

Original Article

Chronic renal failure versus acute kidney injury: Clinico-etiological profile and hematological parameters of patients undergoing hemodialysis in central India

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Abstract

Background: Chronic kidney disease (CKD) and acute kidney injury (AKI) are serious public health burden. Patients with advanced stage CKD and severe AKI require hemodialysis (HD). However, their clinico-etiological profile has not been compared yet. Thus, we compared the clinico-etiological and hematological profile of patients with CKD and AKI undergoing HD.

Materials and Methods: This prospective, observational, cross-sectional study involved 425 adult patients with CKD (n=328) and AKI (n=97) that underwent HD, between January 2020 and June 2021, in a tertiary care hospital. Data related to clinico-demographic characteristics, and laboratory investigations were recorded.

Results: Hypertension (75.9%) and sepsis (49.48%) were predominant etiological factors of CKD and AKI, respectively. The patients with CKD had significantly greater mean age (p=0.005) and BMI (p<0.0001). Significantly greater proportion of the patients with AKI had anorexia (p=0.003), breathlessness (p=0.018), nausea and vomiting (p=0.019), and icterus (p=0.002). Significantly greater proportion of the patients with CKD had impaired cognition (p=0.043), swollen feet (p=0.008), and edema feet (p=0.012). Significantly greater proportion of the patients with AKI and CKD had hyperkalemia (p=0.034) as well as 3+ proteinuria (p<0.0001); and 2+ proteinuria (p<0.0001) as well as moderate anemia (p=0.045), respectively.

Conclusion: Hypertension and sepsis are predominantly implicated in CKD and AKI, respectively. CKD is significantly associated with advancing age and higher BMI. Though AKI is significantly associated with hyperkalemia and high-grade proteinuria, CKD is significantly associated with moderate-grade proteinuria and moderate anemia.

Key Words: Acute kidney injury, Chronic kidney disease, Hypertension, Kidney dysfunction, Sepsis

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I. Introduction

Chronic kidney disease (CKD), a global public health issue, is a result of constant renal parenchyma injury resulting in progressive worsening of kidney function that may steadily evolve to end-stage renal disease.^[1] Over the last three decades (1990-2019), number of cases (147.6 million to 697.3 million) and deaths (0.6 million to 1.4 million) attributed with CKD have increased more than two-folds.^[2] Moreover, between 1990 and 2017, availability of renal replacement therapy (RRT) has increased tremendously. Globally, in 2017, around 3.9 million patients with kidney failure required RRT. Hemodialysis (HD) is the most frequent form of RRT, amounting for about 69% of all RRT and 89% of all dialysis.^[3]

Contrary to CKD, acute kidney injury (AKI) develops over a few hours or days, and is marked by rapid decline in kidney function with or without kidney damage.^[4] It affects around 13 million individuals and leads to 1.7 million deaths per year worldwide. Even a mild AKI is linked to 50% greater chances of death.^[5] Currently, proportion of patients with AKI requiring HD is increasing.^[4] However, a considerable proportion of these patients recover spontaneously without HD. In severe cases, though HD is life-saving, it is associated with various complications, including infection, bleeding, or hypotension, and a substantial economic burden.^[6]

With rising burden of CKD and AKI, the Government of India has initiated free dialysis services for marginalized sections of the society.^[7] Though clinico-etiological profile of CKD and AKI are well described in literature,^[8-11] they are yet to be compared. Thus, we compared the clinico-etiological and hematological profile of patients with CKD and AKI undergoing HD.

II. Materials and Methods

This prospective, observational, cross-sectional study involved patients with CKD and AKI that underwent HD, between January 2020 and June 2021, in a tertiary care hospital. The study protocol was approved by the Institutional Ethics Committee and patients were enrolled after obtaining written informed consent.

The study included adult patients aged 18 to 80 years, of either sex, and required HD for first time due to any reason, including CKD and AKI. While, pediatric patients, patients on maintenance HD, and those that required peritoneal dialysis were excluded from the study.

