Spectrum of Glomerular Diseases Among Adult Multinational Patients from a Single Center in the United Arab Emirates: A Case Series

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Abstract- This case series presents a retrospective analysis of glomerular diseases among adult multinational patients from a single center in the United Arab Emirates (UAE). Glomerular diseases encompass a wide range of kidney disorders that can progress to chronic kidney disease and end-stage renal failure if not appropriately managed. The study aims to characterize the types and clinical features of glomerular diseases within the UAE, taking into account the diverse population residing in the country. By reviewing medical records of adult patients diagnosed with glomerular diseases during a specified time frame, comprehensive data regarding demographic information, clinical presentation, laboratory findings, and kidney biopsy results were collected and analyzed. The study population consisted of multinational patients, effectively reflecting the multicultural composition of the UAE. Through this investigation, the case series provides valuable insights into the spectrum of glomerular diseases in the UAE and contributes to enhancing the understanding and management of these conditions within a diverse patient population.

Index Terms- Glomerular diseases, Adult patients, Multinational patients, incidence of kidney disease.

I. INTRODUCTION

Kidney diseases, particularly glomerular diseases, pose a significant burden on global health. Understanding the diverse spectrum of glomerular diseases is crucial for accurate diagnosis and appropriate management. This case series aims to shed light on the prevalence and characteristics of glomerular diseases among a multinational adult patient population from a single center in the United Arab Emirates (UAE).

A total of 20 kidney biopsies performed at the Iranian Hospital were retrospectively reviewed. Each biopsy was conducted using sterile technique and ultrasound guidance, with local anesthesia administered through lidocaine (20 mg/ml). A semiautomatic biopsy needle gauge 18 from Bard company was used to obtain two or sometimes three specimens. One core needle biopsy sample was fixed in 10% formalin for Hematoxylin and Eosin (H&E) staining and subsequent pathology review. The second sample was placed in Michel's solution for immunofluorescence studies, while the third sample was fixed using Glutaraldehyde for electron microscopy analysis. Furthermore, special stains including Periodic Acid-Schiff (PAS), Jone's, Masson, and Amyloid stains were performed on the formalin-fixed specimens, in addition to H&E staining.

The primary objective of this case series is to provide insights into the spectrum of glomerular diseases among adult patients(8,9) from diverse nationalities who sought care at a single center in the UAE. By examining the histopathological findings, immunofluorescence patterns, and electron microscopy features, we aim to enhance our understanding of the prevalence, clinical presentations, and diagnostic challenges associated with glomerular diseases in this region.

This case series will contribute valuable information to the existing knowledge base on glomerular diseases in the UAE, focusing on the adult population and encompassing patients from various nationalities. The comprehensive evaluation of kidney biopsies, including histopathology, immunofluorescence, and electron microscopy, along with the application of special stains, will facilitate a thorough characterization of the glomerular diseases encountered. The findings from this study may aid in optimizing diagnostic approaches, tailoring treatment strategies, and improving patient outcomes in the context of glomerular diseases in the UAE and beyond.

II. CASE PRESENTATION

Patient 1

A 63-year-old male presented with clinical symptoms of nephrotic syndrome, including proteinuria of 21gms/24 hr urine, microscopic hematuria, hypertension, and renal impairment. The patient tested negative for ANA. Microscopic examination revealed normal cellularity, absence of sclerosis, crescent, cellular proliferation, and basement membrane thickening, as well as no deposits, interstitial inflammation, or blood vessel thickening. Occasional hyaline cast was observed. Congo red stain test was negative. Immunofluorescence assay showed no glomerular deposits of IGG, IgA, IgM, or C3. Electron microscopy was not performed. The diagnosis was minimal change disease. Electron microscopic findings indicated enlarged glomeruli, irregular proliferation of endothelial and mesangial cells with neutrophilic infiltration, and mild presence of epithelial foot process effacement without electron dense deposits.

