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A narrative review of acute post-streptococcal glomerulonephritis in Nepali children

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Abstract

Background Acute post-streptococcal glomerulonephritis (APSGN) is the primary cause of acute glomerulonephritis in children in Nepal and contributes significantly to paediatric hospitalisations in the country. This review discusses the current status of streptococcal infections, epidemiological trends, and the challenges in diagnosing and managing APSGN in Nepalese children. This study aimed to develop local data on acute post-streptococcal glomerulonephritis to help compare epidemiological trends and patterns with regions where this disease is less prevalent.

Methods A targeted literature review was conducted in PubMed, Google Scholar, and Nepal Journals Online (a local database) to identify relevant literature published between 1 January 2000 and 31 December 2024. Additional searches of conference abstracts and reviews were performed using Google. The collected literature was analysed to determine the kidney disease patterns, current status of Group A Streptococcal infection, epidemiological trends, clinical manifestations, management, and outcomes of APSGN in Nepali children aged < 16 years.

Results Thirty-four articles were selected for in-depth review. A synthesis of local hospital studies revealed significant differences in the application of diagnostic criteria for APSGN owing to the inaccessibility of serological tests and complement testing. Children over five years of age, particularly those aged 8 to 11 years and predominantly male, were more severely affected. The disease was present year-round, with pyoderma identified as the main route of preceding streptococcal infection rather than throat infection, particularly affecting economically disadvantaged children. The classical manifestations were oedema, hypertension, gross haematuria, and oliguria, whereas complications included acute kidney injury, rapidly progressive glomerulonephritis, hypertensive emergency, congestive cardiac failure, and the need for kidney replacement therapy. The anti-streptolysin O titre was positive in 34-72.7% of patients, while complement C3 levels were depressed in 61.9–100% of cases. Urinalysis showed haematuria in 67–100% of patients and pyuria in 7.9–37%. Kidney ultrasonography indicated increased echogenicity in 37–78% of the cases. Most patients were managed conservatively with diuretics and anti-hypertensives. Atypical cases and those with a progressive disease course were further managed with steroids, kidney biopsies, or kidney replacement therapy. Most patients exhibited favourable short-term kidney outcomes. There was low mortality among patients with rapidly progressive glomerulonephritis and those who required kidney replacement therapy.

Conclusions This review highlights that acute post-streptococcal glomerulonephritis remains a common cause of hospitalisation in Nepal. It remains a diagnostic difficulty owing to the inaccessibility of serological and complement

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tests. The disease has distinct clinical manifestations, demographic patterns, histological findings and outcomes in Nepali children.

Keywords Acute post-streptococcal glomerulonephritis (APGSN), Clinical profile, Epidemiology, Nepali children, Outcome

Background

APSGN is a significant health problem and one of the leading causes of hospitalisation and outpatient clinic visits among children in Nepal. This high incidence of APSGN is primarily attributed to the prevalence of streptococcal pyoderma and pharyngitis in Nepali children [1, 2]. However, the lack of comprehensive data on APSGN in paediatric populations from various geographical locations (plains, hills, and mountains), diverse clinical severities (severe, mild, and subclinical cases), and different clinical settings (tertiary or peripheral hospitals) complicates the task of accurately determining the actual prevalence of APSGN in Nepal.

Epidemiological data on APSGN in Nepali children were primarily gathered from hospital records at tertiary care facilities. These centres mainly manage severe and complicated cases of APSGN, meaning that milder cases often do not reach these facilities. Furthermore, there are significant information gaps regarding cases that resolve without medical intervention, frequency of subclinical diseases, predominant routes of preceding infections, and specific serotypes of Streptococcus responsible for APSGN in Nepal. Additionally, many health centres in Nepal do not have access to streptococcal serological tests, complement analysis, and kidney biopsy [3, 4], making it difficult to diagnose APSGN.

This narrative review aimed to analyse hospital-based data on APSGN from different regions of Nepal in order to estimate the epidemiological burden of APSGN in Nepali children. This review also collected information on various clinical manifestations, diagnostic findings, complications, and treatment outcomes of APSGN in different regions. The results of this review can help healthcare professionals manage and understand the different disease patterns among populations in different regions of Nepal.

Methods

Geographical location of the study population

Nepal is a landlocked South Asian country bordered largely by India to the east, south, and west and China to the north [5]. The country is divided into three central geographical regions: the Himalayan, Hilly, and Terai lowlands [5], with distinct topographical features according to attitude (Fig. 1) [6]. The total population of Nepal is 29 million as of November 2021, of which 15 million are females (51.1%) and about 14 million are males (48.9%) [7]. Furthermore, the proportion of the population aged 14 years and younger was 27.8%. The average household size was 4.37 people per household, and the population density (number of people per square kilometre of area) was 198 in 2021 [7]. According to the 2021 census, the literacy rate in Nepal is 76.2% (men: 83.6% and women: 69.4%).

Nepal's healthcare system consists of private and public hospitals, with most people receiving healthcare services in public hospitals. The public health sector emphasises preventive care and curative services and plans the healthcare system. In contrast, the private sector focuses primarily on preventive and curative services. The government implements several public health programs, including financial assistance in kidney replacement services and health insurance, to improve the health of its citizens. Despite recent improvements in the healthcare infrastructure, health status and education status, several challenges persist. These include issues related to the performance of the healthcare system, the shortage of the healthcare workforce, the need for external financing to support healthcare expenditures, and the challenge of maintaining minimum quality standards for care at the point of service delivery [8].

Climate significantly influences human health and well-being as it is linked to air quality, sanitation, and infectious disease outbreaks. The climate of Nepal varies significantly with altitude. The southern lowlands experience a tropical-subtropical climate, whereas the lower mountain ranges or hilly areas have a temperate climate. In contrast, the Himalayas are characterised by a cold climate [5]. Due to these geographical differences, Nepal has five distinct seasons (spring, summer, monsoon, autumn, and winter), unlike many Western countries [9]. The changing climate and temperature during these seasons make Nepalese children more susceptible to recurrent skin and respiratory infections.

