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GLOMERULAR DISEASE: IRAQI DATA 2020-2024

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ABSTRACT

Background: Glomerular diseases represent a diverse group of renal pathologies with significant morbidity. Understanding their demographic distribution is crucial for early diagnosis and effective management. **Aim:** To assess the prevalence and patterns of glomerular diseases in Iraq over a five-year period (2020–2024) with respect to age, sex, and temporal trends. **Patients and Methods:** A retrospective analysis was conducted on 1,377 patients who underwent renal biopsy and were diagnosed with various types of glomerular disease. Data were categorized and analyzed based on age, sex, and year of diagnosis. Histopathological patterns were classified according to standard diagnostic criteria. **Results:** Focal Segmental Glomerulosclerosis (FSGS) was the most common diagnosis (28.54%), followed by Membranous Glomerulonephritis (21.79%), Minimal Change Disease (16.63%), and IgA Nephropathy (16.34%). IgA Nephropathy showed a significant male predominance ($p=0.009$) and an increasing trend over time ($p=0.088$). Minimal Change Disease was more frequent in younger patients ($p<0.001$), while Membranous GN increased with age ($p<0.001$). Lupus Nephritis was significantly more common in females ($p=0.001$). A statistically significant rise was noted in C3 Glomerulopathy ($p=0.006$) and Pauci-immune GN ($p=0.012$) in recent years. Several diseases, such as MPGN and monoclonal-related nephropathies, showed strong age associations, and the "Other" category of diagnoses significantly declined over time ($p=0.028$), indicating improved diagnostic specificity. **Conclusion:** The spectrum of glomerular diseases in Iraq shows significant age- and sex-based variation, with emerging trends in immune-complex and complement-mediated nephropathies. These findings underscore the need for improved diagnostic infrastructure, national registry development, and tailored clinical strategies to address evolving disease patterns.

KEYWORDS: Glomerular disease, Iraq, sex-wise association, age-wise association.

INTRODUCTION

1.1 Background

Glomerular diseases are a common cause of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) in children and adult. They can present as part of a systemic disease, with multiple symptoms, or with only renal findings and perhaps no symptoms. Glomerular diseases are generally divided into two classic syndromes: nephrotic and nephritic syndromes. Nephrotic syndrome is generally defined by >3.5 g of protein in the urine per 1.73 m^2 of body surface area and the constellation of findings that result from the spilling of a large amount of protein in the urine, namely, edema, hypoalbuminemia, hyperlipiduria, and hyperlipidemia.[1] Nephritic syndrome is defined by hematuria, proteinuria, and kidney damage, with glomerular inflammation (glomerulonephritis) resulting in the clinical findings. While this division into the syndromes provides an organized approach to the different glomerular diseases, overlap of the two syndromes does occur. For example, glomerulonephritis (GN) from a vasculitis can also cause nephrotic range proteinuria. Likewise, a patient with nephrotic syndrome can present with some hematuria and a rising creatinine. Sometimes, pathology consistent with both nephritic and nephrotic syndrome can be present on the same kidney biopsy specimen, as is often found in a systemic disease like systemic lupus erythematosus.[2]

1.2 Epidemiology

Glomerular diseases contribute significantly to the global burden of CKD, affecting around 10–15% of adults worldwide. In pediatric populations, glomerular diseases account for roughly 25– 50% of CKD cases, with minimal change disease and focal segmental glomerulosclerosis (FSGS) being more common in children, and conditions like diabetic nephropathy and hypertensive nephrosclerosis prevalent in adults.[3,4] Key risk factors include genetic predispositions (e.g. Alport syndrome), autoimmune conditions such as systemic lupus erythematosus (SLE) and vasculitis, and infections like human immunodeficiency virus and hepatitis B. Additionally, lifestyle factors – such as obesity and high sodium intake – exacerbate risks, particularly in cases of diabetes and hypertension, which are major contributors to glomerular injury. The primary types of glomerular diseases include FSGS, membranous nephropathy, minimal change disease, and diabetic nephropathy, each with distinct risk profiles and associations. The high prevalence of diabetic and hypertensive nephropathy underscores the importance of managing metabolic and cardiovascular risk factors to reduce the burden of glomerular disease.[5,6]

1.3 Nephrotic Syndrome

1.3.1 Minimal Change Disease

Minimal change disease typically presents with nephrotic syndrome, more commonly in children but also seen in adults. Light microscopy shows no changes, while electron microscopy reveals diffuse effacement of foot processes. Minimal change disease can be primary (idiopathic) or secondary. In secondary cases, evaluation for paraproteinemia, cancer (especially lymphoma), and a review of medications is necessary. Currently, there are no established serologic tests or urinary markers to diagnose idiopathic minimal change disease. Research has shown that podocytes may express CD80, with urinary CD80 levels higher in minimal change disease compared to other nephrotic syndromes. Although promising, urinary CD80 testing is not yet standard. Newer approaches suggest minimal change disease exists on a spectrum with FSGS, with minimal change disease being steroid-responsive.[2]

1.3.2 Membranous Nephropathy

Membranous nephropathy presents with nephrotic syndrome but may overlap with nephritic syndrome if serum creatinine is elevated or hematuria is present. It can also occur in association with glomerulonephritis, such as lupus nephritis.[7] Like minimal change disease, membranous nephropathy can be primary or secondary. When secondary membranous nephropathy is suspected, evaluation should include cancer screening, autoimmune disease evaluation, review of medications, and assessment for infections like hepatitis B. Histological features such as IgG4 staining are more typical of primary membranous nephropathy, while other immunoglobulins and immune deposits in different locations suggest secondary causes.[8] The discovery of Phospholipase A2 Receptor (PLA2R) antibodies has been significant, showing high sensitivity and specificity for primary membranous nephropathy. PLA2R antibody levels correlate with disease activity and treatment response. In patients negative for PLA2R, anti-thrombospondin type-1 domain-containing 7A (THSD7A) antibodies may be present. These findings have revolutionized the diagnostic approach to membranous nephropathy.[2]

1.3.3 Focal Segmental Glomerulosclerosis

FSGS is diagnosed via histology and categorized into five subtypes. Its pathogenesis is complex, and mechanisms may vary between lesions. There are currently no validated serologic tests for FSGS diagnosis. Although Serum soluble urokinase receptor (suPAR) levels were initially promising as a marker, their specificity has proven unreliable. suPAR levels are influenced by disease activity and glomerular filtration rate (GFR). Genetic causes of nephrotic syndrome, including FSGS, are increasingly recognized, and genetic testing may become more common.[9]

1.3.4 Systemic Diseases Causing Nephrotic Syndrome

Evaluation of nephrotic syndrome should include an investigation for systemic causes, including infections, medications, diabetes, and paraproteinemias. Protein electrophoresis and serum free light chain assays are valuable for identifying paraprotein-related kidney diseases.[10]

1.4 Nephritic Syndrome

Diagnoses presenting with nephritic syndrome often require a kidney biopsy for definitive diagnosis. Classification is based on immunofluorescence and electron microscopy findings, dividing glomerulonephritis into immune complex, pauci-immune, and anti-GBM categories.

