

Clinical Profile and Outcome of Acute Glomerulonephritis in a Tertiary Care Centre in the Eastern Nepal

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Abstract

Introduction: Acute glomerulonephritis (AGN) generally presents as a constellation of findings that includes hematuria, edema, and often hypertension. The study aimed to determine the clinico-biochemical profile and outcome of acute glomerulonephritis in children at a tertiary care centre in the Eastern Part of Nepal.

Methods: A prospective study was conducted on all the cases found to have acute glomerulonephritis, and sample size constituted all children between 1 month to 15 year, attending the Renal Special OPD and those hospitalized in pediatric department of BPKIHS from Feb. 2012 to Jan. 2013 with acute glomerulonephritis.

Results: There were total of 94 cases of acute glomerulonephritis (AGN), which comprise 3.1% of annual pediatric admissions. 52.1% were female and male were 47.8%. Mean age of presentation was 9.2 yrs. \pm 3.1 SD. Aetiology of AGN was post infectious glomerulonephritis (PIGN) 84.0%, lupus nephritis 10.6%, mumps nephritis 3.1% and HSP nephritis 2.1%. ASO titer was raised in 34.0%. 47.8% had raised urea, 43.6% had raised creatinine level, >2+proteinuria was present in 26.6% and pyuria in 34.0%. The common clinical findings at presentation were microscopic hematuria (95.7%), hypertension (86.2%), edema (85.1%), fever (63.8%), oliguria (22.3%), pain abdomen (21.3%) and others. History of sore throat and pyoderma was present in 25.5% and 19.1% respectively. Complications at presentation were hypertensive encephalopathy (9.5%), cardiac failure (9.5%) and acute kidney injury (6.3%). Of 94 cases, 92 cases (97.8%) improved and 2 cases (2.1%) expired due to acute kidney injury.

Conclusion: Acute glomerulonephritis is a significant renal problem in children and is one of the common causes of hospital admission. Early identification and comprehensive monitoring and management is required to prevent morbidity and mortality.

Key words: Children, hematuria, Acute glomerulonephritis

Introduction

Acute glomerulonephritis (AGN) generally presents as a constellation of findings that includes hematuria, edema, and often hypertension. Although the pathogenesis is not fully understood, current evidence supports that most cases of AGN are due to an immunologic response to a variety of different etiologic agents. The immunologic response, in turn, activates a number of biological processes that

result in glomerular inflammation and injury.^{1,2} AGN may be isolated to the kidney (primary glomerulonephritis) or be a component of a systemic disorder (secondary glomerulonephritis). Although Post streptococcal glomerulonephritis (PSGN) continues to be the most common cause of AGN globally, it primarily occurs in developing countries. Of the estimated 470,000 new annual

cases of PSGN worldwide, 97 percent occur in developing countries, with an annual incidence that ranges from 9.5 to 28.5 per 100,000 individuals.^{3,4} The risk of PSGN is increased in children between 5 and 12 years of age.⁵

Methods

This was a prospective study conducted over one year from February 2012 to January 2013. All the cases in the age group of 1 month to 15 years, reporting to pediatric renal clinic and / or admitted to pediatric wards, with features suggestive of renal system involvement were subjected to a detailed history, clinical examination and investigations and data were recorded in a pre-designed proforma. Those cases found to have acute glomerulonephritis were included and rest was excluded from the study. The investigations included hemoglobin, total and differential leukocyte counts, ESR, gross and microscopic examination of urine and culture, 24 hr urinary protein, serum total protein, albumin, cholesterol, urea, creatinine, sodium, potassium, calcium and phosphate. Serum Antistreptolysin O, C3, C4, ANA and anti-Ds DNA were done wherever indicated. Radiological investigations included ultrasonography of kidney, ureter and bladder.

Acute glomerulonephritis were categorized as post infectious glomerulonephritis (PIGN), which included post streptococcal glomerulonephritis (PSGN), mumps nephritis and other infectious aetiology. Other causes of AGN included lupus nephritis and Henoch Schonlein purpura nephritis. AGN was defined and categorized using standard definitions.⁶⁻⁸ They were treated as per our hospital protocols. Each patient was followed during hospital stay and was kept under regular follow up in pediatric renal clinic for their future course. The study protocol was approved by Institute Ethics Committee. Informed written consent was taken from parent or authorized representative of each patient.

Statistical analysis

The data were analyzed using SPSS 20.0 version. Descriptive analysis was made using count, mean, median, range, proportion, percentage and standard deviation.