Data sources and search strategy

This narrative review employed a targeted literature review methodology to identify relevant studies published in English in both local and international peer-reviewed journals. The databases searched included PubMed, Nepal Journals Online (NepJOL), Google Scholar, Web of Science, Hinari, and various local journals that were not indexed in these databases. Additional searches for conference abstracts and reviews were conducted using Google searches. The keywords used for the search included acute glomerulonephritis

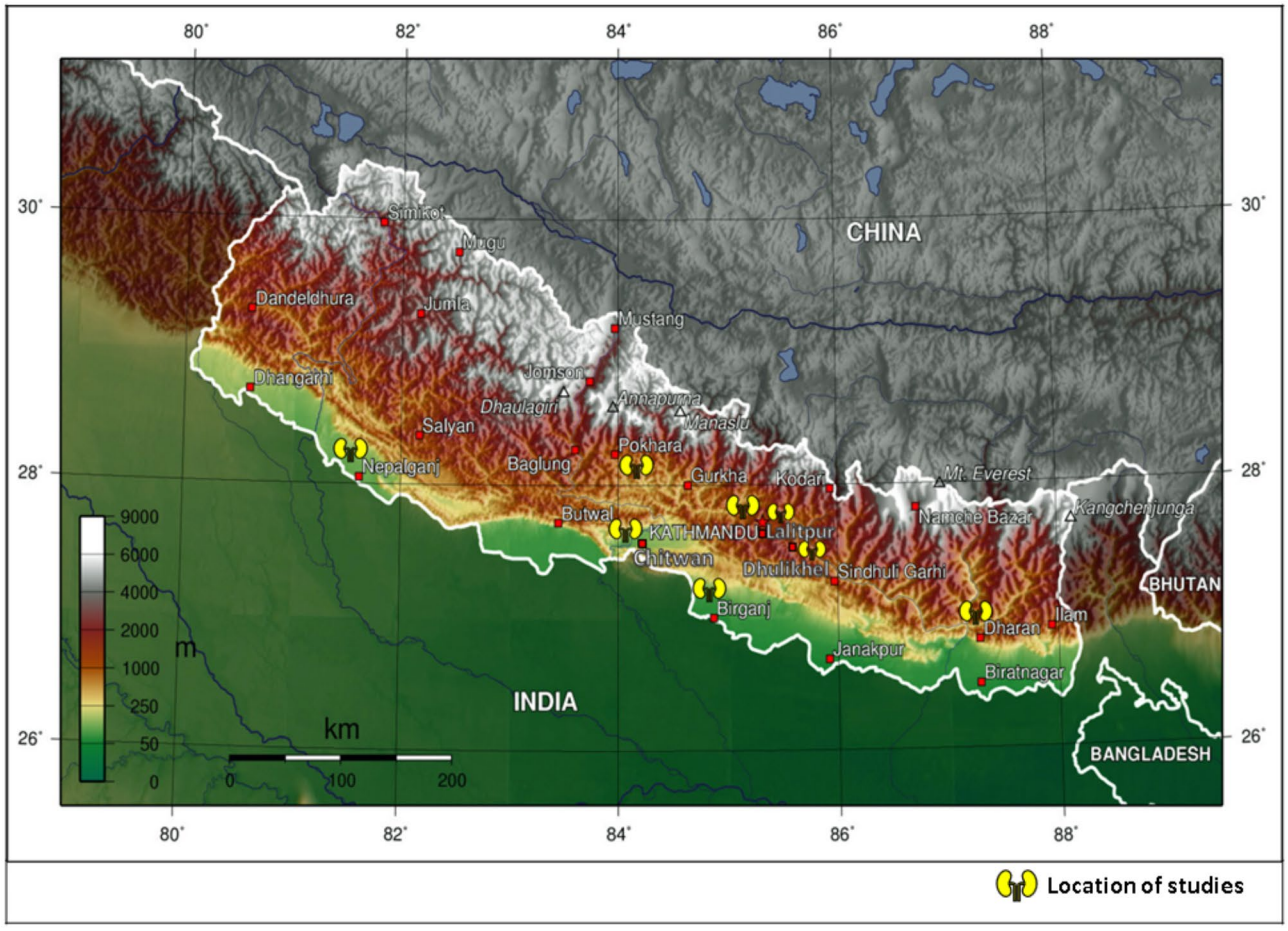

Fig. 1 Topographical map of Nepal (adapted from map available at https://www.mapsland.com/asia/nepal/large-topographical-map-of-nepal)

(AGN), post-streptococcal glomerulonephritis (PSGN), acute post-streptococcal glomerulonephritis (APSGN), post-infectious glomerulonephritis (PIGN), infectious glomerulonephritis, Nepal, and children. All studies conducted between 1 January 2000 and 31 December 2024 on APSGN or PIGN in Nepali children under 16 years of age were included in the review. The literature search was restricted to studies published since 2000 because relevant studies on APSGN before this period could not be found in local databases or printed journals. We excluded studies that focussed on the prevalence of streptococcal skin and throat infections in adults, APSGN in adults, and articles for which the abstract or full text could not be retrieved. All the authors searched the research databases separately and reviewed the studies independently and collaboratively.

Study selection and data extraction

The search was conducted in the databases using keywords; duplicates were removed, and the titles, abstracts, and full texts of the remaining publications were reviewed for a comprehensive analysis. During screening, the inclusion and exclusion criteria focused on the pattern of kidney disease in Nepalese children, the current status of Group A Streptococcal infection, epidemiological trends, clinical manifestations, management, and outcomes of acute post-streptococcal glomerulonephritis in Nepalese children.

The studies included in this review were conducted at tertiary referral facilities across various regions of Nepal. The literature search identified no randomised controlled trials, cohort studies, or case-control studies on APSGN in Nepal. Consequently, this review was based on the analysis of case reports, cross-sectional studies, and descriptive studies on APSGN. The identified studies exhibited significant variability in terms of study type, geographical location, demographic characteristics, and diagnostic definitions of PIGN and APSGN. Most of the studies included in the analysis in this review were from lowland and hilly regions, where the temperature and climate fluctuated throughout the year and skin and throat infections were common. Very few studies have been conducted in mountainous regions. This review did not use the PICOS (Patient/Population, Intervention,

Compare, Outcome and Study Design) framework for formulating eligibility criteria in systematic reviews and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) diagram.

Thirty-four full-text articles were selected for in-depth review following the screening and selection process. The findings of the study are summarised and presented in tabular form to highlight the different study characteristics of APSGN in the paediatric population of Nepal.

Critical appraisal of studies

All publications included in this study were descriptive observational studies. The quality of these studies was assessed by three researchers using the CASP checklist designed for systematic reviews and meta-analyses of observational studies [10].

Results

Data on the incidence of subclinical APSGN, cases that resolve without medical treatment, and cases treated at primary health centres are limited. Furthermore, many patients with infection-related glomerulonephritis were labelled as having PIGN because of the unavailability of diagnostic tests to confirm APSGN and other infections at many centres.

