Review Article

Glomerulonephritis among Saudi Children: Current Status Evaluation

Shehab Ahmed Alenazi[1]

ABSTRACT

Glomerulonephritis (GN) is a common renal problem affecting children and is manifested by hematuria, proteinuria, fluid retention, and hypertension with altered glomerular filtration. This condition may be primary or developed secondary to other systemic diseases, immune disorders, infections, or drugs induced with different histopathological patterns and a wide range of clinical manifestations. The current study helps record and comprehend GN's epidemiology in Saudi Arabia. Additional research is recommended that includes a more thorough examination of the prognostic factors, therapies, illness progression, and outcomes. While there are several advantages to having a GN registry that collects data in the future, resource constraints frequently prevent the establishment of comprehensive registries. Expanding research endeavors will play a significant role in fostering a healthy society and may provide insight into the underlying pathology of fascinating glomerulopathies.

KEYWORDS: Nephropathy, glomerulonephritis, nephritis, lupus nephritis, Saudi Arabia

INTRODUCTION

A diverse set of conditions known as glomerulonephritis (GN) primarily affects the kidney but can impact multiple organ systems. Infections, medications, malignancies, monoclonal gammopathy, or multisystemic diseases can bring it on[1]. It is mainly presented by haematuria, proteinuria, fluid retention, hypertension, and a decrease in the glomerular filtration rate (GFR), some of the symptoms that indicate glomerular disease. The most frequent symptoms are proteinuria and asymptomatic haematuria[2].

Glomerulonephritis refers to different immune disorders which cause glomerular inflammation. Numerous immunological and inflammatory processes can lead to glomerulonephritis[3]. It is commonly called secondary When glomerular involvement is a component of a systemic disease, such as polyarteritis nodosa or systemic lupus erythematosus (SLE)[1]. One can categorize primary glomerulonephritis based on the underlying etiology, the histological appearance, or the generated clinical condition. The pathological description and the clinical syndrome caused do not directly correlate[2]. According to the level of affection of the glomeruli, GN can be categorized as widespread, impacting every glomerulus; focal, just influencing a portion of the glomeruli; or segmental, impacting only a part of the glomerulus in a segment. Many glomerulonephritis patients have a moderate or asymptomatic disease, which is mainly missed to be diagnosed[1].

[1]*Pediatrics Department, Faculty of Medicine, Northern Border University, Arar, Saudi Arabia*
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Clinical diagnosis of GN is mainly based on the urine sediment microscopic examination, serum and urine biochemical assays, glomerular filtration rate studies (GFR), as well as serological studies, including anti-neutrophil cytoplasmic antibodies (ANCA) or anti-glomerular basement membrane (GBM)[4]. A kidney biopsy, which reveals the presence of glomerular inflammation and different patterns of elevated glomerular cellularity, is the cornerstone in diagnosing glomerulonephritis. A biopsy is mainly indicated in cases with nephrotic syndrome (NS), prolonged acute kidney injury (AKI), rapidly progressive glomerulonephritis (RPGN), systemic diseases involving renal dysfunction, isolated microscopic hematuria, and unexplained renal impairment [5].

Data from kidney biopsies can be used to determine the frequency of specific renal diagnoses. Light microscopy is the primary method for pathological classification; however, immunofluorescence and electron microscopy offer supplementary data and may offer insights into the etiology[6].

Management of GN of different causes and pathological patterns is mainly based on a low-sodium (<2 g daily) diet and control of hypertension (systolic blood pressure is kept below 120 mm Hg) with blockers of the renin-angiotensin-aldosterone system (RAAS) blockage using maximally permitted or patient tolerated dosages[7]. The quantity of kidney function and the degree of proteinuria should determine how much protein should be consumed through diet to replace nephrotic losses. Lifestyle alteration is mandatory, decreases blood pressure, and lowers cardiovascular risk through weight reduction and daily sports activities. Renal replacement therapy must be started as soon as possible when kidney function declines despite active treatment; this process must prioritize patient empowerment and life participations[6].

METHODOLOGY & DISCUSSION

Common types of GN:

Minimal change disease (MCD):

It most frequently manifests in children older than one year, with a dramatic decrease in incidence by puberty. It is responsible for about 70–90% of childhood NS[8]. It is mainly shown as nephrotic syndrome with selective proteinuria, normal blood pressure, kidney function, and complement levels; elevated predisposition to infections, particularly urinary tract infection and pneumococcal peritonitis[2]. Electron microscopy of GN samples reveals increased fusion of the foot processes of the epithelial cells to the outside the basement membranes of the glomeruli, while light microscopy is essentially routine. Generally, immunofluorescence is negative[6].