1.4.1 Immune Complex Glomerulonephritis

The main types include lupus nephritis, postinfectious glomerulonephritis (PIGN), IgA nephropathy, and membranoproliferative glomerulonephritis (MPGN).

1.4.1.1 Lupus Glomerulonephritis

Systemic lupus erythematosus presents varied kidney involvement and is divided into six classes. Diagnosis is clinical, supported by American College of Rheumatology and Systemic Lupus International Collaborating Clinics criteria, including urinary findings. Immunologic tests such as ANA and anti-dsDNA support SLE diagnosis but are not reliable for predicting lupus nephritis. Complement levels and anti-dsDNA are monitored for disease activity, but their predictive value is limited. Urinary biomarkers like monocyte chemoattractant protein-1 (MCP-1), TNF-like weak inducer of apoptosis (TWEAK), and neutrophil gelatinase-associated lipocalin (NGAL) are promising but not validated.[11]

1.4.1.2 Post-infectious Glomerulonephritis

Postinfection glomerulonephritis usually follows infections, notably streptococcal throat infections. Diagnosis involves light microscopy, immunofluorescence (C3 and IgG deposits), and electron microscopy showing subepithelial humps. PIGN caused by staphylococcus shows IgA dominance. Low C3 levels typically normalize after infection resolution. Persistent low C3 suggests alternative diagnoses like MPGN or C3 glomerulopathy. C3 glomerulopathy requires complement system evaluation and may benefit from complement inhibitors.[12]

1.4.1.3 Membranoproliferative Glomerulonephritis

Membranoproliferative glomerulonephritis refers to a histologic pattern characterized by immune complex deposition. Classification is now based on etiology rather than electron microscopy findings. Positive immunoglobulin findings prompt evaluation for paraproteinemias and systemic diseases. C3-dominant cases suggest C3 glomerulopathy or dense deposit disease. Lack of immune deposits raises suspicion for thrombotic microangiopathies.[2]

1.4.1.4 IgA Nephropathy

IgA nephropathy, the most common primary GN globally, is identified by mesangial IgA deposits. It is caused by galactose-deficient IgA1 molecules forming pathogenic immune complexes. Although blood assays for galactose-deficient IgA1 have been explored, they are not reliable for diagnosis. Antibodies to galactose-deficient IgA1 and urine proteomics are being researched for future diagnostic utility.[13]

1.4.2 Pauci-immune Glomerulonephritis

This category involves glomerulonephritis with minimal immune deposition. Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitides, including microscopic polyangiitis, granulomatosis with polyangiitis, and eosinophilic granulomatosis with polyangiitis, fall under this category. ANCA immunofluorescence patterns (C-ANCA and P-ANCA) correspond to anti-PR3 and anti-myeloperoxidae (MPO) antibodies, respectively. Atypical ANCAs, like anti-human neutrophil elastase, are linked to conditions like cocaine-induced vasculitis.[14]

1.4.3 Anti-glomerular Basement Membrane Disease

Anti-GBM disease is caused by antibodies targeting the non-collagenous domain of type IV collagen in the glomerular and alveolar basement membranes, leading to glomerulonephritis and alveolar hemorrhage. Kidney biopsy shows linear immunofluorescence patterns. Diagnosis involves detecting serum and/or tissue anti-GBM antibodies along with clinical features. Serum tests can sometimes be negative due to antibody disappearance, and false positives may occur. Tissue antibodies tend to persist longer than serum antibodies.[15]

1.5 Diagnostic modalities for glomerular diseases

Noninvasive diagnostics form the cornerstone in evaluating glomerular diseases, with urine and blood tests playing a crucial role. Proteinuria, excess protein in the urine, is a sensitive marker of glomerular damage. Its quantification via 24-hour urine collection or a spot urine protein-to-creatinine ratio is essential for diagnosing conditions such as nephrotic syndrome and FSGS. Persistent proteinuria above 3.5 g/day strongly suggests nephrotic syndrome, while lower levels may indicate other forms of glomerulonephritis.[16] Hematuria, or the presence of blood in the urine, is another key indicator, particularly in nephritic syndrome. Microscopic hematuria and dysmorphic red blood cells often indicate a glomerular origin. Red blood cell casts, pathognomonic for glomerulonephritis, underscore the need for prompt evaluation.[17]

Immunological markers add further precision to the diagnostic process. Serum levels of complement proteins (C3 and C4) can help distinguish between glomerular pathologies. For example, lupus nephritis is commonly associated with low levels of both C3 and C4, while isolated low C3 levels are more characteristic of post-infectious glomerulonephritis.[18] Specific autoantibodies, such as anti-glomerular basement membrane (GBM) or ANCAs, further narrow the differential diagnosis. Anti-GBM antibodies diagnose Goodpasture's syndrome, while ANCA positivity, especially in combination with renal impairment suggests ANCA-associated vasculitis, including granulomatosis with polyangiitis and microscopic polyangiitis.[19]

Renal imaging is an essential adjunct to biochemical tests, especially when structural abnormalities are suspected. Ultrasound, a widely available modality, can assess kidney size, cortical thickness, and echogenicity. Chronic glomerular diseases may show small, shrunken kidneys with increased echogenicity, indicating scarring, while acute diseases often present with normal-sized kidneys. Doppler ultrasound can also provide information on renal blood flow, helping to distinguish glomerular from vascular causes of renal dysfunction.[4,20] Advanced imaging techniques like MRI and CT are typically reserved for detecting masses or complex cysts, ruling out other causes of renal dysfunction, such as obstructive uropathy or renal artery stenosis.[21]

Emerging biomarkers are being explored to enhance diagnostic accuracy in glomerular diseases. NGAL, for instance, correlates with the severity of kidney injury and may serve as an early marker for acute kidney injury. Similarly, even in the microalbuminuria range, urinary albumin levels predict early glomerular injury, particularly in diabetic nephropathy. These biomarkers show promise for detecting glomerular diseases earlier, potentially leading to better outcomes through earlier intervention.[5,6]

1.5.1 Renal biopsy

Renal biopsy has remained an essential tool for diagnosing and characterizing glomerular diseases for decades owing to the lack of validated and available substitute diagnostic biomarkers with high sensitivity and specificity. The most promising biomarkers have not yet been implemented in routine clinical practice because of insufficient validation in large cohorts, or because limited access or high costs prevent global implementation.[22] Consequently, most of the proposed biomarkers have not been incorporated into the current Kidney Disease: Improving Global Outcomes (KDIGO) guidelines for managing glomerular diseases. Consequently, renal biopsy has maintained its relevance, and its clinical utility remains highly valuable, providing definitive histological data that can guide the diagnosis, management, and prognostication of kidney diseases. Moreover, renal histology can lead to changes in treatment decisions in approximately 40% of cases.[23]

