🧯 **Diabetic Nephropathy :** → #1c growing disease worldwide

🔷**Definition**

- **Persistent albuminuria** (>300 mg/**day** or >200 µg/**min**) = (hallmark) of diabetic nephropathy.
  - **Meaning:** If a diabetic patient keeps losing *more than 300 mg of albumin* every day in urine → suspect diabetic kidney disease.
- **Diagnosis is clinical** when:
  - **1.** The patient also has **diabetic retinopathy**
  - **2.** There's **no evidence** of other kidney or urinary diseases (like infection, stones, etc.)

🧠 **Memory Tip**:

Diabetic nephropathy = albuminuria + retinopathy + no other cause.

- **Clinically:** The patient will show:
  - **Progressive proteinuria**
  - **Decreasing GFR**
  - **HTN**
  - **Very high risk of CVS disease** (heart attack, stroke).
- **~40%** of DM pt →will develop nephropathy.
  - **Type 2 diabetes** is more common, so *most cases* of diabetic nephropathy are **Type 2**.

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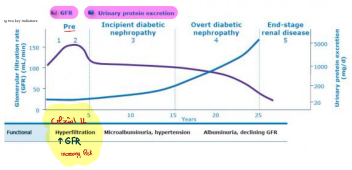
🔷**Progression Timeline**

- **High GFR in dm pt → not good sign**
- **Microalbuminuria** →appears **5–10 years after diabetes starts**.




🧠 **Important: Screening recommendations**

- **Type 1 Diabetes**: Start screening **5 years** after diagnosis, then **every year**.
- **Type 2 Diabetes**: Screen **immediately** when diagnosed, then **every year**.

🧠 **Memory Tip**:

Type 1 = 5 years → start.
Type 2 = start now → every year.

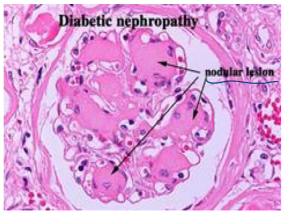



Fig 2. Diabetic nephropathy is one of the causes of nodular glomerulosclerosis, illustrated here, with large nodules of matrix within mesangial areas with lesser increase in mesangial cellularity. The glomerular basement membrane is thick without apparent deposits. (Periodic acid-Schiff stain, original magnification X400).

🔷**Early Kidney Changes**

- **1-2 years** after clinical diabetes → **changes** start in the kidneys:
  - **GBM thickens**.--> sensitive indicator for dm
  - But GBM thickening **alone** doesn't mean severe disease yet.

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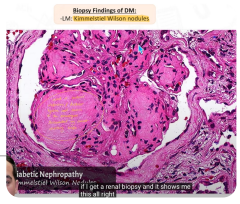
🔷**Pathology Changes**

- **Loss of heparin sulfate** from GBM → removes negative charge → allows **negatively charged albumin** to leak into urine.
  *(Normal GBM repels albumin; when you lose the charge, albumin escapes.)*
- **Mesangial expansion** (middle part of glomeruli swells with extracellular matrix).
  - *This swelling = clinical diabetic nephropathy symptoms.*
- **Diffuse diabetic glomerulosclerosis** = when this swelling becomes generalized across glomeruli.
- **Nodular glomerulosclerosis** (Kimmelstiel-Wilson nodules) = special finding:
  - Big, round, mesangial nodules
  - Compress nearby capillaries badly.
- **Arteriolar hyalinosis** (thickening of arteries around glomeruli) shows up within 3–5 years.

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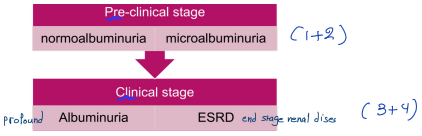
🔷**Clinical Course (Natural Phases) → dec renal fx**

1. **Hyperfiltration Phase**:
   - **GFR is high** at first (kidneys work harder).
   - No hypertension or albuminuria yet.
   - Intensified insulin tx and control near normal blood glu levels → red GFR to nl range after (days to week) in both dm types (1+2)

- 2.71x → progress to micro

2. **Microalbuminuria Phase**:
   - Urinary albumin = **30–300 mg/day**.--> Confirm at least by testing *2 out of 3* sterile samples(persist microalbum.)
   - **Urinary albumin/creatinine ratio** can also be used (30-300 mg/g = microalbuminuria).
   - Earliest marker of kidney damage.
3. **Overt Nephropathy**:
   - Full-blown proteinuria (>300 mg/day).
   - May develop nephrotic syndrome.
4. **End-Stage Renal Disease (ESRD)**:
   - Complete kidney failure.
   - Need dialysis or transplant.

