

Nephrology Cases

Greenlane Medical Specialists

12 Oct 2026

Case 1

- 54 years old female presented with painless macroscopic haematuria for 3 weeks and deteriorating renal function
- Previously investigated for microscopic hematuria in 2021 – normal cystoscopy, nil urine albumin, eGFR >90 ml/min
- Drinks 2 litres of fluid a day, no leg swelling or oedema/headache/cough/haemoptysis/SOB/viral illness/sore throat/cellulitis/joint pain/weight loss/change in bowel habits
- Non-smoker, non-drinker for years, avoids caffeine
- Father has one small kidney, nil else

Case 1

- Afebrile, BP 110/70 mmHg, Pulse 70 per min
- Normal examination
- Labs at initial presentation – Creatinine 197 $\mu\text{mol/L}$, eGFR 24 ml/min, urine RBC >100, no growth, repeat eGFR 4 days later 19 ml/min, urine ACR 233 mg/mmol – refer for admission
- Labs at admission – urine rbc >1000, serum creatinine 250 $\mu\text{mol/L}$, CRP 47 mg/L

Case 1

What would you do next?

- Ultrasound renal tract
- CT KUB
- GN screen and repeat renal function
- Refer for admission to Urology
- Refer for admission to renal

Case 1

- Admitted under medical team
- Urine PCR 429 mg/mmol, Serum creatinine e 250 umol/L, urine rbc >1000
- CT KUB – no obstruction, no calculi
- GN screen – ANCA – MPO 62.9 CU, IgA 4.2, ANA 320, Complements – normal
- TTE – no vegetations, Creatinine 286 umol/L
- Consistent with RPGN started IV methylprednisolone 500 mg daily
- Urgent renal biopsy inpatient –next day

Case 1

- 8 sclerosed glom, 21 not sclerosed, cellular crescents 14 gloms, rbc casts in tubules, 20% fractional scarring – pauci-immune GN
- IF – 2 + IgA and C3
- Final diagnosis – necrotizing crescentic GN with coexisting IgA nephropathy – EM awaited

Case 1

- Started IV methylprednisolone 500 mg for 3 days (after +ANCA result)
- IV cyclophosphamide as vasculitis protocol with oral steroids
- Serum creatinine started to improve to 225 $\mu\text{mol/L}$ at discharge
- Follow up arranged

