Original Research Article

Study of clinical profile of acute kidney injury in children with idiopathic nephrotic syndrome

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Abstract

Background: Acute kidney injury (AKI) in childhood nephrotic syndrome is an uncommon but serious complication resulting from intravascular volume depletion, acute tubular necrosis, interstitial nephritis, bilateral renal venous thrombosis or rapid progression of underlying glomerular disease. Present study was aimed to study clinical profile of acute kidney injury in children with idiopathic nephrotic syndrome. **Material and Methods:** Present study was retrospective, case record-based study, conducted children who had discharge diagnosis of acute kidney injury in idiopathic nephrotic syndrome. **Results:** In present study 64 children admitted as acute kidney injury in idiopathic nephrotic syndrome were studied. Majority were of 8-11 years age (39.06 %) followed by 12-15 years (23.44 %). Boys (65.63 %) outnumbered girls (34.38 %) and boys: girls ratio was 1.9:1. According to histopathology findings common lesion were Focal segmental glomerulosclerosis (32.81 %), Minimal change disease (20.31 %) and Mesangioproliferative GN (6.25 %), while biopsy was not performed in 19 cases (29.69 %) Most common factor associated with AKI in idiopathic nephrotic syndrome was infection (46.88 %) followed by shock/ hypovolemia (26.56 %), mechanical ventilation (20.31 %), congestive cardiac failure (12.50 %), nephrotoxic medication exposure (9.38 %), diuretic use (7.81 %) and prerenal cause (6.25 %), **Conclusion:** Acute kidney injury (AKI) is an alarming complication of idiopathic nephrotic syndrome. Common causes of AKI were infection, aggravation of NS, dehydration, and nephrotoxic agents.
Keywords: Acute kidney injury (AKI), idiopathic nephrotic syndrome. Infection, dehydration, nephrotoxic agents.

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INTRODUCTION

The most common cause of nephrotic syndrome in children is idiopathic nephrotic syndrome (INS), also called nephrosis.[1] INS is defined by the combination of a nephrotic syndrome (massive proteinuria, hypoalbuminemia, hyperlipidemia, and edema) and nonspecific histological abnormalities of the glomeruli including minimal changes, focal and segmental glomerular sclerosis (FSGS), and diffuse mesangial proliferation. Acute kidney injury (AKI) is an important and alarming complication of idiopathic nephrotic syndrome (INS).[2,3] AKI, usually reversible, has been observed in children with normal or minimally altered glomeruli on renal biopsy. It may occur either at the time of presentation or much later.[4] Acute kidney injury (AKI) in childhood nephrotic syndrome is an uncommon but serious complication resulting from intravascular volume depletion, acute tubular necrosis, interstitial nephritis, bilateral renal venous thrombosis or rapid progression of underlying glomerular disease.[2] AKI is generally known to be associated with high mortality and morbidity, including risk of progression to chronic kidney disease.[5] Present study was aimed to study clinical profile of acute kidney injury in children with idiopathic nephrotic syndrome.

MATERIAL AND METHODS

Present study was retrospective, case record-based study, conducted in Department of Pediatrics, Surabhi Institute of

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Medical Sciences, Mittapally Village, Siddipet Mdl And Dt, India. Study was approved by institutional ethical committee. All hospitalizations for children <16 years of age between January 2017 and December 2021 with a discharge diagnosis of acute kidney injury in idiopathic nephrotic syndrome were studied. Children with NS who at admission had CKD Stage >III, known secondary NS (e.g., systemic lupus erythematosus, Henoch–Schoenlein purpura, etc.) and history and investigations suggestive of rapidly progressive glomerulonephritis were excluded from the study. Detailed clinical history regarding demographic data, infections, nephrotoxic drugs and hypertension were noted. Detailed general physical examination and systemic examinations finding were noted. Laboratory investigations like CBC, Serum creatinine and blood urea, USG abdomen, etc. were noted. Treatment details and clinical outcome noted. All details were entered in a pre-designed proforma. Statistical analysis was done using descriptive statistics.

RESULTS

In present study 64 children admitted as acute kidney injury in idiopathic nephrotic syndrome were studied. Majority were of 8-11 years age (39.06 %) followed by 12-15 years (23.44 %). Boys (65.63 %) outnumbered girls (34.38 %) and boys: girls ratio was 1.9:1.

Table 1: Age and gender

Age (years)	No. of children	Percentage
0–3	11	17.19%
4–7	13	20.31%
8–11	25	39.06%
12–15	15	23.44%
Gender		
Boys	42	65.63%
Girls	22	34.38%

According to histopathology findings common lesion were Focal segmental glomerulosclerosis (32.81 %), Minimal change disease (20.31 %) and Mesangioproliferative GN (6.25 %), while biopsy was not performed in 19 cases (29.69 %)

Table 2: Histopathology

Histopathology	No. of children	Percentage
Biopsy not done	19	29.69%
Focal segmental glomerulosclerosis	21	32.81%
Minimal change disease	13	20.31%
Mesangioproliferative GN	4	6.25%
Mesangiocapillary GN	2	3.13%
Membranous nephropathy	2	3.13%
IgM nephropathy	2	3.13%
IgA nephropathy	1	1.56%

Most common factor associated with AKI in idiopathic nephrotic syndrome was infection (46.88 %) followed by shock/ hypovolemia (26.56 %), mechanical ventilation (20.31 %), congestive cardiac failure (12.50 %), nephrotoxic medication exposure (9.38 %), diuretic use (7.81 %) and prerenal cause (6.25 %),

