.Glomerular Diseases

Glomerular Diseases include many conditions, but they fall into two major Types:

- **Glomerulonephritis** describes the inflammation of the membrane tissue in the kidney that serves as a filter, separating wastes and extra fluid from the blood.
- **Glomerulosclerosis** describes the scarring or hardening of the tiny blood vessels within the kidney.

Clinical Syndromes:

- **Nephritic syndrome**. (Oliguria, Haematuria, Proteinuria, Oedema.)
- **Nephrotic syndrome**. (Gross proteinuria, hyperlipidemia)
- **Acute renal failure** (Oliguria, loss of Kidney function - within weeks)
- **Chronic renal failure**. Over months and years – Uremia.

T Lymphocyte mediated Renal Injury:

The immune system is among the key pathogenic factors in acute kidney injury . Various immune cells, including dendritic cells, natural killer T cells, T and B lymphocytes, neutrophils and macrophages are involved. Conventional CD4+ lymphocytes are well established to participate in early injury,T lymphocytes accumulate in the kidney within a few hours after Acute kidney inlammmation, and CD4+CD25+FoxP3 regulatory T cells are protective and can accelerate repair

Primary glomerulonephritis

1-Membranous glomerulonephritis…..(MGN)

A slowly progressive disease of kidney affecting people between ages of 30 to 50 years old. It is the second common cause of nephrotic syndrome in adults.

Signs and symptoms : proteinuria, edema with or without renal failure.

Causes and classification :Primary/idiopathic :The disease is idiopathic (of unknown origin or cause).

Secondary :autoimmune conditions (e.g., systemic lupus erythematosus) & infections (e.g., syphilis, malaria, hepatitis B, hepatitis C) drugs (e.g., captopril, NSAIDs, penicillamine, probenecid). inorganic salts (e.g. gold, mercury).

Treatment; immunosuppressive drugs and cyclophosphamide alternating with a corticosteroid.

2-Post-infectious glomerulonephritis

It is a disorder of the glomeruli or small blood vessels in the kidneys. It is a common complication of bacterial infections, typically skin infection by *Streptococcus* bacteria types 12, 4 and 1 **(impetigo**) but also after streptococcal pharyngitis, for which it is also known as *poststreptococcal glomerulonephritis*. .

Signs and symptoms Hematuria , Oliguria ,Edema ,Hypertension ,Fever (headache, malaise, anorexia, nausea).

Diagnosis

Kidney biopsy ,Complement profile ,Imaging studies ,Blood chemistry studies

Serologically, diagnostic markers can be tested; specifically, the **streptozyme test** is used and measures multiple streptococcal antibodies: antistreptolysin, antihyaluronidase, antistreptokinase, antinicotinamide-adenine dinucleotidase, and anti-DNAse B antibodies.

Treatment: blood pressure (BP) control: A low-sodium diet may be needed when hypertension is present. In individuals with oliguric acute kidney injury, the amount of potassium should be controlled.

3-IgA nephropathy (IgAN)

also known as **IgA nephritis**, **Berger disease** or **synpharyngitic glomerulonephritis** , specifically it is a form of glomerulonephritis.

IgA nephropathy is the **most common glomerulonephritis worldwide.** Primary IgA nephropathy is characterized by **deposition of the IgA antibody in the glomerulus**.

Signs and symptoms

hematuria, which usually starts **within a day or two** of a non-specific upper respiratory tract infection, as opposed to post-streptococcal glomerulonephritis, which occurs some time (**weeks**) after initial infection, **Groin pain**, **proteinuria**, the patients may **not** have any symptoms .

Diagnosis

1-Ultrasound of the kidney and cystoscopy These tests would rule out kidney stones and bladder cancer, two other common urological causes of hematuria.

2- A kidney biopsy is necessary to confirm the diagnosis.. **3- A urinalysis** will show red blood cells, usually as red cell urinary casts. Proteinuria.

4-Other blood tests done to aid in the diagnosis include **CRP** or **ESR**, **complement levels**, **ANA**,. **Protein electrophoresis** and **immunoglobulin levels** can show increased IgA .

2- Glomerulonephritis associated with systemic disease and Vasculitis

1-Henoch–Schönlein purpura (HSP)

It is also called **IgA vasculitis**, **anaphylactoid purpura**, **HSP**, is a disease of the skin and mucous membranes that most commonly affects **children**. The skin, show palpable purpural) and there are joint pain & abdominal pain. When kidney involvement, there may be (hematuria and proteinuria). HSP is often preceded by **an infection, such as throat infection.**

HSP is **a systemic vasculitis** (inflammation of blood vessels) and is characterized by **deposition of immune complexes** containing the antibody (IgA) complement component 3 (C3) are deposited on arterioles, capillaries, and venules.; the exact cause for this phenomenon is **unknown**.

Diagnosis of HSP : No specific diagnostic laboratory test is available for HSP. The following general laboratory tests may be helpful:

Antinuclear antibody (ANA) and rheumatoid factor (RF) ,,,Factors VIII and XIII

Urinalysis and Blood urea nitrogen (BUN) and creatinine Complete blood count (CBC) and Platelet count ,,Erythrocyte sedimentation rate (ESR) and CRP,, Level of C3 and C4

Stool guaiac test:- detects the presence of fecal occult blood.

Plasma D-dimer:- (a small protein fragment present in the blood after a blood clot is degraded by fibrinolysis. It is so named because it contains two D fragments of the fibrin .

2- Goodpasture syndrome (GPS)

Is a rare autoimmune disease in which antibodies attack the basement membrane in lungs and kidneys, leading to bleeding from the lungs and kidney failure. It is thought to attack the subunit of collagen, which has therefore been referred to as Goodpasture's antigen.often leading to **death** . In Goodpasture's disease, the (anti-glomerular basement antibody disease) , or anti-GBM disease) attack the basement membrane in lungs and kidneys, leading to bleeding from the lungs and to kidney failure.

Cause: unknown, but exposure to tobacco smoke, certain gene mutations (***HLA-DR15***), infection (such as influenza A), cocaine inhalation.

Pathophysiology: GPS causes the abnormal production of anti-GBM antibodies, by the plasma cells of the blood These antibodies bind their reactive epitopes to the basement membranes and activate the complement cascade, leading to the death of tagged cells. T cells are also implicated. **It is generally considered a type II hypersensitivity reaction.**

Diagnosis: often difficult, biopsy, especially from the kidney, **serum anti-GBM antibodies,** cytoplasmic antineutrophilic antibodies ANCA are positive.