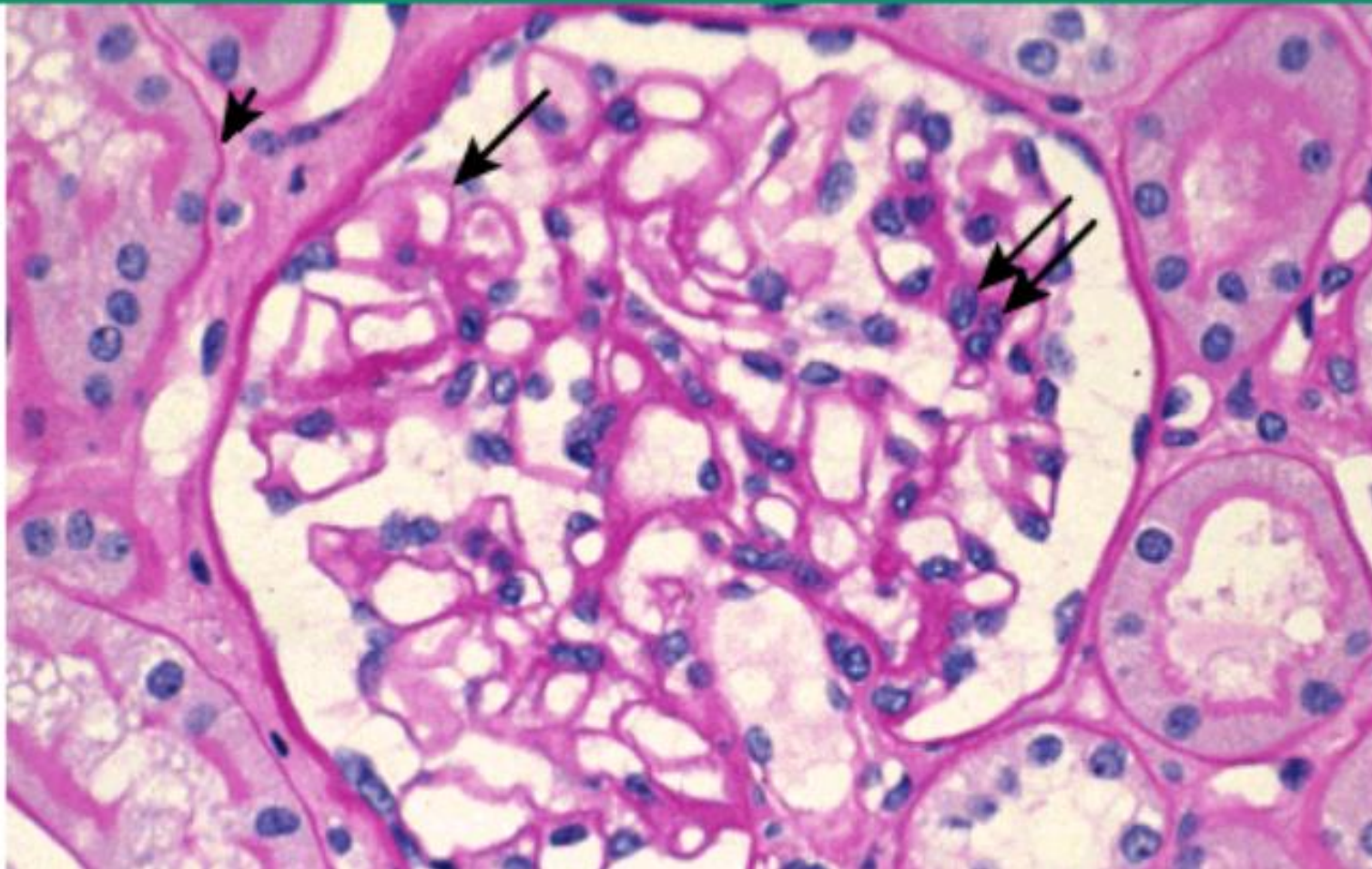
RPGN: Core Definition

- Clinical syndrome of:
 - Evidence of glomerular disease in urine (haematuria, proteinuria)
 - Progressive loss of renal function in a short amount of time (days-months)
- - Usually has extensive **crescent** formation
 - Crescent formation -> Severe glomerular inflammation
 - Pathological process:
 - Rupture of glomerular capillary wall ☐ allows inflammatory cells and mediators to enter Bowman's space
 - macrophage/ T cells/ cytokines IL-1, TNF-alpha / plasma proteins
 - These contents enter the interstitium and makes peri-glomerular inflammation worse
 - crescents form by: fibrin, collagen deposition from fibroblasts