The indications for renal biopsy vary depending on the physicians, centers, and even the times. Not all countries have the same criteria, and often the availability of study options or the interpretation of results influences the timing and selection of patients for renal biopsy.[24]

The general indications for renal biopsy can be summarized as fellows.[26]

- Adult nephrotic syndrome: Excepting new-onset nephrotic syndrome with evidence of PLA2-R-Abs.
- Proteinuria >1–2 g/24 h with or without hypertension: Excepting proteinuria/albuminuria associated with diabetes mellitus in the presence of proven diabetic retinopathy.
- Progressive increase in serum creatinine with microscopic evidence of acanthocytes and/or red blood cell casts.
- Systemic diseases (immunological or paraneoplastic) with suspected renal involvement: e.g., clinical/serologic evidence of systemic vasculitis or p-ANCA positive and proteinase3-/MPO-Abs positive, clinical/serologic evidence of SLE, and serologic evidence of monoclonal gammopathy.
- Impaired renal function of unclear etiology (if kidneys are of normal size on ultrasound) with or without sterile pyuria/white blood cell casts/low-grade proteinuria, e.g., drug-induced interstitial nephritis and interstitial nephritis related to autoimmune diseases (sarcoidosis, IgG4-related disease).
- Repeated biopsy to examine severity of damage or progression of an already-known kidney disease.
- Nonresponse to an established therapy**:** e.g., steroid resistance with glomerular minimal lesions.
- Therapy monitoring**:** e.g., clarification of whether immunosuppressive therapy needs to be intensified or can be suspended in individual cases of SLE or ANCA-associated vasculitis.
- Recurrent disease activity: e.g., evaluation of active/chronic (scarring) lesions prior to resumption of immunosuppressive therapy.
- Graft dysfunction inpatient undergone kidney transplant.

The contraindications for renal biopsies can be summarized as follows.[25]

- Clotting disorder
- Thrombocytopenia (platelet count $<120 \times 10^3/\mu L$)
- Medication uncontrolled hypertension (>140/90 mmHg)
- Small hyperechoic kidneys on ultrasound
- Patient's inability to follow instructions
- Patient's inability to provide informed consent: Anatomic anomaly/horseshoe kidney
- Multiple bilateral cysts
- Hydronephrosis
- Active kidney infection/pyelonephritis or perirenal abscess
- Skin infection at the site of needle insertion

1.6 Aim of the study

The present study aimed to determine the prevalence and spectrum of glomerular **diseases** diagnosed by renal biopsy in Iraq over a five-year period (2020–2024).

Patients and Methods

2.1 Settings and Design

This is a retrospective study which included 1377 biopsy samples from symptomatic patients of glomerular disease sent to the histopathology departments all over Iraq with complete relevant clinical information and other laboratory findings during the years form 2021-2024. The study was approved by the Iraqi Council for Medical Specializations.

2.2 Inclusion criteria

- All patients, males and females diagnosed with glomerular diseases.

2.3 Exclusion criteria

- Non glomerular disease
- Any patient with glomerular disease with serology positive tests without kidney biopsy
- Kidney transplant patient

2.4 Ethical consideration

A written consent from each participant was obtained prior to data collection after explaining the aim of study. The confidentiality of data throughout the study was guaranteed and the patients were assured that data will be used for research purpose only.

2.5 Data Collection

Sociodemographic data including the age, gender and year of examination were collected from patient's reports.

2.6 Biopsy examination

All the samples were evaluated using light microscopy and immunofluorescence. For light microscopy, tissue specimens were fixed in 10% neutral buffered formalin, Processed for 12 hours in semi-automated processor by providing the medium (Xylene, Alcohol, Formalin & Paraffin wax) manually. Embedded the fixed renal biopsy core in paraffin wax to make block for further proceedings. Several serial sections were cut (at a thickness of 2mm) on microtome, stained by hematoxylin– eosin (HE) and special stains, like periodic acid-Schiff (PAS) and silver stains (Gomori's Methenamine Silver, GMS) for optimal evaluation of the morphological details.

For Immunofluorescence studies, tissue cores stained by the direct method using fluorescein isothiocyanate (FITC)-conjugated antisera monospecific for immunoglobulin (Ig)G, IgA, IgM, C3 and C1q (Dako, Glostrup, Denmark). The slides were visualized under an Olympus BX41-fluorescence microscope and graded semi-quantitatively as 0 to 3+ (on a scale of 0 to 3+, where 0 = absent and 3+ = brightest) and distribution will be described as membranous or mesangial in a granular or linear pattern.

2.8 Statistical Analysis

Data were expressed as mean and SD for continuous and normally distributed variables, or frequencies (%) for categorical variables. Chi square test was used to explore the association of glomerular disease with age, sex and year, The level of statistical significance was set at p less than or equal to 0.05. Statistical analysis was carried out using the Statistical Package for Social Sciences, version 22 (SPSS Inc., Chicago, Illinois, USA).

RESULTS

3.1 Demographic Characteristics of the patients

The dataset comprises 1,377 patients diagnosed with glomerular diseases in Iraq over a five-year period from 2020 to 2024. The mean age of the patients was 33.24 years with a standard deviation of 16.3 years, and the age ranged from 0.5 to 95 years. Patients were distributed across four age categories: 336 (24.40%) were aged 0.5–20 years, 602 (43.72%) were 21–40 years, 361 (26.22%) were 41–60 years, and 78 (5.66%) were older than 60.

The sex distribution showed a male predominance: 772 males (56.06%) and 605 females (43.94%). The yearly distribution was as follows: 204 patients (14.81%) in 2020, 221 (16.05%) in 2021, 352 (25.56%) in 2022, 273 (19.83%) in 2023, and 327 (23.75%) in 2024.In terms of histopathological diagnosis, FSGS was the most common (28.54%), followed by membranous GN (21.79%), MCD (16.63%), and IgA Nephropathy (16.34%). Less frequent diagnoses included AA and AL Amyloidosis, Lupus Nephritis, C3 GN, Pauci-immune GN, Light Chain Deposition Disease, Diabetic Nephropathy, MPGN, Crescentic GN, and other rare conditions (Table 3-1).

