EDITORIAL

Acute kidney damage in primary nephrotic syndrome patients

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INTRODUCTION

In patients with primary nephritic syndrome, Acute Kidney Damage (AKI) is a common and dangerous consequence (PNS). The goal of this study was to determine the factors that influence AKI in patients with PNS, with implications for clinical management and nursing care of patients with PNS. In patients with primary nephritic syndrome, acute kidney damage (AKI) is a common but dangerous consequence (PNS). According to studies, the incidence of AKI in children with PNS ranges from 1.28 percent to 38.26 percent, while it can reach 44.9 percent in adults. According to the Kidney Disease Improving Global Outcomes Guidelines, the prior criteria for diagnosing acute renal failure overlooked some patients in the early stages of AKI (KDIGO). Furthermore, AKI is an independent risk factor for the progression of nephritic syndrome to chronic renal disease. As a result, early detection and prevention of AKI is critical for PNS patients' prognosis. The exact mechanism of secondary AKI in patients with PNS is currently unknown. AKI is thought to be caused by intracranial ischemia, renal interstitial edema, glomerular lesions, renal tubular necrosis, drug-related interstitial nephritis, and other factors. The clinical features and prevalent pathological kinds of children with PNS are considerably different from those of adults with PNS, as most of the reported studies are focused on the adult population [1]. There are few studies that look at the link between PNS and the risk of adult AKI in the kidneys' degenerative characteristics As a result, we retrospectively examined the clinic pathological characteristics of patients with PNS in our hospital with the goal of analyzing the factors that influence AKI in patients with PNS and providing evidence for clinical management and nursing care of patients with PNS.

Acute kidney injury (AKI), also known as acute renal failure (ARF), is a brief period of kidney failure or damage that occurs over a period of hours or days [2]. AKI causes a build-up of waste products in your blood, making it difficult for your kidneys to maintain the proper fluid balance in your body. Other organs, such as the brain, heart, and lungs, can be affected by AKI. Acute kidney injury is common in hospitalized patients, especially in intensive care units, and in the elderly. AKI treatment frequently necessitates a stay in a hospital. recover. Dialysis may be required in more serious cases to help replace renal function until your kidneys heal. Your healthcare provider's main goal is to treat the underlying cause of your acute kidney injury. Until your kidneys recover, your healthcare practitioner will treat all of your symptoms and consequences. Following AKI, your risks of developing other health issues (such as renal disease, stroke, or heart disease) or developing AKI again are increased. Every time AKI develops, the odds of developing renal disease and kidney failure increase [3]. To protect yourself, you should keep track of your kidney function and recovery with your healthcare professional. Preventing acute kidney injury or detecting and treating it as soon as possible are the greatest approaches to reduce your chances of developing kidney damage and preserving renal function.

The global burden of AKI-related death much outweighs that of breast cancer, heart failure, or diabetes, and mortality rates have remained high over the last 50 years. The occurrence of AKI is often classified as either community-acquired or hospital-acquired AKI [4]. AKI is mostly hospital-acquired in high-income countries (HIC), whereas community-acquired AKI is more common in low-income nations. These patterns can be found in both adults and children around the world. Patients with AKI in HIC are generally older, have several comorbidities, and have access to dialysis and intensive care if necessary. The main causes of AKI in HIC include post-surgical or diagnostic treatments, as well as iatrogenic events. In low-income areas, however, there are a variety of community-acquired causes, such as sepsis, volume depletion, toxins (bites, treatments), and pregnancy. A meta-analysis of 154 studies that defined AKI according to the 2012 Kidney Disease Improving Global Outcomes (KDIGO) classification gathered data from 3,585,911 people from mostly north of the Equator (84 percent HIC) and found community-acquired AKI in 8.3% of ambulatory patients and 20.0%-31.7% of patients at various levels of in-hospital care [5]. Others report substantially lower rates, which could be due to differences in AKI classifications and local conditions. The average pooled death rate was 23%, but

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those requiring KRT had a rate of 49.4%. The following were the diagnostic criteria for PNS: (1). The patient had: (1) proteinuria (quantitative urine protein > 3.5 g/L); (2) hypo albuminuria (plasma protein less than 30g/L); (3) edema; and (4) hyperlipidemia. Item 1 and 2 were both required for PNS diagnosis. We ruled out Henoch-Schonlein purpura, systemic lupus erythematous, hepatitis B-related nephropathy, and other illnesses as causes of secondary nephritic syndrome [6].