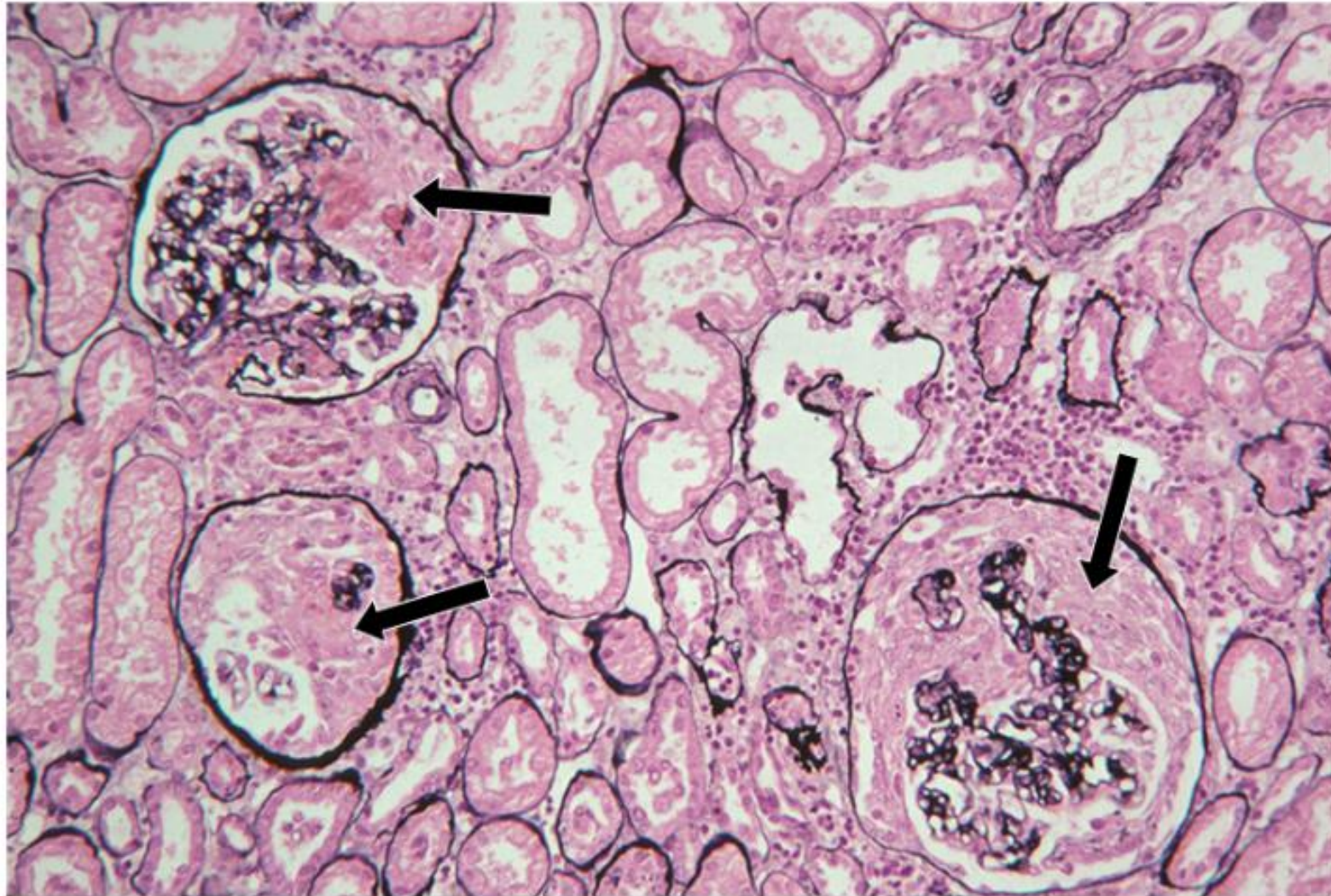
Diagnostic Algorithm

- Initial Labs
 - ANCA (PR3/MPO ELISA), anti-GBM antibodies
 - Complements (C3, C4)
 - ANA ± anti-dsDNA, cryoglobulins
 - Hepatitis B, C, HIV serology
 - Serum free light chains, immunofixation if myeloma suspected
- Urinalysis & Microscopy
 - Dysmorphic RBCs, RBC casts → confirms glomerular source
- Imaging
 - Chest X-ray/CT ± bronchoscopy if pulmonary hemorrhage suspected
- Early Renal Biopsy (unless contraindicated)
 - Look for crescents → Immunofluorescence is key
 - Linear IgG → Anti-GBM disease
 - Granular immune deposits → Immune-complex GN (e.g., lupus, IgA, infection-related)
 - Minimal/absent deposits → Pauci-immune GN (ANCA-associated vasculitis)

