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Incidence and Risk Factors of Acute Kidney Injury in Patients Hospitalized with Pneumonia: A Prospective Observational Study

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Abstract

Background: Acute kidney injury (AKI) is frequent in hospitalized patients with critical illness and presents in up to one-quarter of patients with non-severe community-acquired pneumonia (CAP), resulting in increased short and long-term mortality. There is a paucity of literature from resource-limited settings regarding the incidence and risk factors for AKI in patients with CAP. In this study, we looked at the incidence and risk factors for AKI in patients hospitalized with CAP in a resource-limited setting

Methods: This prospective observational study conducted over 1 year period included patients ≥ 18 years of age diagnosed with CAP admitted to a tertiary care center. The differences in baseline characteristics between hospitalized CAP patients with and without AKI; and risk factors for AKI and the need for renal replacement therapy (RRT) were analyzed using Chi-square test, t-test, Mann-Whitney U test, and logistic regression with p-value <0.05 considered statistically significant.

Results: We observed 27.6 % (58/210) of patients had AKI in our study. Patients with AKI had significantly higher baseline comorbidities of chronic kidney disease (p=0.005) and coronary artery disease (p=0.032), and significantly higher uric acid (p=0.002), lower albumin (p=0.005), lower total protein (p=0.015), higher bilirubin (p=0.001), higher LDH (p=0.041), and higher CURB-65 score (p<0.001) in addition to elevated creatinine, BUN (p<0.001) compared to the no-AKI group. The patient group requiring RRT had significantly more males (p=0.019), with significantly higher phosphorus (p=0.038), lower ALT (p=0.022), and expectedly higher creatinine (p<0.001) and blood urea nitrogen (p=0.016). The adjusted logistic regression analysis revealed that patients with higher CURB-65 scores were at increased odds of undergoing RRT (OR 8.74, 95% CI 5.27 to 12.21, p=0.039).

Conclusion: There is a high incidence of AKI in patients hospitalized for CAP in developing countries. Clinicians should be alert for the prevention and early detection of AKI in CAP patients.

Keywords: Kidney Injury, Pneumonia, Dialysis, CURB-65

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Introduction

Acute Kidney Injury (AKI, previously acute renal failure) is a clinical syndrome of rapid onset renal impairment, with a decrease in excretory function and accumulation of products of nitrogen metabolism (including urea, creatinine, and other unmeasured product s) (1, 2). AKI has been reported to be present in up to 22% of hospitalized adults, with a much higher incidence (40-60%) reported in critically ill adults (3). There are multiple definitions used for AKI, with the criteria proposed by the Acute Kidney Injury Network (AKIN) group in 2007 widely accepted (2). An acute decrease in kidney function is classified as AKI if there is an absolute increase in se-

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↑What is "already known" in this topic:
Community-acquired pneumonia leads to Acute Kidney Injury (AKI) through various mechanisms. AKI increases hospital stay, morbidity and mortality.

→What this article adds:
As there are limited data evaluating the incidence and risk factors for AKI in patients of community-acquired pneumonia from resource-limited countries, our study is an effort to bridge this knowledge gap.

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rum creatinine of ≥ 0.3 mg/dL, an increase in serum creatinine of ≥ 50% (1.5-fold from baseline), or a reduction in urine output (<0.5 ml/kg per hour for >6 hours). AKI in hospitalized patients is an independent adverse prognostic factor, with a reported mortality of 24%, and associated with more than twice the twice mortality rate in critically ill patients without AKI (3). Various risk factors associated with hospital-acquired AKI (HAAKI) include infection, sepsis, use of nephrotoxic medications, intravenous contrast administration, major surgeries, and underlying kidney disease (1, 4). Community-acquired pneumonia (CAP) is one of the most common reasons for hospital admission, and previous studies have suggested that the development of AKI during hospitalization for pneumonia substantially increases mortality (5, 6). The mechanism of development of AKI in these patients is presumed to be multifactorial, with inflammation induced by an infectious process thought to play a prominent role (5, 7). The pro-inflammatory cytokines could cause or exacerbate impaired renal function by direct renal endothelial injury, vasoplegia, altered mitochondrial function, direct tubular injury, or causing cell cycle arrest in addition to other mechanisms (8-12). AKI is not uncommon even in patients with non-severe CAP (up to 25 % of patients), resulting in increased short- and long-term mortality (1, 7).

