



Original Article

CLINICAL PROFILE, ETIOLOGY, AND OUTCOMES OF ACUTE KIDNEY INJURY IN PATIENTS WITH ACUTE FEBRILE ILLNESS: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

Dr Shubham Sharma¹, Dr Neha Sharma², Dr Prashant Sharma³, Dr Diksha Taneja⁴, Dr Jatin Prajapati⁵, Dr Nandini Verma⁶

¹ Medical Officer (MD General Medicine), Government of Rajasthan, Ramsar, Ajmer, Rajasthan, India.

² Senior Resident, Department of General Medicine, J.L.N. Medical College, Ajmer, Rajasthan, India.

³ Post Graduate Resident, Department of Radiation Oncology, RNT Medical College, Udaipur, Rajasthan, India.

⁴ Post Graduate Resident, Department of Obstetrics and Gynaecology, RNT Medical College, Udaipur, Rajasthan, India.

⁵ Senior Resident, Department of Community Medicine, World College of Medical Sciences & Research, Jhajjar, Haryana, India.

⁶ Post Graduate Student, Department of Obstetrics and Gynaecology, Maulana Azad Medical College, New Delhi, Delhi, India.

 OPEN ACCESS

Corresponding Author:

Dr Neha Sharma

Senior Resident, Department of General Medicine, J.L.N. Medical College, Ajmer, Rajasthan, India.

Received: 21-03-2026

Accepted: 27-04-2026

Available online: 11-05-2026

Copyright © International Journal of Medical and Pharmaceutical Research

ABSTRACT

Background: Acute kidney injury (AKI) is a common and serious complication of acute febrile illness (AFI), particularly in tropical and resource-limited settings. Infectious diseases such as dengue, malaria, and scrub typhus frequently contribute to the development of AKI and are associated with increased morbidity and mortality.

Objectives: To evaluate the clinical profile, etiological spectrum, and outcomes of AKI in patients presenting with acute febrile illness.

Methods: This hospital-based cross-sectional analytical study was conducted over a period of 12 months at a tertiary care center in Udaipur, Rajasthan. A total of 100 adult patients (≥ 18 years) presenting with acute febrile illness of less than 7 days duration were included and evaluated for the development of AKI. Detailed clinical history, physical examination, and laboratory investigations including complete blood count, renal and liver function tests, and serological tests for tropical infections were performed. AKI was diagnosed based on standard clinical and laboratory criteria and classified according to the RIFLE criteria. Patients were followed during hospitalization to assess outcomes including recovery, need for hemodialysis, and mortality. Statistical analysis was performed using SPSS version 25.0, with $p < 0.05$ considered statistically significant.

Results: Among the study population, AKI was observed in a substantial proportion of patients with AFI. Dengue was the most common etiological factor, followed by malaria and scrub typhus. Platelet counts showed a significant increasing trend during hospitalization, while serum creatinine levels initially increased and subsequently decreased, indicating recovery in most patients. Patients with adverse outcomes had significantly higher total leukocyte counts, liver enzyme levels, blood urea, and serum creatinine levels, along with lower platelet counts. A subset of patients required hemodialysis, and mortality was observed predominantly among patients with dengue infection.

Conclusion: AKI is a frequent and clinically important complication of acute febrile illness, with outcomes closely related to disease severity and laboratory parameters. Early diagnosis, regular monitoring of renal function, and timely intervention are essential to improve patient outcomes and reduce mortality.

Keywords: Acute kidney injury; Acute febrile illness; RIFLE criteria; Dengue; Tropical infections; Hemodialysis; Mortality.

INTRODUCTION

Acute kidney injury (AKI) is a sudden decline in renal function characterized by an acute increase in nitrogenous waste products such as blood urea nitrogen and serum creatinine, occurring over hours to days. It reflects a rapid deterioration in the kidney's ability to maintain fluid, electrolyte, and acid–base balance, often accompanied by reduced urine output. In 2002, the Acute Dialysis Quality Initiative (ADQI) introduced the RIFLE criteria to standardize the definition and classification of AKI, categorizing it into Risk, Injury, and Failure, along with outcome parameters such as Loss and End-stage renal disease [1]. Subsequently, the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines further refined the diagnostic criteria, emphasizing changes in serum creatinine and urine output as key indicators [2].