A total of 425 patients were enrolled and divided into two groups: CKD (n=328) and AKI (n=97). Subsequently, data related to demographic (age, gender, body mass index (BMI)), clinical characteristic (presenting symptoms and signs, stage of CKD, comorbidities, and etiology of CKD and AKI), vital parameters (systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse rate (PR), and respiratory rate (RR)), and laboratory investigations (hemoglobin (Hb), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), serum urea, serum creatinine (SCr), serum sodium, serum potassium, and proteinuria) were recorded.

CKD and AKI were defined by Kidney Diseases: Improving global Outcomes (KDIGO) guidelines.^[12] The term CKD suggested a glomerular filtration rate (GFR) less than 60 mL/min per 1.73 m² for more than 3 months. While, the term AKI was defined as an increase in SCr by 50% within 7 days, or by 0.3 mg/dL within 2 days, or presence of oliguria (urine

volume less than 0.5 mL/kg/hr) for more than 6 hrs. The consensus statement for diagnosis of obesity among Asian Indians was used to categorize the patients according to BMI.^[13] While, severity of anemia was assessed as per the classification recommended by the Indian Council of Medical Research.^[14]

Statistical analyses

The data was analyzed with SPSS (IBM, Armonk, NY, USA) version 23.0 for windows. The categorical and continuous variables are represented as frequency (percentage) and mean (standard deviation, SD), respectively. Between group comparison of categorical and continuous variables was performed with Chi-square test and independent sample t-test, respectively. A probability value of < 0.05 was considered as statistically significant.

III. Results

In both the groups, majority of the patients were male (CKD vs AKI: 73.5% vs 69.1%). However, there was no significant difference between the groups with respect to gender ($p = 0.394$). The mean age ($p = 0.005$) and BMI ($p < 0.0001$) were significantly greater in the CKD group relative to the AKI group. Additionally, significantly lesser proportion of the patients in the CKD group belonged to 21 – 40 years age group (24.7% vs 38.1%; $p = 0.014$). Similarly, significantly lesser proportion of the patients in the CKD group had below normal ($< 18.5 \text{ kg/m}^2$; 7.3% vs 20.6%; $p < 0.0001$) and normal BMI (18.5 – 22.9 kg/m^2 ; 46.9% vs 65.9%; $p = 0.001$). While, significantly greater proportion of the patients in the CKD group were overweight (23 – 24.9 kg/m^2 ; 24.4% vs 9.3%; $p = 0.001$) and obese ($\geq 25 \text{ kg/m}^2$; 21.3% vs 4.1%; $p < 0.0001$) (Table 1).

Table 1. Comparison of demographic characteristics: CKD (n=328) and AKI (n=97)

Characteristics	CKD n (%)	AKI n (%)	p
Age (years)			
Mean \pm SD	50.04 \pm 12.18	45.91 \pm 13.60	0.005
Range	19 – 80	21 – 74	
≤ 20	4 (1.2)	0 (0)	NA
21 – 40	81 (24.7)	37 (38.1)	0.014
41 – 60	182 (55.5)	45 (46.4)	0.115
> 60	61 (18.6)	15 (15.5)	0.479
Gender			
Male	241 (73.5)	67 (69.1)	0.394
Female	87 (26.5)	30 (30.9)	
Body mass index (kg/m^2)			
Mean \pm SD	23.37 \pm 4.57	20.39 \pm 2.43	< 0.0001
Range	14.04 – 42.86	14.52 – 26.67	
< 18.5	24 (7.3)	20 (20.6)	< 0.0001
18.5 – 22.9	154 (46.9)	64 (65.9)	0.001
23 – 24.9	80 (24.4)	9 (9.3)	0.001
≥ 25	70 (21.3)	4 (4.1)	< 0.0001

Evaluation of the presenting symptoms suggested that significantly greater proportion of the patients in the AKI group, relative to the CKD group, had anorexia (97.9% vs 87.8%; $p = 0.003$), breathlessness (87.6% vs 76.5%; $p = 0.018$), and nausea and vomiting (41.2% vs 28.7%; $p = 0.019$). While, significantly greater proportion of the patients in the CKD group, relative to the AKI group, had impaired cognition (22.9% vs 13.4%; $p = 0.043$), and swollen feet (76.5% vs 62.9%; $p = 0.008$). Similar evaluation of presenting signs suggested that significantly greater proportion of the patients in the CKD group, relative to the AKI group, had edema feet (65.5% vs 51.5%; $p = 0.012$). While, significantly greater proportion of the patients in the AKI group, relative to the CKD group, had icterus (13.4% vs 4.6%; $p = 0.002$). Additionally, significantly greater proportion of the patients in the CKD group, relative to the AKI group, had coexisting hypertension and diabetes (21.9% vs 8.2%; $p = 0.022$) (Table 2).

