IgA Nephropathy (Berger's Disease)

Berger’s disease is a nephritic kidney disease, also known as IgA glomerulopathy, that is characterized by the presence of IgA deposits in the mesangium of the kidneys, which can be detected via immunofluorescence microscopy and light microscopy. Berger’s disease is a common cause of recurrent gross or microscopic hematuria and is the most common cause of nephritic syndrome. IgA nephropathy can present as an isolated renal disease but similar IgA deposits are present in the systemic disorder Henoch Schonlein purpura. Henoch Schonelin purpura affects children and has many overlapping features with IgA nephropathy. This disease can affect people of any age but older children and young adults are most often affected. Common presentation of disease is gross hematuria after an upper respiratory infection or less commonly an acute gastroenteritis. While some patients present with only microscopic hematuria, a fraction develop a typical acute nephritic syndrome. The hematuria typically lasts for several days and then subsides and can reoccur. The clinical course is highly variable. Many patients maintain normal renal function for several decades but slow progression to renal failure can occur over a period of 20 years.

PLAY PICMONIC

Classification

Nephritic
Nerd-cricket
IgA nephropathy falls under nephritic syndrome, which means the kidneys are inflamed. You’ll mainly see blood in the urine (hematuria), sometimes red blood cell casts, and mild to moderate proteinuria instead of the heavy protein loss seen in nephrotic syndrome. Kidney function can dip too because that inflammation affects filtration.

Epidemiology

Most Common Glomerulonephritis
#1 Foam-finger Glow-mare with Kidney-on-fire
This is the number one cause of primary glomerular disease worldwide, especially in younger patients. If an exam asks for the most common primary glomerulonephritis, it’s IgA nephropathy.

Higher Prevalence in East Asians
Map of East Asia
IgA nephropathy shows up more often and tends to be more severe in East Asian populations. It’s less common in Caucasians and is rare in people of African descent, which helps with epidemiology questions.

Clinical Presentation

Synpharyngitic Hematuria after URI or GI Infection
Pharaoh Coughing and Sitting on a Toilet and Red-urinal
The classic presentation is hematuria that starts at the same time or shortly after a sore throat, cold, or GI infection. This timing is key. It helps you tell it apart from post-strep GN, which shows up weeks later, not days.

Proteinuria
Mr. Protein Urinal
Of all the things to monitor, protein in the urine tells you how the disease will progress. The more proteinuria, the higher the risk of long-term kidney damage. Persistent proteinuria is a red flag that the disease isn’t staying mild.

Pathogenesis

Alternative Complement Pathway Activation (C3)
Alternative Computer (3) Tree Path Activated
The inflammation isn’t just from IgA itself. The alternative complement pathway, especially C3, gets activated and adds fuel to the fire. This complement involvement helps explain the kidney damage seen on biopsy.

Increased Synthesis of IgA

Increased Synthesis of IgA Up-arrow Apple-goblin

The body makes more IgA during respiratory or GI infections, which contributes to IgA immune complex buildup in the kidneys. In patients with IgA nephropathy, levels of plasma IgA are increased, and there is prominent deposition of IgA immune complexes in the mesangium.

Pathology

Mesangial IgA Immune Complex Deposition on Biopsy

Maze-angel and Apple-goblin In-moon Complexes

The immune complexes made of IgA end up depositing in the mesangium, which is the central support area inside the glomerulus. Their presence is one of the hallmark features of this disease.

Treatment

RAAS Blockade is First Line

Raspberries Blocks

ACE inhibitors or ARBs are the first treatment choice. They help lower proteinuria and protect kidney function. KDIGO guidelines recommend starting RAAS blockade early as part of supportive care.

Steroids for High-risk

Steroid-stairs

If a patient still has more than 1 g/day of proteinuria after proper supportive care, systemic corticosteroids may be added. They’re not for everyone because of side effects, so they’re reserved for those with higher-risk or progressive disease.