Focal Segmental glomerulosclerosis:

This pattern includes foot process fusion and segmental scarring in some glomeruli[6]. In older children and younger adults, it is commonly seen in cases of nephrotic syndrome; it may also be linked to hypertension, haematuria, and reduced renal function. While the majority of cases are idiopathic, SLE, hepatitis B, cancer, or the use of gold or penicillamine may also be secondary causes[9].

Membranous nephropathy:

The glomerular basement membrane thickens widely. Immunofluorescence exposes complement and immunoglobulin granules. While idiopathic causes account for the majority of cases with membranous nephropathy, it can also be due to Systemic lupus, hepatitis B, cancers, or gold or penicillamine intake. Adult men are more likely to have it, and it is mainly manifested as nephrotic syndrome. Hypertension, nephritic syndrome, or proteinuria may also present[10].

Mesangial proliferative nephritis:

This type is characterized by Mesangial cell proliferation with matrix expansion and IgA deposition in most cases (known as IgA nephropathy). It mainly occurs within a few days after upper respiratory tract infections and is manifested by hematuria. The patient may also be presented with microscopic haematuria and/or NS with proteinuria. The condition may progress to impaired kidney function (20% of cases) or even end-stage kidney disease (3% of cases)[11].

Diffuse proliferative glomerulonephritis:

It is characterized by widely speeding hypercellularity due to infiltrating inflammatory infiltration and diffuse cellular proliferation with immunoglobulins and complement deposition. Most cases are secondary to streptococcal infection[12].

Focal segmental glomerulosclerosis:

It is mainly associated with capillary loops segmental necrosis predisposing to crescent formation. It occurs secondary to viral infections such as Hepatitis C, or as a part of systemic diseases manifesting as lupus nephritis or may be predisposed by certain drugs[13].

Crescentic glomerulonephritis:

It may occur in the progression of IgA nephropathy or mesangiocapillary glomerulonephritis cases of primary GN or in the context of Goodpasture's syndrome and widely spreading vasculitis. Idiopathic crescentic GN is categorized into five forms based on the associated immune disorders. Type 1 is characterized by linear deposits of immunoglobulin G (IgG) in the basement membrane, Immune-complex granular deposits cause type 2, Type 3 is pauci-immune that may be renal-limited or part of a systemic disease, Type 4 is a combination of types 1 and 3. In contrast, Type 5 is mainly a pauci-immune renal vasculitis (5% to 10% of cases). Crescentic GN is clinically manifested as rapidly progressive glomerulonephritis (RPGN) characterized by oliguria and deteriorating renal functions within weeks to months[14]. Rapidly Progressive Glomerulonephritis cases are described by widely spreading crescents due to the proliferation of Bowman's capsule parietal epithelial cells and inflammatory infiltrates with detected anti-glomerular basement membrane antibodies in 95% of cases[15].

Lupus Nephritis (LN):

It is a common renal complication among children with SLE. It was divided into six main groups with definite sub-groups according to the site of the complement and immunoglobulins deposition, with a close correlation between histological data and clinical presentations. LN is characterized as WHO Class II if deposits are localized in the mesangium. In contrast, cases with deposits involving both the mesangium and capillary loops are considered WHO Class III, IV or V[16].

Post-streptococcal glomerulonephritis:

A bacterial infection following a streptococcal infection that triggers an inflammatory response quickly deteriorates kidney function, leading to post-streptococcal glomerulonephritis (PSGN); the most usual times for PSGN to manifest in kids are one to two weeks after a skin infection caused by streptococcal bacteria or six weeks after a throat infection[2]. When PSGN is symptomatic, it usually manifests as hematuria, oliguria, hypertension, edema, and sometimes substantial proteinuria. These are characteristics of the nephritic syndrome. In addition to reviewing the genesis, assessment, and treatment of PSGN, this activity clarifies the function of the interprofessional team in diagnosing and managing individuals with this illness[1].

Glomerulonephritis among Saudi children current status:

The published data related to the topic were collected from databases such as Google, PubMed, Web of Sciences, and Scopus from 2013 to 2023 to evaluate the current status of GN among Saudi children. After article screening and revision, ten articles were selected and studied as they are related to the scope of the current review article. The location of the studies, sample size, aim, and primary outcomes are shown in **Table I**.