Massive proteinuria is the most common symptom of PNS, which is a set of clinical disorders. The most significant complication of PNS is acute renal damage. Renal interstitial edema, glomerular pathology, hypo perfusion, renal tubular epithelial cells necrosis, and renin–angiotensin–aldosterone system (RAAS) activation are all linked to AKI. PNS has a rather high incidence of AKI. It will not only influence the patient's prognosis, but it will also increase the patient's suffering and financial load, and in more extreme circumstances, it could be life-threatening [7]. As a result, it's critical to address AKI risk factors as soon as possible. Diabetes, pulmonary infection, albumin 24g/L, serum creatinine 90mol/L, blood urea nitrogen 6.5mmol/L, uric acid 390mol/L, and renal tubular casts are independently affecting variables of AKI in PNS patients, according to our findings.

Mild glomerular disease, Ig A nephropathy, and membranous nephropathy are the most common pathological forms of AKI, according to the findings of this study. Previous research has found that adults with mild glomerular illness are the most vulnerable to AKI, with an AKI incidence ranging from 24.11% to 38.42%. Previous researchers collected 13 publications on mild glomerular disease and AKI published between 1993 and 2017 [8], finding that the incidence of AKI in mild glomerular disease patients is 33%, which is comparable with the findings of this study. Only occasional data on the association between certain renal pathological characteristics and PNS secondary AKI exist at the moment. Renal tubular avascular necrosis with pathological injury has been identified as a risk factor for AKI in studies. Tubulin casts were found to be an independent risk factor for AKI in this study's pathological investigation of the kidneys. As a result, in the clinical environment, early alarms on the development of AKI are required for those patients with protein casts who have a greatly elevated risk of AKI. According to previous research, the risk of AKI increases by 4.97 times for every 10g/L fall in albumin level. Proteinuria is directly linked to renal injury, which can enhance the development of AKI in PNS patients [9]. Hypo albuminuria is common in PNS patients, owing to the leakage of a substantial amount of proteinuria. Urine protein may cause endoplasmic reticulum stress, cell apoptosis, and renal tubule damage by activating complement, promoting chemo taxis, and cytokine production. Furthermore, patients with PNS are more likely to develop hyper uremia as a result of low blood volume, diuretic use, poor renal function, and lipid metabolism issues. Hyper uremia has been linked to an increased risk of AKI in patients with PNS in previous investigations. This study also found that a blood uric acid level of 390mol/L at the time of admission in PNS patients is a risk factor for AKI. Hyper uremia can activate the RASS, alter renal hemodynamics, and damage endothelial cells and the renal interstitial, resulting in renal ischemia. Furthermore, the renal tubules can produce a significant increase in blood uric acid. Uric acid crystals can obstruct the renal tubules or compress the distal renal blood vessels, resulting in AKI. Infections are common in PNS patients due to the lack of cellular immunodeficiency, immunoglobulin Ig G, and complement factors. In patients with PNS, lung infection is also a risk factor for AKI, according to this study. The majority of PNS patients' kidneys are edematous and ischemic [10]. As a result, infection may exacerbate renal ischemia and hypoxia, renal tubule damage, and impact kidney repair through immune inflammatory reactions, oxidative stress damage, and other processes, leading to AKI in PNS patients. Hypertension and diabetes have been linked to the development of AKI in previous research; however the influence of hypertension and diabetes on AKI in PNS patients is currently unknown.