Table 2. Comparison of clinical characteristics: CKD (n=328) and AKI (n=97)

Characteristics	CKD n (%)	AKI n (%)	p
Symptoms			
Decreased urine output	320 (97.6)	95 (97.9)	0.830
Easy fatiguability	309 (94.2)	95 (97.9)	0.136
Anorexia	288 (87.8)	95 (97.9)	0.003
Swollen feet	251 (76.5)	61 (62.9)	0.008
Breathlessness	251 (76.5)	85 (87.6)	0.018
Nausea and vomiting	94 (28.7)	40 (41.2)	0.019
Impaired cognition	75 (22.9)	13 (13.4)	0.043
Altered sensorium	43 (13.1)	11 (11.3)	0.646
Signs			
Pallor	314 (95.7)	89 (91.8)	0.120
Edema feet	215 (65.5)	50 (51.5)	0.012
Encephalopathy	30 (9.1)	11 (11.3)	0.520
Icterus	15 (4.6)	13 (13.4)	0.002
Comorbidities			
Hypertension	248 (75.6)	67 (69.1)	0.022
Hypertension and diabetes	72 (21.9)	8 (8.2)	

In CKD group, the most common etiology was hypertension (75.9%) followed by coexisting hypertension and diabetes (21.9%). While, in AKI group, sepsis (49.48%) and gastroenteritis (19.59%) were mostly implicated. Also, the patients predominantly had stage V (68.29%) followed by stage IV CKD (24.39%) (Table 3).

Table 3. Etiologies of CKD (n=328) and AKI (n=97)

Etiologies	n	%
CKD		
Hypertension	249	75.9
Hypertension and diabetes	72	21.9
Obstructive uropathy	2	0.6

Focal segmental glomerulonephritis	2	0.6
Chronic glomerulonephritis	2	0.6
P-ANCA vasculitis	1	0.3
CKD stage		
II	7	2.13
III	17	5.18
IV	80	24.39
V	224	68.29
AKI		
Sepsis	48	49.48
Gastroenteritis	19	19.59
Chronic liver disease	9	9.28
Cardiogenic shock	8	8.25
Snake bite	5	5.15
Unknown	5	5.15
Obstructive uropathy	3	3.09

Comparison of the vital parameters revealed that the mean DBP (97.17 ± 10.01 mmHg vs 92.04 ± 15.46 mmHg; $p = 0.003$), and RR (22.02 ± 3.73 /minute vs 20.02 ± 5.88 /minute; $p = 0.002$) were significantly greater in the CKD group than the AKI group. Further evaluation revealed that significantly greater proportion of the patients in the CKD group, relative to the AKI group, had systolic hypertension (SBP > 120 mmHg; 98.2% vs 85.6%; $p < 0.0001$), diastolic hypertension (DBP > 80 mmHg; 93.6% vs 78.4%; $p < 0.0001$), and tachypnea (RR > 20 /minute; 61.3% vs 25.8%; $p < 0.0001$) (Table 4).

Table 4. Comparison of vital parameters: CKD (n=328) and AKI (n=97)

Characteristics	CKD n (%)	AKI n (%)	p
Systolic blood pressure (mmHg)			
Mean \pm SD	163.97 ± 18.29	162.23 ± 31.73	0.606
Range	110 – 230	90 – 260	
90 – 120	6 (1.8)	14 (14.4)	< 0.0001
> 120	322 (98.2)	83 (85.6)	
Diastolic blood pressure (mmHg)			
Mean \pm SD	97.17 ± 10.01	92.04 ± 15.46	0.003
Range	70 – 140	50 – 140	
< 60	0 (0)	1 (1.0)	NA
60 – 80	21 (6.4)	20 (20.6)	< 0.0001
> 80	307 (93.6)	76 (78.4)	
Pulse rate (/minute)			
Mean \pm SD	90.49 ± 11.42	91.61 ± 11.15	0.398
Range	60 – 128	68 – 126	
60 – 100	277 (84.5)	84 (86.6)	0.604