Normal glomerulus

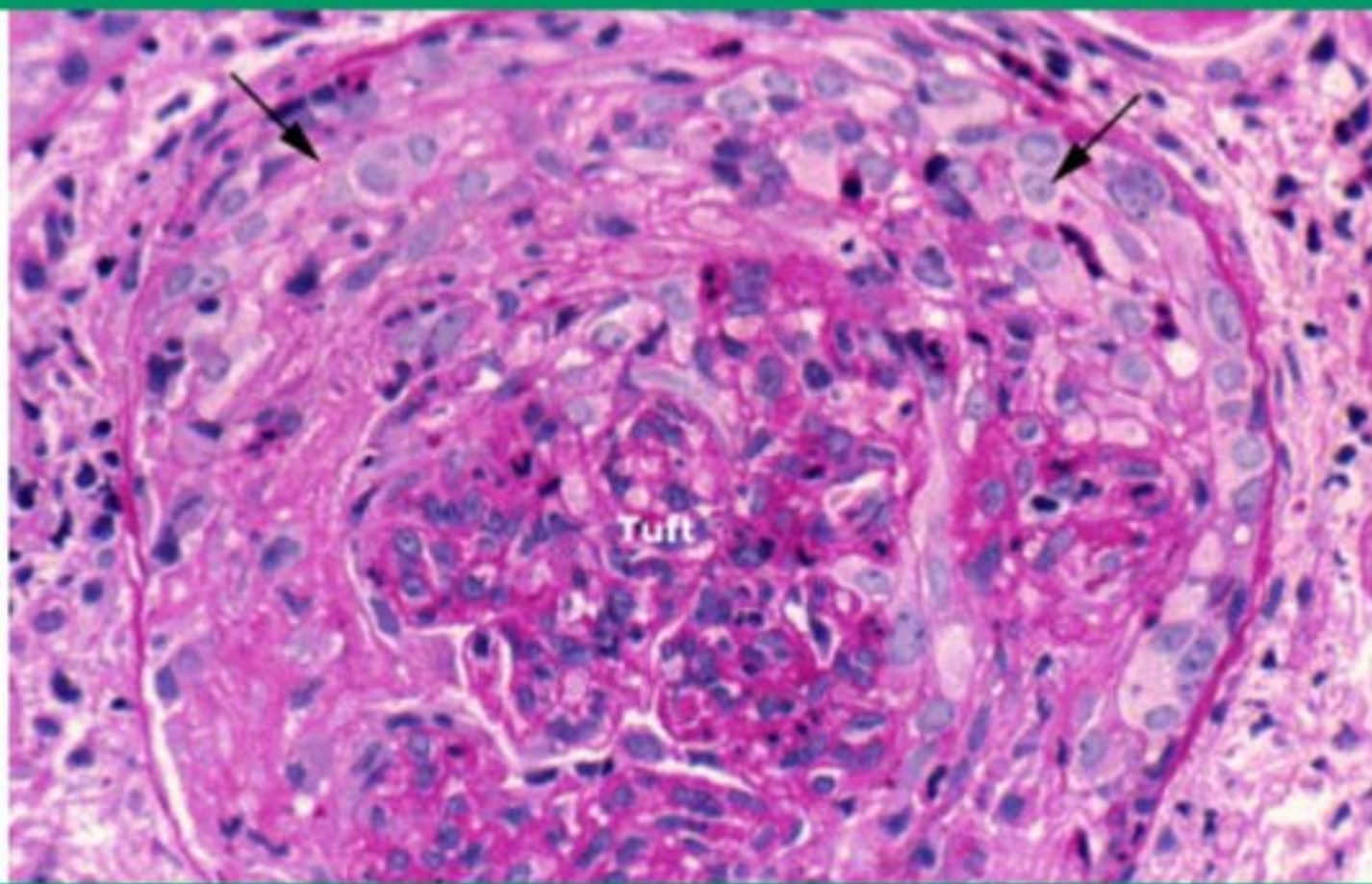


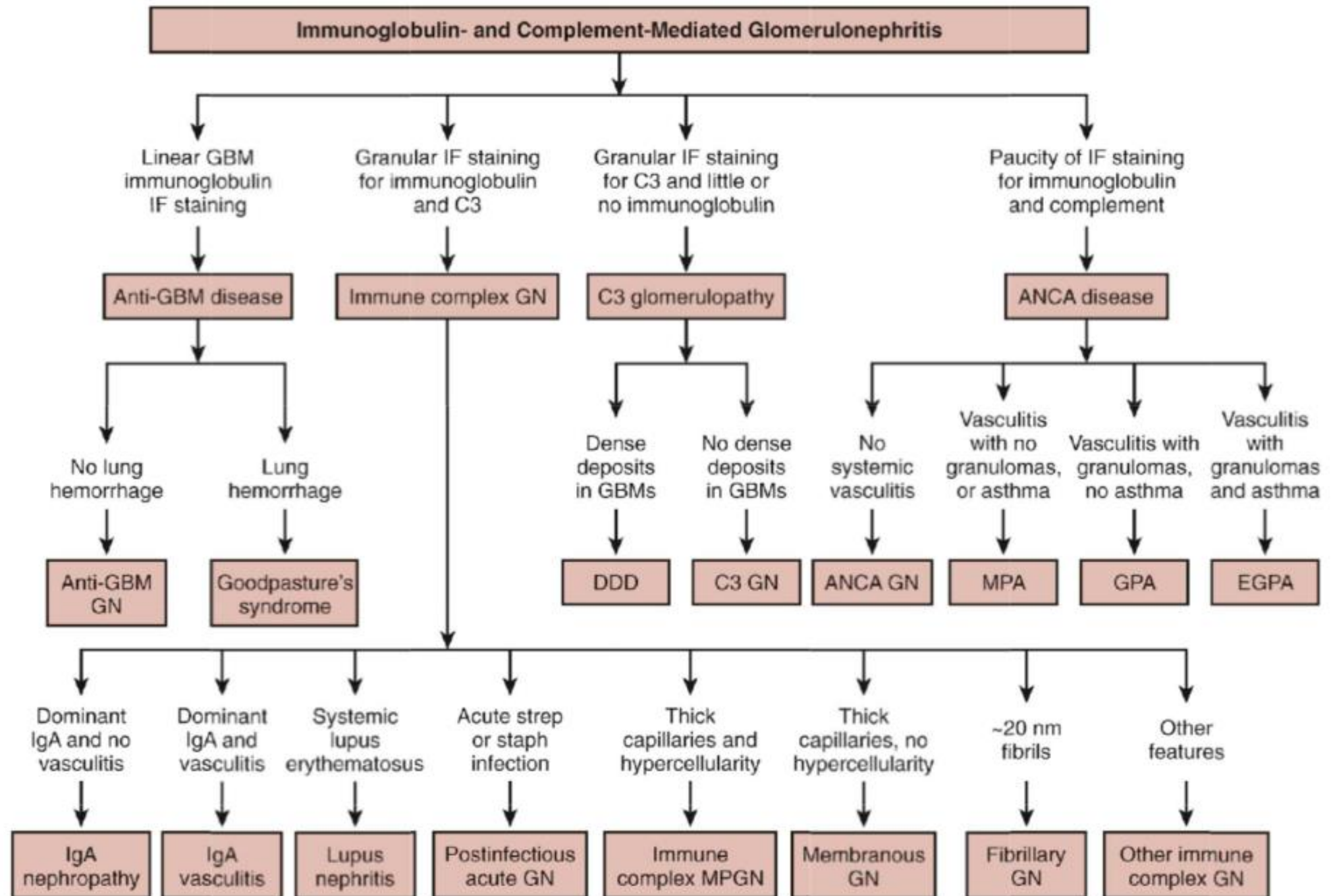
Light micrograph (silver stain) showing crescentic glomerulonephritis