Prevalence of kidney disease in Nepalese children

Kidney diseases, such as acute glomerulonephritis (AGN), nephrotic syndrome, AKI (acute kidney injury), congenital anomalies of kidney and urinary tract (CAKUT), and urinary tract infection (UTI), pose a significant burden in terms of morbidity and mortality among children in Nepal (Table 1). Hospital data show that kidney disease accounted for 6–7% of all annual paediatric hospital admissions between 2002 and 2007, with some centres reporting figures as high as 12% in 2019 [1, 2, 11]. A study conducted in 2024 found that the prevalence of kidney disease was 5.6% [12].

Historically, the prevalence of asymptomatic kidney disease detected through urinalysis screening among seemingly healthy Nepalese schoolchildren was only 0.71% [13]. However, recent studies have reported a prevalence range of 11.1 -12.08%, suggesting an increasing burden of kidney disease in asymptomatic children [14, 15]. In contrast, one study observed a gradual decline in the number of patients attending a nephrology clinic over a ten-year period [16], which differs from the findings of other studies. During this period, the spectrum of kidney diseases shifted from CAKUT to acute glomerulonephritis and nephrotic syndrome [16]. Additionally, the younger age group is primarily affected by CAKUT, while children over the age of five years tend to present with acquired kidney diseases [16].

Current situation of streptococcal infection in Nepali children

A cross-sectional study conducted in 2001 found that 7.2% of children aged between 3 and 10 years had Group A beta-haemolytic Streptococcus (GABHS) pharyngitis [17]. In a separate study involving 487 throat swabs to assess the rate of asymptomatic throat carriage among schoolchildren, Group A Streptococci were isolated from 9.2% of the samples [18]. By 2013, the prevalence of GABHS in throat swabs in asymptomatic preschool children had decreased to 5% [19]. Furthermore, there was an increase in cases of streptococcal pneumonia and APSGN in Nepal following the 2015 earthquake, affecting both the adult and paediatric populations [20].

Incidence of APSGN

Incidence among hospitalised children

Most hospitalisations related to kidney disease are due to AGN, with the incidence rates varying between 3.1% and 37.7% across different geographical regions of Nepal [2, 4, 16, 21]. A 2022 study examining 48 AGN cases identified PIGN as the most common cause (40 cases, 83.3%) [22]. Similarly, another study showed that 73% of acute nephritic syndrome cases were caused by PIGN [23]. However, a recent 2024 study reported that the prevalence of PIGN was 4.05%, the lowest documented rate in various studies [24]. In a study published in 2005 that investigated the causes of AKI in admitted patients between 1998 and 1999, PIGN was an important cause of AKI in children below 13 years of age [25]. PIGN was identified as the most common cause of AGN in most of the studies (Table 1). However, only a few studies have reported the frequency of APSGN in patients with PIGN. One study involving 92 patients diagnosed with AGN revealed that 50% had a positive anti-streptolysin O titre (ASOT), suggesting a high incidence of APSGN [3]. Another study of 94 patients with AGN found that PIGN was the primary cause in 84% of cases, and within that number, 34% were attributed to APSGN [4]. Another study indicated that APSGN accounts for 48% of PIGN cases [26]. In a ten-year review of patients attending a nephrology clinic, AGN (32.7%) was predominantly (94.7%) due to APSGN [16]. In an analysis of patients admitted between 2002 and 2007, APSGN accounted for 28.7% of hospitalisations [1]. Although APSGN (63%) is recognised as the most common type of PIGN [23], other causes, such as mumps, pneumonia, and scrub typhus, have also been documented to cause PIGN [4, 26]. Moreover, studies suggest that the aetiological agents of glomerulonephritis remain unidentified in 30.4–48% of patients, even when an infection is suspected [23, 26].

Table 1 Spectrum of kidney problems in Nepali children

Diseases	Bhatta et al. [1]	Malla et al. [21]	Adhikari et al. [46]	Yadav et al. [2]	Adhikari et al. [47]	Joshi et al. [11]	Kansakar et al. [16]	Agrawal et al. [22]	Gautam et al. [12]
Study year	2002–2007	2000–2007	2014 (6 months)	2012–2013	2015–2016	2017	2008–2018	2014–2015	2022–2023
Population	651	228	48	206	115	607	352	120	63
Glomerular disease	AGN: 46.5% -PIGN: 28.7% -HSP: 4% -LN: 3.7% NS (34.1%) -MCD: 32% -MesPGN:1.2% -FSGS: 0.6% -MPGN: 0.3%	AGN: 30.7% NS: 17.5%	NS: 37.5% APSGN: 25%	AGN: 45.6% IgAN: 0.4% NS: 31.5%	APSGN: 17.3%	AGN: 28.3% NS: 36.4%	AGN: 32.7% -APSGN: 31% -LN: 1.1% -MesPGN: 0.3% - HSP nephritis: 0.3% NS: 23.6%	AGN: 40% IgAN: 0.8% NS: 26.6%	AGN: 15.8% NS: 6.3%
AKI/CKD/HUS	AKI: 3.9% CKD: 4.2% HUS: 10.1%	AKI: 2.2%	NA	AKI: 17.9% CKD: 1.4% HUS: 0.4%	NA	NA	AKI: 0.3% HUS: 0.3%	AKI: 25% CKD: 1.6% HUS: 1.6%	AKI: 6.3% CKD: 1.5%
CAKUT	CAKUT: 7.8% -PUV: 3.4% -VUR: 2.3% -Prune belly syndrome: 0.9%	CAKUT: 6.1%	NA	Obstructive uropathy: 1.9%	NA	NA	CAKUT: 39% -VUR: 28.7% -Hydronephrosis: 4.5% -Renal agenesis: 1.4% -Other: 4.4%	Obstructive uropathy: 1.6%	CAKUT: 3.1%
UTI	3.5%	39.5%	37.5%	21.3%	37.3%	24.8%	1.1%	NA	63.4%
Nephrolithiasis	NA	2.6%	NA	0.4%	NA	NA	1.1%	1.6%	NA
Miscellaneous	Polycystic kidney disease: 0.6% Wilm's tumour:0.6%	Unexplained recurrent haematuria: 1.3%	NA	Polycystic kidney disease: 0.4%	Others (unspecified): 45%	Others(unspecified): 10.4%	Renal tubular acidosis: 1.1% Nocturnal enuresis: 0.6% Voiding dysfunction: 0.3%	Polycystic Kidney disease: 0.8%	Haemorrhagic cystitis: 3.1% Bladder problem: 1.5%

VUR: Vesicoureteral reflux, HUS: Haemolytic uremic syndrome, IgAN: IgA nephropathy, LN: Lupus nephritis, MesPGN: Mesangial proliferative glomerulonephritis, NS: Nephrotic Syndrome

Incidence among asymptomatic children

In a survey of 2,243 healthy schoolchildren, out of 10 patients with kidney problems, five (50%) had GN, which included four cases of lupus nephritis and one case of APSGN [13]. In another recent study, screening of children aged 6–15 years revealed that 96 (11.13%) of 862 children had urinary abnormalities at the first screening [14]. However, only eight children (0.92%) had specific urinary abnormalities after the second screening, including one case of APSGN [14].