Patient 2

A 63-year-old female presented with clinical symptoms of nephrotic syndrome, including positive proteinuria (9.5gm/24 hr urine), negative microscopic hematuria, negative hypertension, and positive renal impairment. The patient tested negative for ANA. Microscopic examination revealed increased cellularity, absence of sclerosis and crescents, increased cellular proliferation of endothelial and mesangial cells with some neutrophil infiltration, presence of basement membrane thickening and blood vessel thickening. Immunofluorescence assay showed no glomerular deposits of IgA and IgM, but 2+ mesangial and membrane deposits of IgG, and 1+ mesangial and membrane deposits of C3. The diagnosis was membranous glomerulonephritis. Electron microscopic findings indicated enlarged glomeruli, irregular proliferation of endothelial and mesangial cells with neutrophilic infiltration, diffuse foot process flattening of all loops, and subepithelial and intermembranous electron-dense deposits. No sclerosis was observed.

Patient 3

A 22-year-old female presented with clinical features consistent with nephrotic syndrome, including proteinuria of less than 3 grams in 24-hour urine collection and positive microscopic hematuria. Hypertension, renal impairment, and antinuclear antibody (ANA) were negative. Microscopic examination revealed increased cellularity without sclerosis or crescent formation. Cellular proliferation was observed in the mesangial region, but basement membrane thickening was absent. Mesangial deposits were detected, along with hyaline and few red blood cell casts. Mild tubular atrophy and focal moderate acute inflammation in the interstitium were noted, while blood vessel thickening was absent. Immunofluorescence assay showed no glomerular deposit of IgG, strong positive granular mesangial deposition of IgA, focal weak positive granular mesangial deposition of IgM, and weak positive granular mesangial deposition of C3. The diagnosis was IgA nephropathy with mesangioproliferative pattern and acute interstitial nephritis. Electron microscopic findings were not performed.

Patient 4

A 23-year-old male presented with nephrotic syndrome characterized by proteinuria of 17.5gms in 24-hour urine, positive microscopic hematuria, hypertension, renal impairment, and various clinical manifestations. Microscopic examination revealed the presence of global and segmental sclerosis, moderate interstitial fibrosis, mild tubular atrophy, the presence of crescents, mesangial expansion, increased mesangial cellularity, and absence of glomerular basement membrane abnormalities. Mild to moderate interstitial inflammation was also observed. Immunofluorescence assay indicated diffuse positive staining for IgA and C3, while IgG and IgM were absent. Fibrinogen staining was negative in areas of crescents. The diagnosis was IgA dominant glomerulonephritis with crescentic features. Electron microscopy showed a normal basement membrane, prominent mesangial electron dense deposits, increased mesangial matrix, and large subepithelial and subendothelial deposits. The effacement of epithelial foot processes was moderate to severe.

Patient 5

An 18-year-old male presented with nephrotic syndrome characterized by microscopic proteinuria but negative findings for hematuria, hypertension, impaired renal function, and ANA. Microscopic examination revealed the presence of global sclerosis, absence of hypercellularity, segmental sclerosis, crescents, fibrinoid necrosis, interstitial fibrosis, tubular atrophy, interstitial inflammation, and casts. The arteries appeared unremarkable, but tubular cytoplasmic swelling was present. Immunofluorescence assay showed negative staining for IgG, IgM, IgA, C3, C1Q, fibrinogen, kappa, and lambda. The diagnosis was minimal change disease. Electron microscopy demonstrated a normal basement membrane with the absence of electron-dense deposits and widespread effacement of epithelial foot processes. The tubular basement membrane appeared normal.

Patient 6

A 33-year-old female presented with positive microscopic hematuria but negative findings for nephrotic syndrome, hypertension, renal impairment, and ANA. Microscopic examination revealed normal cellularity, segmental sclerosis, absence of crescents and cellular proliferation, no basement membrane thickening, segmental mesangial deposits, hyaline casts, mild tubular atrophy, mild interstitial inflammation with focal acute inflammation, mild interstitial fibrosis, and moderate hyaline arteriolar sclerosis. Immunofluorescence assay indicated no glomerular deposits of IgG, absence of glomerular deposits for IgM, C3, and fibrinogen, and granular mesangial deposits of IgA (3+). C1Q staining was negative. The diagnosis was IgA nephropathy with a focal segmental pattern and acute interstitial nephritis. Electron microscopy was not performed in this case.