High-power light micrograph showing crescentic glomerulonephritis. The hypercellular circumferential crescent (arrows) is compressing the glomerular tuft in the center of the glomerulus and closing the capillary lumens.

Rapidly progressive glomerulonephritis





Treatment of ANCA associated vasculitis

- Organ Threatening or Life threatening disease
 - Active GN
 - Pulmonary haemorrhage
 - Cerebral vasculitis
 - Progressive peripheral or cranial neuropathy
 - GI bleeding due to vasculitis
 - Cardiac disease due to vasculitis
 - Orbital pseudotumor

Organ Threatening or Life threatening disease

Induction

Glucocorticoids with Rituximab or cyclophosphamide

- CYC is preferred for severe kidney disease or pulmonary haemorrhage
- Rituximab is preferred for frail older adults, concerns for fertility, alopecia, malignancy or previously treated with CYC

Rituximab 1 G followed by another 1 G 14 days later

CYC – IV preferred – protocol available, usually two weekly over 3 months adjusted for age and eGFR

Organ Threatening or Life threatening disease

Glucocorticoid dosing and taper

- IV methylprednisolone for RPGN, Pulmonary haemorrhage, neurological involvement for three doses
- 1 mg/kg with max of 60 to 80 mg per day – PEXIVAS protocol – 7.5 mg daily by 12 to 18 weeks depending on body weight

Maintenance Therapy

- Usually 3 to 6 months after induction therapy
- Choice – depends on severity of disease and patient specific factors

Rituximab

Azathioprine

MMF

Methotrexate

- Duration – 12 to 24 months

Non-organ and Non-life threatening disease

- Glucocorticoids with methotrexate or azathioprine

Case 2

- 32 years old Samoan female, referred to rheumatology clinic for facial rash with positive ANA, dsDNA, positive Ro, anti-SM and RNP, normal eGFR, urine microscopy – 37 rbc in contaminated urine, urine ACR not available – DNA rheumatology clinic – advised to start HCQ, reschedule
- What would you do?
 - Call patient and re-refer
 - Follow up for HCQ to start
 - Monitor labs with urine
 - Do nothing

Case 2

- Presented with SOB on exertion and chest pain 3 months later
SOB worse with exertion, central lower chest pain worse with lying flat and improved with sitting upright
- Facial rash +, HR 111 per min, T 37.3C, Cr 168 umol/L, CRP 12, Hb 88 G/L, ANA + anti-dsDNA >300, SM, RNP, Ro positive
- ECHO – normal LV and RV function, trivial pericardial effusion
- US renal tract – normal, urine 86 rbc, ACR 74 mg/mmol (contaminated)
renal biopsy – Class 3 lupus nephritis with 3 cellular crescents
- Single, sexually not active, works 7 days per week