Table 3-1: Baseline characteristics of the patients with glomerular disease (n=1377)

Variables	Category	Value
Age, years	Mean±SD	33.24±16.3
	Range	0.5-95
	0.5-20	336(24.40%)
	21-40	602(43.72%)
	41-60	361(26.22%)
	>60	78(5.66%)
Sex	Male	772(56.06%)
	Female	605(43.94%)
Years	2020	204(14.81%)
	2021	221(16.05%)
	2022	352(25.56%)
	2023	273(19.83%)
	2024	327(23.75%)
Final diagnosis	FSGS	393(28.54%)
	IgA nephropathy	225(16.34%)
	MCD	229(16.63%)
	Membranous GN	300(21.79%)
	AA amyloidosis	33(2.40%)
	Lupus nephritis	22(1.60%)
	C3 GN	32(2.32%)
	Pauci immune GN	33(2.40%)
	Light chain deposition disease	16(1.16%)
	MPGD	36(2.61%)
	AL amyloidosis	15(1.09%)
	Diabetic nephropathy	24(1.74%)
	Crescent GN	6(0.44%)
	Others	13(0.94%)

Other diagnosis include 5 cases of C1q nephropathy, 4 cases of thrombotic microangiopahty and one case of each of post-infcetion GN, monoclonal IgM nephropathy, rapid progressive GN, fibronectin glomerulopathy.

3.2 Age-wise distribution of glomerular disease

FSGS was the most common glomerular disease across all age groups. It affected 102 patients (30.36%) aged 0.5–20 years, 179 (29.73%) aged 21–40, 95 (26.32%) aged 41–60, and 17 (21.79%) over 60 years. Although there was a gradual decline in prevalence with increasing age, this difference was not statistically significant (p=0.310).

IgA Nephropathy showed a significant age-related distribution (p<0.001). It was most common among patients aged 21–40 years, affecting 129 individuals (21.43%). Its frequency dropped to 36 (10.71%) in the 0.5–20 group, 53 (14.68%) in the 41–60 group, and just 7 (8.97%) in those older than 60 years.

MCD demonstrated a very strong inverse relationship with age (p<0.001). It was highly prevalent in the 0.5–20 age group (115 patients; 34.23%), but much less so in older groups: 79 (13.12%) in the 21–40 group, 31 (8.59%) in the 41–60 group, and only 4 (5.13%) in those over 60 years.

Membranous GN increased progressively with age (p<0.001). It was present in 31 children and adolescents (9.23%), 135 patients (22.43%) aged 21–40, 106 (29.36%) aged 41–60, and 28 (35.90%) in the >60 age group, reflecting its known association with aging.

AA Amyloidosis was rare in children (2 patients; 0.60%), but increased in older groups: 15 (2.49%) in the 21–40 group, 13 (3.60%) in the 41–60 group, and 3 (3.85%) in those over 60 years. However, the p-value (0.054) indicates this trend approached but did not reach statistical significance.

Lupus Nephritis was predominantly seen in younger age groups—7 (2.08%) in the 0.5–20 group and 14 (2.33%) in the 21–40 group. It was rare in the 41–60 group (1 case; 0.28%) and absent in those older than 60, showing borderline significance (p=0.050), consistent with the known epidemiology of systemic lupus erythematosus.

C3 Glomerulopathy had a higher prevalence in children and young adults—14 (4.17%) in the 0.5–20 group, 11 (1.83%) in the 21–40 group, 6 (1.66%) in the 41–60 group, and only 1 (1.28%) in the oldest group. This trend did not reach statistical significance (p=0.080).

Pauci-immune GN showed a significant increase with age (p=0.007). It was diagnosed in 4 children (1.19%), 10 patients (1.66%) aged 21–40, 14 (3.88%) aged 41–60, and 5 (6.41%) in those over 60 years, reflecting its known association with vasculitic syndromes in older adults.

Light Chain Deposition Disease (LCDD) was absent in children, but increased with age: 2 (0.33%) in the 21–40 group, 10 (2.77%) in the 41–60 group, and 4 (5.13%) in the elderly group. This trend was highly significant (p<0.001), consistent with its typical presentation in older adults with plasma cell dyscrasias.

MPGN was significantly more common in the youngest age group, affecting 20 patients (5.95%) aged 0.5–20 years, compared to 12 (1.99%) in the 21–40 group, 3 (0.83%) in the 41–60 group, and 1 (1.28%) in those over 60 (p<0.001).

AL Amyloidosis, a disease strongly associated with aging, was not diagnosed in any patients under 41 years of age. It was found in 10 patients (2.77%) aged 41–60 and in 5 (6.41%) aged >60 (p<0.001), consistent with its well-documented prevalence in older adults.

Diabetic Nephropathy followed a similar trend. It was absent in individuals younger than 41 years, but found in 17 (4.71%) of the 41–60 group and 1 (1.28%) in those older than 60, with a statistically significant association (p<0.001).

Crescentic GN was a rare finding across all age groups, with 1 case (0.30%) in 0.5–20 years, 3 (0.50%) in 21–40, 1 (0.28%) in 41–60, and 1 (1.28%) in >60. No significant age-related trend was observed (p=0.637).

Other Glomerular Diseases, which include rare or unclassified entities, were seen sporadically: 1 (0.94%) in 0.5–20 years, 7 (1.16%) in 21–40, 1 (0.28%) in 41–60, and 1 (1.82%) in >60, with no significant difference across age groups (p=0.285) as shown in able 3-2.

Table 3-4: Age-wise distribution of glomerular diseases.

Glomerular disease	Age groups				p-value
	0.5-20 yrs (n=336)	21-40 yrs (n=602)	41-60 yrs (n=361)	>60 yrs (n=78)	
FSGS	102(30.36%)	179(29.73%)	95(26.32%)	17(21.79%)	0.310
IgA nephropathy	36(10.71%)	129(21.43%)	53(14.68%)	7(8.97%)	**<0.001**
Minimal change disease	115(34.23%)	79(%13.12)	31(8.59%)	4(5.13%)	**<0.001**
Membranous GN	31(9.23%)	135(22.43%)	106(29.36%)	28(35.90%)	**<0.001**
AA amyloidosis	2(0.60%)	15(2.49%)	13(3.60%)	3(3.85%)	0.054
Lupus nephritis	7(2.08%)	14(2.33%)	1(0.28%)	0(0%)	0.050

C3 GN	14(4.17%)	11(1.83%)	6(1.66%)	1(1.28%)	0.080
Pauci immune GN	4(%1.19)	10(1.66%)	14(3.88%)	5(6.41%)	**0.007**
Light chain deposition disease	0(0.00%)	2(0.33%)	10(2.77%)	4(5.13%)	**<0.001**
Membranous proliferative disease	20(5.95%)	12(1.99%)	3(0.83%)	1(1.28%)	**<0.001**
AL amyloidosis	0(0.00%)	0(0.00%)	10(2.77%)	5(6.41%)	**<0.001**
Diabetic nephropathy	0(0.00%)	0(0.00%)	17(4.71%)	1(1.28%)	**<0.001**
Crescent GN	1(0.30%)	3(0.50%)	1(0.28%)	1(1.28%)	0.637
Others	1(0.94%)	7(1.16%)	1(0.28%)	1(1.82%)	0.285