Results

There were total of 94 cases of acute glomerulonephritis (AGN), comprising 3.1% of annual pediatric admissions. As shown in **Table 1**, 52.1% were female and male were 47.8%, with almost equal female and male proportion. Majority of the cases, 91.5% were above 5 years (school going age), age range was from 3 to 14 years. Mean age of presentation was 9.2 yrs. \pm 3.1 SD. Aetiology of

AGN as shown in **Figure 1**, was PIGN including PSGN 84.0%, lupus nephritis 10.6%, and HSP nephritis 2.1%. Among PIGN, PSGN was 34.0%, mumps nephritis 3.1% and in remaining the aetiology could not be determined. Clinical findings at the time of presentation (**Table 2**) were hypertension 86.2%, edema 85.1%, fever 63.8%, gross hematuria 41.5%, oliguria 22.3%, pain abdomen 21.3%, dyspnea 11.7%, encephalopathy 10.6% and joints pain 10.6%. History of sore throat and pyoderma was present in 25.5% and 19.1% respectively. Urine analysis (**Table 1**) at presentation showed hematuria in 95.7%, proteinuria ($>2+$) in 33.9%, proteinuria ($\leq 2+$) in 66.0%, and pyuria in 34.0%. Thus in nephritis hematuria is almost always accompanied by some degree of proteinuria and in some cases there is pyuria. UTI was present in 5.3%, the culture isolates were E.coli 1%, streptococcus 2.1% and acinetobacter 2.1%. Anemia as per WHO reference value was present in 91.4%. Renal function was impaired in almost half of the patient. High urea was found in 47.8%, similarly creatinine was high in 43.6%. Electrolytes were also deranged in some cases. Hyponatremia (<135 meq/L) was present in 4.2% and hypernatremia (>145 meq/L) in 12.7%. Similarly hyperkalemia (>5 meq/L) was seen in 10.6% and remaining were normokalemic. ASO titre was raised in 34.0%. About 31% cases with AGN also presented with complications of which hypertensive encephalopathy and cardiac failure 9.5%. Other complications were acute kidney injury in 6.3% and nephrotic range proteinuria 5.3%. All the cases were managed as per the hospital protocol, 98% cases improved and 2% expired. Expired cases were those who presented with acute kidney injury.

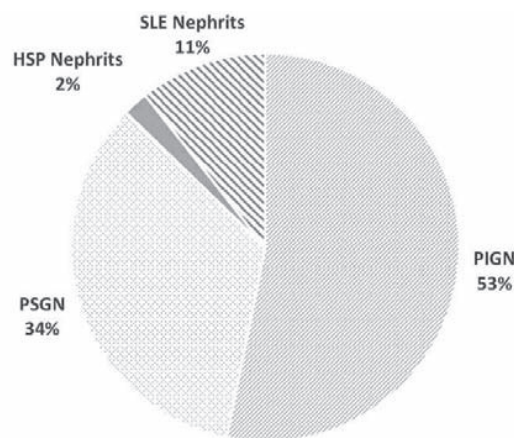


Figure 1 Aetiology acute glomerulonephritis

Table 1: Clinical Profile of Glomerulonephritis (N=94)

Characteristics	Frequency	Percentage
Common Age Group (5-15) yrs	87	91.5%
Mean Age = 9.29 yr \pm 3.13 SD	Range (3-15yrs)	
Sex		
Male	45	47.87 %
Female	49	52.12 %
Hypertension (>95 th Centile)	81	86.17 %
Urine Analysis		
≤ 2 (protein)	136	66.01%
>2+ (protein)	70	33.98%
Hematuria	90	95.74%
Pyuria	32	34.04%
Renal Function Test		
High Urea	45	47.87%
High Creatinine	41	43.61%
Complications at presentation		
Acute Kidney Injury	6	6.38 %
Cardiac failure	9	9.57 %
Encephalopathy	9	9.57 %
Nephrotic range proteinuria	5	5.31 %

Table 2: Signs and symptoms at presentation

S.No.	Symptoms	Number (%)
1	Fever	60 (63.8%)
2	Edema	80(85.1%)
3	Hematuria	39(41.5%)
4	Oliguria	21 (22.3%)
5	Pain abdomen	20 (21.3%)
6	Dysuria	4(4.3%)
7	Sore throat	24(25.5%)
8	Pyoderma	18 (19.1%)
9	Dyspnea	11 (11.7%)
10	Encephalopathy	10 (10.6%)
11	Joints pain	10 (10.6 %)
12	BP (>95 th Centile)	81 (86.2 %)