Case 2

- Treated with IV methyl prednisolone X 3 days while awaiting biopsy
- Treated with oral MMF induction dose of MMF 3 G/day, prednisone 60 mg daily as per PEXIVAS protocol
- Serum creatinine peak to 207 $\mu\text{mol/L}$, improved to 136 $\mu\text{mol/L}$ at discharge
- Clinic review 4 weeks – resolution of all symptoms, serum creatinine further improved to 75 $\mu\text{mol/L}$, urine PCR 54 mg/mmol
- Clinic review at 6 months – Serum creatinine 78 $\mu\text{mol/L}$, urine ACR 13 mg/mmol
- MMF 1 g BD as maintenance, prednisone 5 mg od, HCQ 400 mg daily, colecalciferol 1.25 mg monthly

Case 2

- At 1 year post biopsy – doing well but developed shingles –GP treated her with Valciclovir – improved
- At 15 months – rheumatology clinic – doing well, advised to have Shingrix vaccine (funded)
- At 21 months Rheumatology clinic – hair loss ?active lupus, anti dsDNA level rising, complements decreasing – refer to renal
- Labs done after a few phone calls – serum creatinine 107 $\mu\text{mol/L}$, urine PCR 107 mg/mmol
- Seen at renal clinic – still feeling well, told she did not miss MMF
- MMF increased to 1.5 G BD with oral prednisone 60 mg od reducing dose
- Outpatient biopsy arranged

Case 2

- Biopsy was delayed for 3 weeks – was done on 25/05/2026 – Class 4 lupus nephritis
- - Test safe – no script between Dec 2025 to Mar 2026!
- Elected to treat with IV CYC by Euro lupus protocol – 6 cycles, again some delay due to no space at infusion center. Last dose on 23 September
- Urine ACR 25 mg/mmol, serum creatinine – 109 umol/L, Hb 97 g/L
- Maintenance with MMF and low dose prednisone planned

Lupus Nephritis

- Most patients with SLE will have clinical evidence of kidney disease, usually abnormal urinalysis up to 50% of patients – 10 to 20% ESRD
- Immune complex GN and can involve variety of mechanisms-expression of genes neutrophils activation, interferon and proinflammatory cells with complement activation.
- Most kidney abnormalities emerge soon after diagnosis of SLE (with 6 months to 3 years)
- Less commonly patients may have silent LN with no clinical signs of kidney involvement. Usually benign outcome

Evaluation and Diagnosis of LN

- Suspects if patient has active urinary sediments – blood and protein with elevated serum creatinine
- Elevated anti-dsDNA and low complements levels often indicate active SLE particularly LN although utility of serological assessment differs among patients.
- Establishing diagnosis requires kidney biopsy – individualized risk – benefit assessment is required
- Generally biopsy is indicated – urine protein >500 mg/day, persistent hematuria (dysmorphic rbc or casts), rising serum creatinine that is not clearly attributable to another mechanism.

Treatment

- Hydroxychloroquine for all patient unless contraindicated
- Supportive measures for kidney disease

Dietary sodium and protein restriction

Anti-hypertensive

RAS blockage

SGLT2 inhibitors – lacking evidence

Lipid lowering

Treatment of LN

- Mycophenolate based regimen
- Cyclophosphamide based regimen
- Triple immunosuppressive regimens
 - Belimumab plus MMF or CYC
 - Calcineurin inhibitor plus MM
 - Obinutumab plus MMF
- Steroid dosing and taper – no consensus for best steroid regimen
 - Low dose regimens are preferred – 0.3 to 0.5 mg/kg (max 40 mg) and taper to <7.5 mg od by 3 to 6 months. Individualize for age, severity (lupus class), comorbid

Monitoring response to therapy

- Follow up evaluation

Urine protein and microscopy

Serum creatinine

FBC

Complements levels

CRP/ESR

Tacrolimus or cyclosporin levels

Definitions of response

Complete remission

- Decrease in proteinuria by 25% at 3 m, 50% at 6 m, 0.5 to 0.7 g/day by 12m
- patients with nephrotic range proteinuria may take 6 to 12 months
- Improvement or stabilization of eGFR
- Improvement of urinary sediments – rbc or casts

Partial remission

- Less stringent – reduction in proteinuria <50% or less than nephrotic range
- Improvement in eGFR
- Improvement in urinary sediments

Treatment related toxicities and prophylaxis

Steroids

- Infectious & Non-infectious complications

Cyclophosphamide

- Haematological, infection, gonadal toxicity, malignancy, alopecia and bladder toxicity