TABLE I: PUBLISHED SAUDI DATA REGARDING GLOMERULONEPHRITIS AMONG CHILDREN IN YEARS FROM 2013-2020

Study	Location	Number of cases	Aim	Main outcomes
Al-Hussain et al. (2017)[17]	King Faisal Specialist Hospital and Research Center	78 cases	Evaluation of the Patterns of Crescentic GN from January 2003 to February 2014	- Lupus nephritis was the most common cause of crescentic GN. - Immune complex-mediated GN was the most common cause - Cases of pauci-immune GN showed fibrinoid necrosis, disrupted GBM, and increased serum c-ANCA with Bowman's capsule.
AlMatham et al. (2017)[18]	King Fahad Medical City, Riyadh	102 biopsies	Studying the Patterns of glomerulonephritis	- Primary GN was focal, and segmental glomerulosclerosis was the most common pattern. - LN grade IV was the most common, followed by grade III in cases with LN.
Mosaad et al. (2018)[19]	Department of Pediatric Medicine at KAUH	19 case	Studying the causes and outcomes of RPGN	- post-infectious GN was the most typical cause (2/3 of enrolled cases). Poor prognosis was reported in (31.6%) of cases.
Nawaz et al. (2013)[20]	the Armed Forces Hospital, Riyadh	348 native renal biopsies	Pattern of glomerular disease	- The most common pattern was focal and segmental glomerulosclerosis. - SLE was the most common cause of secondary GN
Al-Moaigel et al. (2020)[21]	King Fahad University Hospital in Eastern Province	15 cases	Evaluation of Acute Post -infectious Glomerulonephritis (PIGN) in children	- Edema, eye puffiness and gross hematuria were the main manifestations. - Laboratory investigations showed microscopic hematuria in all cases. Urinary protein/creatinine ratio was elevated, with elevated C3 found in most cases, while ANA, IgA and C4 were normal in all tested subjects.
Alhasan et al. (2020)[22]	Four tertiary health centers in Jeddah and Riyadh	326 cases	To identify the trends in patterns of glomerular disease by renal biopsies of Saudi children over 20 years.	- Secondary glomerulonephritis was diagnosed in 42.3% of the cases, and LN was the most common cause. - Over the study years, the MCD and FSGS were the most common patterns with decreasing trends for membranoproliferative glomerulonephritis and mesangioproliferative glomerulonephritis.
Jalalah (2020)[23]	western region of Saudi Arabia	448 1ry GN and 263 2ry GN	Studying the frequency of GN over 26 years within two periods, period 1 (1988–19999) and 2 (2000–2013).	- FSGSC was the most common 1ry GN in both periods, while SLE was the most typical cause for the secondary GN - PIGN increased over the years, while MCD decreased among the diagnosed cases.
Al-Mayouf et al. (2017)[24]	Lupus Clinic at King Faisal Specialist Hospital and Research Center	84 cLN patients	Studying cases of SLE between January 2000 and June 2015	- Proliferative GN was the most typical pattern, followed by membranous nephritis with mean activity and chronicity indices of 5.9 (±3.9) and 2.9 (±2.2) for both types, respectively.
Mokhtar et al. (2014)[25]	King Abdulaziz University, in Saudi Arabia	750 percutaneous native renal biopsies	To analyze cases of Mesango-proliferative (MesPGN)	- MesPGN was diagnosed in 14% of cases with more dominance of the focal pattern. - IgM nephropathy was shown in most cases - Cases were mainly manifested by NS, followed by hematuria.
Gomaa et al. (2014)[26]	King Abdulaziz University Hospital and King Faisal Specialist Hospital and Research Centre	148 cases	Studying of lupus nephritis (LN) in the Western province of Saudi Arabia f(1995 to 2011)	- Class IV (51.4%) was the most typical pattern - Positive fluorescence staining for IgG and C3 in almost all cases.

According to publicly available Saudi statistics, males between the ages of 4 and 18 are the majority of diagnosed cases of GN. The average age in the literature varies greatly, ranging from 9 years [27] to 15 years old[28]. The average age was 13.5±4.1 years in one of the studies conducted in China[29]. Consistent with previous research, adolescents constituted a significant portion of our patient population (72.3%); this is primarily because kidney biopsies on older children are more widely accepted and have lower thresholds[29].