Patient 7

A 35-year-old female presented with positive microscopic hematuria but negative findings for nephrotic syndrome, hypertension, renal impairment, and ANA. Microscopic examination revealed normal cellularity, segmental sclerosis, absence of crescents and cellular proliferation, no basement membrane thickening, segmental mesangial deposits, absence of polymorphs, hyaline casts, mild tubular atrophy, mild interstitial inflammation with focal acute inflammation, mild interstitial fibrosis, and moderate hyaline arteriolar sclerosis. Immunofluorescence assay indicated no glomerular deposits of IgG, absence of glomerular deposits for IgM, C3, and fibrinogen, and granular mesangial deposits of IgA (3+). C1Q staining was negative. The diagnosis was IgA nephropathy with a focal segmental pattern and acute interstitial nephritis. Electron microscopy was not performed in this case.

Patient 8

A 26-year-old male presented with negative findings for nephrotic syndrome, proteinuria, microscopic hematuria, hypertension, renal impairment, and ANA. Microscopic examination revealed normal cellularity, absence of sclerosis, crescents, and cellular proliferation, diffuse basement membrane thickening, subendothelial deposits, absence of polymorphs, no tubular atrophy or casts, mild acute interstitial inflammation, absence of interstitial fibrosis, and normal blood vessel thickening. Immunofluorescence assay indicated granular 3+ staining for IgG in the glomerular basement membrane, absence of glomerular deposits for IgA and IgM, granular 2+ staining for C3 in the glomerular basement membrane, and negative staining for C1Q and fibrinogen. The diagnosis was membranous nephropathy. Electron microscopy was not performed in this case.

Patient 9

An 18-year-old male presented with nephrotic syndrome characterized by positive findings for proteinuria (22.5 grs/24 hr urine) but negative results for microscopic hematuria, hypertension, and renal impairment. Microscopic examination revealed normal cellularity, absence of sclerosis, hypercellularity, crescents, basement membrane thickening, interstitial inflammation, tubular atrophy, casts, interstitial fibrosis, and blood vessel thickening. Immunofluorescence assay indicated no glomerular deposits of IgG, IgA, IgM, C3, C1Q, fibrinogen, kappa, and lambda. The diagnosis was minimal change disease. Electron microscopy showed a normal basement membrane, absence of electron-dense deposits, widespread effacement of epithelial foot processes, and microvillous transformation of the podocytes. No deposits were observed in the tubular basement membrane.

Patient 10

A 20-year-old male presented with clinical manifestations of nephrotic syndrome, including urinary protein excretion of 11.2 gms/24 hrs, microscopic hematuria, hypertension, renal impairment, and positive ANA indicative of lupus. Microscopic examination revealed increased cellularity, diffuse mesangial and endocapillary proliferation, absence of sclerosis, increased polymorphs in intracapillary and mesangial regions, presence of crescents, patchy thickening of capillary basement membrane, subendothelial deposits, mild focal tubular atrophy and interstitial fibrosis, hyaline and granular casts in tubular lumina, and focal chronic inflammation. Immunofluorescence assay showed granular deposition of IgA, IgG, IgM, C3, C1Q, kappa light chain, and intense deposition of lambda light chain in capillary wall and mesangial regions. The diagnosis was crescentic and diffuse proliferative glomerulonephritis. Electron microscopic findings were not performed.

Patient 11

A 15-year-old female presented with clinical findings indicative of nephrotic syndrome. However, further analysis revealed negative results for nephrotic syndrome, including proteinuria (<3 grams/24hr), microscopic hematuria, hypertension, renal impairment, and basement membrane thickening. The patient tested positive for antinuclear antibodies (ANA) associated with lupus. Microscopic examination showed normal cellularity, focal and segmental sclerosis, absence of crescents and cellular proliferation, and no thickening of the basement membrane. Deposits were observed, specifically mesangial deposits, while polymorphs and casts were absent. Additionally, there were no signs of interstitial inflammation, fibrosis, or blood vessel thickening. Immunofluorescence assay indicated the presence of IgG, IgA, IgM, C3, C1Q, and fibrinogen deposits in a mesangial, coarse granular pattern. The final diagnosis was focal segmental glomerulosclerosis, while electron microscopic findings were not performed.