Acute febrile illness (AFI) is a common clinical presentation in tropical and subtropical regions, defined as a sudden onset of fever exceeding 38°C lasting for less than 14 days without an identifiable source at presentation. In countries like India, AFI constitutes a significant proportion of hospital admissions, with common etiologies including dengue fever, malaria, scrub typhus, leptospirosis, and enteric fever. These infections often occur in outbreaks and vary geographically, posing diagnostic and therapeutic challenges, especially in resource-limited settings [3-5].

AKI is increasingly recognized as a frequent and serious complication of AFI, particularly among hospitalized and critically ill patients. The incidence of AKI in AFI varies depending on the underlying etiology and severity of illness. Studies have reported AKI in up to 26% of patients with febrile illness, often associated with multi-organ dysfunction and increased mortality [3]. In malaria, AKI occurs in approximately 13–17.8% of cases, while in leptospirosis, it may be seen in 16–40% of severe infections [6,7]. Similarly, dengue fever, which is hyperendemic in Southeast Asia, can lead to AKI primarily due to acute tubular necrosis secondary to hypotension and shock.

The pathophysiology of AKI in tropical acute febrile illness is complex and multifactorial. Proposed mechanisms include direct renal invasion by infectious agents, immune-mediated injury, hemolysis, rhabdomyolysis, and intravascular coagulation. Additionally, systemic factors such as hypovolemia, hypotension, and sepsis contribute to renal hypoperfusion and ischemic injury. In diseases such as malaria, cytoadherence of parasitized erythrocytes to the vascular endothelium leads to microvascular obstruction, while in leptospirosis and scrub typhus, direct tubular injury and interstitial inflammation are prominent features [8,9]. Furthermore, the use of certain antibiotics and nephrotoxic drugs in the management of infections may exacerbate renal injury.

Globally, AKI remains a major contributor to morbidity and mortality, even in developed healthcare settings [10]. However, the burden is disproportionately higher in developing countries due to delayed diagnosis, limited access to healthcare, inadequate sanitation, and lack of awareness. In tropical regions, AKI is often underreported and poorly characterized due to variability in diagnostic criteria and limited availability of laboratory facilities [10]. Early identification and timely management are crucial, as AKI is potentially reversible if treated promptly.

The clinical profile of AKI in AFI is influenced by multiple factors, including the type of infection, severity of illness, presence of comorbidities, and access to healthcare. Patients may present with a wide spectrum of manifestations ranging from mild renal dysfunction to severe multi-organ failure. In addition to renal involvement, complications such as hepatic dysfunction, pulmonary involvement, and central nervous system manifestations may coexist, further worsening prognosis [11]. Importantly, AKI in the setting of AFI has also been associated with long-term sequelae, including progression to chronic kidney disease in some patients [12].

Despite the significant burden and clinical implications of AKI in acute febrile illness, there is a relative paucity of data on its clinical profile, laboratory characteristics, and outcomes, particularly in resource-limited settings. Most available studies focus on individual etiologies rather than providing a comprehensive evaluation across different causes of AFI. Understanding the patterns of presentation, progression, and outcomes of AKI in AFI is essential for early risk stratification and appropriate management.

Therefore, the present study was undertaken to assess the clinical profile and outcomes of acute kidney injury in patients presenting with acute febrile illness in a tertiary care hospital setting.

MATERIALS AND METHODS

Study Design and Setting: This was a hospital-based cross-sectional analytical study conducted in the Department of General Medicine, including medical wards and intensive care units (ICUs), at RNT Medical College and its associated hospitals in Udaipur, Rajasthan, India.

Study Duration: The study was carried out over a period of 12 months after obtaining approval from the Institutional Ethics Committee.

Study Population: The study included adult patients aged ≥ 18 years presenting with acute febrile illness (AFI) of less than 7 days duration. These patients were evaluated for the development of acute kidney injury (AKI) during hospitalization.