There are limited data evaluating the incidence and risk factors for AKI in patients hospitalized with CAP and we could not identify any studies from resource-limited countries looking at this subset of patients in our literature review. We examined the incidence and risk factors for AKI in patients hospitalized with CAP at a tertiary care hospital in India.

Methods

This prospective observational study conducted over 1 year period included 210 patients. Patients ≥ 18 years were included in the study if they had three or more of the following symptoms or signs: fever, new-onset cough with or without sputum production, pleuritic chest pain, dyspnea, or altered breath sound on auscultation, plus a chest radiograph showing a new pulmonary infiltrate compatible with the presence of acute pneumonia (13). Exclusion criteria included hospital-acquired pneumonia (development of symptoms 48 hours following admission); or active thoracic malignancy. Demographic data were collected, and past history was noted for any underlying comorbid illnesses such as diabetes mellitus, chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD), coronary artery disease (CAD), chronic liver disease (CLD), or hypertension (HTN). The study was approved by the Institutional Ethics Committee and all the participants consented to the study. The severity of pneumonia at presentation was assessed by the CURB-65 score (14). All the patients underwent routine investigations in the form of complete blood count, routine serum biochemistry, radiography of the chest, electrocardiogram, and computed tomography of the chest when indicated. The patients were managed as per the protocol of the treating unit. We classified patients with acute kidney injury if they met the criteria at any time during hospitalization as per Acute kidney injury network (AKIN) criteria. In patients with no history of CKD or pre-hospital creatinine value, we estimated premorbid creatinine using the Modification of Diet in Renal Disease (MDRD) equation and subsequently selected the lower creatinine value from either the MDRD creatinine or hospital admission creatinine as the baseline value (15). Differences in baseline characteristics between hospitalized CAP patients with and without AKI were evaluated. We also assessed laboratory predictors for the development of AKI and the requirement for renal replacement therapy. The decision to proceed to renal replacement therapy (RRT) was at the discretion of the treating unit.

Statistical Analyses

The results were analyzed using SPSS statistical software version 19.0 (IBM Corporation; Armonk, NY, USA). After analyzing the data for distribution and skewness, descriptive statistics were applied and discrete variables were analyzed using the Chi-square test for categorical data. Normally distributed continuous variables were compared using Welch's t-test for independent samples, while variables showing non-Gaussian distribution were subjected to the Mann-Whitney U test. Variables were subjected to binary logistic regression to examine the association with AKI, using odds ratios (OR). We considered p-value <0.05 as statistically significant.

Results

We analyzed 210 patients with CAP who met the study inclusion criteria. There was a slight male preponderance (n=110, 52.3%), with the median age of the study population being 59 years (Range 18 to 95 years, SD= 15.6). A total of 58 (27.6%) patients developed AKI. The demographic characteristics and comorbidities for patients in AKI and no-AKI groups are compared in Table 1. Patients in the AKI group had a significantly higher proportion of patients with co-morbidities like CKD (p=0.005) and CAD (p=0.032).

A comparison of laboratory characteristics revealed significantly higher uric acid (p=0.002), lower albumin levels (p=0.005), lower total protein (p=0.015), higher bilirubin (p=0.001), and higher LDH levels (p=0.041) in the AKI group in addition to the elevated creatinine, BUN (p<0.001) compared to the no-AKI group (Table 2). We observed that the CURB-65 score was higher in the AKI compared to the non-AKI group (p<0.001).

