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# Epidemiology And Outcomes Of Community Acquired Acute Kidney Injury: A Prospective Single Center Study

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***Abstract***
***Purposes***
*Community Acquire AKI (CAAKI) develops outside the hospital settings and imposes significant burden on healthcare with its high mortality rate and long-term consequences in selected cases. Infective causes are commonly incriminated and thus CAAKI incidence can be curtailed with social and preventive aspect of modern medicine. We studied patients of CAAKI admitted at our institute with follow-up for 6 months.*
***Methods***
*All patients aged >18yrs, with diagnosis of CAAKI admitted to our Institute, a tertiary care centre located in northern part of India, was included in the study for a period of 3 years (2019-2022). The etiological spectrum and renal and patient outcomes of CA-AKI at the index visit and at 1-month, 3-month and 6 month follow-ups were analyzed.*
***Findings***
*Sepsis (38.8%) was the most common cause of AKI followed by Acute Gastroenteritis. Drug induced AKI was the third most common cause with 9.4% of total cases. Mortality at the index admission was 8.5% and Complete recovery at discharge was observed in 14% of patients. Remaining patients (77.5%) had partial recovery on discharge. On follow Up at 6 Months, 13 patients (5.5%) had persistent renal dysfunction of which 2 (0.85%) were on Renal replacement therapy.*
***Conclusion***
*Infective causes remain the common culprits of CAAKI with large contribution from Drug induced AKI. Significant number of patients progress to CKD and/or ESRD. Improving Socio-economic status is likely to lower the incidence of CAAKI.*

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## I. Introduction

Acute kidney injury is a significant cause of morbidity and mortality worldwide and more so in developing countries. Community acquired AKI (CAAKI) develops outside of a hospital in home settings and has significantly different etiologies than hospital acquired AKI (HAAKI). AKI can also lead to CKD in significant group of patients which has further healthcare implications in developing countries like India. We aimed to evaluate the etiologies of CAAKI admitted to us over a period of 3 years (2019- 2022) with follow up of individual patients over the course of 6 months.

## II. Methods

This is a single centre prospective observational study where we studied adults with AKI with follow up for 6 months post hospitalization.Baseline characteristics that included eGFR, hemogram, ultrasonography of KUB of each patient was noted. Specific etiologies of AKI were determined after a detailed history and physical examination. All patients received standard treatment as per the existing guideline including renal replacement therapy (RRT). All participants had assessments of kidney function (eGFR) at one, three and six months after the index hospitalization. This study was approved by the institutional ethics committee.

**Inclusion and exclusion criteria**

All patients >18 years of age with CA-AKI, defined as AKI occurring outside the hospital setting—typically in the community or home setting—and admitted to inpatient department of the institute were included in the study. Patients who had HA-AKI, developed AKI at any time after 48 h of hospitalization, preexisting CKD, renal transplant recipients, and those with incomplete records were excluded.

**Data collection**

The following data were collected: clinical ; risk factors and etiology of AKI; comorbidities; laboratory investigation results; management; and patient outcomes. Patients with pre-existing diabetes mellitus, hypertension, or other cardiovascular diseases were also recorded. CKD was defined as kidney function and/or structural abnormalities persisting >3 months. Demographic and clinical data included age, sex, BMI, blood pressure, and general and systemic physical examinations. Laboratory parameters included baseline and follow-up hemoglobin levels and leukocyte counts, blood urea nitrogen (BUN), serum creatinine, proteinuria, serum uric acid, alkaline phosphatase, and any other special investigation(s) required for diagnosis at the discretion of the treating clinician.

Height and weight measurements were collected from each patient to calculate BMI using the following equation: BMI = weight (kg)/height (m2 ). Patients were categorized as underweight (BMI < 18.kg/m2), normal weight (BMI 18.5–22.9 kg/m2 ), overweight (BMI 23–24.9 kg/m2 ), and obese (BMI ≥25 kg/m2 ), as per the Indian Council of Medical Research guidelines for the categorization of BMI

Renal function assessment AKI was defined and classified using the KDIGO guidelines, which used serum creatinine (increase in serum creatinine by ≥0.3 mg/dl within 48 h or increase in serum creatinine to ≥1.5 × baseline) and urinary output <0.5ml/kg/hr.For patients with serum creatinine values before admission, the most recent value was considered the baseline level. For patients without baseline creatinine in the 7–365 days before admission, baseline creatinine was imputed by back calculation using the Modification of Diet in Renal Disease (MDRD) equation and glomerular filtration rate (GFR) of 75 ml/min/1.73 m2 , as suggested by Pickering et al.16 Staging of AKI was based on the following: an elevation of serum creatinine level 1.5–1.9 × baseline or ≥0.3 mg/dl elevation (stage 1); elevation of serum creatinine 2.0–2.9 × baseline (Stage 2); and 3.0 × baseline or increase in serum creatinine to ≥4.0 mg/dl or the initiation of renal replacement therapy (Stage 3).