Inclusion Criteria

- Patients aged ≥ 18 years
- History of fever of less than 7 days duration
- Patients willing to participate and provide informed consent

Exclusion Criteria

- Fever duration more than 7 days
- Known chronic kidney disease
- History of organ transplantation
- Presence of chronic illnesses such as tuberculosis, connective tissue disorders, malignancy, or immunocompromised states
- Post-traumatic or post-surgical cases
- History of nephrotoxic drug intake

Sample Size Calculation: The sample size was calculated using the formula for estimating a proportion in a cross-sectional study:

$$n = \frac{Z^2 \times p \times q}{d^2}$$

where p was taken as 40% based on previous studies reporting the proportion of acute kidney injury among patients with acute febrile illness, $q = 1 - p$, $Z = 1.96$ at 95% confidence interval, and $d = 10\%$ allowable error. The calculated sample size was 93.58, which was rounded off to 100 participants.

Study Procedure: After obtaining informed consent, all eligible participants were evaluated at the time of admission. A detailed history including demographic profile, duration of fever, and associated clinical symptoms was recorded. A thorough general physical examination and systemic examination were performed. Laboratory investigations included complete blood count (CBC), liver function tests (LFT), renal function tests (RFT), serum electrolytes, arterial blood gas (ABG), and blood culture where indicated. Fever profile and serological tests for common tropical infections such as dengue, malaria, scrub typhus, leptospirosis, and enteric fever were performed. Urine analysis including routine microscopy and urine culture was carried out. Imaging investigations such as chest X-ray, electrocardiography (ECG), and ultrasonography (USG) abdomen were performed as clinically indicated.

Definition and Classification of AKI: Acute kidney injury was diagnosed based on clinical and laboratory parameters and classified using the RIFLE criteria into Risk, Injury, and Failure categories based on changes in serum creatinine and urine output. Outcomes were categorized as Loss and End-stage renal disease.

Outcome Measures

The primary outcomes assessed were:

- Clinical profile of patients with AKI in AFI
- Etiological distribution of AFI
- Requirement of hemodialysis
- Patient outcomes (recovery or death)

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean \pm standard deviation, and categorical variables as frequency and percentage. Comparisons between categorical variables were performed using the Chi-square test or Fisher's exact test, as appropriate. Changes in laboratory parameters over time (Day 1, Day 3, and Day 5) were analyzed using repeated measures ANOVA. A p -value of <0.05 was considered statistically significant. Normality of data was assessed before applying parametric tests.

Ethical Considerations: Ethical approval was obtained from the Institutional Ethics Committee prior to the commencement of the study. Written informed consent was obtained from all participants. Confidentiality and anonymity of patient data were strictly maintained throughout the study.

RESULTS

A total of 100 patients with acute febrile illness were included in the study and were evaluated for the presence of acute kidney injury. The majority of patients belonged to the younger age group, with a predominance of males. Most patients had a duration of acute kidney injury between 4 to 8 days (**Table 1**).

Table 1: Socio-demographic and clinical characteristics of study participants (n = 100)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	18–30	58	58.0
	31–40	20	20.0
	41–50	22	22.0
Gender	Male	82	82.0
	Female	18	18.0
Duration of AKI (days)	4–8	76	76.0
	9–12	24	24.0

Dengue was the most common etiology of acute febrile illness in the study population, followed by malaria, scrub typhus, and undiagnosed cases. Dialysis was required in a proportion of patients across different etiologies. Mortality was observed predominantly among patients with dengue, while all patients in the other etiological groups recovered (**Table 2**).

Table 2: Etiology, dialysis requirement, and outcomes among patients with AKI in acute febrile illness

Disease	Total (n=100) n (%)	Dialysis required n (%)	Death n (%)	Improved n (%)
Dengue	56 (56.0)	6 (10.7)	4 (7.1)	52 (92.9)
Malaria	14 (14.0)	2 (14.3)	0 (0.0)	14 (100)
Scrub typhus	12 (12.0)	6 (50.0)	0 (0.0)	12 (100)
Undiagnosed	18 (18.0)	0 (0.0)	0 (0.0)	18 (100)

Percentages for dialysis and outcomes are calculated within each disease category. The requirement of dialysis and mortality varied across etiological groups, indicating differences in disease severity and clinical outcomes.