> 100	51 (15.5)	13 (13.4)	
Respiratory rate (/minute)			
Mean \pm SD	22.02 \pm 3.73	20.02 \pm 5.88	0.002
Range	14 – 44	16 – 60	
12 – 20	127 (38.7)	72 (74.2)	< 0.0001
> 20	201 (61.3)	25 (25.8)	

Comparison of the laboratory parameters suggested that the mean serum potassium levels were significantly higher in the AKI group than the CKD group (5.08 \pm 1.23 mEq/L vs 4.78 \pm 0.95 mEq/L; $p = 0.012$). Significantly greater proportion of the patients in the CKD group had normal serum potassium levels (56.1% vs 43.3%; $p = 0.026$). While, significantly greater proportion of the patients in the AKI group had hyperkalemia (49.5% vs 37.5%; $p = 0.034$). Additionally, significantly greater proportion of the patients in the AKI group, relative to the CKD group, had nil (4.1% vs 0.6%; $p = 0.010$) and 3+ proteinuria (39.2% vs 20.7%; $p < 0.0001$). While, significantly greater proportion of the patients in the CKD group had 2+ proteinuria (31.4% vs 8.2%; $p < 0.0001$). Similarly, significantly greater proportion of the patients in the CKD group had moderate anemia (49.7% vs 38.1%; $p = 0.045$) (Table 5).

Table 5. Comparison of laboratory parameters: CKD (n=328) and AKI (n=97)

Parameters	CKD n (%)	AKI n (%)	p
Hemoglobin (gm/dl)			
Mean \pm SD	7.34 \pm 1.75	7.31 \pm 2.16	0.900
Range	3 – 12.8	3.2 – 13.1	
≥ 11	10 (3.0)	7 (7.2)	0.066
10 – 10.9	16 (4.9)	1 (1.0)	0.089
7 – 9.9	163 (49.7)	37 (38.1)	0.045
4 – 6.9	133 (40.5)	50 (51.5)	0.055
< 4	6 (1.8)	2 (2.1)	0.822
MCV (fL)			
Mean \pm SD	81.94 \pm 8.81	81.21 \pm 9.93	0.490
Range	31.6 – 109.5	60 – 110.8	
< 75	65 (19.8)	21 (21.6)	0.693
75 – 100	260 (79.3)	73 (75.3)	0.399
> 100	3 (0.9)	3 (3.1)	0.110
MCH (pg)			
Mean \pm SD	27.11 \pm 3.24	26.68 \pm 3.52	0.257
Range	13.01 – 36.6	17 – 36.5	
< 25	74 (22.6)	29 (29.9)	0.139
25 – 35	253 (77.1)	66 (68.0)	0.069
> 35	1 (0.3)	2 (2.1)	0.069
MCHC (g/dL)			
Mean \pm SD	32.52 \pm 3.44	32.36 \pm 2.83	0.659

Range	22 – 82.8	11.1 – 38	
< 31	70 (21.3)	14 (14.4)	0.133
31 – 38	255 (77.7)	83 (85.6)	
> 38	3 (0.9)	0 (0)	NA
Serum urea (mg/dL)			
Mean ± SD	149.16 ± 66.04	149.02 ± 63.19	0.986
Range	29 – 371	8.4 – 389	
≤ 40	3 (0.9)	2 (2.1)	0.357
> 40	325 (99.1)	95 (97.9)	
Serum creatinine (mg/dL)			
Mean ± SD	10.71 ± 5.01	9.80 ± 3.68	0.053
Range	3.04 – 34.51	3.1 – 17.94	
≤ 1.2	0 (0)	0 (0)	NA
> 1.2	328 (100)	97 (100)	
Serum sodium (mEq/L)			
Mean ± SD	136.45 ± 6.22	135.63 ± 4.28	0.140
Range	102 – 169	124 – 147	
< 135	112 (34.1)	27 (27.8)	0.244
135 – 145	207 (63.1)	69 (71.1)	
> 145	9 (2.8)	1 (1.0)	
Serum potassium (mEq/L)			
Mean ± SD	4.78 ± 0.95	5.08 ± 1.23	0.012
Range	2.05 – 8.4	2.8 – 9.7	
< 3.5	21 (6.4)	7 (7.2)	0.776
3.5 – 5	184 (56.1)	42 (43.3)	0.026
> 5	123 (37.5)	48 (49.5)	0.034
Proteinuria			
Nil	2 (0.6)	4 (4.1)	0.010
1+	41 (12.5)	15 (15.5)	0.448
2+	103 (31.4)	8 (8.2)	< 0.0001
3+	68 (20.7)	38 (39.2)	< 0.0001
4+	114 (34.8)	32 (32.9)	0.748