Age and sex distribution of APSGN patients

Table 2 shows the age and sex distributions of the patients with APSGN. Most patients were older than five years, with a mean age of 8–12 years. Most of those affected were male (Table 2).

Seasonal variations in APSGN/PIGN

Infectious glomerulonephritis was most common in the autumn, summer, and winter (Table 2). One study noted that kidney diseases were particularly prevalent in July and March, especially during the first six months [12].

Evidence of preceding infections/other infections in APSGN

In Nepal, pyoderma was the most common cause of APSGN, and its prevalence varied from 20 to 45.6% compared to throat infections (10-33.3%) in different studies (Table 3). Other uncommon causes of PIGN included mumps (3.1–4.3%), scrub typhus (2%), and pneumonia (2.2%) [4, 23, 26]. Notably, no specific cause was found in 30.4-62.9% of patients [4, 23, 26].

APSGN by socioeconomic strata and geographical locations

A review of the burden of APSGN and socioeconomic status indicated that 51% of the affected children were from socioeconomically disadvantaged backgrounds [27]. Furthermore, a significant majority of the patients (64.6%) belonged to rural areas [26]. Approximately 50% were from hilly regions, 17.4% from mountainous areas, 17.4% from outer Terai, and 15.2% from inner Terai [23].

Clinical presentation of APSGN and PIGN

The clinical features of APSGN and PIGN showed significant variations across different studies (Table 4). Few

Table 2 Age-sex distribution and seasonal variation in patients with APSGN and PIGN at presentation

Studies	Patient population	Data period	Published year	Mean Age (± SD) in yrs	Age group	Sex (M: F)	Seasons
Malla et al. [3]	92	2000–2007	2008	NA	5–15 yrs: 95.7% < 5 yrs: 4.3%	1.6:1	Not studied
Shah GS et al. [4]	94	2012–2013	2014	9.2 (± 3.13)	5–15 yrs: 91.5% < 5 yrs: 8.5%	0.91:1	Not studied
Poudel et al. [35]	30	6 months (2014)	2014	11.5 (± 3.3)	5–15 yrs: 80% < 5 yrs: 20%	2.3:1	Win: 40% Aut:30% Rest:30%
Shah et al. [27]	41	2013–2016	2017	9.5	5–15 yrs: 97% < 5 yrs: 3%	1.7:1	Not studied
Yadav et al. [36]	74	2017–2018	2020	8.9 (± 3.06)	NA	2.2:1	Not studied
Kansakar et al. [16]	115*	2008–2018	2021	9.8 (± 2.9)	< 5 yrs: 6.1% 5–10 yrs: 34.8% 10–15 yrs: 59.1%	1.8:1	Not studied
Adhikari et al. [26]	48	2018–2021	2022	9.5 (± 3.72)	> 5 yrs: 83.3% < 5 yrs: 16.7%	1.5:1	Aut: 54.2% Sum: 18.8% Win: 16.7% Spr: 10.4%
Agrawal et al. [22]	48*	2014–2015	2022	NA	< 5 yrs: 10.4% 5–10 yrs: 37.5% 10–15 yrs: 52.1%	0.9:1	Not studied
Dhakal et al. [28]	77	2015–2022	2023	10.1 (± 2.8)	< 5 yrs: 5.2% 5–12 yrs: 72.7% > 12 yrs: 22.1%	1.9:1	Not studied
Kalakheti et al. [23]	46	2020–2021	2023	11.2 (± 3.2)	< 5 yrs: 2.2% 5–10 yrs: 26.1% 10–15 yrs: 54.3% > 15 yrs: 17.4%	1.5:1	Aut: 37% Sum: 32.6% Win: 17.4% Spr: 13%
Shrestha et al. [24]	63	2020–2023	2024	9 (± 3.4)	< 5 yrs: 19% 6 to 10 yrs: 39.7% 11 to 14 yrs: 41.3%	1.8:1	Not studied

M: Male, F: Female, SD: Standard deviation, Yrs: Years, Aut: Autumn, Win: Winter, Sum: Summer, Spr: Spring, *Only patients with glomerulonephritis

Table 3 Incidence of preceding skin infections and throat infections in different studies

Study author with reference	Study population	Skin infection (%)	Throat infection (%)	Common
Malla et al. [3]	92	45.6	30.4	Skin Infection
Shah GS et al. [4]	94	19.1	25.5	Throat infection
Poudel et al. [35]	30	40	33.3	Skin infection
Shah et al. [27]	41	31.7	19.5	Skin infection
Yadav et al. [36]	74	18.9	32.4	Throat infection
Adhikari et al. [26]	48	12.5	10.4	Skin infection
Dhakal et al. [28]	77	31.2	16.9	Skin infection
Kalakheti et al. [23]	46	37	26	Skin infection
Shrestha et al. [24]	63	30.1	14.2	Throat infection
Gautam et al. [12]	10*	20	10	Skin infection

*Only acute glomerulonephritis patients were included out of total patients

studies have focused exclusively on APSGN, as most have generally focused on PIGN or AGN. Most children commonly had clinical signs and symptoms of acute glomerulonephritis. Typical findings were hypertension (87.5%), haematuria (85.4%), AKI (39.28%), and mild to moderate proteinuria > 2+ (39.5%) [22]. However, one study found that 64.1% of patients presented to healthcare facilities with atypical symptoms and signs involving multiple systems [3]. Very few patients also had uncommon symptoms at the presentation, such as malena in 1% [3], haemoptysis in 1.3% [28], epistaxis in 1.3% [28] to 2.1% [3], upper respiratory tract infection in 2.6% [28], diarrhoea in 3.2% [3], and blurring of vision in 8.7% [23].