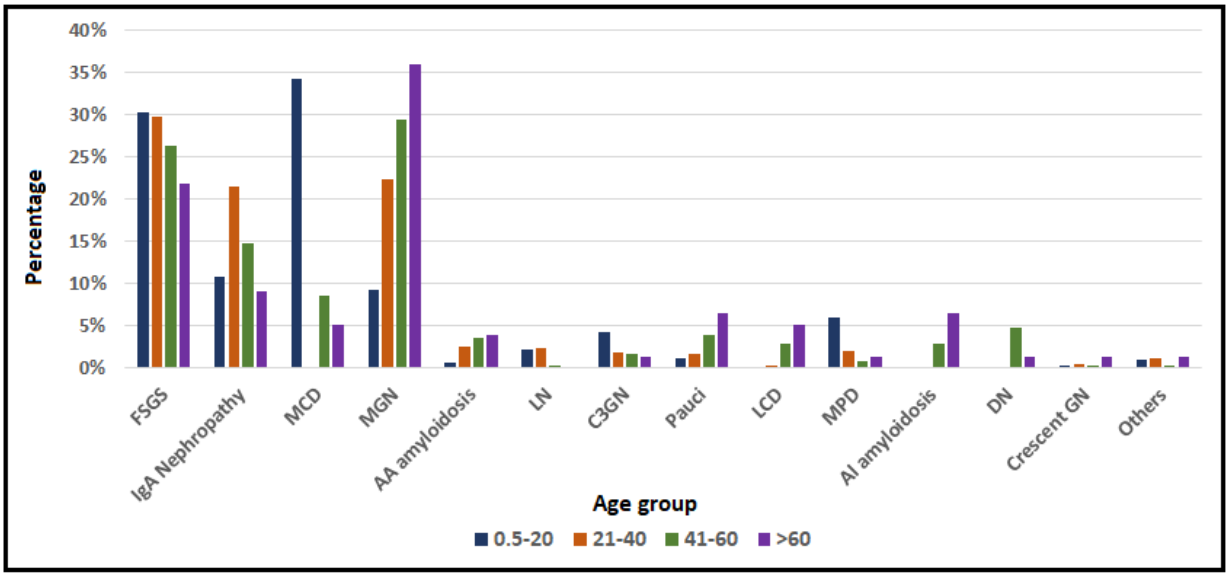

Figure 3-1: Age-wise distribution of glomerular diseases.

3.3 Sex-wise distribution of glomerular diseases

FSGS was the most common glomerular disease among both sexes, diagnosed in 208 males (26.94%) and 185 females (30.58%). Although the percentage was slightly higher in females, the difference was not statistically significant (p=0.138).

IgA Nephropathy showed a statistically significant male predominance, being more frequent in males (144 patients; 18.65%) compared to females (81 patients; 13.39%) with a **p-value of 0.009**, suggesting a sex-linked susceptibility in favor of males.

MCD was similarly distributed between sexes, affecting 125 males (16.19%) and 104 females (17.19%) with no significant difference (p=0.621).

Membranous GN also showed comparable rates: 159 males (20.60%) and 141 females (23.31%), with a non-significant (0.227). **AA Amyloidosis** was slightly more common in males (2.72%) than females (1.98%), but this difference was not significant (p=0.375). **Lupus Nephritis** had a strong female predominance, diagnosed in 17 females (2.81%) versus only 5 males (0.65%). This difference was statistically significant (p=0.001).

C3 Glomerulopathy and **Pauci-immune GN** were slightly more frequent in males (each 2.72%) than in females (1.82% and 1.98%, respectively), but neither difference was statistically significant (p=0.270 and p=0.375).

LCDD was diagnosed in 10 males (1.3%) and 6 females (1.0%), with no significant difference (p=0.602).

MPGN was **significantly more common in males**, affecting 26 males (3.37%) versus 10 females (1.65%), with a p-value of 0.048**.**

AL Amyloidosis was slightly more common in females (9 cases; 1.49%) than males (6 cases; 0.78%), but this was not statistically significant (p=0.208).

Diabetic Nephropathy showed nearly identical distributions in both sexes: 13 males (1.68%) and 11 females (1.82%) with a p-value of 0.850.

Crescentic GN was rare, with 4 male cases (0.52%) and 2 female cases (0.33%), showing no significant sex difference (p=0.600).

The **"Others"** category included various less common or unclassified glomerular lesions. It was found in 9 males (1.17%) and 4 females (0.66%), again without statistical significance (p=0.337) as shown in Table 3-3.

Table 3-3: sex-wise distribution of glomerular disease

Glomerular disease	Male (N=772)	Female (N=605)	p-value
FSGS	208(26.94%)	185(30.58%)	0.138
IgA nephropathy	144(18.65%)	81(13.39%)	**0.009**
Minimal change disease	125(16.19%)	104(17.19%)	0.621
Membranous GN	159(20.6%)	141(23.31%)	0.227
AA amyloidosis	21(2.72%)	12(1.98%)	0.375
Lupus nephritis	5(0.65%)	17(2.81%)	**0.001**
C3 GN	21(2.72%)	11(1.82%)	0.270
Pauci immune GN	21(2.72%)	12(1.98%)	0.375
Light chain deposition disease	10(1.3%)	6(1%)	0.602
Membranous proliferative disease	26(3.37%)	10(1.65%)	**0.048**
AL amyloidosis	6(0.78%)	9(1.49%)	0.208
Diabetic nephropathy	13(1.68%)	11(1.82%)	0.850
Crescent GN	4(0.52%)	2(0.33%)	0.600
Others	9(1.17%)	4(0.66%)	0.337