PJP prophylaxis

Bone prophylaxis

Case 3

- 66 years old with long standing microscopic haematuria and renal dysfunction
 - Long standing proteinuria (sub-nephrotic range) dated back to 2017 with eGFR 70 ml/min/1.73m³
 - Long standing hypertension, hypothyroidism, right mastectomy for Ca breast in 1990, raised BMI
 - Seen at renal clinic in 2017 for first time
- eGFR 70 ml/min/1.73m³, Urine ACR 48 mg/mmol, no RBC in urine, negative myeloma screen – GN screen requested but lost follow up

Case 3

- Referred back to renal clinic in 2021 for increased proteinuria 196 mg/mmol and some decline eGFR 64 ml/min/1.73m²
- Repeat GN screen unremarkable, urine no rbc, proteinuria improved on repeat 79 mg/mmol for follow up
- Arranged SMO follow up – attended but no clinic letter – lost follow up
- Refer to urology clinic for microscopic haematuria in May 2026, urine rbc >100, eGFR 40 ml/min/1.73m³ – less contaminated urine normal CTU and cystoscopy – refer to renal clinic

Case 3

- Seen on 28/07/2026

Patient is asymptomatic

History and lab reviewed, proteinuria is long standing, but haematuria is new since Feb 2026 rbc >100 with only a few epi cells.

eGFR started to decline in late 2025 less than 60 ml/min and less than 50 ml/min in 2026