**Etiological assessment**

The settings and specific etiologies of CAAKI were noted. When AKI was multifactorial, the factor attributed by the treating clinician as the most contributory factor was considered the etiology of AKI. If no clear single etiology was ascertained, it was considered undetermined. Sepsis was defined according to the Third International Consensus Definition as a documented source of infection with a quick Sequential Organ Failure Assessment (qSOFA) score ≥2.

**Follow-up and outcome measures**

Each patient was followed up for renal outcomes at discharge and at 1 ,3 and 6 months after the onset of AKI. Patient outcome(s) were classified as complete Recovery, partial recovery and and non-recovery. Among the survivors, complete recovery (CR) was defined as adequate urine output (>1 ml/kg/h) with serum creatinine < 1.4mg/dl without persistent proteinuria or microscopic hematuria. Partial recovery was defined as decrease in serum creatinine by >50% with improved urine output (>0.5ml/kg/h) and no further need of dialysis. Dialysis dependency was defined as need for any form of dialysis for >28 days. Proteinuria was defined as urine protein excretion of ≥1+ on urine dipstick examination. Microscopic hematuria was defined as >5 red blood cells/high-power field on spun urine evaluation.

## III. Results

Total of 255 patients were enrolled in the study over a period of 2 years. 20 patients did not return for follow up. Thus data from 235 patients with AKI were analyzed.

**Demographics**

The baseline characteristics of the patients are summarized in table 1. The mean age of the study population was 50.6 yrs. AKI severity was not related to the age or gender of population. Mean BMI of patients was 22.24 kg/m2. Majority of the patients belong to KDIGO AKI Stage 3(N=192, 75%).

| | Total N=255 | KDIGO stage 1 N=30 | KDIGO stage 2 N=33 | KDIGO stage 3 N=192 | P value |
|---|---|---|---|---|---|
| Age (mean ±SD) years | 50.60(18.27) | 52.00 (17.64) | 49.64 (19.81) | 50.55 (18.17) | 0.93 |

| | | | | | |
|---|---|---|---|---|---|
| Gender Male, n (%)<br>Female, n (%) | 144 (56.5)<br>111 (43.5) | 18 (60.0)<br>12 (40.0) | 13 (39.4)<br>20 (60.6) | 112 (58.9)<br>78 (41.1) | 0.18<br>0.13 |
| BMI | 22.24 (3.89) | 21.02 (4.04) | 22.51 (3.488) | 22.38 (3.906) | 0.64 |
| Hypertension, n (%) | 26 (10.2) | 3 (10) | 3 (9.1) | 20 (10.4) | 0.83 |
| Diabetes Mellitus, n (%) | 28 (11.0) | 6 (20) | 2 (6.1) | 20 (10.4) | .001 |
| CLD, n (%) | 15 (5.9) | 00 (00) | 1 (3.0) | 14 (7.3) | 0.13 |
| CAD n (%) | 8 (3.1) | 00 (00) | 2 (6.1) | 6 (3.1) | 0.98 |
| Haemoglobin, g (%) Mild<br>Moderate<br>Severe | 68 (26.7)<br>99 (38.8)<br>32 (12.5) | 5 (16.7)<br>8 (26.7)<br>4 (13.4) | 9 (27.3)<br>18 (54.5)<br>3 (9.1) | 53 (27.9)<br>73 (38.11)<br>25 (13.2) | 0.07<br>0.07<br>0.07 |
| TLC (*103) per cu mm | 12 (8-17) | 9.9 (5.9-15.7) | 12.5 (7.7-18.9) | 12.1 (8.7-18) | 0.75 |
| Platelet count (lac/cu mm) | 1.34 (.92-2.1) | 1.1 (.7-1.8) | 1.36 (.9-2.24) | 1.34 (.98-2.1) | 0.695 |
| S. Creatinine, mg (%) | 4.9 (2.9-7.3) | 1.5 (1.2-1.7) | 2.4 (2.2-2.5) | 6 (4.3-8.3) | 0.001 |
| Sodium, mg (mmol/L) | 133.9 (130-138) | 133.1(129.7-137.5) | 135.59 (132.5-139.0) | 133.8 (130-138) | 0.21 |
| Potassium, (mmol/L) | 4.3 (3.4-4.3) | 4 (2.0-4.7) | 4.5 (3.7-5.1) | 4.3 (3.4-5.1) | 0.56 |
| AST (IU /L) | 79 (41-86) | 70(32-87) | 74 (66-91) | 80 (41-86) | 0.84 |
| ALT (IU/L) | 53 (26-77) | 59 (21-81) | 54 (25-88) | 52 (26-87) | 0.81 |

