IJIMM, Volume 2, Issue 6, 2024 ISSN: 2995-5319
http://medicaljournals.eu/index.php/IJIMM/issue/view/3

INTERNATIONAL JOURNAL OF INTEGRATIVE AND MODERN MEDICINE

Risk Factors, Prevalence, and Epidemiology of Glomerulonephritis

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Abstract: The term glomerulonephritis encompasses a group of kidney diseases characterized by immune-mediated damage to the basement membrane, mesangium, or capillary endothelium, resulting in hematuria, proteinuria, and azotemia. Acute forms of glomerulonephritis (GN) can occur as a result of a primary renal cause or a secondary disease causing renal manifestations.

Keywords: Nephrotic glomerulonephritis, Nephritic glomerulonephritis, Epidemiology, Histopathology, Pathophysiology.

Introduction and Main Content:

The structural and functional unit of the kidney is the nephron, consisting of the renal corpuscle (glomerulus surrounded by Bowman's capsule) and renal tubules. Each adult kidney contains about 1 million nephrons. The fenestrated endothelium forms the inner layer of the glomerulus, followed by the glomerular basement membrane (GBM), a mesh-like layer made up of various extracellular matrix proteins. The outer layer consists of visceral epithelial cells (podocytes) and mesangial cells. This complex design establishes the basis for continuous volumetric filtration of plasma at the glomerular level. The term glomerulonephritis (GN) encompasses a group of kidney diseases caused by immune-mediated damage to the basement membrane, mesangium, or capillary endothelium, leading to hematuria, proteinuria, and azotemia. Acute forms of GN can occur due to primary renal causes or secondary diseases manifesting in the kidneys. For example, acute post-streptococcal glomerulonephritis (PSGN) is a typical example of acute GN secondary to streptococcal infection; similarly, Staphylococcus aureus infection can also lead to GN. However, unlike the recent decline in PSGN in the United States and many developed countries, the incidence of staphylococcus-associated GN has increased. Most forms of GN are progressive diseases. Without timely treatment, chronic glomerulonephritis develops (characterized by progressive damage with glomerular and tubulointerstitial fibrosis, leading to a decrease in glomerular filtration rate). This progression, along with cardiovascular diseases, leads to chronic kidney disease (CKD) and end-stage renal disease (ESRD), resulting in the retention of uremic toxins.

Etiology:

The etiological classification of glomerulonephritis can be made based on clinical presentation, ranging from severe proteinuria (>3.5 g/day) and edema typical of nephrotic syndrome to hematuria, hypertension, and proteinuria more characteristic of nephritic syndrome.

Nephrotic Glomerulonephritis:

- ✓ Minimal change disease
- ✓ Focal segmental glomerulosclerosis
- ✓ Membranoproliferative glomerulonephritis
- ✓ Membranous nephropathy
- ✓ HIV-associated nephropathy
- ✓ Diabetic nephropathy
- ✓ Amyloidosis

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Nephritic Glomerulonephritis:

- ✓ IgA nephropathy
- ✓ Henoch-Schönlein purpura (HSP)
- ✓ Post-streptococcal glomerulonephritis
- ✓ Anti-glomerular basement membrane disease
- ✓ Rapidly progressive glomerulonephritis
- ✓ Wegener's granulomatosis
- ✓ Eosinophilic granulomatosis with polyangiitis
- ✓ Polyarteritis nodosa
- ✓ Idiopathic crescentic glomerulonephritis
- ✓ Goodpasture syndrome
- ✓ Lupus nephritis
- ✓ Hepatitis C-associated glomerulonephritis

A more modern and widespread method for classifying GN is based on the primary immune processes, dividing GN into five forms:

1. Immune complex GN - e.g., IgA nephropathy, IgA vasculitis, infectious GN, systemic lupus erythematosus (SLE) with fibrillar GN.
2. Pauci-immune GN - e.g., PR3-ANCA GN, MPO-ANCA GN, ANCA-negative GN.
3. Anti-GBM GN - e.g., Anti-GBM GN.
4. Monoclonal Ig GN - e.g., proliferative GN with monoclonal Ig deposits, fibrillary GN with monoclonal Ig deposits.
5. C3 glomerulopathy - e.g., C3 glomerulonephritis, dense deposit disease.