Approximately 14.6–42% of patients present to the hospital with APSGN-related complications [24, 26–28]. The most common complications included hypertensive encephalopathy/emergency, congestive heart failure, pulmonary oedema, acute kidney injury, and rapidly progressive glomerulonephritis (Table 4). Children sometimes present with unusual symptoms of posterior reversible encephalopathy syndrome [29]. Furthermore, PIGN has been identified as a significant cause of acute

Table 4 Clinical features and complications of APSGN and PIGN at presentation

Studies with references	Malla et al. [3]	Shah GS et al. [4]	Poudel et al. [35]	Shah et al. [27]	Yadav et al. [36]	Adhikari et al. [26]	Dhakal et al. [28]	Kalakheti et al. [23]	Shrestha et al. [24]
Number of cases (n)	92	94	30	41	74	48	77	46	63
Classical manifestations									
Oedema (%)	100	85.1	100	100	59.5	81.3	93.5	100	71.4
HTN (%)	86.9	86.2	93.3	100	100	72.9	87.0	89.2	65
Gross haematuria (%)	100	41.5	46.6	46.3	94.6	25	67.5	58.7	57.1
Oliguria (%)	54.3	22.3	56.6	51.2	14.9	6.3	54.5	63	19
Complications									
AKI/elevated creatinine (%)	41[$]	43.6	50	31.7	4.1	6.3	58.4	41.3	3.1
RPGN (%)	5.4[$]	NA	10	NA	NA	NA	6.5	2.2	1.5
Hypertensive Emergency (%)	11.9[$]	9.5	3	9.7	6.8	6.3	11.7	6.5	7.9
CCF (%)	10.8[$]	9.5	17	17	12.2	2.1	23.4	17.4	11.1
Miscellaneous									
Fever (%)	65.2	63.8	36.7	80.4	NA	25	14.3	21.7	39.6
Pain abdomen (%)	33.6	21.3			NA	4.2	16.9	2.2	44.4
Epistaxis (%)	2.1	NA			NA	NA	1.3	NA	NA
Shortness of breath (%)	19.5	11.7			12.2	29.2	25.9	47.9	14.2
Seizures (%)	22.8	NA			6.8	2.1	1.3	4.3	7.9
Headache (%)	63	NA	36.6	19.5	13.5	2.1	2.6	23.9	6.3
Altered sensorium (%)	22.8	10.6	NA	NA	NA	NA	NA	2.2	NA
Vomiting (%)	35.8	NA	NA	NA	13.5	NA	24.7	17.4	NA
Palpitations (%)	NA	NA	NA	NA	12.2	NA	NA	23.9	NA
Cough (%)	15.2	NA	NA	NA	NA	22.9	5.1	2.2	41.2
Joint pain (%)	2.1	10.6	NA	NA	NA	NA	Nil	NA	1.5
Rashes (%)*	2.1	NA	NA	NA	NA	NA	1.3	NA	6.3

NA: Not documented/Data not available, HTN: Hypertension, RPGN: Rapidly progressive glomerulonephritis, CCF: Congestive cardiac failure, KRT: Kidney replacement therapy, [$]recalculated and expressed as a percentage of total patients, [*]Rashes other than healing pyoderma

kidney injury in patients requiring admission to paediatric intensive care [30]. Dyselectrolytemia was a common problem in patients with APSGN and PIGN, and both hyponatremia and hypernatremia were frequently reported (Table 5).

A striking feature of these studies was the high incidence of proteinuria at the time of presentation; however, very few studies have conducted longitudinal follow-ups in these patients. In one study, all 48 patients exhibited proteinuria, with 60.41% showing up to 2 + protein, 39.5% more than 2 + protein, and 8.3% presenting with nephrotic-range proteinuria [22]. The different biochemical, urinalysis, and kidney ultrasonography findings in the APSGN and PIGN groups are shown in Tables 5 and 6.

Serological evidence of Streptococcal infection

ASOT was positive in 34-72.7% of the patients with APSGN or PIGN (Table 6). A recent study found positive anti-DNase B and ASOT results in 86.9% and 72.7% of patients, respectively [28]. In the above study, streptococcal throat infections were mainly associated with positive ASOT, while skin infections were associated with positive anti-DNase B titres [28]. A patient who presented with PIGN features and positive anti-nuclear antibodies (ANA) was diagnosed with lupus nephritis after a kidney biopsy [27]. Similarly, another PIGN patient with positive ANA and nephrotic-range proteinuria underwent a kidney biopsy that revealed features consistent with PIGN [24]. Few studies have examined concurrent throat swab cultures for the detection of streptococcal throat infections at presentation. One study found that half (50%) of six PIGN patients with throat swabs tested positive for streptococcal infection at presentation [24].

Histopathological finding

In a study of patients who underwent kidney biopsies for various indications of glomerular disease, PIGN was identified as a common condition, with 18 of 97 patients (18.6%) diagnosed with PIGN [31]. In Nepalese studies, kidney biopsies were performed in complex cases of APSGN/PIGN with rapid deterioration of kidney function or delayed resolution of clinical or laboratory parameters, diagnostic dilemmas and positive ANA status. Recent studies indicate that approximately 7.9–15.6% of patients require kidney biopsy to confirm their diagnosis or investigate persistent clinical symptoms to rule out other conditions [28, 32]. These recent findings are further supported by additional studies indicating that only a small number of patients with APSGN or PIGN require kidney biopsy (Table 7). In most studies, diffuse proliferative glomerulonephritis (DPGN) was the characteristic histopathological feature of APSGN [33]. Among the 12 children who underwent kidney biopsy

Table 5 Laboratory findings in APSGN and PIGN at presentation: haematology, albumin, cholesterol, electrolyte and kidney ultrasound