**Table 1- Baseline characteristics of patients according to AKI staging (KDIGO 2012)**

**Associated Comorbidities**

Hypertension (HTN) and diabetes mellitus (DM) were the most common comorbidities followed by chronic liver disease (CLD) and coronary artery disease (CAD) . Majority of patients with CLD presented in KDIGO stage 3.

**AKI evaluation and staging**

Of 255 patients enrolled in the study, 192 patients were classified as AKI stage 3 with remainder being in Stage 2 (N=33) and stage 1 (N=30). Most patients,irrespective of their AKI staging, had transaminitis [Mean-79(AST), 56(ALT) ].

Total 255 patients with AKI were diagnosed and further subdivided according to their etiology. Most common etiology was sepsis (N= 99, 39%) followed by Acute Gastroenteritis (N=28, 11%). Drug induced AKI was found in 24 patients (9.4%) with ACE inhibitors/ ARBs , NSAIDsand Rifampicinbeing the most common culprit. Herbal medications as causative agent wasfound in 3 patients (N=1.2%). Other commonly encountered causes were Nephrotic syndrome(N=23, 9%) , Tropical fever (N=17,6.7%) and Cardiorenal syndrome (N=12, 4.7%). See Table 2

**Aki Etiologies**

| | | Frequency | Percent |
|---|---|---|---|
| | **Sepsis** | 99 | 38.8 |
| | **Gastroenteritis** | 28 | 11.0 |
| | **Drug induced** | 24 | 9.4 |
| | **Nephrotic Syndrome** | 23 | 9.0 |
| | **Tropical fever** | 17 | 6.7 |
| | **Cardiorenal Syndrome** | 12 | 4.7 |
| | **Obstructive Uropathy** | 11 | 4.3 |
| | **Multiple myeloma** | 9 | 3.5 |
| | **Glomerulonephritis** | 8 | 3.1 |
| | **Acute Pancreatitis** | 7 | 2.7 |
| | **Hepatorenal Syndrome** | 5 | 2.0 |
| | **Thrombotic microangiopathy** | 4 | 1.6 |
| | **Herbal Medication** | 3 | 1.2 |
| | **Hypercalcemia** | 2 | 0.8 |
| | **Snake bite** | 2 | 0.8 |
| | **Rhabdomyolysis** | 1 | 0.4 |
| | **Total** | 255 | 100.0 |

Table 2 – Etiological spectrum of acute kidney Injury

**Clinical Outcomes**

Out of 235 patients ,33 (14%) had complete recovery at the time of discharge. Mortality at the index admission was 8.5% (N=20). Remaining patients (77.5%) had partial recovery at discharge. No patients were dialysis dependent at the time of discharge. On subsequent follow up at 1 months ,additional 104 patients (44.2%) showed complete recovery and another 22 patients died with a total mortality of 17.8% . At the follow

up 3 months , complete recovery was noted in 70.6 % patients. 15 patients showed persistent renal dysfunction (Serum Cr.>1.4mg/dl)

At 6 months, additional 2 patients had complete recovery . No additional mortality was noted. 13 patients (5.5%) had persistent renal dysfunction. 2 patients were dialysis dependent (0.85%).

