sepsis, Hemodialysis.

Introduction:

Sepsis is a serious medical condition characterized by a whole-body inflammatory state (systemic inflammatoryresponse syndrome) and the presence of a known or suspected infection that has severe consequences, including multiple organ failure including renal involvement in the form of acute kidney injury. [1]

Acute kidney injury (AKI) is a frequent and serious complication of sepsis in intensive care unit (ICU) patients particularly in the elderly. Moreover, there is strong evidence that sepsis and septic shock are the most important causes of AKI in critically ill patients, account for 50% or more of cases of AKI in ICUs, and associate with a very high mortality. [1]

Acute kidney injury (AKI) is described by rapid (over hours to days) decrease in glomerular filtration rate, maintenance of nitrogenous waste items and annoyance of the extracellular liquid volume, electrolytes and corrosive base homeostasis [2]. AKI causes diminished renal perfusion without cell harm, an ischemic, lethal or obstructive affront to the renal tubule, a tubulointerstitial procedure with irritation and edema or essential decrease in the separating limit of the glomerulus. The death rate among patients with AKI approaches half and has changed minimal in the course of recent years. There is an expansion in the rate of surgical and sepsis-related AKI [3]. Sepsis is the most common contributing factor for the development of AKI. In adult and pediatric data, sepsis accounts for 26% to 50% of all AKI in developed nations, compared with 7% to 10% of primary kidney disease associated AKI. Clinical and basic science evidence indicate that sepsis-associated AKI (SA-AKI) is distinct from AKI without sepsis, driven by a number of characteristic pathophysiological mechanisms, carrying a unique profile of timing (onset, duration), and being associated with different short-and long-term outcomes.[4]

This study was conducted in nephrology department in KIMS hospital and research center, a tertiary care centre in Bengaluru, India, to assess clinico-etiological aspects, risk factors, clinical course and outcome of sepsis induced AKI.

Methods:

It is a prospective study, done over a period of 2 years in a tertiary care hospital during October 2014 to November 2016. All adult (>18 years) patients admitted to this hospital who had AKI secondary to sepsis were included. 234 cases with sepsis, clinical (uremic symptoms or oliguria or anuria of recent onset) and laboratory evidence of azotemia (urea and creatinine above 40 mg/dl and 1.5 mg/dl, respectively) were eligible. Patients with chronic renal failure, acute on chronic renal failure, renal transplant and those not willing to participate were excluded from this study. The patients included in the study were explained in detail about the purpose of the study and an informed consent was taken.

Evaluation included a detailed history, physical examination and laboratory investigations. Patients were followed up until discharge or death. Each patient was looked for the complications of AKI-like fluid overload, hypertension, electrolyte abnormalities, metabolic acidosis, uremic complications, bleeding, neurological abnormalities and infections. Hemodialysis was instituted as and when required. Kidney injury was listed as a cause of death if patient exhibited evidence of severe uremia, hyperkalemia or volume overload secondary to oliguria. Patients were classified as oliguric (urine output<500 ml/day) and nonoliguric (urine output>500 ml/day) during the azotemic phase. The study has been approved by institutional ethics committee. Chi-square test was used for statistical analysis.

All patients aged above 18 years with features of AKI (secondary to sepsis) as per AKIN (Acute Kidney Injury Network) criteria [5] which is defined as an increase in serum creatinine of 0.3 mg/dl or more within 48 hours of observation or 1.5 times baseline or greater, which is known or presumed to have occurred within 7 days, or a reduction in urine volume below 0.5 ml/kg/h for 6 hours. Both inpatients and out patients during the period of October 2014 to November 2016 were included in our study.

Results:

 Table 1: Source of sepsis which causing aki in our study

 group

SOURCE	NO OF PATIENTS
URINARY TRACT	88 (37.60%)
RESPIRATORY TRACT	59 (25.21%)
CELLULITIS	45 (19.23%)
GI TRACT	24 (10.25%)
DEVICE RELATED	9 (3.84%)
BLOOD	6 (2.56%)
UNKNOWN	3(1.28%)

A total of 234 patients were enrolled in the study. The mean age of these patients was 51.26 ± 11.3 (range 18 to 83) years. The number of males was 129 (55.12%) and 105 were females (44.87%).

Most common source of sepsis which resulted in AKI was urinary tract infection in 88 (37.60%)patients, Respiratory tract infection in 59 (25.21%) patients, cellulitis 45 (19.23%) patients, GI tract in 24 (10.25%), Device related in 9 (3.84%) patients, blood related sepsis in 6 (2.56%) patients and unknown in 3(1.28%).