BP 120/80 mmHg, weight 88 kilo, height 1.51 metre

Case 3

- Medications

Levothyroxine 125 mcg daily

Losartan 50 mg daily

Losartan plus 1 daily

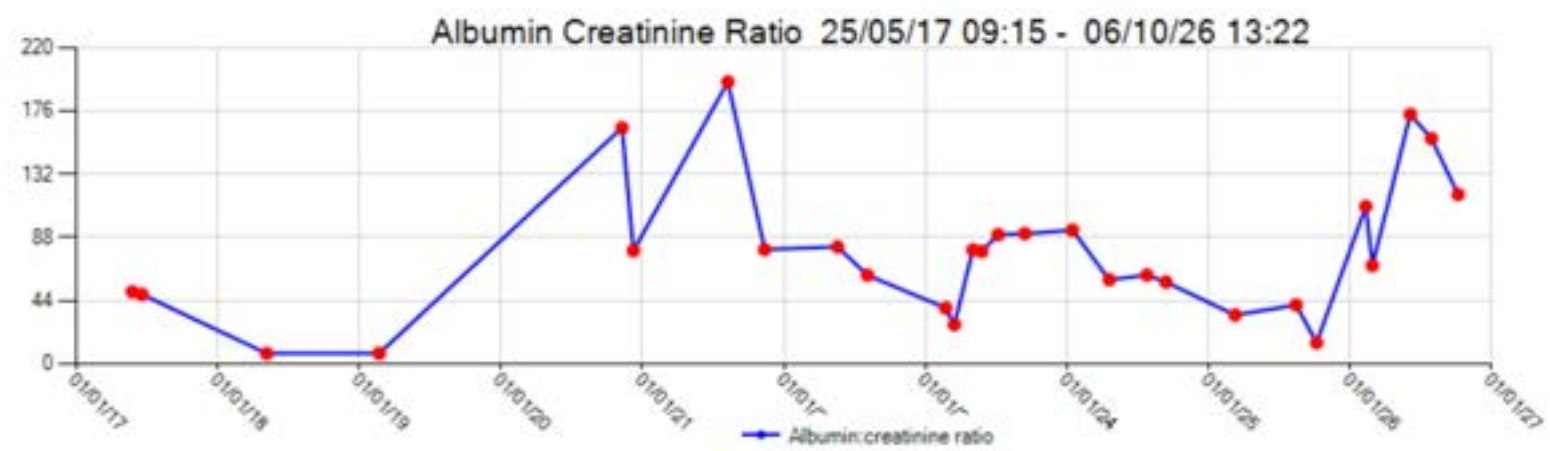
Atorvastatin 20 mg daily

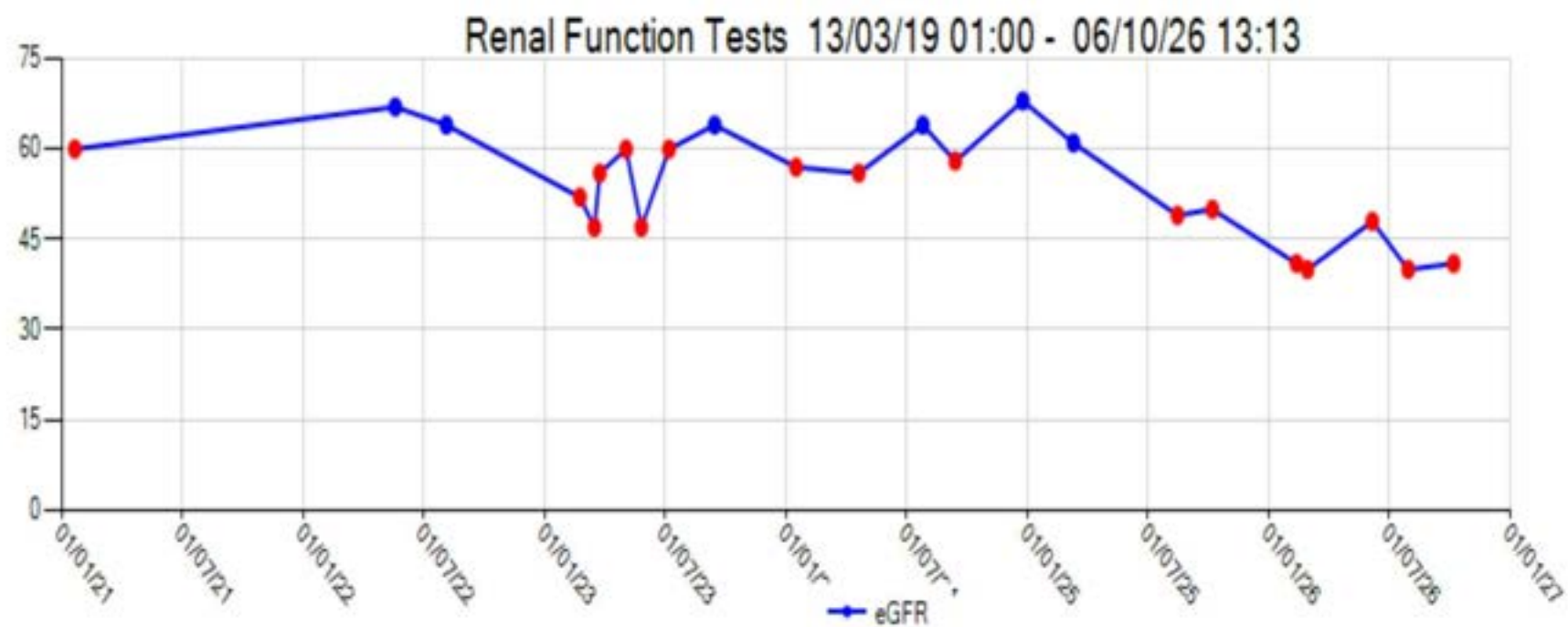
Famotidine 20 mg daily

Cholecalciferol 1.25 mg monthly

Case 3

- ANCA, Strep serology, Hep C, Hep B – all normal
- ANA 80, ENA negative, normal anti –dsDNA , HbA1c 43 mmol/mol
- No paraprotein in protein electrophoresis
- Immunoglobulins – slightly high IgA 4.8 g/L, normal IgG and IgM
- Urine microscopy rbc >100, epi cell 17, urine ACR 157 mg/mmol





Case 3

What would you do next?

Case 3

- Renal biopsy

Gloms total, 3 sclerosed, 18 non-sclerosed show increased mesangial hypercellularity, increased mesangial matrix,

Immunofluorescence - 3+ IgA and 3+ C3 mesangial positivity

Fractional scarring 10%

- IgA nephropathy, Oxford score M1, E0, S1, T0, C0

IgA Nephropathy Treatment

- Goal – To prevent disease progression and ESRD
- Supportive or immunosuppressive therapy can reduce disease progression

Risk assessment – 3 or more below – high risk of progression to ESRD

Persistent proteinuria >1 g/day (0.5 g/day for some expert)

Persistent microscopic haematuria

Progressive eGFR decline - <60 ml/min or >3 ml/min per year – consider as active IgA

One or more active lesions in Oxford scores, M1, E1, C1 or C2, S1

Supportive management

- RAS blockage
- BP control
- Life style modification including dietary restriction
 - Sodium and protein restriction
 - Weight control
 - Cessation of smoking
 - Physical exercise
 - Lipid lowering agents

Immunosuppressive Treatment

- Very high risk patients or progressive disease

Choice of immunosuppressive therapy

- Predominantly act on glomerular inflammation – Steroids, MMF, complement inhibitors
- Targeting pathogenic IgA or immune complex deposition -
Glucocorticoids, TRF-Budesonide, A proliferation-Inducing Ligand (APRIL) inhibitor, Combined B cell activating factor and April inhibitors (BAFF)

Thank You!