See Table 3 and 4

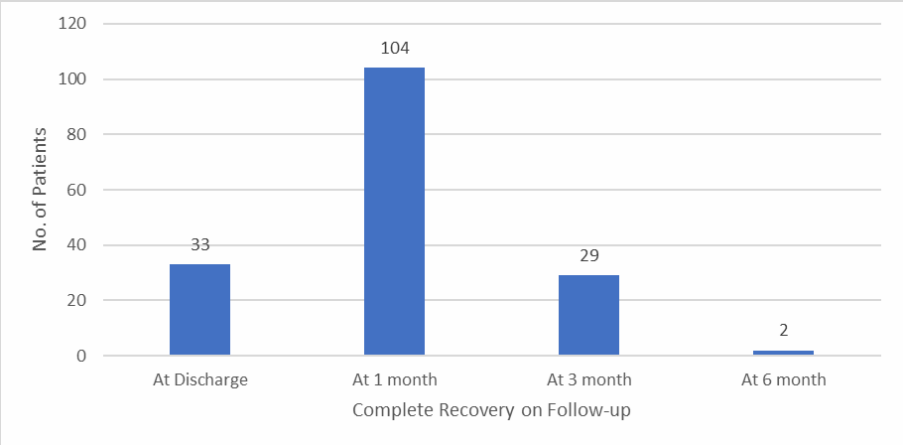


**Table 3-Complete Recovery of patients at discharge and Follow-up**

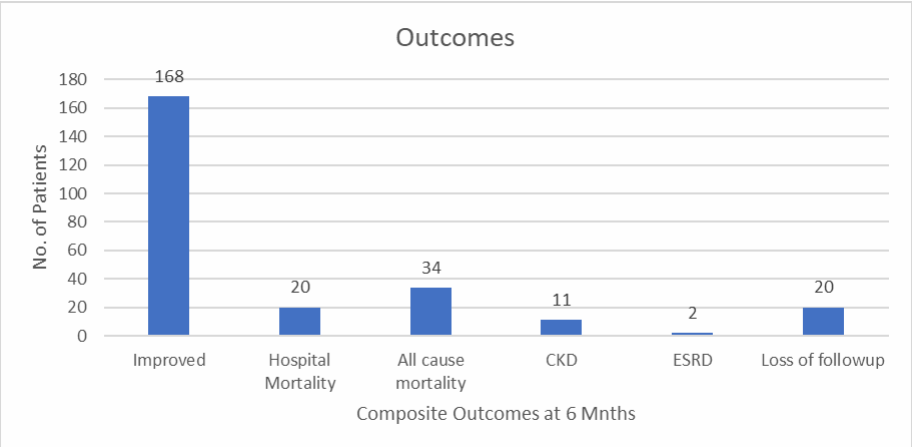


**Table 4- Outcomes of patients at discharge and Follow-up in terms of Mortality and adverse renal outcomes.**

## IV. Discussion

The reported incidence of AKI in different regions of the world is widely variable . In developed countries, AKI primarily develops in hospitalized patients, while it is mainly community-acquired in developing countries [1,2]. Challenges facing healthcare systems in LMICs (Low and Middle income countries), such as India, the most populous country in the world with varied climates in different regions, are addressing both forms of AKI [3,4]. One of the largest descriptions of CA-AKI comes from nationwide survey of 2.2 million hospitalized patients in China [5]. The study confirmed the association between epidemiology and outcomes of CA-AKI with socio-economic factors and geo-graphical location.

In this study, we found that mean age of patients were 50.6 yrs (+_18.2) and majority of patients were AKI stage 3. Only a minority of patients (14%) had complete recovery at discharge and this probably reflects our policy of early discharge on noting clinical improvement.

**Aki Spectrum**

The most common cause for AKI in our study was Sepsis. Other studies have also reported sepsis as one of the leading cause of AKI.[6,7].Recent trend have also shown a rise in sepsis related AKI[8]. and occurrence of AKI represents an independent risk factor for longer intensive care unit (ICU) and hospital stays, higher mortality, increased rate of long-term disability and reduced quality of life in adult and paediatric populations in these patients[9,10]. This is likely attributed to simultaneous involvement of other organs and

hemodynamic alterations during the hospital course. In one study[11].sepsis accounts for 45%–70% of all AKI events . Meanwhile, around 60% of patients with sepsis have AKI[12].

A single center study from Northern part ofindia have suggested diarrhoeal disease as the most common cause of CAAKI[8]. In our study it was found as the second most common cause after sepsis.