Hematological parameters were assessed at different time intervals during hospitalization. Platelet counts showed an increasing trend from Day 1 to Day 5 and this change was statistically significant. Total leukocyte count demonstrated a decreasing trend over the same period; however, this change was not statistically significant (**Table 3**).

Table 3: Hematological parameters over time among study participants

Parameter	Day 1 (Mean ± SD)	Day 3 (Mean ± SD)	Day 5 (Mean ± SD)	p-value*
Platelet count (cells/mm ³)	48,857 ± 21,500	77,568 ± 28,200	105,615 ± 34,800	<0.001
Total leukocyte count (cells/mm ³)	11,294 ± 3,250	10,107 ± 2,980	8,672 ± 2,540	0.078

*Repeated measures ANOVA applied.

Biochemical and renal parameters were also evaluated over time. Serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) levels decreased from admission to Day 5 and at discharge. Blood urea levels increased during hospitalization and decreased at discharge. Serum creatinine levels increased initially and subsequently decreased at discharge. The change in serum creatinine over time was statistically significant, whereas changes in other parameters were not statistically significant (**Table 4**).

Table 4: Biochemical and renal parameter trends during hospitalization

Parameter	Day 1 (Mean ± SD)	Day 3 (Mean ± SD)	Day 5 (Mean ± SD)	Discharge (Mean ± SD)	p-value*
SGOT (U/L)	265.1 ± 110.4	228.6 ± 95.2	186.5 ± 82.6	142.3 ± 60.8	0.058
SGPT (U/L)	171.8 ± 72.5	152.4 ± 64.1	139.0 ± 58.3	118.6 ± 49.7	0.072
Blood urea (mg/dL)	26.3 ± 9.6	33.8 ± 11.2	40.6 ± 13.4	28.2 ± 10.1	0.067
Serum creatinine (mg/dL)	0.94 ± 0.32	1.08 ± 0.41	1.13 ± 0.46	0.71 ± 0.28	0.021

*Repeated measures ANOVA applied.

On comparison between outcome groups, patients who died had higher total leukocyte counts, higher liver enzyme levels, higher blood urea, and higher serum creatinine levels, along with lower platelet counts, compared to those who improved. These differences were statistically significant (**Table 5**).

Table 5: Comparison of laboratory parameters between outcome groups

Parameter	Death (n=4) Mean ± SD	Improved (n=96) Mean ± SD	p-value**
Total leukocyte count (cells/mm ³)	14,850 ± 3,200	10,920 ± 2,850	0.018
Platelet count (cells/mm ³)	32,400 ± 12,500	82,600 ± 29,400	0.011
SGOT (U/L)	398.2 ± 120.6	248.3 ± 98.4	0.022
SGPT (U/L)	265.5 ± 88.2	158.4 ± 64.7	0.027
Blood urea (mg/dL)	52.6 ± 14.2	31.4 ± 10.8	0.015
Serum creatinine (mg/dL)	1.84 ± 0.52	1.02 ± 0.36	0.009

**Independent t-test / Mann–Whitney U test applied as appropriate.

Dialysis requirement was observed in patients with varying etiologies and was more frequent in certain disease categories as shown in **Table 2**.

DISCUSSION

Acute kidney injury (AKI) continues to be a major concern in tropical countries, where infectious diseases constitute a significant proportion of its etiology. The burden of AKI in acute febrile illness (AFI) remains substantial, requiring prompt recognition and integrated management of both the underlying infection and renal dysfunction. AKI associated with AFI is a serious clinical condition and an important contributor to mortality among hospitalized patients [13]. In resource-limited settings, delayed presentation and empirical treatment further complicate the clinical course of such patients.

Acute febrile illness is a common clinical presentation in tropical regions and forms a significant proportion of hospital admissions. Many patients initially receive empirical treatment at peripheral centers and are later referred to tertiary care hospitals due to inadequate response. AFI has been identified as one of the most common causes of AKI in hospitalized patients, with etiologies including dengue, malaria, scrub typhus, leptospirosis, and undifferentiated febrile illness [13].