IV. Discussion

Over the last few decades, burden of both CKD and AKI has increased significantly. Severe case of AKI and patients with higher stages of CKD require RRT. This resulted in increased demand for HD. Following the global trend, rise in the cases CKD and AKI led Government of India to provide free HD services for people living below poverty line. To the best of our knowledge, this is the first study to compare CKD and AKI regarding clinico-etiological profile and hematological parameters in patients undergoing HD.

The findings of the study suggested that significantly higher proportion of patients with CKD had impaired cognition, and swollen feet due to fluid overload. While, significantly higher proportion of patients with AKI had anorexia, breathlessness, nausea, vomiting, and icterus. While, the groups did not differ regarding decreased urine output, easy fatiguability, altered sensorium, pallor, and encephalopathy. In patients with CKD, cognitive impairment is one of the causes of chronic alterations of mental status, and results from chronic CNS injury. Around 30-60% patients on dialysis have cognitive impairment, and its prevalence as well as progression are indirectly related to the kidney function with a higher risk of cognitive decline in patients with moderate CKD.^[15] Moreover, fluid retention is a complication associated with advanced CKD.^[16] In patients with AKI, the presentation depends on disease severity. Though anorexia, nausea, vomiting, malaise, and altered mental state are common symptoms; symptoms, including seizures and myoclonic jerks, are observed in advanced stage.^[17] The symptoms such as anorexia, nausea, and vomiting are a result of accumulation of various toxins. While breathlessness is due to pulmonary edema resulting from kidney dysfunction.

Hypertension, and coexisting hypertension and diabetes were predominantly implicated in CKD. While, in patients with AKI, sepsis and gastroenteritis were the most common etiological factors. Moreover, among comorbidities, coexisting hypertension and diabetes was present in significantly greater proportion of the patients with CKD. Comorbidities, including hypertension and diabetes, are a risk factor for CKD, and account for 23.26% and 50.62% CKD cases globally, respectively. Coexisting hypertension and diabetes act synergistically leading to CKD. Their coexistence complicates preexisting diseases, including obesity and metabolic syndrome; leads to upregulation of the renin- angiotensin- aldosterone system and sodium transporters; and these patients have higher risk of peripheral arterial resistance. These factors act together to cause renal dysfunction.^[18] Regarding AKI, sepsis- and gastroenteritis-associated hypovolemia is a preventable risk factor.^[19] During sepsis, the inflammatory response leads to downregulation of the tubular epithelial cell function, to limit energy demand and to promote cell survival, thereby reducing kidney function.^[20] Moreover, hypovolemia causes reversible hypoperfusion of the kidney causing transient reduction in GFR in absence of parenchymal injury.^[21]

The patients with CKD had significantly higher age and BMI. Increase in age leads to increase in prevalence of CKD (from 4% at age <40 years to 47% at age \geq 70 years). This group of patients have higher prevalence of comorbidities. This is marked by progression to more severe CKD stages associated with lower estimated GFR and worse outcomes.^[22] Similar to CKD, increasing age is a risk factor for AKI. This is mainly attributed to higher risk of infection owing to reduced immunity, age-related decline in kidney function, greater comorbidities, and polypharmacy.^[17] It is further reported that obesity is associated with higher risk of CKD and AKI. Obesity leads to compensatory hyperperfusion and hyperfiltration to fulfill the raised metabolic demands of increased BMI. Obesity-associated raised hemodynamic and metabolic load on each glomerulus reduces the number of functional nephrons and leads to glomerular hypertrophy and glomerulosclerosis due to raised capillary pressure on the available functional nephrons. Furthermore, activated

inflammatory cytokines and oxidative stress are produced by adipocytes.^[23] Over a period of time, these changes have a detrimental effect on the glomeruli leading to renal dysfunction. In the present study, higher age and BMI in patients with CKD may be an incidental finding.

