

Nephritic Syndrome

Practical bedside approach • Adults

01 RECOGNISE THE SYNDROME

- **Glomerular haematuria with variable proteinuria**, often accompanied by reduced kidney function, hypertension, oedema and oliguria.
- Urine may appear tea- or cola-coloured. **Dysmorphic red blood cells, especially acanthocytes, and red cell casts** support a glomerular source of bleeding.
- Glomerular inflammation reduces filtration; sodium and water retention cause hypertension and oedema.
- Proteinuria is often below the nephrotic range, but **nephritic and nephrotic features can overlap**.

02 IDENTIFY THE CAUSE

- Consider **IgA nephropathy, infection-related glomerulonephritis, lupus nephritis, ANCA-associated vasculitis and anti-GBM disease**.
- Ask about recent or ongoing infection, rash, joint pain, sinus symptoms, haemoptysis, medications and systemic illness.
- **Rapidly worsening kidney function suggests rapidly progressive glomerulonephritis (RPGN)** and requires urgent nephrology assessment.

03 INITIAL ASSESSMENT

- Assess blood pressure, oedema, oxygen saturation, volume status, urine output and the creatinine trend.
- Perform urinalysis and **urine microscopy**; quantify proteinuria using a urine protein-to-creatinine ratio.
- Check creatinine, potassium, bicarbonate, albumin and full blood count. Ultrasound helps exclude obstruction.
- Select serology according to presentation: **C3/C4, ANA/anti-dsDNA, MPO-/PR3-ANCA and anti-GBM antibodies**. Add infection and monoclonal protein studies when indicated.
- Kidney biopsy establishes the cause and activity; suspected RPGN usually requires an urgent biopsy.

04 TREATMENT AND MONITORING

- **Treat the underlying cause.** Infection-related GN requires treatment of the infection; immunosuppression is not routine for every nephritic presentation.
- Use disease-specific immunosuppression under specialist guidance. Anti-GBM disease usually requires urgent plasma exchange and immunosuppression.
- In strongly suspected organ-threatening ANCA vasculitis or anti-GBM disease, **treatment must not be delayed solely while awaiting biopsy**.
- Control blood pressure and fluid overload with sodium restriction and diuretics as appropriate. Individualise fluid intake.
- Monitor kidney function, potassium, acid-base status and urine output. Avoid nephrotoxins; reconsider ACE inhibitors/ARBs during AKI or hyperkalaemia.

05 COMPLICATIONS AND RED FLAGS

- **Rapid creatinine rise, oliguria/anuria, severe hyperkalaemia, pulmonary oedema or hypertensive emergency** requires urgent hospital assessment.
- **Haemoptysis, hypoxaemia or an unexplained haemoglobin fall** may indicate alveolar haemorrhage and a pulmonary-renal syndrome; haemoptysis can be absent.
- Kidney replacement therapy may be needed for refractory hyperkalaemia, acidosis, fluid overload or uraemic complications. **Do not dialyse a creatinine value alone.**

KEY MESSAGE

Nephritic syndrome suggests glomerular inflammation. Confirm glomerular haematuria, assess kidney function and identify the cause. A rapid creatinine rise or pulmonary involvement is an emergency.

REFERENCE

KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases. Kidney Int. 2021;100(Suppl):S1-S276. KDIGO 2024 Clinical Practice Guideline for ANCA-Associated Vasculitis. Kidney Int. 2024;105(Suppl):S71-S116.