In the present study, the mean age of patients was 29.98 years, with the majority belonging to the 18–30 years age group. This finding is consistent with observations from tropical countries, where AKI tends to affect a younger population compared to developed regions. Similar findings were reported by Niveditha N V et al [14], who observed a mean age of 36.5 years, and by Mathew M K et al [15], who reported a mean age of 49.6 years. In contrast, Deshpande N S et al [16] reported a higher mean age of 51.7 years, with a predominance of elderly patients. Studies from developed countries indicate that AKI is more common in older age groups (60–70 years), highlighting geographic and demographic variations [17].

A significant male predominance was observed in the present study. This finding is consistent with previous studies by Niveditha N V et al [14], Mathew M K et al [15], and Gopal Basu et al [18], which also reported higher male involvement. Similar trends have been documented by Deshpande N S et al [16] and other studies [19-21]. The higher prevalence in males may be attributed to increased exposure to environmental and occupational risk factors, as well as differences in healthcare-seeking behavior [22].

Dengue was identified as the most common etiology of AFI-associated AKI in the present study, followed by malaria and scrub typhus. This pattern is comparable to findings by Niveditha N V et al [14] and Cherian J et al [23], who also reported dengue as a leading cause. However, variations exist across regions, as demonstrated by J.J Nair et al [6] and Gopal Basu et al [18], where malaria and scrub typhus were more predominant. These differences reflect geographical variation in disease prevalence and seasonal patterns.

Hematological parameters in the present study showed trends consistent with infectious etiologies of AFI. Previous studies have also demonstrated significant alterations in platelet count and leukocyte count in patients with AKI. Aggarwal H K et al [24] reported significant differences in platelet counts between groups, while Badge R P et al [25] observed elevated leukocyte counts and altered hematological parameters in AKI patients. Thrombocytopenia has been widely reported in tropical infections and is considered a common laboratory abnormality [26].

Biochemical parameters in the present study demonstrated changes consistent with renal and hepatic involvement. Elevated urea and creatinine levels have been consistently reported in patients with AKI. Studies by Aggarwal H K et al [24] and Badge R P et al [25] also demonstrated significantly higher urea and creatinine levels in AKI patients compared to non-

AKI groups. Elevated liver enzymes have similarly been reported in association with tropical infections and multi-organ involvement.

The requirement of dialysis in the present study varied across different etiologies, with a significant proportion of patients requiring renal replacement therapy. Comparable findings have been reported by Badge R P et al [25] and Omar F D et al [27]. Studies have shown that the need for dialysis ranges widely depending on disease severity, from 7.9% to 44.8% in patients with tropical AFI-associated AKI [28-29]. In malaria-associated AKI, higher dialysis requirements have been reported [30], whereas lower rates have been observed in dengue [31].

Mortality in the present study was low and was observed only in dengue cases. Similar findings have been reported in previous studies, where dengue-associated AKI was associated with increased mortality [32]. However, mortality rates vary widely across studies, ranging from 10% to over 60% depending on severity and healthcare access [30,33-35]. Increased mortality has been associated with higher stages of AKI and presence of multi-organ dysfunction [18,36].

Overall, the findings of the present study are consistent with existing literature and highlight the importance of early diagnosis and timely management of AKI in patients with acute febrile illness.

CONCLUSION

Acute kidney injury (AKI) is a significant and potentially life-threatening complication of acute febrile illness (AFI) in tropical settings. The present study highlights that AKI commonly occurs in young adults and is frequently associated with infectious etiologies such as dengue, malaria, and scrub typhus. The severity of AKI, as reflected by clinical and laboratory parameters, has a direct impact on patient outcomes, including the need for renal replacement therapy and risk of mortality. Patients with higher leukocyte counts, elevated liver enzymes, increased blood urea and serum creatinine, and lower platelet counts were found to have poorer outcomes. Early recognition of AKI and close monitoring of renal function in patients with AFI are crucial for timely intervention. Strengthening diagnostic facilities and implementing prompt management strategies can significantly reduce morbidity and mortality associated with AKI in AFI.