The association of AKI with uric acid (OR 1.101, 95% CI 0.97 to 1.24, p=0.134), albumin (OR 0.629, 95% CI 0.29 to 1.32, p=0.221), total protein (OR 0.809, 95% CI 0.48 to 1.35, p=0.421), and bilirubin (OR 1.722, 95% CI 0.96 to 3.07, p=0.066) levels were not statistically significant in binary logistic regression analysis, after adjusting for creatinine and BUN levels. Increased LDH levels minimally increased odds for AKI (OR 1.001, 95% CI 1.0001 to 1.002, p=0.036), while higher CURB-65 score (OR 1.794, 95% CI 1.25 to 2.55, p=0.001) continued to be significantly associated with AKI (Table 3).

Out of the 58 patients who developed AKI, 8 (13.8%) needed RRT. All the patients needing RRT were male

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Table 1. Baseline characteristics in AKI and no-AKI groups (* SD: Standard deviation, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, COPD: Chronic obstructive pulmonary disease, CKD: Chronic kidney disease, CAD: Coronary artery disease, CLD: Chronic liver disease)

Variable	AKI (n=58)	No-AKI (n=152)	p-value
Age in years, Mean (SD)	60.2 (13.2)	58 (16.4)	0.356
Gender, n (%)			0.091
Male	36 (17.1)	74 (35.2)	
Female	22 (10.5)	78 (37.2)	
SBP in mmHg, Mean (SD)	108 (17.4)	113.2 (18)	0.062
DBP in mmHg, Mean (SD)	70.2 (11.7)	71.3 (11)	0.518
COPD (n)			
Yes	21	58	0.874
No	37	94	
CKD (n)			
Yes	13	12	0.005
No	45	140	
CAD (n)			
Yes	9	9	0.032
No	49	143	
CLD (n)			
Yes	1	2	1.000
No	57	150	
Hypertension (n)			
Yes	33	79	0.540
No	25	73	

Table 2. Laboratory values for AKI and no-AKI groups

Variable	AKI (n=58)	No-AKI (n=152)	p-value
Creatinine in mg/dL, Mean (SD)	2.77 (1.71)	1.12 (0.35)	<0.001
BUN in mg/dL, Mean (SD)	104.5 (69.9)	51.0 (23.3)	<0.001
Calcium in mg/dL, Mean (SD)	8.8 (0.8)	9.0 (0.7)	0.094
Uric acid in mg/dL, Mean (SD)	6.1 (2.7)	4.8 (2.4)	0.002
Albumin in g/dL, Mean (SD)	3.04 (0.51)	3.28 (0.57)	0.005
Phosphorus in mg/dL, Mean (SD)	11.2 (41.3)	3.0 (0.8)	0.137
Total Bilirubin in mg/dL, Mean (SD)	1.2 (0.7)	0.9 (0.5)	0.001
Total Protein in g/dL, Mean (SD)	5.9 (0.7)	6.2 (0.7)	0.015
ALT/SGPT in IU/L, Mean (SD)	88.1 (111.9)	58.7 (67.7)	0.067
LDH in IU/L, Mean (SD)	492.8 (492.0)	353.9 (205.4)	0.041
Hemoglobin in g/dL, Mean (SD)	11.1 (2.4)	11.8 (2.3)	0.051
WBC count x 10^9/L, Mean (SD)	12.1 (11)	10.7 (4.3)	0.180
CURB-65 score	2.3 (0.9)	1.6 (1)	<0.001
ICU admission (n)			
Yes	4	17	0.447
No	54	135	
Mortality (n)			
No	55	142	0.494
Yes	3	10	

Table 3. Results of regression analysis of biochemical parameters and CURB 65 score vis a vis AKI

Variable	Beta coefficient	SE (beta coefficient)	Lower limit of CI	Upper limit of CI	Odds Ratio	p-value
Uric acid	0.0965	0.0645	0.970	1.249	1.101	0.134
Albumin	-0.4632	0.3791	0.299	1.322	0.629	0.221
Bilirubin	0.5436	0.2958	0.964	3.075	1.722	0.066
Total protein	-0.2110	0.2623	0.484	1.354	0.809	0.421
LDH	0.0013	0.0006	1.0001	1.002	1.001	0.036
CURB-65 score	0.5847	0.1808	1.259	2.557	1.794	0.001