Studies	Number of cases	Haematology	Serum Biochemistry	Serum electrolytes (Na, K)	Kidney Ultrasonography (USG)
Malla et al. [3]	92	NA	Albumin (< 2.5gm/dl): 17.4% Cholesterol (> 200 mg/dl): NA	Sodium level: NA Potassium level: NA	USG: Normal: 22% Increased echogenicity: 78%
Shah GS et al. [4]	94	Anaemia: 91.4%	Albumin: NA Cholesterol: NA	Hyponatremia: 4.2% Hypernatremia: 12.7% Hyperkalaemia: 10.6%	NA
Poudel et al. [35]	30	NA	Albumin: NA Cholesterol: NA	Sodium level: Normal Potassium level: Normal	USG ($n = 5$) Normal: 100%
Shah et al. [27]	41	NA	Albumin: NA Cholesterol: NA	Dyselectrolytemia (Na^+, K^+): 17.1%	USG ($n = 27$) Normal: 63% Increased echogenicity: 37%
Yadav et al. [36]	74	Mean Hb: 10.7 gm/dl	Albumin: NA Cholesterol: NA	Dyselectrolytemia: 19%	NA
Adhikari et al. [26]	48	NA	Albumin: NA Cholesterol: NA	Sodium level: NA Potassium level: NA	NA
Dhakal et al. [28]	77	NA	Albumin (< 2.5gm/dl): 9.1% Cholesterol (> 200 mg/dl): 15.6% Hypoalbuminemia + NRP: 6.5%	Hyponatremia: 9.1% Potassium level: Normal	USG ($n = 77$) Normal: 54.6% Increased echogenicity: 41.5% Increased echogenicity and Loss of CMD: 3.9%
Kalakheti et al. [23]	46	NA	Albumin: NA Cholesterol: NA	Sodium level: NA Hyperkalaemia: 32.6%	NA
Shrestha et al. [24]	63	NA	Albumin: NA Cholesterol: NA	Sodium level: Normal Potassium level: Normal	NA

Hb: Haemoglobin, NRP: Nephrotic range proteinuria

Table 6 Laboratory findings in APSGN and PIGN at presentation: urinary findings, serum complements, serological evidence of Streptococcal infection and ANA status

Studies	Number of cases	Urinary findings	Serum Complement level	Serological evidence of strep infection	ANA status
Malla K et al. [3]	92	Haematuria: 100% NRP: 16.9% RBC casts: NA Pyuria: 15.2%	C3 level: NA C4 level: NA	Positive ASOT: 50% Positive Anti-DNase B: NA	NA
Shah GS et al. [4]	94	Haematuria: 95.7% NRP: 5.3% RBC casts: 0% Pyuria: 34%	C3 level: NA C4 level: NA	Positive ASOT: 34% Positive Anti-DNase B: NA	NA
Poudel et al. [35]	30	Haematuria: 67% NRP: 6.7% RBC casts: NA	Low C3: 97% C4 level: NA	Positive ASOT: 60% Positive Anti-DNase B: NA	ANA ($n=9$) Normal = 9 (100%)
Shah et al. [27]	41	Haematuria: 90.2% NRP: 17% RBC cats: NA Granular casts: 24.3%	Low C3: 85.3% C4 level: NA	Positive ASOT: 65.8% Positive Anti-DNase B: NA	ANA ($n=12$) Normal: 11 (91.6%) Positive:1 (8.3%)*
Yadav et al. [36]	74	Haematuria: 94.6% Sub-NRP: 52% RBC casts: NA Pyuria: 16%	Low C3: 100% C4 level: NA	Positive ASOT: NA^{+} Positive Anti-DNase B: NA	NA
Adhikari et al. [26]	48	Haematuria: 81.2% Proteinuria:62.5% RBC casts: NA	Low C3: 72.9% C4 level: NA	Positive ASOT: 45.8% Positive Anti-DNase B: NA	NA
Dhakal et al. [28]	77	Haematuria: 100% NRP: 27.3% Sub-NRP: 20.8% Pyuria: 35% RBC casts: NA	Low C3: 96.1% Low C4: 7.8%	Positive ASOT: 72.7% Positive Anti-DNase B: 86.9%	ANA ($n=5$) Normal = 5 (100%)
Kalakheti et al. [23]	46	Haematuria: 78.2% Proteinuria: 67.3% NRP: 10.9% Pyuria: 37% RBC casts: NA	Low C3: 95.7% C4 level: NA	Positive ASOT: 50% Positive Anti-DNase B: NA	NA
Shrestha et al. [24]	63	Haematuria: 93.6% NRP: 3.1% Sub-NRP: 28.5% Pyuria: 7.9% RBC casts: NA	Low C3: 61.9% C4 level: NA	Positive ASOT: 54% Positive Anti-DNase B: Done in one patient, which was positive	ANA ($n=9$) Normal: 8 (88.9%) Positive: 1 (11.1%)$^{\#}$

NRP: Nephrotic range proteinuria, CXR: chest X-ray, *Lupus nephritis, $^{+}$Expressed as mean ASO titre (395 IU/ml), $^{\#}$DPGN

for proteinuria due to APSGN, 10 had features of DPGN [34]. These findings highlighted that DPGN was the most prevalent histopathological finding in the clinical context of APSGN in Nepali children, although other histopathological findings may be infrequently present (Table 7). The most common indications for kidney biopsy in cases of APSGN/PIGN included rapidly progressive glomerulonephritis [35], persistent nephrotic-range proteinuria, hypertension, and impaired renal function [28].

In a study of 97 paediatric kidney biopsies, 10 (9.7%) children underwent biopsy for rapidly progressive glomerulonephritis, with post-infectious glomerulonephritis accounting for half of these cases (five patients) [31]. All cases of rapidly progressive glomerulonephritis due to PIGN showed crescentic glomerulonephritis in kidney biopsies [35]. However, another study found that only one in three patients with PIGN with rapidly progressive

Table 7 Treatment pattern, frequency and findings of kidney biopsy and outcomes of children with PIGN and PSGN