DECLARATIONS

Ethical Approval and Consent to Participate: The study was approved by the Institutional Ethics Committee of RNT Medical College, Udaipur. Written informed consent was obtained from all participants prior to inclusion in the study.

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Funding: No external funding was received for this study.

Conflict of Interest: The authors declare that they have no conflict of interest.

Acknowledgements: The authors acknowledge the support of the Department of General Medicine and all study participants for their cooperation.

REFERENCES

1. Kellum JA, Mehta RL, Angus DC, Palevsky P, Ronco C. The first international consensus conference on continuous renal replacement therapy. *Kidney Int.* 2002;62(5):1855-63.
2. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Vol. 120, *Nephron - Clinical Practice.* 2012;179-84.
3. Andrews M.A, Ittyachen A.M. Aetiology of acute febrile illness: A multicentre study from the province of Kerala in southern India. *Trop.*2018;48: 322-325.
4. Luvira V, Silachamroon U, Piyaphanee W, Lawpoolsri S. Etiologies of Acute Undifferentiated Febrile Illness in Bangkok, Thailand. *Am. J. Trop. Med. Hyg.* 2019;100:622-629.
5. Nair J.J, Bhat A, Prabhu M.V. A Clinical Study of Acute Kidney Injury in Tropical Acute Febrile Illness. *J. Clin. Diagn. Res.* 2016;10: OC01-OC05.
6. Mehta KS, Halankar AR, Makwana PD, Torane PP, Satija PS, Shah VB. Severe acute renal failure in malaria. *J Postgrad Med.* 2001;47(1):24.
7. Daher ED, Abreu KL, Junior GB. Leptospirosis associated acute kidney injury. *Brazilian J Nephrol* 2010;32(4):408-15.
8. Attur R.P, Kuppasamy S, Bairy M, Nagaraju S.P et al. Acute kidney injury in scrub typhus. *Clin. Exp. Nephrol.* 2013;17:725-729.
9. Watanabe T. Renal complications of seasonal and pandemic influenza A virus infections. *Eur. J. Pediatr.* 2013;172:15-22.
10. Jeyachandran Dhanapriya, T.D. Ramanathan Sakthirajan, Natarajan Gopalakrishnan. Acute Kidney Injury in Tropical Countries. *EMJ* 2017;5: 66-74.
11. Mehta K, Pajai A, Bhurke S, Shirkande A, Bhadade R, D'Souza R. Acute Kidney Injury of Infectious Etiology in Monsoon Season: A Prospective Study Using Acute Kidney Injury Network Criteria. *Indian J Nephrol.* 2018;28(2):143-52.