The patients with CKD had significantly higher DBP and RR. Moreover, significantly greater proportion of the patients with CKD had systolic hypertension, diastolic hypertension, and tachypnea. In younger adults aged 20 – 39 years, isolated systolic hypertension (ISH), isolated diastolic hypertension (IDH), and systolic diastolic hypertension (SDH) are reported to be associated with a higher risk of CKD relative to those with normal BP. The risk of CKD is comparable among patients with ISH and IDH, but lower than the patients with SDH.^[24] In the present study, patients with CKD had higher prevalence of hypertension than those with AKI. This finding may be attributed to significantly higher age of the patients with CKD. Available literature suggests high prevalence of hypertension in patients with AKI (70%), and its prevalence depends on origin, including pre-renal, renal, and post-renal.^[25] In patients with kidney dysfunction, tachypnea is frequently observed. It occurs as a compensatory response to pulmonary edema and stressed cardiovascular system. Moreover, tachypnea helps by blowing away excess carbon dioxide as a buffer mechanism.^[26]

The patients with AKI had significantly higher serum potassium levels. Moreover, significantly greater proportion of the patients with AKI had hyperkalemia as well as nil and 3+ proteinuria. However, significantly greater proportion of the patients with CKD had 2+ proteinuria and moderate anemia. Hyperkalemia is a frequent complication of AKI. In these patients, sudden decline in the GFR is a limiting factor for K⁺ secretion. In patients with oligoanuria, decreased distal transport of salt and water further adds to reduced distal K⁺ secretion. Contrarily, in CKD, there is progressive loss of nephrons leading to decrease in number of collecting ducts to secrete K⁺. As this is a chronic process, it permits compensatory response in the available nephrons, permitting the amount of K⁺ excreted per unit GFR to rise. This explains significantly higher hyperkalemia observed in patients with AKI.^[27] Moreover, AKI is reported to be a risk factor for incident or worsening proteinuria, highlighting a possible relation between AKI and future CKD. In CKD, de novo proteinuria promotes inflammation and oxidative stress resulting in fibrosis and scarring. It is further suggested that higher proteinuria is linked to accelerated deterioration of kidney function and a greater chances of developing end-stage renal disease.^[28] Thus, both CKD and AKI are associated with proteinuria, and significantly higher grade of proteinuria in both CKD (2+) and AKI (3+) may be an incidental finding.

In CKD, anemia is of multifactorial origin, including decline in endogenous erythropoietin (EPO) levels, decrease in bone marrow response to EPO (due to uremic toxins), systemic inflammation resulting from CKD and associated comorbidities, decreased red cell life span, iron deficiency (from blood losses or reduced impaired iron absorption), inefficient utilization of iron stores (due to raised hepcidin levels), or vitamin B12/folic acid deficiency.^[29] Most of the patients with AKI have anemia, associated with oliguria and uremia level. In AKI, EPO level generally rises in the first 48 hrs, and then gradually declines.^[30] It should be highlighted that anemia, reduces oxygen transport and subsequently alters brain metabolism, plays a pivotal role in onset of cognitive decline in patients with CKD.^[15] This hypothesis

justifies significantly higher cognitive impairment in patients with CKD included in the present study.

Though this is a novel study, it had certain limitations. First, due to cross-sectional nature of the study, outcome was not evaluated. Second, baseline SCr levels were not available in all the patients with AKI, thus stages of AKI could not be determined. Third, single center study with limited sample size does not allow us to generalize the findings.

V. Conclusion

To conclude, hypertension and sepsis were predominantly implicated in CKD and AKI, respectively. Significantly higher proportion of patients with CKD and AKI presented with impaired cognition, and swollen feet due to edema; and anorexia, breathlessness, nausea and vomiting, and icterus, respectively. The patients with CKD had significantly greater mean age and BMI. Significantly greater proportion of the patients with AKI and CKD had hyperkalemia as well as nil and 3+ proteinuria; and 2+ proteinuria and moderate anemia.