(Table 4). We also analyzed the data for laboratory values associated with RRT in patients with AKI. As expected, the RRT group had significantly higher creatinine (p<0.001) and blood urea nitrogen (p=0.016). The number of male patients was significantly higher in the RRT group compared to the no-RRT group (p=0.019). Also, patients in the RRT group were observed to have significantly higher phosphorus (p=0.038), and lower ALT (p=0.022) levels. Although the CURB-65 score showed a trend for higher scores in the RRT group, the difference did not attain statistical significance (p=0.064). In the regression analysis, ALT (OR 0.98, 95% CI 0.97 to 0.99, p=0.031) levels were marginally associated with a need for RRT, while phosphorus levels did not show a statistically significant association (OR 5.5, 95% CI 0.85 to 10.14, p=0.083). Pertinently, the adjusted logistic regression analysis revealed that patients with higher CURB-65 scores were at higher odds of undergoing RRT (OR 8.74, 95% CI 5.27 to 12.21, p=0.039).

Discussion

We report an incidence of AKI in 27.6% of patients hospitalized for CAP in our study. Factors associated with AKI in these patients include past history of CKD or

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Table 4. Comparison of patients requiring RRT with no-RRT group

Variable	RRT (n=8)	No-RRT (n=50)	p-value
Age in years, Mean (SD)	63.4 (16.5)	59.7 (12.7)	0.560
Gender (n)			
Male	8	28	0.019
Female	0	22	
Creatinine in mg/dL, Mean (SD)	6.37 (2.06)	2.18 (0.63)	<0.001
BUN in mg/dL, Mean (SD)	216.3 (116.3)	86.6 (37.2)	0.016
Calcium in mg/dL, Mean (SD)	8.7 (0.8)	8.8 (0.8)	0.813
Uric acid in mg/dL, Mean (SD)	8.1 (4.3)	5.8 (2.3)	0.255
Albumin in g/dL, Mean (SD)	3.1 (0.5)	3.0 (0.5)	0.908
Phosphorus in mg/dL, Mean (SD)	5.1 (2.1)	3.2 (0.9)	0.038
Total Bilirubin in mg/dL, Mean (SD)	2.05 (1.2)	1.17 (0.5)	0.083
Total Protein in g/dL, Mean (SD)	6.3 (0.5)	5.9 (0.7)	0.09
ALT/SGPT in IU/L, Mean (SD)	42.6 (39.6)	95.5 (118.2)	0.022
LDH in IU/L, Mean (SD)	340 (150.3)	517.3 (523)	0.477
Hemoglobin in g/dL, Mean (SD)	10.83 (2.62)	11.17 (2.43)	0.742
WBC count x 10^9/L, Mean (SD)	13.72 (2.65)	11.95 (11.82)	0.359
CURB-65 score	3.0 (0.9)	2.2 (0.9)	0.064

CAD, certain laboratory parameters (high uric acid, low albumin, low total protein, high bilirubin, and high LDH levels), and higher CURB-65 score (i.e., more severe pneumonia). However, the laboratory parameters of uric acid, albumin, total protein, and bilirubin levels were not seen to increase the odds for AKI independently in the regression analysis, and the relation for LDH levels was also attenuated in the adjusted analysis (a unit increase in LDH levels increases odds of AKI by 0.1%). Pneumonia severity was significantly associated with AKI independently, with an increase of CURB-65 score by a single digit resulting in 79.4% higher odds of AKI.