12. Kumar V, Kohli HS. Renal involvement in tropical infections. In: Bhalla A, eds. Association of Physicians of India Indian College of Physicians Update on Tropical Fever. Maharashtra; 2015; 50-9.
13. Bhattacharjee K, Roy S, Balamurali P, Paul N. The Revelations of Acute Kidney Injury in Cases of Acute Febrile Illness – A Hospital Based Observational Study from North Eastern India. *Adv J Uro Nephro*, 2020;2(1):16-19.
14. Niveditha N.V, Kumar P, Sanjay H K. Clinical Profile of Acute Kidney Injury in Acute Febrile Illness with Thrombocytopenia. *Int. J. Adv. Res*(2023); 11(02): 100-104.
15. Mathew M, Radha T R. Etiological Factors and Clinical Profile of Acute Kidney Injury in Medical Intensive Care Unit. *Journal of Current Medical Research and Opinion CMRO* 2019; 02 (11):350–360.
16. Deshpande NS, Harkut J. Clinical profile and etiologic spectrum of patients with acute kidney injury at a tertiary care hospital. *MedPulse International Journal of Medicine*. September 2020; 15(3): 69-73.
17. Wijewickrama E, Ratnayake G, Wikramaratne C, Sheriff R, Rajapakse S. Incidences and clinical outcomes of acute kidney injury in ICU: a prospective observational study in Sri Lanka. *BMC Research Notes*. 2014;7(1):305–305.
18. Basu G, Chrispal A, Boorugu H, Gopinath KG, Chandy S, Prakash JA, et al. Acute kidney injury in tropical acute febrile illness in a tertiary care centre RIFLE criteria validation. *Nephrol Dialysis Transplant*. 2011;26(2):524-31.
19. Patel U, Pasari A, Balwani M, Bhawane A, Tolani P, Acharya S. Clinical profile of acute kidney injury in a tertiary care center in the Tropical Region. *J Integr Nephrol Androl*. 2018;5(4):130.
20. Rajesh K, Rajak M, Seshagirirao Y. Clinical manifestations of Acute kidney injury : A Prospective Observational Study. 2017;16(8):22–8.
21. Eswarappa M, Gireesh MS, Ravi V, Kumar D, Dev G. Spectrum of acute kidney injury in critically ill patients: A single center study from South India. *Indian J Nephrol*. 2014;24(5):280–5.
22. Cerdá J, Bagga A, Kher V, Chakravarthi RM. The contrasting characteristics of acute kidney injury in developed and developing countries. *Nature Rev Nephrol*. 2008;4(3):138.
23. Cherian J, Deodhar D. A study of incidence and outcome of acute kidney injury in common undifferentiated febrile illnesses. *Int J Res Med Sci* 2020;8:497-502.
24. Aggarwal HK, Jain D, Kundu M, Bishnoi A. Evaluation of Renal Functions in Tropical Acute Febrile Illness. *JACM* 2020; 21(1-2): 20-24.
25. Badge RP, Babu VS, Rathore V. Spectrum of acute kidney injury in patients of tropical acute febrile illness in a tertiary hospital. *Int J Res Med Sci* 2023;11:2621-5.
26. Anghan H, Sethi P, Soneja M, Mahajan S, Wig N. Clinical and laboratory features associated with acute kidney injury in severe malaria. *Indian J Crit Care Med* 2018;22:71822.
27. Omar F.D, Phumratanaprapin W, Silachamroon U, Hanboonkunupakarn B et al. Clinical Characteristics of Acute Kidney Injury Associated with Tropical Acute Febrile Illness. *Trop. Med. Infect. Dis*. 2023; 8:147.
28. Vikrant S, Gupta D, Singh M. Epidemiology and outcome of acute kidney injury from a tertiary care hospital in India. *Saudi J. Kidney Dis. Transpl*. 2018;29: 956–966.
29. Barber B.E, Rajahram G.S, Grigg M.J, William,T, Anstey N.M. World Malaria Report: Time to acknowledge *Plasmodium knowlesi* malaria. *Malar. J*. 2017;16:135.
30. Wilairatana P, Westerlund EK, Aursudkij B, Vannaphan S, Krudsood S, Viriyavejakul P, et al. Treatment of malarial acute renal failure by hemodialysis. *Am J Tropical Med Hygiene*. 1999;60(2):233-7.
31. Mei-Chuan Kuo , Lu PL, Chang JM, Lin MY, Tsai JJ, Chen YH, et al. Impact of renal failure on the outcome of dengue viral infection. *Clin J Am Soci Nephrol*. 2008;3(5):1350-6.
32. Lizarraga KJ, Nayer A. Dengue-associated kidney disease. *J Nephropathol*. 2014;3(2):57-62.
33. Kute VB, Shah PR, Munjappa BC, Gumber MR, Patel HV, Jain SH, et al. Outcome and prognostic factors of malaria-associated acute kidney injury requiring hemodialysis: a single center experience. *Ind J Nephrol*. 2012;22(1):33.
34. Khalil MA, Sarwar S, Chaudry MA, Maqbool B, Khalil Z, Tan J, et al. Acute kidney injury in dengue virus infection. *Nephrology Dialysis Transplantation Plus*. 2012;5(5):390-4.
35. Daher E.F, Vieira A.P, Jacinto C.N, Lima R.S et al. Differences among children, adolescents and adults with severe leptospirosis: A comparative analysis. *Indian J. Nephrol*. 2014;24:166–170.
36. Thanachartwet V, Desakorn V, Sahassananda D, Win KKYK, Supaporn T. Acute renal failure in patients with severe falciparum malaria: using the who 2006 and rifle criteria. *Int J Nephrol*. 2013;1-6.