ThoughDrug induced AKI is commonly encountered in the hospital settings, we noted this entity in 9.4% of our CAAKI patients.NSAIDs, ACEI/ARBs and Rifampicin were most commonly encountered.Drugs are associated with AKI in 14%–26% of adults in prospective cohort studies[13,14] and 37.5% in a cross-sectional survey[15]. Although medications induce various forms of kidney injury, drug-induced injury to the tubulointerstitial compartment is a common cause of AKI[16] .Previous studies also have showed an increasing trend of Nephrotoxic drugs related AKI[17]. This is especially important in our clinical setup where access to healthcare is limited, allowing Indiscriminate use of over the counter medications, commonly NSAIDs.

Tropical fever (Malaria, Leptospirosis and Dengue fever being the most frequently encountered) remains a common cause in ur setup.Previous studies have also reported similar incidence rate[17]. A large retrospective study on epidemiological trends in CA-AKI between 1990 and 2014 from Pakistan has shown that numbers due to rhabdo-myolysis, malaria, and dengue increased over time[18] .In a large hospital in Chandigarh, India that was seeing many cases with AKI in the setting of an undifferentiated febrile illness, the availability of a specific and sensitive test allowed the identification of scrub typhus as the cause in a quarter of cases[19] . This has important implications since scrub typhus can be easily treated with doxycycline and thus avoiding many nephrotoxic IV medications. In contrast to previous study[20], snake bite as causative agent was uncommon in our study.This is quite expected as this entity is heavily dependent on local geography and environmental factors. We also noted lower incidence of acute GN (3.1%) and this may partly be explaineddue to lack of pediatric patients in our study.Amongst the surgical cause, Bladder outlet obstruction was the most common cause followed by Nephrolithiasis.

**Patient Outcome**

Mortality rate at index admission was 8.5% . Though mortality in CAAKI can vary widely because of underlying health conditions, access to healthcare, treatment options, and demographics, this also means a significant role of socio-economic conditions in determining overall mortality. A recent Multi-centrestudy has reported mortality of around 10.8%[7]. A study from Gujarat in India reported a mortality rate of 11.8%. In contrast, a United-Kingdom-based Study by Wonacott et al. reported a mortality rate of 43.7% in CA-AKI [21].

A minority of patients in our cohort showed complete recovery at index admission but on follow up at 1months, significant majority had recovered. Those patients who still had renal dysfunction at 1 months were labeled as AKD and this has been found to be of prognostic importance[22]. In our study, AKD was noted to be in 23.8% patients.However, on further follow up, majority of these patients showed complete recovery. At 3 months, 6.3% of the patients were labeled as CKD because of the persistent renal dysfunction. Narayan Prasad et al reported 8% of CKD incidence following an episode of AKI[7]. There are limited data regarding CKD progression from CA-AKI, and most studies have focused on HA-AKI progression to CKD. Better and closer follow-up protocols by nephrologists may help improve the short- and long-term outcomes of AKI.

## References


[1] LameireN, BiesenWv, Vanholder R. The Changing Epidemiology Of Acute Renal Failure, Nat Clin Pract Nephrol, 2006, Vol. 2 (Pg. 364-377)

[2] Jha V. ,Parameswaran S. Community-Acquired Acute Kidney Injury In Tropical Countries.Nat Rev Nephrol. 2013; 9: 278-290

[3] Cerdá J, Bagga A, Kher V, Chakravarthi R.M.The Contrasting Characteristics Of Acute Kidney Injury In Developed And Developing Countries. Nat Rev Nephrol. 2008; 4: 138-153

[4] State-Wise Percentage Of Population Below Poverty Line By Social Groups, 2004-05 | Department Of Social Justice And Empowerment - Government Of India. ([Cited 2023 May 3]. Available From:)Https://Socialjustice.Gov.In/Common/76672

[5] Wang Y, Wang J, Su T, Et Al. Community-Acquired Acute Kidney Injury: A Nationwide Survey In China. Am J Kidney Dis. 2017;69:647–657.

[6] Vikrant S., Gupta D., Singh M. Epidemiology And Outcome Of Acute Kidney Injury From A Tertiary Care Hospital In India. Saudi J Kidney Dis Transplant. 2018;29(4):956–966

[7] Narayan Prasad Et Al. Community-Acquired Acute Kidney Injury In India: Data From Isn-Acute Kidney Injury Registry.Lancet Reg Health Southeast Asia. 2024 Feb; 21: 100359.

[8] Kaul A., Sharma R.K., Tripathi R., Suresh K.J., Bhatt S., Prasad N. Spectrum Of Community-Acquired Acute Kidney Injury In India: A Retrospective Study. Saudi J Kidney Dis Transplant. 2012;23(3):619

[9] Poston, J. T. &Koyner, J. L. Sepsis Associated Acute Kidney Injury. Bmj 364, K4891 (2019).