Table 2: Potential risk factors for sepsis seen in ourstudy group

Risk factors	Number of patients, n (%)
Diabetes mellitus	154(65.81%)
Hypertension	128(54.70%)
Alcohol	69 (29.4%)
Congestive heart failure	58(24.78%)
COPD/Smoking	53(22.64%)
Chronic liver disease	36(15.38%)
Malignancy	22(9.41%)
Retroviral diseae	20(8.54%)

Potential risk factors observed in our study includes Diabetes mellitus in 154(65.81%), Hypertension in 128(54.70%), Alcohol in 69 (29.4%), Congestive heart failure in 58(24.78%), COPD/Smoking in 53(22.64%), Chronic liver disease in 36(15.38%), Malignancy 22(9.41%) and Retroviral disease in 20(8.54%) patients.

Table 3:	Renal	replacement	therapy	and	outcome
		- opinioni	· · · · · · · · · · · · · · · · · · ·	*****	04000

Type of RRT	Outcome	Percentage
Hemodialysis	88	37.60%
SLED	26	11.11%
No Dialysis	120	51.28%

Out of 234 patients 114 patients underwent renal replacement therapy, 88 (37.60%) cases had undergone normal Hemodialysis whereas 26(11.11%) cases undergone SLED because of low blood pressure.

Table 4: Outcome

Death	Recovered
63(26.93%)	171(73.07%)

Outcome wise 63 (26.93%) patients were died and 171 survived (73.07%).

Discussion:

Acute Kidney injury (AKI), the most common renal manifestation of sepsis, is not an isolated event but often a component of MODS that may complicate sepsis, indicating that similar mechanisms are operative in inducing the dysfunction of various organ systems. Sepsis and particularly septic shock are important risk factors for the development of Acute Kidney injury (AKI). [08]

During severe sepsis, in addition to overwhelming production of inflammatory humoral mediators and activation of cellular system, there is activation of sympathico-adrenal axis with increased plasma levels of (nor) epinephrine, of renin-angiotensin aldosterone system (RAAS) with elevated levels of angiotensin II and a rise in vasopressin levels are often part of host response. These mechanisms are largely responsible for the clinical manifestations of sepsis, including the hemodynamic alterations that are characterized by vasodilation, a hyperdynamic circulation and microcirculatory changes contributing to inefficient oxygen extraction.[05]

The mortality is high in patients with multiorgan failure as in other studies.[6,7,8] Mortality was high in patients having AKI due to medical causes as compared to patients with AKI resulting from surgical and obstetrical causes. Multiorgan failure in our study was seen in 106 (16.98%) patients. Among them 60 (63.6%) patients were expired. Vasopressor support were needed for 134 patients to maintain hemodynamic stability. Among them 29 (40%) died within 48 hours of admission. Whereas, only 5(3.73%) patients who were not supported with vasopressors had mortality.

Mortality was seen higher in patients who required vasopressor support and amongst those who required dialysis therapy. Vasopressor supports as well as dialysis support are considered a prognostic factor for outcome in AKI patients.[09]

Conclusion:

Sepsis was the most common cause of AKI in our study. The judicious and timely management of sepsis and prompt initiation of renal replacement therapy will certainly reduce the mortality in AKI.

References:

- Sepsis and Acute Kidney Injury Abolfazl Zarjou and Anupam Agarwal J Am Soc Nephrol 22: 999 – 1006, 2011. doi: 10.1681/ASN.2010050484
- [2] S. Kumar, S. Raina, S. Vikrant, and R. K. Patial. Spectrum of acute kidney injury in the Himalayan region. Indian J Nephrol. 2012 Sep-Oct; 22(5): 363–366.
- [3] Umesh L. Acute kidney injury: Experience from a state run tertiary care centre in Southern India. International Journal of Medical Research & Health Sciences, 2016, 5, 5:83-87
- [4] Alobaidi R, Basu RK, Goldstein SL, Bagshaw SM. Sepsis-Associated Acute Kidney Injury. Seminars in nephrology. 2015; 35(1):2-11. doi:10.1016/j.semnephrol.2015.01.002.
- [5] Sepsis and the Kidney SC Tiwari*, Sanjay Vikrant** Journal of Indian Academy of Clinical Medicine Vol. 5 No. 1

- [6] Prasher PK, Verma PP, Chauhan SS. Spectrum of ARF in hospital setting. Indian J Nephrol 1996;6:114
- [7] Frost L, Pederson RS, Bentzon S, Billeh H, Hansen HE. Short and long term outcome in a consecutive series of 419 patients with acute dialysis-requiring renal failure. Scand J UrolNephrol 1993; 27:453-62.
- [8] Brivet FG, Kleinknecht DJ, Loirat P, Landais PJ. Acute renal failurein intensive care units- causes, outcome and prognostic factors of hospital mortality; a prospective multicentre study. French Study Group on Acute Renal Failure. Crit Care Med 1996; 24:192-9.
- [9] Liaño F, Garcia-Martin F, Gallego A, Orte L, Teruel JL, Marcén R, et al. Easy and early prognosis in acute tubular necrosis: a forward analysis of 228 cases. Nephron 1989;51:307-13