Studies with ref	Study population/ Number(*N*)	Medications	Immunosuppressants	KRT: HD/PD	Kidney biopsy-Indications	Kidney biopsy (frequency/ biopsy findings)	Duration of hospital stay	Mortality	Outcomes
Malla et al. [3]	AGN *N* = 92	Antibiotics: NA Diuretics: NA Anti-HTN: NA	NA	HD: 5 (5.4%)	RPGN course	3 (5%) • CresGN = 3	NA	2 (2.1%)	Persistent AKI (15.2%) on follow up
Shah GS et al. [4]	AGN *N* = 94	Antibiotics: NA Diuretics: NA Anti-HTN: NA	NA	None	NA	NA	NA	2 (2%)	*Complete recovery (98%)
Poudel et al. [35]	PIGN *N* = 30	Antibiotics: NA Diuretics: 97% Anti-HTN: 77% Both diuretics and anti-HTN: 77%	Steroid: 3 (10%)	HD: 1 (3%)	RPGN course	3 (10%) • CresGN = 3	Mean (SD) 7.86 + 3.84 days (range: 3–15 days)	None	NA
Shah et al. [27]	PIGN *N* = 41	Antibiotics: NA Diuretics: 24% Anti-HTN: 3% Both diuretics and anti-HTN: 73%	NA	HD: 1 (2.4%)	Not documented	2 (5%) • DPGN = 1 • Lupus = 1	Mean: 7.5 days (range: 2–38 days)	None	NA
Yadav et al. [36]	APSGN *N* = 74	Antibiotics: NA Diuretics: NA Anti-HTN: NA	NA	None	No biopsy	NA	Mean (SD) 3.38 days (+/- 0.753 days)	None	*Complete recovery (100%)
Adhikari et al. [26]	AGN *N* = 48	Antibiotics: NA Diuretics: NA Anti-HTN: NA	NA	None	No biopsy	NA	NA	None	*Complete recovery (100%)
Dhakal et al. [28]	APSGN *N* = 77	Antibiotics: NA Diuretics: NA Anti-HTN: NA	Steroid: 9 (11.5%)	HD: 1 (1.3%)	NS: 1 RPGN: 1 PGH: 1 RPGN + NRP: 4 Persistent NRP: 4 PGH + NRP + HTN: 1	12 (15.6%) • DPGN = 7 • MCPG = 3 • Increase mesangial cellularity = 2	NA	None	At 3 months, 6.5% of patients had persistent hypertension, AKI, and proteinuria in isolation or combination
Kalakheti et al. [23]	PIGN *N* = 46	Antibiotics: NA Diuretics: NA Anti-HTN: NA	Cyclophosphamide: 1 (2.2%)	None	RPGN course	1 (2.2%) • CresGN	NA	None	*Complete recovery (85%) 8 weeks: haematuria (10.9%), proteinuria (8.3%), AKI (4.4%)
Shrestha et al. [24]	PIGN (*N* = 63)	Antibiotics: NA Diuretics: NA Anti-HTN: NA	NA	None	NRP and positive ANA	1 (1.5%) • DPGN	NA	None	*Complete recovery (100%)

NA- Not available /Not documented in the study, *At discharge, HTN: Hypertensive, CresGN: Crescentic GN, MCPG: Mesangial endocapillary proliferative glomerulonephritis, DPGN: Diffuse proliferative glomerulonephritis, KRT: Kidney replacement therapy, HD: Haemodialysis, PD: Peritoneal dialysis, PGH: Persistent gross haematuria, NRP: Nephrotic range proteinuria, NS: Nephrotic syndrome

glomerulonephritis had crescentic glomerulonephritis, while the remaining patients had DPGN [33].

Treatment pattern of APSGN

Medical management (supportive care, diuretics, and anti-hypertensives) was the standard of care, and only a small percentage of patients needed kidney replacement therapy. There was no documentation of antibiotic use in any of the studies. Immunosuppressive medications (primarily corticosteroids and one patient received cyclophosphamide) were required in only a few patients (Table 7). In one study, seven of 50 patients (14%) with suspected PIGN required immunosuppressive therapy for various indications at presentation. These patients showed diffuse proliferative glomerulonephritis (DPGN) without crescents on kidney biopsy, and steroid therapy was stopped [33]. Other studies have indicated that 10-11.6% of patients required steroids (Table 7). Patients with different grades of disease severity were mainly managed in general wards rather than intensive care units [23, 36].

Outcomes of APSGN and PIGN

The mean length of hospital stay was between 4 and 8 days, ranging from two to 15 days, although some patients were hospitalised for up to 38 days (Table 7). Many studies have reported rapid resolution of proteinuria within weeks, and only 3.9% had persistent nephrotic-range proteinuria after three months of follow-up [28]. However, the limited long-term follow-up beyond three months in most studies makes it difficult to predict the overall resolution rate of proteinuria in these patients. Other abnormalities observed during a two-month follow-up included microscopic haematuria, persistent proteinuria, and elevated creatinine levels, seen in 10.9%, 8.3%, and 4.4% of cases, respectively [23]. At the three-month follow-up, 6.5% of patients continued to have persistent proteinuria, hypertension, and impaired renal function, either alone or in combination [28]. Approximately 1.3 to 17.8% of patients required kidney replacement therapy at presentation or within three months of illness. Studies have demonstrated that most patients have favourable kidney outcomes, experience minimal complications, and have low mortality rates in the short term (Table 7). No studies have followed these patients for long-term complications, such as hypertension, microscopic haematuria, and CKD, over three months of illness. None of the studies examined recurrence or its occurrence in other siblings.

Discussion

The review identified AGN as one of the leading causes of hospitalisation among children in Nepal. In the Nepalese context, the primary cause of AGN is post-infectious glomerulonephritis, with APSGN being the predominant cause. APSGN is typically linked to skin infections and can occur in any season. Most APSGN children are admitted to hospitals with the characteristic symptoms of acute glomerulonephritis, and only a minority of these patients develop complications. The review concluded that these children are usually treated conservatively and have favourable short-term renal outcomes.

APSGN is the leading cause of acute nephritis globally, with most cases occurring in developing countries [37]. The exact prevalence of APSGN in Nepal remains unknown. In developed countries such as the USA, the prevalence is 0.13, and the incidence is 0.78, while in similar environments in neighbouring countries in India, the prevalence is 10.14, and the incidence is 39.24 per 100,000 person-years [37]. APSGN is particularly prevalent in regions where streptococcal infections are common due to poor hygiene, malnutrition, and overcrowding [37]. GABHS sometimes causes community outbreaks and epidemics in natural disasters, resulting in high rates of throat and skin infections, with consequences such as rheumatic fever, APSGN, and other invasive infections. The situation was similar in Nepal following the 2015 earthquake, when an increased incidence of streptococcal pneumonia and APSGN was reported in many regions [20]. Furthermore, pharyngitis (5-7.2%) [17, 19], asymptomatic carrier conditions (9.2%) [18], and skin infections caused by GABHS are common among school-aged Nepali children, similar to other regions of the world [38]. Additionally, previous research from Nepal indicated that invasive pneumococcal disease rarely presents as glomerulonephritis [39]. Therefore, Streptococcal infection remains a significant public health problem in Nepal and many developing countries because of the aforementioned factors [37]. Patients with medical conditions that weaken the immune system, such as chronic conditions or immunosuppressants, are also susceptible to GABHS infections [37]. Recently, the role of genetic factors in increasing susceptibility to APSGN after GABHS infection has been studied, although no specific gene has been identified [40]. APSGN was found to be present in 12–25% of glomerular problems among young, asymptomatic school-going children in Nepal during urine screenings [13, 14]. Additionally, of the 83 patients who experienced glomerular haematuria, 36.3% were diagnosed with APSGN, the most common glomerular cause worldwide [41]. These findings suggest that subclinical APSGN is prevalent not only among Nepalese children but also among children from other countries.