Patient 12

A 45-year-old female presented with nephrotic syndrome, including positive proteinuria, microscopic hematuria, and hypertension. Microscopic findings revealed reduced cellularity, segmental and global sclerosis, absence of crescents and cellular proliferation, and no basement membrane thickening. Deposits were observed in the mesangial region, and acute focal interstitial nephritis was present. Immunofluorescence assay showed mesangial and glomerular basement membrane deposits of IgA and IgM, while IgG deposits were absent. The diagnosis was IgA nephropathy with focal segmental glomerulosclerosis and acute focal interstitial nephritis. Electron microscopy was not performed.

Patient 13

A 40-year-old female presented with nephrotic syndrome, characterized by positive proteinuria (5.2 grams/24 hours) and microscopic hematuria. The patient did not exhibit hypertension, renal impairment, or ANA positivity. Microscopic analysis revealed normal cellularity with focal and segmental sclerosis, the absence of crescen bts and cellular proliferation, and segmental mesangial deposits. Polymorphs and casts were absent, while mild focal acute and chronic interstitial inflammation was observed. Interstitial fibrosis was not present, but there was moderate hyalinization of arterioles. Immunofluorescence assay showed no glomerular deposits of IgG, IgA, IgM, or C3. The diagnosis was focal segmental glomerulosclerosis, with electron microscopic findings not performed.

Patient 14

A 53-year-old female presented with clinical features not indicative of nephrotic syndrome, as evidenced by negative results for proteinuria, microscopic hematuria, hypertension, renal impairment, and ANA. Microscopic examination revealed normal cellularity with the absence of sclerosis, crescents, cellular proliferation, basement membrane thickening, and deposits. Polymorphs were absent, but hyaline casts and focal acute tubular necrosis with tubulitis were observed. Mild lymphocytic interstitial inflammation was present, while interstitial fibrosis and blood vessel thickening were absent. Immunofluorescence assay showed no glomerular deposits of IgG, IgA, IgM, C3, C1Q, or fibrinogen. The diagnosis was unremarkable glomeruli with acute tubulo-interstitial inflammation, and electron microscopic findings were not performed.

Patient 15

A 51-year-old male presented with clinical features not suggestive of nephrotic syndrome, as indicated by negative results for proteinuria, microscopic hematuria, hypertension, and ANA. However, renal impairment was observed. Microscopic examination revealed normal cellularity with the absence of sclerosis, crescents, cellular proliferation, and basement membrane thickening. Focal mild segmental mesangial matrix deposits were observed, while polymorphs and interstitial inflammation were absent. No interstitial fibrosis was observed, but blood vessel thickening was present. Immunofluorescence assay showed no glomerular deposits of IgG, IgA, IgM, C3, C1Q, or fibrinogen. The diagnosis was minimal change disease, with electron microscopic findings not performed.

Patient 16

A 45-year-old female presented with clinical features not indicative of nephrotic syndrome, as demonstrated by negative results for proteinuria and microscopic hematuria. However, the patient exhibited hypertension and renal impairment. Microscopic examination revealed normal cellularity with segmental and global sclerosis present (2/5 and 1/5, respectively). There were no crescents or cellular proliferation, and basement membrane thickening was absent. Segmental mesangial deposits were observed, while polymorphs were absent. Mild focal interstitial inflammation and no interstitial fibrosis were present. Blood vessel thickening included hyaline arteriolosclerosis and mild fibrointimal hyperplasia. Immunofluorescence assay showed no glomerular deposits of IgG, IgA, IgM, C3, C1Q, or fibrinogen. The diagnosis was focal segmental glomerulosclerosis, with electron microscopic findings not performed.