Epidemiology:

Glomerulonephritis (GN) is a common cause of kidney failure, accounting for 10-15% of end-stage renal disease (ESRD) cases in the United States. Without timely intervention, the disease often progresses, leading to chronic glomerulonephritis, which is the third most common cause of ESRD after diabetes and hypertension. GN represents 25-30% of all cases of end-stage renal failure. IgA nephropathy is the most common cause of GN worldwide. The incidence of post-streptococcal GN has declined in most developed countries, while the incidence of GN associated with Staphylococcus aureus has increased. Geographic and seasonal variations in PSGN incidence are more pronounced in GN associated with pharyngeal infections compared to skin-related GN. Acute nephritis can occur at any age, but PSGN typically affects children aged 5-15, with only 10% of cases occurring in patients aged 40 and above. Acute GN is more frequent in males, with a male-to-female ratio of 2:1. Post-infectious GN does not show a racial or ethnic predisposition.

Pathophysiology:

The primary pathogenic mechanism common to all types of GN is immune-mediated, involving both humoral and cellular pathways. Subsequent inflammatory responses often lead to fibrosis. The targets of immune-mediated damage vary with the type of GN. For example, in staphylococcus-associated GN, complement IgA and C3 deposits are observed. The immune responses activate common inflammatory pathways, such as the complement system and blood coagulation cascade. Cytokines from platelets, such as platelet-derived growth factor (PDGF), contribute to glomerulosclerosis.

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Structural Changes:

Cellular proliferation increases the cellularity of the glomerular tuft due to the proliferation of endothelial, mesangial, and epithelial cells. Proliferation can be endocapillary (within glomerular capillary tufts) or extracapillary (involving parietal epithelial cells forming crescents). Thickening of the glomerular basement membrane (GBM) appears as thickened capillary walls under light microscopy, and electron microscopy reveals electron-dense deposits in or adjacent to the GBM.

Functional Changes:

- ✓ Proteinuria
- ✓ Hematuria
- ✓ Decreased creatinine clearance, oliguria, or anuria
- ✓ Active urinary sediment with red blood cells and red blood cell casts

These changes lead to intravascular volume overload, edema, and systemic hypertension.

Histopathology:

Histopathological analysis of GN typically reveals diffuse endocapillary proliferative changes. The most common histological patterns include diffuse, focal, and mesangial proliferative glomerulonephritis. Other features can include normal glomerular morphology under light microscopy, with electron microscopy showing podocyte effacement, hypercellular glomeruli, and thickened GBM with periodic acid-Schiff (PAS) stain positivity. Glomerular sclerosis can result from various causes of glomerular damage.

References:

1. Stahl R.A., Xoxa E. [Glomerulonefrit]. Dtsch Med Wochenschr. 2016.
2. Vang XY, et al. Clinical, pathological, and prognostic features of staphylococcus-associated GN. Medicine (Baltimore). 2016.
3. Khalighi M.A., et al. Staphylococcus-associated GN with cryoglobulinemia features. International Kidney Representative. 2018.
4. Satoskar A.A., et al. Epidemiology, pathogenesis, treatment, and outcomes of infection-associated GN. Nat Rev Nephrol. 2020.
5. Ven Y.K., Chen M.L. Significance of atypical morphology in the spectrum of post-infectious GN. Clin Nephrol. 2010.
6. Politano S.A., et al. Nephrotic syndrome. Primary Care. 2020.
7. Davin JC, Coppo R. Henoch-Schönlein purpura nephritis in children. Nat Rev Nephrol. 2014.
8. Ozkok A., Yildiz A. Hepatitis C virus-associated glomerulopathies. World J Gastroenterol. 2014.
9. Kauser W.G., Jonson R.J. Etiology of glomerulonephritis: role of infection and autoimmunity. Kidney Int. 2014.
10. Seti S, Fervenza. Standardized classification and reporting of glomerulonephritis. Nephrol Transplant. 2019.
11. Wetmore JB, et al. Prevalence, incidence, and outcomes of glomerulonephritis. Kidney Int. 2016.
12. Shchena F.P., Nistor I. Epidemiology of IgA nephropathy: global perspective. Semin Nephrol. 2018.
13. Usui J, et al. Temporal changes in post-infectious GN in Japan (1976-2009). PLOS One. 2016.
14. Anochi I, et al. Acute glomerulonephritis in children in Port Harcourt, Nigeria

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