Discussions

Acute glomerulonephritis is one of the common renal problems encountered in paediatric emergency or outpatient clinic. Glomerulonephritis (GN) typically presents as the sudden onset of hematuria (either gross or microscopic) with or without proteinuria, decreased glomerular filtration rate, and retention of sodium and water, which usually results in an elevated blood pressure and edema. In children, the most common cause of acute AGN is poststreptococcal GN.⁹⁻¹¹ Acute nephritis also has been associated with other infectious agents (PIGN). Other causes of AGN include secondary GN (eg, Henoch-Schönlein purpura [HSP], nephritis associated with subacute bacterial endocarditis, and shunt nephritis). In addition, several causes of chronic GN may present as acute nephritis syndrome and may be initially indistinguishable clinically from acute disorders. These chronic conditions include primary GN (eg, IgA nephropathy and membranoproliferative GN [MPGN]) and secondary GN (e.g., lupus nephritis).

In the developed countries Acute post infectious (most often post streptococcal) GN has almost been wiped out but in Asia including Nepal, it still accounts for a large number of cases.¹² Group A streptococcal diseases are more common in children than in adults with diseases ranging from pharyngitis and impetigo to invasive infections and the post-streptococcal sequelae: acute rheumatic fever and acute post-streptococcal glomerulonephritis.¹³ We found PIGN in 84.0%, of which about 40% of the cases were PSGN, 4% were mumps and remaining infectious aetiology were probably unknown viral aetiology. Lupus nephritis and HSP nephritis were 10.6% and 2.1% respectively. Similarly in a study by A.Y. Elzouki et al, in Eastern Libya, the major renal diseases were post-streptococcal glomerulonephritis (116 of 343 renal cases over a study period of 2 yrs).¹⁴

In a study by Zhongguo 15.8% as HSP nephritis, and 7.3% has hepatitis B virus-associated nephritis.¹⁵ We did not find any nephritis associated with hepatitis in our study. In our study almost equal proportion of female and male were affected (1.08:1), however in a study by Anochie I¹⁶ male were more with male to female ratio of (1.1:1), the proportion of male was still higher in a study by Malla K¹⁷ with the ratio 1.6:1. The reasons for this gender variations are not known. As in other studies,¹⁸ most children were school going age group.

Common modes of presentation of AGN were gross and microscopic hematuria (96%), pyoderma (19%), encephalopathy (11%), hypertension (86%), oedema (85%) and oliguria (22%). 30% of AGN presented with complications commonest being hypertensive encephalopathy (10%), followed by CCF (9%) and AKI (6%). Ocheke IE¹⁹ documents hypertension (92.3%) as commonest presentation followed by oliguria (88.5%), Edema (84.6%), Hypertensive encephalopathy (23.1%)

and CCF(15%). The findings suggest that modes of presentation though similar, our study showed relatively lower complications rate at presentation. UTI was present in 5.3%, the culture isolates were E.coli 1%, streptococcus 2.1% and acinetobacter 2.1%. In the study by Malla K⁸ 17% has associated UTI. AGN complicated by UTI was also observed in 20% cases in a study in Nigeria.²⁰

Anochie I¹⁶ found UTI in 22.6% patients; klebsiella being the commonest organism isolated. We thus have less number of UTI complicating AGN.

In our study almost half of the cases had renal impairment, elevated urea and creatinine in 47.8% and 43.6% respectively. Malla K¹⁷ noted that impairment of urea was seen in 56% and impairment of creatinine in 41%, which was almost similar to our study. Renal impairment observed in another study was 33.3% and 5.5% respectively.²¹ ASO titre was raised in 34.0%. In other studies rise in the ASO titre was observed up to 50%.^{22,23}

In our study all the cases of AGN was treated symptomatically and all the available supportive measures were provided. Majority of the cases 98% improved, the results are almost similar in comparison to other studies.^{16,17}

The cases that expired possibly developed crescentic glomerulonephritis. Poor investigations facilities and laboratory and pathology back up are major constraints in developing countries for further workup. Hence the confirmatory diagnosis could not be made in these cases. However a good outcome from our study suggests that early admission and meticulous treatment in majority of the cases minimizes the fatal outcome.

Conclusion

In conclusion, AGN is a common renal disease requiring hospital admission in school going children in eastern region of Nepal. They often have atypical presentations, hence early diagnosis and prompt management are required for better outcome. Adverse outcomes can be annulled with more detailed laboratory and diagnostic methods which are limited at present. Renal biopsy in complicated cases not improving by supportive measures is mandatory, especially in allowing rational use of corticosteroids and other immunosuppressive drugs. Lack of specialised centres and financial deprivation are a major drawbacks in a developing country settings. Nevertheless, as majority of cases improves with symptomatic treatment and careful supportive care, it is justified to give the benefit of treatment to these patients before considering advanced diagnostic procedures.