Patient 17

A 37-year-old male presented with nephrotic syndrome, characterized by positive proteinuria (7 grams/24 hours) but negative microscopic hematuria and hypertension. Renal impairment was observed while ANA test yielded negative results. Microscopic analysis revealed mild increased cellularity with the presence of focal segmental and mesangial sclerosis. There were no crescents or cellular proliferation, and basement membrane thickening was absent. Mesangial deposits were present, while polymorphs were absent. Mild focal interstitial inflammation consisting of mononuclear cells was observed, but interstitial fibrosis was absent. Blood vessel thickening included the presence of hyaline arteriolosclerosis. Immunofluorescence assay showed no glomerular deposits of IgG, IgA, IgM, C3, C1Q, or fibrinogen. The diagnosis was focal segmental glomerulosis, and electron microscopic findings were not performed.

Patient 18

A 17-year-old male presented with clinical features not indicative of nephrotic syndrome, as indicated by negative results for proteinuria and hypertension. However, the patient exhibited positive results for microscopic hematuria. Renal impairment and ANA test yielded negative results. Microscopic examination revealed normal cellularity with the absence of sclerosis, crescents, cellular proliferation, basement membrane thickening, deposits, polymorphs, casts, interstitial inflammation, interstitial fibrosis, and blood vessel thickening. Immunofluorescence assay showed no glomerular deposits of IgG, IgA, IgM, C3, C1Q, or fibrinogen. The diagnosis was unremarkable glomeruli, and electron microscopic findings revealed normocellular glomeruli with intact foot processes, normal basement membrane, absence of sclerosis, and no electron-dense deposits.

Patient 19

A 32-year-old male presented with clinical features not indicative of nephrotic syndrome, as demonstrated by negative results for proteinuria and hypertension. However, the patient exhibited positive results for microscopic hematuria. Renal impairment and ANA test yielded negative results. Microscopic examination revealed mild increased cellularity with globally present sclerosis. There were no crescents, but cellular proliferation of mesangial cells was observed. Basement membrane thickening and deposits were absent, and polymorphs were absent as well. Mild focal tubular atrophy and the presence of hyaline casts were observed. Mild focal interstitial inflammation consisting of mononuclear cells and interstitial fibrosis were present. Blood vessel thickening included mild medial thickening, subintimal sclerosis, and subendothelial hyalinosis of arterioles. Immunofluorescence assay showed no glomerular deposits of IgG and IgM, but mesangial deposits of IgA (3+), C3 (2+), C1Q, kappa light chain (1+), and lambda light chain were observed. The diagnosis was IgA nephropathy with mild focal and segmental increased in mesangial matrix, and electron microscopic findings were not performed.

Patient 20

A 38-year-old male presented with nephrotic syndrome characterized by proteinuria (<3 grams/24 hours) and microscopic hematuria (1+). Hypertension was negative, and impaired renal function was not present. Microscopic examination revealed normal cellularity with focal segmental sclerosis and absence of crescents and cellular proliferation. Basement membrane thickening was absent, while mesangial deposits were present. Polymorphs were absent, and hyaline casts were observed. Mild focal interstitial inflammation consisting of mononuclear cells was present, but interstitial fibrosis was absent. Blood vessel thickening included the presence of hyaline arteriolosclerosis. Immunofluorescence assay showed no glomerular deposits of IgG and C3, but mesangial deposits of IgM (2+) were observed. C1Q and fibrinogen yielded negative results. The diagnosis was focal segmental glomerulosclerosis, and electron microscopic findings were not performed.

III. DISCUSSION

In this case series we analyzed the clinical and pathological characteristics of 20 patients with glomerular diseases. Our aim was to determine the most frequent type of glomerular disease and compare it with the commonly reported types in textbooks. Additionally, we explored the reasons for any differences in the prevalence of glomerular diseases in our study population, including geographic area, race, and the potential influence of special human leukocyte antigen (HLA) typing studies. We also compared our findings with similar studies conducted in other centers in different countries.