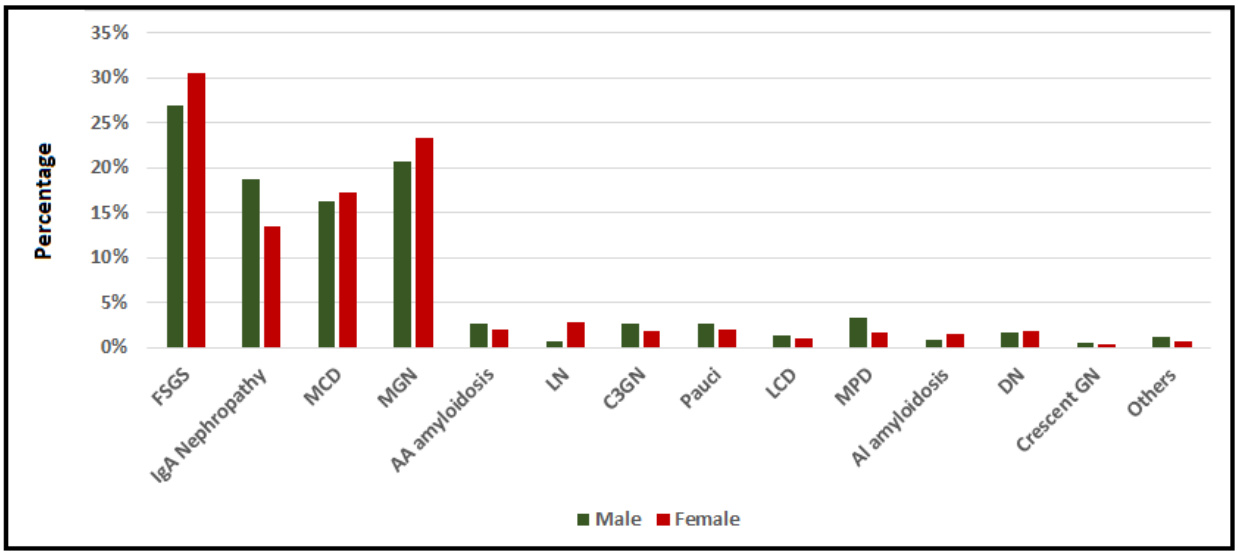

Figure 3-2: Sex-wise distribution of glomerular diseases.

3.4 Year-wise distribution of glomerular disease

FSGS was consistently the most frequently diagnosed glomerular disease, with slight fluctuations across years: 26.96% (2020), 31.22% (2021), 29.83% (2022), 25.64% (2023), and 29.05% (2024). Despite these variations, the changes were not statistically significant (p=0.649).

IgA Nephropathy showed an increasing trend over the five years, from 12.75% in 2020 to 20.8% in 2024. Intermediate rates were observed in 2021 (13.57%), 2022 (15.63%), and 2023 (18.85%). Although the p-value (0.088) was just above the conventional threshold for statistical significance.

MCD peaked in 2021 at 21.27% but generally declined thereafter, reaching 14.37% in 2024. It accounted for 13.24% in 2020 and remained within a modest range of 16–18% in other years. These year-to-year changes were not statistically significant (p=0.137).

Membranous GN maintained a relatively steady distribution throughout the study period: 23.04% (2020), 23.08% (2021), 20.74% (2022), 21.98% (2023), and 21.1% (2024), with no significant difference (p=0.948).

AA Amyloidosis remained consistently rare over the five years, ranging between 1.70% and 3.17%, without any significant temporal change (p=0.797).

Lupus Nephritis also remained low, fluctuating between 0.73% and 2.45%, with no statistically significant trend (p=0.497).

In contrast, **C3 Glomerulopathy** showed a statistically significant increase over time (p=0.006), with a marked rise in 2023 (13 cases; 4.76%) and sustained elevation in 2024 (11 cases; 3.36%), compared to ≤3 cases per year in 2020–2022.

Pauci-immune GN also showed **significant year-to-year variability** (p=0.012). It was relatively high in

2020 (4.9%) and 2023 (3.66%), but low in 2021 (1.36%) and especially in 2024 (0.61%).

LCDD remained uncommon, with a peak of 2% in 2022, but dropped sharply to 0.31% in 2024. The observed variation was not statistically significant (p=0.142).

MPGN remained relatively stable, fluctuating between 1.36% and 3.67%, with a slight rise in 2024. The differences, however, were not significant (p=0.214).

AL Amyloidosis, another rare condition, showed sporadic distribution: 1.96% in 2020, none in 2021, and modest frequencies thereafter. The p-value (0.094) indicates a possible trend but without statistical confirmation.

Diabetic Nephropathy showed no consistent pattern—rising from 0% in 2021 to 2.84% in 2022, then declining again to 1.22% in 2024. These variations were not statistically significant (p=0.121). **Crescentic GN** remained very rare and showed no meaningful annual variation (p=0.927), with 0–2 cases per year throughout the study.

The **"Other"** glomerular diseases category showed a **statistically significant decline** over time (p=0.028), dropping from 2.94% in 2020 to just 0.73% in 2023 and 2024 (Table 3-4).

Table 3-4: year-wise distribution of glomerular diseases.

Glomerular disease	Years					p-value
	2020 (n=204)	2021 (n=221)	2022 (n=352)	2023 (n=273)	2024 (n=327)	
FSGS	55(26.96%)	69(31.22%)	105(29.83%)	70(25.64%)	95(29.05%)	0.649
IgA nephropathy	26(12.75%)	30(13.57%)	55(15.63%)	46(18.85%)	68(20.8%)	0.088
MCD	27(13.24%)	47(21.27%)	64(18.18%)	44(16.12%)	47(14.37%)	0.137
Membranous GN	47(23.04%)	51(23.08%)	73(20.74%)	60(21.98%)	69(21.1%)	0.948
AA amyloidosis	6(2.94%)	7(3.17%)	6(1.7%)	7(2.56%)	7(2.14%)	0.797
Lupus nephritis	5(2.45%)	2(0.9%)	7(2%)	2(0.73%)	6(1.83%)	0.497
C3GN	2(0.98%)	3(1.36%)	3(0.85%)	13(4.76%)	11(3.36%)	0.006
Pauci immune GN	10(4.9%)	3(1.36%)	8(2.27%)	10(3.66%)	2(0.61%)	0.012
LCDD	4(1.96%)	3(1.36%)	7(2%)	1(0.37%)	1(0.31%)	0.142
MPD	7(3.43%)	3(1.36%)	5(1.42%)	9(3.3%)	12(3.67%)	0.214
AL amyloidosis	4(1.96%)	0(0%)	7(2%)	1(0.37%)	3(0.92%)	0.094
Diabetic nephropathy	4(1.96%)	0(0%)	10(2.84%)	6(2.2%)	4(1.22%)	0.121
Crescent GN	1(0.49%)	1(0.45%)	1(0.28%)	2(0.73%)	1(0.31%)	0.927
Others	6(2.94%)	2(0.9%)	1(0.28%)	2(0.73%)	2(0.73%)	0.028