[10] Schuler, A. Et Al. The Impact Of Acute Organ Dysfunction On Long-Term Survival In Sepsis. Crit. Care Med. 46, 843–849 (2018).

[11] Uchino, S., Kellum, J. A., Bellomo, R., Doig, G. S., Morimatsu, H., Morgera, S., Et Al. (2005). Acute Renal Failure In Critically Ill Patients: A Multinational, Multicenter Study. Jama 294 (7), 813–818. Doi:10.1001/Jama.294.7.813

[12] Bagshaw, S. M., Lapinsky, S., Dial, S., Arabi, Y., Dodek, P., Wood, G., Et Al. (2009). Acute Kidney Injury In Septic Shock: Clinical Outcomes And Impact Of Duration Of Hypotension Prior To Initiation Of Antimicrobial Therapy. Intensive Care Med. 35 (5), 871–881. Doi:10.1007/S00134-008-1367-2

[13] Mehta Rl, Pascual Mt, Soroko S, Savage Br, Himmelfarb J, Ikizler Ta, Paganini Ep, Chertow Gm; Program To Improve Care In Acute Renal Disease: Spectrum Of Acute Renal Failure In The Intensive Care Unit: The Picard Experience. Kidney Int 66: 1613–1621, 2004
[14] Hoste Ea, Bagshaw Sm, Bellomo R, Cely Cm, Colman R, Cruz Dn, Edipidis K, Forni Lg, Gomersall Cd, Govil D, Honoré Pm, Joannes-Boyau O, Joannidis M, Korhonen Am, Lavrentieva A, Mehta Rl, Palevsky P, Roessler E, Ronco C, Uchino S, Vazquez Ja, Vidal Andrade E, Webb S, Kellum Ja: Epidemiology Of Acute Kidney Injury In Critically Ill Patients: The Multinational Aki-Epi Study. Intensive Care Med 41: 1411–1423, 2015
[15] Liu C, Yan S, Wang Y, Wang J, Fu X, Song H, Tong R, Dong M, Ge W, Wang J, Yang H, Wang C, Xia P, Zhao L, Shen S, Xie J, Xu Y, Ma P, Li H, Lu S, Ding Y, Jiang L, Lin Y, Wang M, Qiu F, Feng W, Yang L: Drug-Induced Hospital-Acquired Acute Kidney Injury In China: A Multicenter Cross-Sectional Survey. Kidney Dis (Basel) 7(2):143–155, 2021
[16] Perazella Ma: Pharmacology Behind Common Drug Nephrotoxicities. Clin J Am Soc Nephrol 13: 1897–1908, 2018
[17] Jai Prakash, TakhellambamBrojen Singh, Biplab Ghosh, Vinay Malhotra, Surendra Singh Rathore, Rubina Vohra, Rabindra Nath Mishra, Pramod Kumar Srivastava, Usha, Changing Epidemiology Of Community-Acquired Acute Kidney Injury In Developing Countries: Analysis Of 2405 Cases In 26 Years From Eastern India, Clinical Kidney Journal, Volume 6, Issue 2, April 2013, Pages 150–155
[18] Naqvi R. Epidemiological Trends In Community Acquired Acute Kidney Injury In Pakistan: 25 Years Experience From A Tertiary Care Renal Unit. Pak J Med Sci. 2021;37:312–319
[19] Kumar V, Kumar V, Yadav Ak, Et Al. Scrub Typhus Is An Under-Recognized Cause Of Acute Febrile Illness With Acute Kidney Injury In India. PlosNegl Trop Dis. 2014;8:E2605
[20] Kaaviya R., Vadivelan M., Balamurugan N., Parameswaran S., Thabah M.M. Community Acquired Aki: A Prospective Observational Study From A Tertiary Level Hospital In Southern India. Indian J Nephrol. 2019;29(4):254–260.
[21] Wonnacott A., Meran S., Amphlett B., Talabani B., Phillips A. Epidemiology And Outcomes In Community-Acquired Versus Hospital-Acquired Aki. Clin J Am Soc Nephrol. 2014;9(6):1007–1014.
[22] Levey A.S. Defining Akd: The Spectrum Of Aki, Akd, And Ckd. Nephron. 2021;146(3):302–305.