The high prevalence of streptococcal skin infections in Nepal's lower hilly and Terai regions can be attributed to the subtropical climate and climate variability, predisposing the population to recurrent skin infections. The year-round occurrence of APSGN in Nepal is likely related

to the high prevalence of throat and skin infections throughout the year. This review found that skin infections were the most common preceding infections in children with APSGN. We observed a variable yet higher prevalence of pyoderma, ranging from 12.5 to 45.6%, compared to sore throats, which occurred in 10–33.3% of cases. At the same time, global studies indicate that urban or rural environments significantly impact the most common routes of preceding infections [37]. Clinical cases of APSGN were frequently observed in Nepali patients during autumn, summer, and winter. This trend aligns with the increased incidence of throat and skin infections during these seasons. Similar results have been reported in other studies, showing a high prevalence of skin infections and cases of APSGN occurring in fall, with the lowest incidence noted in spring [42].

Regarding diagnosing the preceding streptococcal infection, ASOT is elevated in streptococcal pharyngitis, while anti-DNase B levels are elevated after skin infections. Therefore, in addition to routine measurement of ASOT levels in patients with APSGN, it is essential to assess anti-DNase B levels as part of standard care, especially in regions where skin infections are common [42]. This approach is crucial in Nepal, where skin infections are common in children. However, anti-DNAse B is not routinely available in all healthcare centres in Nepal [28].

Children with APSGN display a range of clinical symptoms. These included nonspecific signs such as malaise, lethargy, abdominal pain, and fever, as well as more severe conditions such as respiratory distress, congestive cardiac failure features, hypertensive encephalopathy, and AKI that necessitated kidney replacement therapy. These findings are consistent with those of the previous studies conducted by Alhamoud et al. [37]. This review highlighted that the clinical characteristics and complication rates of APSGN observed in our study were similar to those reported in other regions worldwide [37].

The incidence of APSGN is slowly decreasing, while that of staphylococcal-associated glomerulonephritis is increasing in Western countries [43]. A similar trend of decreasing incidence of PIGN has also been observed in Nepal, according to a recent study [24]. Few studies have documented scrubs, mumps, and pneumonia as aetiologies of post-infectious glomerulonephritis [4, 23, 26]. PIGN was often used interchangeably with APSGN because of the lack of diagnostic modalities, such as complement levels, serological testing, and kidney biopsy, which are necessary for evaluating various types of glomerulonephritis and potential causes of PIGN [3, 4]. The inaccessibility of these tests to confirm APSGN diagnosis in many health centres in Nepal is not different from that in many developing countries. Fortunately, there have been improvements in access to kidney services and diagnostic facilities in various health centres across major cities in recent years. This has led to an increase in the number of APSGN and other cases of glomerulonephritis cases being diagnosed. A study observed a decline in the number of children seeking nephrology services at a clinic over a ten-year period [16]. This trend may be due to improved access to kidney services in various healthcare settings rather than an actual decrease in the incidence of kidney diseases.

APSGN is generally a self-limiting disease that requires supportive care, monitoring, and early diagnosis and treatment of complications [37]. Most children with mild APSGN can be treated with diuretics and anti-hypertensive medications at a peripheral hospital or by a general paediatrician [37]. This review further confirms that most patients only needed supportive care, whereas only a few required immunosuppressive therapies and KRT. Patients with a severe AKI and RPGN clinical course require referral to a tertiary hospital for possible intensive care, immunosuppressants such as steroids, and the possibility of kidney biopsy, as highlighted in this review and previous studies from other parts of the world [37, 44]. Patients with atypical presentation and persistent severe clinical manifestations, such as persistent nephrotic range proteinuria, hypertension, and persistent AKI, require kidney biopsy, which generally shows diffuse proliferative glomerulonephritis and helps exclude other causes of glomerulonephritis [37]. IgA nephropathy, Henoch Schonlein purpura nephritis, and lupus nephritis can occasionally mimic APSGN, requiring a high index of suspicion to avoid misdiagnosis [45]. A few patients with a typical picture of APSGN also showed ANA positivity in this review and required kidney biopsy to confirm the diagnosis. The present study also demonstrated that most patients responded to conservative treatment with short hospital stays and favourable outcomes [37, 44]. This review also highlighted the low mortality and excellent short-term outcomes among children with APSGN, as shown by previous literature showing excellent short-and long-term prognoses. AKI requiring KRT and crescentic glomerulonephritis had poor long-term outcomes, and long-term follow-up is mandatory in patients with APSGN [37].

Limitations of the study

Several challenges were encountered during this review. These include varying definitions of APSGN, PIGN, and AKI. Additionally, the interchangeable use of APSGN and PIGN, small study populations, limited diagnostic facilities, and research conducted across different centres in various time periods contributed to considerable variability in all aspects of APSGN.

The studies included in this review on APSGN lacked a longitudinal follow-up, specifically on the resolution of proteinuria, microscopic haematuria, hypertension, and

kidney function (serum creatinine/eGFR). As a result, it is challenging to ascertain the long-term prognosis of APSGN in Nepali children and compare our findings with other regional data. Additionally, as this review was based on studies conducted in tertiary referral hospitals, the patient profiles in these studies may not accurately reflect the disease profiles of patients in the community.

Strengths of the study

This review identified various clinical features of APSGN in high-prevalence areas and highlighted trends in the epidemiology of streptococcal infections and APSGN. This review outlined existing gaps in APSGN research and highlighted areas for future research in Nepali children.

Conclusions

APSGN is a common paediatric problem and an important cause of acute glomerulonephritis and has profound public health concerns in developing countries such as Nepal. Long-term kidney outcomes are generally good except in patients with RPGN and those requiring KRT. Improving hygiene and sanitation, reducing overcrowding, and promptly diagnosing and treating pyoderma and sore throat should be strategies for reducing the incidence of APSGN in Nepal.

Recommendations

The uniform standard protocols for diagnosis, treatment, and follow-up should be implemented in all healthcare facilities providing paediatric services in Nepal. These standard protocols will facilitate the collection of robust data, help in comparison among studies and further provide a better understanding of epidemiological trends. Future studies should also document mild cases treated in peripheral hospitals and investigate the incidence of subclinical diseases. Furthermore, an electronic data records system or the establishment of a kidney disease registry will facilitate further documentation.

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