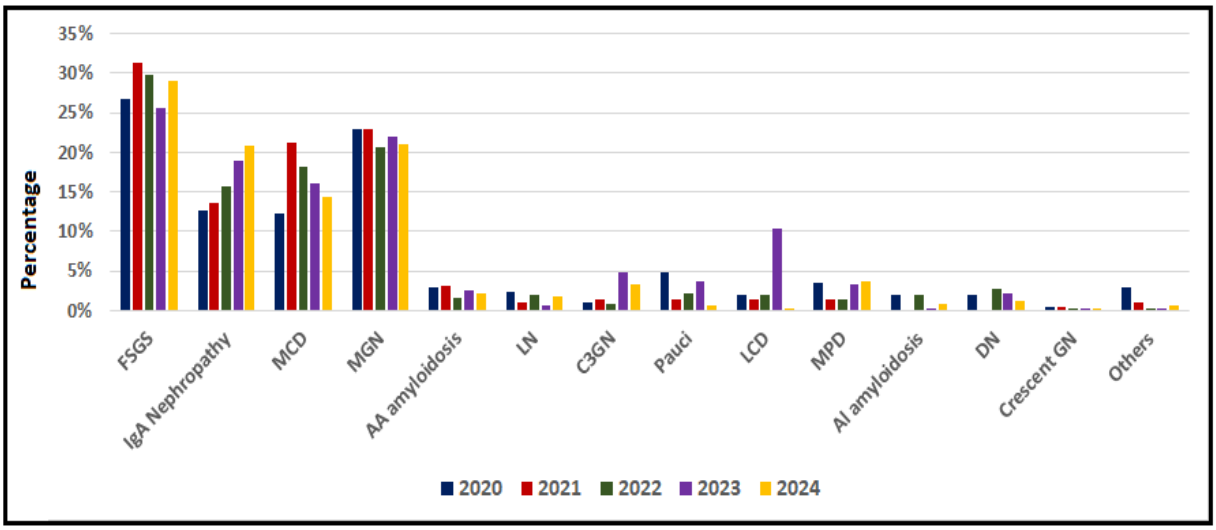

Figure 3-3: Year-wise distribution of glomerular diseases.

DISCUSSION

Glomerular diseases are common causes of CKD. Their epidemiology differs between different age groups, with MCD being the leading cause of NS in childhood, while membranous nephropathy and FSGN are more common in adulthood.[26] The present study aimed to determine the prevalence and spectrum of glomerular diseases diagnosed by renal biopsy in Iraq over a five-year period (2020–2024).

According to the result of the study, the mean age of patients was 33.24±16.3 years. Patients were distributed across four age categories: 336 (24.40%) were aged 0.5-20 years, 602 (43.72%) were 21-40 years, 361 (26.22%) were 41–60 years, and 78 (5.66%) were older than 60. A study from Iraq included 520 patients found an age ranges of 12 to 66 years which is very close to ours.[27] Another Iraqi study included 80 patients with an age range from 17 to 62 years.[28] In Iran, Mohammadhoseiniakbari et al.[29] investigated 400 renal biopsies to determine the pattern of glomerulonephritis among Iranian patients. The mean age of the patients was 31.9±15.9 (range: 3-78 years). In Saudi Arabia, AlFaadhel et al.[30] examined 1760 native renal biopsies. The mean age of the patients was 36.3±12.3 years.

These comparisons suggest that in the Middle East region, including Iraq, Iran, and Saudi Arabia, glomerular diseases are commonly diagnosed in the second to fourth decades of life, often affecting patients in their most productive years. The presence of pediatric cases is also notable, emphasizing the importance of early detection and intervention.

In the current study, male predominance was found against females 56.06% versus 43.94%. Many local and worldwide studies were in agreement with these findings among both children and adults.[27,28,29,30]

In the present study, different types of glomerular disease were encountered, primary glomerulonephritis (PGN) in descending order they were FSGS in 28.54%, MN in 21.79%, MCD in 16.63%, and IgA nephropathy (IgAN) in 16.34%. Secondary glomerulonephritis (SGN) were less frequently encountered such as AA and AL amyloidosis, lupus nephritis (LN), C3 GN, Pauci-immune GN, LCDD, diabetic nephropathy, crescentic GN, and MPGN.

The prevalence of glomerular disease varies significantly in different geographic region. For example, East Asia, Australia, North America, and southern Europe countries, such as Italy, Spain, Hungary, France, the Netherlands, and the Czech Republic have a high prevalence of IgAN (20% to 40%), whereas the US and UK, as well as Canada, South America, and Africa, have a low prevalence (2% to 10%) of IgAN and in whom FSGS is more common.[31-33]

Locally, Shaker et al.[27] reported that in 85.5% biopsies labeled as PGN, 26.3% FSGN, 22.5% of mesangial proliferative glomerulonephritis, 17.1% MCD, 16.2% MPGN, 14.5% of MGN, and 3.4% of rapidly progressive GN. The SGN included 45.5% cases of LN, 27.3% of amyloidosis, 14.5% of DN, 10.9% of hereditary nephritis, and 1.8% hypertensive nephropathy. Likewise, Al-Saedi et al.[28] found MPGN in 40% of the biopsies, FSGS in 20%, MGN in 25%, MCD in 10%. Of the SGN group, renal amyloidosis was seen in 5%.

A recent study from China on 15,146 cases of glomerular diseases found that the most common PGN was IgAN (44.6%), followed by MCD (24.3%) and MN (15.4%).[34] Another study also from China on 9310 biopsy found IgAN (42.83%), MN (19.16%), MCD (12.46%), and FSGS (14.97%) were the 4 most common pathological types.[35]

A study from Korea on 1,924 biopsies retrospectively analyzed for histopathologic results found that IgAN was the most common (37.4%), followed by followed by MCD (8.8%), MN (7.6%), FSGS (6.8%), crescentic GN (2.3%) and others (1.1%). In accordance with our results SGN constituted 10.3% of the total cases. Lupus nephritis was the most common secondary glomerular disease (4.6%), followed by Henoch-Schönlein purpura nephritis (2.0%), DN (1.3%) and others (1.9%).[36]

Regarding the age distribution of glomerular disease in the current study, FSGN was the most common across all age groups. IgAN was most common among patients aged 21-40 years, and least in those older than 60 years. MCD on the other hand, it was highly prevalent in the 0.5-20 age group and much less in older groups. those with GN, show progressive increment with aging. The SGN group as AA Amyloidosis was rare in children but increased in older groups. LN on the other hand, was predominant in younger age groups and absent in those older than 60. DN was absent in individuals younger than 41 years.