Diabetes is an independent risk factor for AKI in patients with PNS, according to our findings. Hyperglycemia can cause kidney cells to produce more endothelin-1, which can exacerbate renal tissue ischemia and raise the risk of AKI in people with PNS. The independent risk factors identified using the above-mentioned multivariate logistic regression analysis can be used to guide early detection, prevention, and active treatment of AKI in patients with PNS, and have a high therapeutic utility for improving patient prognosis [11]. There are some limitations to this study that should be considered. To begin, patients who had not had a kidney biopsy were eliminated, resulting in a discrepancy between the incidence of AKI and the reality. Second, because the study was a single-center retrospective cohort study with a small sample size, it was unable to develop a prediction model for the development of associated AKI. In the future, the findings of this study will need to be confirmed by a large sample of multi-center data. Finally, this study only collected pertinent data during the patient's hospitalization and did not conduct any long-term follow-up analysis. The best AKI treatment and the risk variables that influence prognosis are continuously being researched [12].

In conclusion, we discovered that the incidence of AKI in PNS patients is 28.05 percent, and that PNS patients with diabetes, pulmonary infection, albumin 24g/L, serum creatinine 90mol/L, blood urea nitrogen 6.5mmol/L, uric acid 390mol/L, renal tubular casts, and renal tubular casts may be at higher risk of AKI. Patients with PNS should actively manage pulmonary infections and diabetes, and selecting an appropriate treatment plan is critical for preventing AKI [13].

REFERENCES

1. Zhou YL, XG D. Risk factors of acute kidney injury complicating adult primary nephrotic syndrome. Acta Acad Med Sinic. 2020;42(4):436-443.

2. Rovin BH, Caster DJ, Cattran DC, et al. management and treatment of glomerular diseases (part 2): conclusions from a kidney disease: improving global outcomes (kdigo) controversies conference. Kid Intern. 2019;95(2):281-295.

3. Larpparisuth N, Chanchairujira T, Chawanasuntorapoj R, et al. Acute kidney injury in primary nephrotic syndrome: report of nine cases in siriraj hospital. J Med Asso Thailand. 2011;94(2):125.

4. Lionaki S, Liapis G, Boletis JN. Pathogenesis and management of acute kidney injury in patients with nephrotic syndrome due to primary glomerulopathies. Med. 2019;55(7):365.

5. Chen T, Zhou Y, Chen X, et al. Acute kidney injury in idiopathic membranous nephropathy with nephrotic syndrome. Renal Fail. 2021;43(1):1004-1011.

6. Kodner C. Diagnosis and management of nephrotic syndrome in adults. Am Fam Phy. 2016;93(6):479-485.

7. Shi Y, Huang Y, Zhang TT, et al. Chinese guidelines for the diagnosis and treatment of hospital-acquired pneumonia and ventilator-associated pneumonia in adults (2018 Edition). J Thoracic Dis. 2019;11(6):2581.

8. Ronsin C, Georges M, Chapelet-Debout A, et al. ANCA-negative pauci-immune necrotizing glomerulonephritis: a case series and a new clinical classification. Am J Kidney Dis. 2022;79(1):56-68.

9. Shi C, Li C, Ye W, et al. Nephrotic-range proteinuria and central nervous involvement in typical hemolytic uremic syndrome: a case report. BMC Nephrol. 2020;21(1):1-5.

10. Togashi H, Shimosato Y, Saida K, et al. Childhood nephrotic syndrome complicated by catastrophic multiple arterial thrombosis requiring bilateral above-knee amputation. Front Pediat. 2020;8:107.

11. Kim MY, Cho MH, Kim JH, et al. Acute kidney injury in childhood-onset nephrotic syndrome: Incidence and risk factors in hospitalized patients. Kidney Res Clin Pract. 2018;37(4):347.

12. Mühlbacher T, Amann K, Mahling M, et al. Successful long-term management of recurrent focal segmental glomerulosclerosis after kidney transplantation with costimulation blockade. Clin Kid J. 2021;14(6):1691-1693.

13. Xu X, Hu J, Song N, et al. Hyperuricemia increases the risk of acute kidney injury: a systematic review and meta-analysis. BMC Nephrol. 2017;18(1):1-4.