Funding:

None of the authors received funding for this study.

Competing interest:

There is no competing interest.

Authors contribution:

All authors in our study contributed to the data collection of the patients.

Acknowledgments:

The authors would like to thank Dr. Vikas S. Sharma (MD), CEO, Maverick Medicorum[®] (India), for data analyses and medical writing assistance in the preparation of this article.

References

1. Ke C, Liang J, Liu M, Liu S, Wang C. Burden of chronic kidney disease and its risk-attributable burden in 137 low-and middle-income countries, 1990–2019: results from the global burden of disease study 2019. *BMC Nephrol* 2022;23:17. doi: 10.1186/s12882-021-02597-3
2. Feng X, Hou N, Chen Z, Liu J, Li X, Sun X, et al. Secular trends of epidemiologic patterns of chronic kidney disease over three decades: an updated analysis of the Global Burden of Disease Study 2019. *BMJ Open*. 2023;13(3):e064540. doi: 10.1136/bmjopen-2022-064540.
3. Bello AK, Okpechi IG, Osman MA, Cho Y, Htay H, Jha V, et al. Epidemiology of haemodialysis outcomes. *Nat Rev Nephrol*. 2022;18(6):378-395. doi: 10.1038/s41581-022-00542-7.
4. Pavkov ME, Harding JL, Burrows NR. Trends in Hospitalizations for Acute Kidney Injury — United States, 2000–2014. *MMWR Morb Mortal Wkly Rep* 2018;67:289–293. doi: 10.15585/mmwr.mm6710a2.

5. Abebe A, Kumela K, Belay M, Kebede B, Wobie Y. Mortality and predictors of acute kidney injury in adults: a hospital-based prospective observational study. *Sci Rep*. 2021;11:15672. doi: 10.1038/s41598-021-94946-3.
6. Morzywołek, P., Steen, J., Vansteelandt, S. Decruyenaere J, Sterckx S, Biesen WV. Timing of dialysis in acute kidney injury using routinely collected data and dynamic treatment regimes. *Crit Care* 26, 365 (2022). doi: 10.1186/s13054-022-04252-1.
7. Jha V. Setting up a national dialysis service in India: Change, choice and principles. *Nephrology (Carlton)*. 2016;21(11):913-915. doi: 10.1111/nep.12803.
8. Chetlapalli AK, Prabhu RA, Kolakemar A, Rao IR. Clinical profile and outcomes of acute kidney injury in intensive care units: A prospective single-center study. *Indian J Kidney Dis* 2022;1:8-14. doi: 10.4103/ijkd.ijkd_7_22
9. Bhattacharya PK, Roy A, Jamil M, Barman B, Murti SV, Marak PR. Clinical profile and determinants of short-term outcome of acute kidney injury: A hospital-based prospective study from Northeastern India. *J Lab Physicians* 2019;11:5-10. doi: 10.4103/JLP.JLP_135_18
10. Karan VN, Vishwanath VN. Clinical Profile of Patients with Chronic Kidney Disease. *Acad. J Med*. 2019;2(2):175-78. doi: 10.21276/ajm.2019.2.2.45.
11. Sathyan S, George S, Vijayan P, Jayakumar M. Clinical and epidemiological profile of chronic kidney disease patients in a tertiary care referral centre in South India. *Int J Community Med Public Health* 2016;3:3487-92. doi: 10.18203/2394-6040.ijcmph20164279.
12. Lameire N. Reflections on the KDIGO Definition of Acute Kidney Injury and Its Integration in the Concept of Acute Diseases and Disorders and Chronic Kidney Diseases. *Kidney Dial*. 2022;2:68–79. doi: 10.3390/kidneydial2010008.
13. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al.; Consensus Group. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. *J Assoc Physicians India*. 2009;57:163-70.
14. Indian Council of Medical Research. ICMR evaluation of National Anaemia Prophylaxis Programme. ICMR Task Force Study. New Delhi: ICMR; 1989.
15. Arnold R, Issar T, Krishnan AV, Pussell BA. Neurological complications in chronic kidney disease. *JRSM Cardiovasc Dis*. 2016;5:2048004016677687. doi: 10.1177/2048004016677687.
16. Kalantar-Zadeh K, Regidor DL, Kovesdy CP, Van Wyck D, Bunnapradist S, Horwich TB, et al. Fluid retention is associated with cardiovascular mortality in patients undergoing long-term hemodialysis. *Circulation*. 2009;119(5):671-9. doi: 10.1161/CIRCULATIONAHA.108.807362.
17. Washinger K. Acute Kidney Injury in Adults: An Underdiagnosed Condition. *J Nurse Pract*. 2017;13(10):667-674.e1. doi: 10.1016/j.nurpra.2017.08.005.