Worldwide studies noted the incidence of glomerular disease varies according to age. Previous studies have shown that MN is the most common cause of adult NS whereas MCD is the predominant cause of nephrotic syndrome in children.[31] In elderly patients, the relative proportion of crescentic glomerulonephritis, MN, and FSGS is higher than in younger patients.[37] A Brazilian study found that age distribution of the main glomerular diseases showed FSGS is the most prevalent glomerular disease in the 1st and 2nd decades of life but is surpassed by LN between the 3rd and 5th decade and by MGN in the 7th decade. The prevalence of LN increases from the 1st to the 4th decade and declines from the 5th to the 7th decade. The prevalence of MGN progressively increases from the 1st to the 8th decade and becomes the most common glomerular disease between the 7th and 8th decades of life. The prevalence of MPGN increases from the first to the 5th decade and becomes the 3rd most prevalent glomerulopathy pattern in the 8th decade of life.[38]

In Japan, a nationwide, web-based, registry system on 32,254 records, 3526 (10.9%) biopsies were performed in pediatric patients; 19,658 (60.9%) in younger adults (19–64 years); and 9070 (28.1%) in older adults (≥ 65 years) found the predominant renal biopsy diagnoses were IgAN, MCD, and IgA vasculitis were the predominant diagnoses in pediatric patients (< 19 years),

IgAN, LN and MCD in younger adults (19-64 years), and MN, antineutrophil cytoplasmic antibody-associated vasculitis or anti-glomerular basement membrane glomerulonephritis, and IgAN in older adults (≥ 65 years).[4]

These regional and global patterns reinforce that glomerular disease profiles are strongly age-dependent. The variations reflect underlying immunologic, genetic, and systemic disease processes that evolve with age. The current study's findings are consistent with global trends, particularly the predominance of MCD in children, LN in young adults, and the emergence of secondary and chronic degenerative glomerulopathies in the elderly.

Regarding sex-wise distribution, the present study demonstrated that that among all individuals who underwent kidney biopsy, FSGN, MCD, MN, DN were the most common among both sexes with no significant difference. On the other hand, IgAN and MPGN showed a statistically significant male predominance whereas, LN had a significant strong female predominance. AA Amyloidosis, C3 GN, Pauci-immune GN, and crescentic GN were insignificantly more common in males and AL amyloidosis was insignificantly more common in females.

In general, glomerular disease as a single entity has a marked male preponderance, and males outnumber females affected in all of the PGN.[39] LN is the only glomerular disease in which more females are affected[40], although sex differences in anti–neutrophil cytoplasmic antibody (ANCA)-associated vasculitis may be changing as occupational and recreational exposure to environmental hazards evolve.[39]

Different studies worldwide showed different distribution, nephrosclerosis and DN were diagnosed more frequently in males, whereas LN and thin basement membrane disease were diagnosed more frequently in females; these sex distributions were reported in the US, Poland, Czech Republic, and Japan.[39, 41,42]

Japanese researchers investigated sex differences in GN frequencies stratified by age categories. They noticed MCD and MN were predominant in men aged < 20 and > 40 years, respectively, whereas IgA vasculitis and antineutrophil cytoplasmic antibody-associated vasculitis or anti-glomerular basement membrane GN were predominant in women in their 20s and 30s and aged < 50 years, respectively. IgAN was predominant in men at most ages and in women in their 20s to 40s.[4]

On the contrary, O'Shaughnessy et al.[39] reported that DN and nephrosclerosis were diagnosed more frequently in males.

These difference between male and female could be attributed for several factors such as hormonal, genetic, and autoimmune predispositions that influence the prevalence of certain diseases (e.g., LN in females). Furthermore, environmental and lifestyle factors, including smoking, hypertension, and occupational exposures, may contribute to higher rates of PGN, DN, and nephrosclerosis in males.

In the present study, there was a slight fluctuation in the rates of different GN over the 5 years from 2020 to 2024, yet it was statistically significant. A study from Korea showed ascending rate of prevalence of IgAN but a descending prevalence of MN over the time[43], whereas the prevalence of FSGS has been increasing in the US, Brazil and India.[32, 33,44,45]

In Britain, a significantly increased proportion of cases of IgAN but a decreased proportion of MN and a constant proportion of FSGS cases were reported.[44] On the other hand, the percentages of patients diagnosed with MPGN and IgAN decreased, whereas those with IgA vasculitis and DN increased over the decade.[4]

An American study demonstrated increasing pattern of FSGS and DN over time.[39] The study assumed environmental factors such as air pollution or lifestyle factors such as obesity and diabetes mellitus as risk factors.

The Japanese and Chinese studies demonstrated that the proportion of patients with an IgAN decreased steadily in adults[4,46], whereas the frequency of IgAN diagnoses was stable in the USA[47], and increased in Germany.[48]

In the context of the present study, the statistically significant fluctuations in glomerular disease patterns over five years likely mirror these complex, multifactorial influences. The findings are in line with global evidence showing that the prevalence of individual glomerular diseases is not static, but shifts with time due to demographic, environmental, and epidemiological transitions.

Study limitations

1. The study is retrospective in nature, which limits the ability to establish causality and may be subject to documentation bias.
2. The data is derived solely from patients in Iraq, which may limit the generalizability of findings to other populations with different genetic, environmental, or healthcare system factors.
3. Only patients who underwent renal biopsy were included, potentially excluding milder cases or those managed empirically, leading to selection bias favoring more severe or atypical presentations.
4. The study focuses on histopathological diagnosis and demographic patterns without incorporating patient outcomes, treatment responses, or progression data.

5. CONCLUSIONS AND RECOMMENDATIONS

5.1 CONCLUSIONS

1. Focal segmental glomerulosclerosis is the most common glomerular disease across all demographics and years, with a relatively stable distribution
2. IgA Nephropathy showed a significant male predominance and a rising trend over the years, particularly affecting individuals in the 21–40 age group, highlighting a potentially increasing burden of immune-complex glomerulopathies.
3. Minimal change disease was significantly more common in younger age groups, especially those under 20 years, and showed no sex-based or year-wise differences, supporting its typical pediatric and young adult prevalence.
4. Membranous GN displayed an age-related increase, being more common in older age groups, but was not associated with sex or temporal changes.
5. Lupus nephritis showed a clear female predominance and was largely confined to patients under 40, reflecting the well-known autoimmune pattern of this disease.
6. C3 Glomerulopathy exhibits a statistically significant increase in prevalence over the years, suggesting emerging trends in complement-mediated glomerular diseases.
7. Pauci-immune GN also shows a significant increase with age and fluctuating year-to-year prevalence, pointing toward a growing diagnostic awareness or changing environmental/immune triggers.
8. Monoclonal-related diseases such as LCDD and AL amyloidosis are strongly associated with older age and had low but meaningful representation.
9. Membranoproliferative GN was significantly more frequent in younger males, and diabetic nephropathy appeared predominantly in patients over 40, consistent with known risk factors.

5.2 Recommendations

1. Early screening and biopsy protocols, particularly in young males with hematuria or proteinuria, to catch rising cases of IgA nephropathy and MPGN early.
2. Promote registry development for glomerular diseases in Iraq to track trends over time, analyze treatment outcomes, and guide public health policies.
3. Further research is warranted into the environmental, genetic, and infectious contributors to the increasing trends in immune and complement-mediated glomerular diseases (e.g., IgA nephropathy, C3GN, Pauci-immune GN).

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