Among the 20 patients included in our case series, the most frequent glomerular disease was focal segmental glomerulosclerosis (FSG), accounting for 25% (5/20) of the cases. This was followed by minimal change disease, which accounted for 20% (4/20) of the cases. In comparison, textbooks commonly report focal segmental glomerulosclerosis and membranous glomerulonephritis as the most frequent glomerular diseases in adults. Our study population showed a quite similar distribution, with FSG and minimal change disease being the most prevalent types.

DIAGNOSIS	Count of DIAGNOSIS
Focal segmental glomerulosclerosis	5
Minimal change disease	4
IgA nephropathy with focal segmental pattern and acute interstitial nephritis	2
IgA nephropathy with focal segmental Glomerulosclerosis with acute focal interstitial nephritis	1
Membranous nephropathy	1
Membranous glomerulonephritis	1
Unremarkable glomeruli	1
IgA Dominant glomerulonephritis, crescentic	1
Unremarkable glomeruli with acute Tubulo-intestinal inflammation	1
IgA nephropathy with mild focal and segmental increased in mesangial matrix	1
Crescentic and diffuse proliferative glomerulonephritis	1
IgA nephropathy with mesangioprolifierative pattern with acute interstitial nephritis	1
Grand Total	**20**

These similarities in the distribution of glomerular diseases between our study and textbook reports suggest that our findings align with the expected patterns observed in the literature. However, it is important to acknowledge the potential influence of geographic and racial factors on disease prevalence. While our study population is multinational, the United Arab Emirates is located in a region with a unique genetic and ethnic composition, which could contribute to variations in disease prevalence compared to other populations.

To gain a broader perspective, it is essential to compare our findings with studies conducted in other centers and countries. Such comparative studies allow for a better understanding of the global distribution and characteristics of glomerular diseases. By analyzing data from different populations, we can identify common trends and potential regional variations, which may contribute to our understanding of the pathogenesis and risk factors associated with these diseases.

Regarding the age and gender distribution of glomerular diseases in our case series, we observed a wide age range, with patients ranging from 15 to 63 years old. There was no significant gender bias, as both males and females were equally represented. These findings are consistent with previous reports that show glomerular diseases can affect individuals across a broad age spectrum and are not limited to a specific gender.

The correlation between microscopic, electron microscopic, and immunofluorescence findings and the severity of disease signs and symptoms is an important aspect to consider in the management of glomerular diseases. In our case series, we observed various histopathological findings, including cellularity, sclerosis, crescents, cellular proliferation, basement membrane thickening, and deposits. The presence and severity of these findings were associated with the clinical manifestations of each patient, such as proteinuria, hematuria, hypertension, and renal impairment. These observations highlight the importance of comprehensive pathological evaluation, including immunofluorescence and electron microscopy, in establishing an accurate diagnosis and guiding appropriate treatment strategies.

Screening for glomerular diseases and the potential benefits of early detection are topics of significant interest in the field of nephrology. Identifying individuals at risk and implementing screening programs can lead to the early identification and intervention in glomerular diseases, which may prevent the progression to advanced stages and the development of irreversible kidney damage. Further research is needed to determine the most effective screening strategies, considering the cost-effectiveness and feasibility in different populations.

Lastly, in difficult and complicated cases, electron microscopy plays a crucial role in providing a definite diagnosis. Our case series highlights the significance of electron microscopic findings in confirming and elucidating complex cases. The visualization of glomerular basement membrane integrity, mesangial matrix alterations, and the presence or absence of electron-dense deposits can provide valuable information for accurate diagnosis and guide treatment decisions.

IV. CONCLUSION

In conclusion, our case series on the spectrum of glomerular diseases among adult multinational patients from a single center in the United Arab Emirates demonstrates similarities in the prevalence of glomerular diseases to what is commonly reported in textbooks. The most common types observed in our study were focal segmental glomerulosclerosis and minimal change disease, aligning with the expected patterns. These findings suggest that our study population shares similarities with other populations regarding the distribution of glomerular diseases. Our study emphasizes the importance of comprehensive pathological evaluation, including electron microscopy and immunofluorescence, in establishing accurate diagnoses and guiding treatment decisions. Additionally, further research is needed to explore the potential benefits of screening programs and the necessity of electron microscopic studies in difficult and complicated cases.

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