18. Wang M, Li J, Li Y, Yao S, Zhao M, Wang C, et al. The effects of hypertension and diabetes on new-onset chronic kidney disease: A prospective cohort study. *J Clin Hypertens (Greenwich)*. 2020;22(1):39-46. doi: 10.1111/jch.13768.
19. Mansfield KE, Douglas IJ, Nitsch D, Thomas SL, Smeeth L, Tomlinson LA. Acute kidney injury and infections in patients taking antihypertensive drugs: a self-controlled case series analysis. *Clin Epidemiol*. 2018;10:187-202. doi: 10.2147/CLEP.S146757.
20. Zarbock A, Gomez H, Kellum JA. Sepsis-induced acute kidney injury revisited: pathophysiology, prevention and future therapies. *Curr Opin Crit Care*. 2014;20(6):588-95. doi: 10.1097/MCC.0000000000000153.
21. Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders HJ. Acute kidney injury. *Nat Rev Dis Primers*. 2021;7(1):52. doi: 10.1038/s41572-021-00284-z.
22. Ravani P, Quinn R, Fiocco M, Liu P, Al-Wahsh H, Lam N, et al. Association of Age With Risk of Kidney Failure in Adults With Stage IV Chronic Kidney Disease in Canada. *JAMA Netw Open*. 2020;3(9):e2017150. doi: 10.1001/jamanetworkopen.2020.17150.
23. Ju S, Lee TW, Yoo JW, Lee SJ, Cho YJ, Jeong YY, et al. Body Mass Index as a Predictor of Acute Kidney Injury in Critically Ill Patients: A Retrospective Single-Center Study. *Tuberc Respir Dis (Seoul)*. 2018;81(4):311-318. doi: 10.4046/trd.2017.0081.
24. Bae EH, Lim SY, Jung JH, Oh TR, Choi HS, Kim CS, et al. Chronic Kidney Disease Risk of Isolated Systolic or Diastolic Hypertension in Young Adults: A Nationwide Sample Based-Cohort Study. *J Am Heart Assoc*. 2021;10(7):e019764. doi: 10.1161/JAHA.120.019764.
25. Dylewska M, Chomicka I, Małyszko J. Hypertension in patients with acute kidney injury. *Wiad Lek*. 2019;72(11 cz 2):2199-2201.
26. Malek M, Hassanshahi J, Fartootzadeh R, Azizi F, Shahidani S. Nephrogenic acute respiratory distress syndrome: A narrative review on pathophysiology and treatment. *Chin J Traumatol*. 2018;21(1):4-10. doi: 10.1016/j.cjtee.2017.07.004.
27. Palmer BF, Clegg DJ. Hyperkalemia across the Continuum of Kidney Function. *Clin J Am Soc Nephrol*. 2018;13(1):155-157. doi: 10.2215/CJN.09340817.
28. Bonde SS, Zaman W, Cuomo R, Malhotra R, Macedo E. Risk of de novo proteinuria following hospitalization with acute kidney injury. *BMC Nephrol*. 2023;24:176. doi: 10.1186/s12882-023-03209-y.
29. Portolés J, Martín L, Broseta JJ, Cases A. Anemia in Chronic Kidney Disease: From Pathophysiology and Current Treatments, to Future Agents. *Front Med (Lausanne)*. 2021;8:642296. doi: 10.3389/fmed.2021.642296.
30. Aoun M, Sleilaty G, Boueri C, Younes E, Gabriel K, Kahwaji RM, et al. Erythropoietin in Acute Kidney Injury (EAKI): a pragmatic randomized clinical trial. *BMC Nephrol*. 2022;23(1):100. doi: 10.1186/s12882-022-02727-5.