Conflict of Interest: None declared

References

1. Chadban SJ, Atkins RC. Glomerulonephritis. *Lancet* 2005; 365:1797.
2. Couser WG. Pathogenesis of glomerulonephritis. *Kidney IntSuppl* 1993; 42:S19.
3. Carapetis JR, Steer AC, Mulholland EK, Weber M. The global burden of group A streptococcal diseases. *Lancet Infect Dis* 2005; 5:685.
4. Rodriguez-Iturbe B, Musser JM. The current state of poststreptococcal glomerulonephritis. *J Am SocNephrol* 2008; 19:1855.
5. Blyth CC, Robertson PW, Rosenberg AR. Post-streptococcal glomerulonephritis in Sydney: a 16-year retrospective review. *J Paediatr Child Health* 2007; 43:446.
6. Kliegman RM, Stanton BF, St. Geme III JW, Schor NF, Behrman RE. Nephrology. Chapter XXIII. In: Nelson Textbook of Pediatrics. 19th ed. Philadelphia: Elsevier Saunders; 2011: 1778-1864
7. Mishra OP, Prasad R, Singh UK. Disorders of kidney and urinary tract. In: Text Book of Pediatrics, Ed Gupta P, CBS Publishers, New Delhi; 2013;422-54
8. KDIGO Clinical Practice Guideline for Acute Kidney Injury: Definition and classification of AKI. *Kidney Int (Suppl)* 2012; 2: 19-36
9. Rodriguez-Iturbe B. Postinfectious glomerulonephritis. *Am J Kidney Dis* 2000; 35: XLVI.
10. 4. Zhang Y, Shen Y, Feld LG, Stapleton FB. Changing pattern of glomerular disease at Beijing Children's Hospital. *ClinPediatr (Phila)* 1994; 33:542.
11. Sanjad S, Tolaymat A, Whitworth J, Levin S. Acute glomerulonephritis in children: a review of 153 cases. *South Med J* 1977; 70:1202.
12. Cattran DC. Evidence-Based Recommendations For The Management Of Glomerulonephritis. Introduction. *Kidney IntSuppl* 1999 Jun; 70: S1-2.
13. Steer AC, Danchin MH, Carapetis JR. Group A streptococcal infections in children. *J Paediatr Child Health* 2007; 43:203-213.
14. A.Y. Elzouki, F. Amin, and O.P. Jaiswal. Prevalence and pattern of renal disease in eastern Libya. *Archives of Disease in Childhood*. 1983;(58): 106-109.

15. Zhongguo Dang Dai ErKeZaZhi. Clinicopathologic Characteristics Of 1,316 Children With Renal Disease 2007 Apr;9(2):117-21.
16. Anochie I, Efe K, Okpere A. Childhood acute glomerulonephritis in Port Harcourt, River State, Nigeria. Niger J Med. 2009;18(2):162-7.
17. Malla K, Sharma MS, Malla T, Thaplial A. Varied presentation of Acute Glomerulonephritis in Children Single centre experience from a developing country. Sultan QabosUniver Med. J.2008; 8(2):193-9
18. Ali MS, Rahman S, Siddiqui NI, Talukder SI, UddinMM, Sofiullah M, et al. Studies on clinical pattern ofglomerulonephritis. Mymensingh Med J 2004; 13:33-35.
19. Ocheke IE, Okolo SN, Thomas FB, Agaba EI. Pattern of Childhood Renal Diseases in Jos, Nigeria: A Preliminary Report. Journal of Medicine in the Tropics. 2010; 12: 52-55
20. Michael IO, Gabriel OE Pattern of renal diseases in childrenin Midwestern zone of Nigeria. Saudi J Kidney DisTranspl 2003; 14:539-544.
21. Mitwalli AH, Al Wakeel JS, Al Mohaya SS, Malik HG, Abu-Aisha H, Hassan OS, et al. Pattern of glomerular disease in Saudi Arabia. Am J Kidney Dis 1996; 27:797-802.
22. Travis LB: Acute postinfectious glomerulonephritis. In: Rudolph AM, Hoffman JI, Rudolph CD, eds. Rudolph's Pediatrics. Stamford, CT: Appleton & Lange, 1996.1356-8.
23. Roy S 3rd, Stapleton FB: Changing perspectives in children hospitalized with poststreptococcal acute glomerulonephritis. PediatrNephrol 1990; 4:585-8.