**Glomerulonephritis**

Glomerulonephritis means ‘inflammation of glomeruli’. Most types of glomerulonephritis are often caused by an immune response triggered by an infection or other disease like:

1. Post-streptococcal glomerulonephritis.
2. Infective endocarditis.
3. Systemic lupus erythematous (SLE).

**Clinical features**

1. Haematuria (visible or non-visible) – red cell casts are typically seen in urine microscopy.
2. Proteinuria.
3. Hypertension and oedema.
4. Oliguria.

**Investigations**

1. Urine tests for RBCs, cast and protein.
2. Blood urea, serum creatinine and electrolytes.
3. Serum albumin.
4. Antinuclear (ANA) and anti-DNA antibodies.
5. Hepatitis B surface antigen and hepatitis C antibody.
6. Ultrasound of kidneys.
7. Renal biopsy.

**Nephrotic syndrome**

Nephrotic syndrome is a primary glomerular disease characterized by proteinuria, hypoalbuminemia, diffuse edema, high serum cholesterol, and hyperlipidemia. It is caused by increased glomerular permeability with loss of protein in the urine. It is characterised by:

1. Hypoalbuminaemia (serum albumin <30 g/L) develops as a consequence of heavy proteinuria (>3.5 g/24 h in adults)
2. Edema: commonly occurs around the eyes (periorbital), in dependent areas (sacrum, ankles, and hands), and in the abdomen (ascites).
3. Hyperlipidemia.

**Investigations**: same as investigations of glomerulonephritis:

1. Urine tests for RBCs, cast and protein.
2. Blood urea, serum creatinine and electrolytes.
3. Serum albumin.
4. Antinuclear (ANA) and anti-DNA antibodies .
5. Hepatitis B surface antigen and hepatitis C antibody.
6. Ultrasound of kidneys.
7. Renal biopsy.

**Management**: treatment includes

1. Diuretics for edema.
2. Angiotensin-converting enzyme (ACE) inhibitors to reduce proteinuria.
3. Lipid-lowering agents for hyperlipidemia.

**gout**

Gout is inflammatory conditions related to a genetic defect of purine metabolism and resulting in hyperuricemia. Over secretion of uric acid or a renal defect resulting in decreased excretion of uric acid, or a combination of both. The incidence of gout increases with age and body mass index, and the disorder occurs more commonly in males than in females.

**Primary hyperuricemia may be due to**

* Severe dieting or starvation.
* Excessive intake of foods high in purines.
* Heredity.

**secondary hyperuricemia**

In secondary hyperuricemiathe gout is a clinical feature secondary to any of a number of genetic or acquired processes, including conditions with an increase in cell turnover (leukaemia, multiple myeloma, psoriasis, and some anaemia) and an increase in cell breakdown.

**Clinical Manifestations:**

1. Acute arthritis of gout is the most common early sign.
2. Higher serum concentrations of uric acid are associated with tophus formation.
3. Tophi are generally associated with frequent and severe inflammatory episodes.
4. Joint enlargement may cause loss of joint motion.
5. The acute attack may be triggered by trauma, alcohol ingestion, dieting, medication, surgical stress, or illness.
6. Uric acid deposits may cause renal stones and kidney damage.

**Diagnosis**

A definitive diagnosis of gouty arthritis is established by polarized light microscopy of the synovial fluid of the involved joint. Uric acid crystals are seen within the polymorphonuclear leukocytes in the fluid.

**Management**

1. Colchicine (oral), an NSAID such as indomethacin, or a corticosteroid is prescribed to relieve an acute attack of gout.
2. Uricosuric agents, such as probenecid, correct hyperuricemia and dissolve deposited urate.
3. Allopurinol is effective when renal insufficiency or renal calculi are a risk.
4. Corticosteroids may be used in patients who have no response to other therapy.