Although it is difficult to know the exact incidence of AKI in developing countries due to the paucity of data, it is estimated that the burden of AKI is quite high as it is all over the world. A meta-analysis has suggested a pooled incidence of HAAKI between 7.5- 31% and a mortality of 14-37% in different parts of Asia (3, 16). Most of the data regarding the epidemiology of AKI in India is from single-center studies, with wide fluctuations in incidence and causes of AKI in hospitalized or critically ill patients (17, 18). An incidence of AKI in 6-46% of critically ill patients has been reported in various studies, with sepsis, tropical illnesses (like malaria, scrub typhus, dengue), pneumonia, pyelonephritis, H1N1 influenza, viral hepatitis, leptospirosis, undifferentiated febrile illness, enteric fever, toxins and envenomation as some of the causes reported with widely fluctuating frequencies (4, 17-21). Pneumonia was reported as the most common medical illness in sepsis-associated AKI in a study from Nepal, documented in 50% of the study population with AKI in the intensive care unit (ICU) (21). Viral pneumonia are also associated with high rates of AKI. During the H1N1 pandemic of 2009, AKI was seen as a frequent complication in critically ill patients with incidence rates of 30-50% (22, 23). Similarly, AKI is now recognized as a well-known complication of the novel COVID-19 illness. A recent report observed an incidence of >20% in hospitalized patients, increasing to >50% in ICU patients, causing adverse outcomes with the potential for overburdening already stretched healthcare resources (24).

The observed incidence of AKI in patients with CAP in our study was similar to that noted in previous Western studies (6, 7). Akram et al. observed around 18 % of patients with CAP have AKI at admission in their study (6). Murugram et al. reported an overall 34 % incidence of AKI in patients with CAP, with up to a quarter of non-severe pneumonia patients also developing AKI (7). Also, the higher percentage of AKI seen in severe CAP agrees with our study finding showing AKI is associated with higher CURB-65 scores (6, 7). The risk factors associated with AKI in our study included comorbidities like CKD or CAD, which have been reported previously (6, 7, 25). We also observed laboratory features of higher uric acid, lower albumin and total protein, higher bilirubin, and higher LDH levels in patients with AKI. In their study of elderly patients with AKI, Sevor et al. (26) observed AKI more frequently with altered sensorium and abnormal liver function tests (including hyperbilirubinemia). The incidence of AKI in patients was noted to be higher in males in patients with pneumococcal pneumonia (27). Although we did not note this association for the incidence of AKI, the need for RRT in patients with AKI was significantly higher in males in our study. Also, we observed that around 3.8 % (n=8) of the study population ended up requiring RRT during the hospital stay. This is in close agreement with a previous study, where 2.4 % of patients admitted with CAP required RRT (6).

This study, the first of its kind in a resource-limited setting, highlights the high incidence of AKI in patients hospitalized for CAP, similar to that reported from resource-rich countries. It is recognized that the lower incidence of HAAKI reported from the developing nations could potentially be a result of the under-recognition of AKI due to a lack of awareness among health care providers and limited available resources (28, 29). It cannot be overemphasized that clinicians need to be alert for the prevention and early detection of AKI in patients admitted with this common diagnosis. A few strategies suggested include frequent risk assessment for hospitalized patients (at admission and during stay); optimizing fluid volume, blood pressure and perfusion; correction and maintenance of electrolyte, acid-base, and mineral homeostasis; prompt treatment of hypotension and sepsis; avoiding the use of nephrotoxic drugs especially in those with or at risk of AKI; and if needed they should be administered in dosage

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based on the volume of distribution in the elderly, obese and malnourished patients; and drug level monitoring for nephrotoxic medications (29, 30).

Study Limitations

This was a single-center observational study. As is common with studies of AKI, values of baseline creatinine values were not available for all patients and were estimated based on MDRD guidelines, which is an established methodology. Since the etiopathogenesis of AKI is often multifactorial, confounding factors like patient medications, fluids, and hemodynamic status could have influenced the development of AKI and its course. Future studies could focus on the role of these confounding factors, especially the administration of nephrotoxic medications. Furthermore, we did not have a long-term follow-up for the development of residual kidney disease or other complications.

Conclusion

There is a high incidence of AKI in patients hospitalized for CAP in developing countries, similar to that reported from resource-rich countries. Clinicians should be alert for prevention and early detection of AKI in patients admitted with this common diagnosis. Further studies should be conducted to investigate long-term complications from AKI in these patients and evaluate strategies for prevention and treatment in resource-limited settings

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Conflict of Interests

The authors declare that they have no competing interests.

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