Regarding histopathological patterns, membranous GN is most commonly shown to be the most common type of GN among Saudi children[23] in his study covering two periods (1988 to 1999 and 2000 to 2103) to be around 24% of the enrolled cases for both studied periods. In the more recent period (2000-2013), focal segmental glomerulosclerosis (FSGS) was the second (23%); Ig A nephropathy at 9.6% became the third, and MCD was the last place. Al-Mayouf SM 2017[24] showed that proliferative GN (64.3%) followed by membranous nephritis (27.4%) are the most common histopathological patterns among the study's biopsies reports. Nawaz Z et al. [20] showed that the most common histological lesion FSGS (27.6%), followed by MCD (17.7%) and membranoproliferative glomerulonephritis (MPGN) (13.0%). Al-Hussain T 2017[17], in a twenty-year study, revealed that the MCD and FSGS were the most common GN patterns in 59% of their enrolled cases. Also, they reported that the frequency of membranoproliferative glomerulonephritis and mesangio-proliferative GN significantly decreased from 15% and 17% before 2004 to 3.3% and 1.7% in 2012-2017. AlMatham KI et al. [18] reported that the commonest primary GN was FSGS (35.3%). The contradiction in the current data revealed a shift in the histological patterns of GN among Saudi Children over the years enrolled in the study. In addition, the presence of different histological patterns varies among different studies from other countries. According to an Indian study, MCD was the commonest pattern followed by FSGS[30]. Numerous Iranian investigations Madani A et al.[31]. MCD is the most common pathological diagnosis in renal biopsies in Iran, China[29], a few Arab nations[32], and certain North African countries[27]. Nonetheless, other research from Korea[33] and Croatia[34] has indicated that IgAN is the most typical histopathologic diagnosis. The USA[35] and Serbia[36] have the highest rates of FSGS. MesPGN was the most typical histopathologic diagnosis in a prior study conducted in northwest India.[37]

Some published studies were conducted to evaluate certain histopathological types of GN among Saudi children. Mesangioproliferative GN (MesGN) was studied by Mokhtar GA 2014[25] who found MesGN was diagnosed in 14% with more prevalence of the focal type (seen in 62% of cases).

Al-Hussain T 2017[17] examined 78 cases of crescentic GN and found that SLE was the most common finding in the enrolled cases of crescentic GN. Most cases (71.8%) had crescents caused by GN mediated by immune complexes. Then came anti-GBM antibody GN (7.7%) and pauci-immune GN (20.5%). Cases with anti-GBM antibody GN showed the highest level of glomerular crescents (mean of 93.3%), and Cases with pauci-immune GN showed higher levels of serum anti-neutrophil cytoplasmic antibody (c-ANCA).

Lupus nephritis was reported to be the most common cause of secondary GN among Saudi children[22]. Cases with lupus nephritis were evaluated by Gomaa W et al.[26] and Al-Mayouf SM 2017[24]. Data revealed that the commonest histopathological pattern was proliferative GN (64.3%) followed by membranous nephritis (27.4%) with reported mean activity and chronicity indices of 5.9 (±3.9) and 2.9 (±2.2), respectively. By histological class, there is no discernible difference in ESRD. Class IV is the most common class, followed by class V, according to the enrolled cases' WHO and ISN/RPS classifications. Positive staining for IgG and C3 was around 98% of the enrolled cases. These data are in agreement with the conclusions of the previously published study[16,38,39]. Nevertheless, previous research from Arab nations indicated that class III had a more significant occurrence[40,41]. The different sample sizes employed in these investigations are most likely the cause of this discrepancy in the final findings.

It was stated that post-infection GN (PIGN) was According to Al-Moaigel HM et al.[21] study of recruited 19 cases with RPGN, post-infectious GN (PIGN) was the most common cause (63.2%) among the enrolled cases who were manifested by edema, ocular puffiness, and gross hematuria recorded as the significant symptoms [19] which are comparable to another study.[42] Decreased C3 levels and serum albumin were found in over 80% of cases. Elevated ASO titer was elevated in five subjects (62.5%), urine protein to creatinine ratio was elevated in twelve subjects (median 13), and ANA, IgA, and C4 were normal in all tested subjects. All subjects (100%) had microscopic hematuria. Glomerular hematuria is virtually always seen in PIGN cases worldwide[43]. Hypoalbuminemia and lower hemoglobin were also common findings in previous studies[44,45].

CONCLUSION

In summary, our data help to record and comprehend the epidemiology of GN in the Kingdom; yet, our study bears the drawbacks of retrospective research. It is necessary to conduct additional research that thoroughly examines the prognostic factors, therapies, illness progression, and outcomes. While there are several advantages to having a GN registry that collects data in the future, resource constraints frequently prevent the establishment of comprehensive registries. Retrospective analyses of administrative data, like the studies presented in this report, can offer a significant supplementary method

for researching GN epidemiology. Creating novel treatment plans to preserve glomerular function will probably remain a top research priority. The prevalence, manifestation, and risk factors of the most prevalent illnesses, such as FSGS and LN, will serve as workable models for creating fresh perspectives on pharmacological interventions.

Nonetheless, there is clinical heterogeneity in glomerular disorders. The course and prognosis of renal illness may change if additional conditions and risk factors coexist. Expanding research endeavors will play a significant role in fostering a healthy society and may provide insight into the underlying pathology of fascinating glomerulopathies.

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AUTHOR CONTRIBUTION

Alenazi SA: The idea of study was conceived and conducted by the author from planning till approval of the final version for publication.

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