Table 3: Factors associated with AKI

Associated factor	No. of children	Percentage
Infection,	30	46.88%
· Upper/lower respiratory tract infection	11	17.19%
· Cellulitis	4	6.25%
· Peritonitis	3	4.69%
· Acute gastroenteritis	12	18.75%
Shock/ Hypovolemia	17	26.56%
Mechanical ventilation	13	20.31%
Congestive cardiac failure	8	12.50%
Nephrotoxic medication exposure	6	9.38%
Diuretic	5	7.81%
Prerenal cause	4	6.25%

DISCUSSION

Acute kidney injury/impairment (AKI) includes the entire spectrum from minor changes in markers of renal function to the requirement of renal replacement therapy. It includes patients with functional impairment relative the physiological demand1. It reflects the importance of smaller derangements in kidney function which exert a significant influence on morbidity and mortality.[6,7] Agrawal, A *et al.*,[8] studied 107 cases of nephrotic syndrome, the most common age group was 5–7 years (54.2%). There were 73 (68.2%) males and 34 (31.7%) females with a male-to-female ratio of 2.1:1. It was found that 39 (36.4%) subjects were newly diagnosed and 68 (63.6%) were relapse cases. A total of 95 (88.8%) patients were steroid-sensitive while 12 (11.2%) had initial steroid resistance NS. In study by Prasad BS *et al.*,[9] 73 children (81 admissions) were enrolled; incidence of AKI was 16% (95% CI, 9-23). On multivariate logistic regression analysis, furosemide infusion was observed as an independent risk factor for acute kidney injury (OR 23; 95% CI, 3-141; P<0.001). Out of 13 children with AKI, three died. Acute kidney injury in hospitalized children with nephrotic syndrome has high risk of mortality. Children receiving furosemide infusion should be closely monitored for occurrence of acute kidney injury. Afshan Yaseen *et al.*,[10] noted that the mean age at presentation was 8.8 ± 3.59 years and males were 74 (62.2%). At presentation, 61 (51.3%) children were in Risk category, 43 (36.1%) in Injury category, and 15 (12.6%) in Failure category. Most of them (41.2%) had steroid-resistant nephrotic syndrome (SRNS) and focal segmental

glomerulosclerosis (FSGS) on histopathology (33.6%). Infections were the major predisposing factor for AKI in 67 (56.3%) cases. Drug toxicity was the next common, found in 52 (43.7%) children. A total of 65 (54.6%) children recovered from AKI, while 54 (45.4%) did not. CKD developed in 49 (41.2%) non-recovered cases and 5 (4.2%) children succumbed to acute illness. SRNS, cyclosporine use, FSGS on histology, and drug toxicity were significant factors associated with the development of CKD. Sharma M *et al.*,[11] noted that incidence of AKI in children with NS was 23.66%, 11.24%, 7.95% and 4.48% of children entered Pediatric Risk, Injury, Failure, Loss, End-Stage Renal Disease (pRIFLE) Stages R, I and F, respectively. Infection {odds ratio [OR] 2.53 [95% confidence interval (CI) 1.52–4.22]} and nephrotoxic medication exposure [OR 7.8 (95% CI 4.06–15.01)] were common factors associated with AKI. Children with steroid-dependent NS (SDNS) and steroid-resistant NS (SRNS) were more likely to develop AKI compared with children with steroid-sensitive NS (SSNS). The mean time to recovery for groups pRIFLE Stages R, I and F were 1562, 2263 and 2865 days, respectively. Children with NS who were hypertensive, had higher urinary protein excretion and low serum albumin were more prone to develop AKI. In study by Kumari A *et al.*[12], mean age of the children was 4.7± 2.8 years. Complications were observed in 67% of the cases. The most frequent complication was anemia (25%) followed by infection (21%). The incidence of AKI was 18.6% in hospitalized children with NS. According to the pRIFLE criteria, 11.6% of the children met stage 1 (risk) criteria, 4.6% met stage 2 (injury) criteria, and 2.3% met stage 3 (failure) criteria. Among all NS children, 53% received nephrotoxic drugs during the hospital stay. On applying multivariate logistic regression analysis, only male gender, associated anemia, and vancomycin use were significant independent risk factor for AKI in nephrotic syndrome patients. In study by Kushwah S *et al.*,[13] 115 patients (72.2% boys) enrolled at median (interquartile range) age 64 (36–111) months, 25 (21.7%) developed AKI. The incidence density of AKI was 3.3 (2.2, 4.8) episodes per 100 person-days. Stage 3 AKI comprised 64% of cases. Steroid-resistant illness, hypoalbuminemia, and low baseline eGFR were independently associated with the occurrence of AKI. AKI recovered completely or partially in 48% and 20% cases, respectively; 20% of patients remained dialysis-dependent and 12% of patients died. Patients with AKI had significantly longer hospital stay, and lower median eGFR at 3-month follow-up, than those without AKI. Management of AKI is mostly supportive.[14] Mild AKI that may be seen at the onset of NS along with edema and proteinuria often resolves as the patient goes into remission with steroids. Those presenting with intravascular volume depletion, infection, or nephrotoxic drugs respond to appropriate management of those inciting factors. While volume replacement helps those who have intravascular volume depletion, albumin infusions have not been shown to be beneficial for the management of AKI. It is often lost immediately in the urine and improves neither serum albumin nor renal function.

CONCLUSION

Acute kidney injury (AKI) is an alarming complication of idiopathic nephrotic syndrome. Common causes of AKI were infection, aggravation of NS, dehydration, and nephrotoxic agents. The advances in the understanding of nephrotic syndrome, the changing epidemiology and regional differences in disease pattern, necessitates further studies on clinical types of nephrotic syndrome, management pattern and determination of trends across various settings.

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