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## Mechanisms causing microalbuminuria:

- Loss of glomerular charge barrier
- Podocyte changes (# and morphology)
- Glomerular hypertension +/- sys HTN
- Elevated glomerular filtration pressures (at rest , at exercise)

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## Microalbuminuria Importance

- **Type 1 Diabetes**:
  - Microalbuminuria = big risk (median risk ratio 21) for progressing to nephropathy.
- **Type 2 Diabetes**:
  - Median risk ratio ~8.5.
  - Not as high as type 1, but still significant.
- **If you control blood sugar well**, up to **58%** can return to normal albumin levels!
- **Microalbuminuria predicts**:
  - Cardiovascular disease risk.
  - Death from heart disease.
  - Stroke.
  - Kidney failure.

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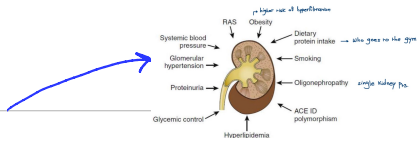
## Diabetic Nephropathy

- **Signs:**
  - Albuminuria >300 mg/day or CR 300mg/g
  - GFR decline.
  - High blood pressure.
  - Enhance CVS morbidity and mortality
- **Symptoms:**
  - **Peripheral edema** (earliest symptom).
  - **First sign → albuminuria**
  - *Seen even when GFR is still relatively normal.*
- **GFR decline rate: variable**
  - 2 to 20 mL/min/year.
  - Average = ~12 mL/min/year.

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## Factors Worsening Progression

- **Systemic hypertension** → glomerular hyperperfusion → inc capillaries pressure → glomerular HTN.
- **Loss of autoregulation** → kidneys can't protect themselves → faster damage → inc vulnerability to HTN or ischemic injury of glomerular cap.

---

## Diabetic Retinopathy Connection

- **Type 1 Diabetes**: → More common
  - 90% of nephropathy cases also have retinopathy.
  - Protienuria and No retinopathy? *Think of another cause*.
- **Type 2 Diabetes**:
  - 60% have retinopathy.

---

🔷**Macrovascular Disease (macroangiopathies)**

- Stroke, coronary artery stenosis , coronary artery disease, peripheral vascular disease → **2-5x more common** in diabetic nephropathy patients.

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🔷**Screening Summary**

🧠**Rules:**

- **Type 2 diabetes:** Screen **immediately** at diagnosis ,, since 7% already have microalbumnuria at the same time
- **Type 1 diabetes:** Screen **5 years later**.
- If microalbuminuria **absent**, **repeat yearly** for both
- Puberty, poor glycemic control and poor lipid control → independent risk factors for microalbuminuria .
- type 1 diabetes, screening for micro albuminuria might be performed 1 year after diabetes diagnosis in these patients or patients with poor glycaemic control

not 5 ✓

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🔷**Diagnosis of Diabetic Nephropathy**

✅ **Biopsy (GOLD) NOT always needed** unless: (not dm nephro)

- Hematuria
- Nephrotic range proteinuria at time of diabetes diagnosis
- Other systemic diseases suspected (autoimmune, hepatitis C, HIV).

✅may be deferred with the assumed diagnosis of diabetic nephropathy in the context of :

• Macro albuminuria (>300 mg/24 hours) that has developed progressively
• Microalbuminuria (30-300 mg/24 h) with retinopathy
• Microalbuminuria in pt with dm > 10 years .

---

🔷**Treatment Goals** 🎯

1. **Control Blood Sugar (Glycemic Control)**
2. **Control Blood Pressure**:
   - Target = **<130/80 mmHg**.
3. **RAAS Inhibition**: ( more BP → more damage / more damage → more BP so it's a cycle).
   - ACE inhibitors or ARBs are first-line treatment for dm pt with microalbuminuria or diabetic nephropathy.
   - inhibition of the RAAS → slows the progression of diabetic nephropathy compared to other
   - antihypertensive drugs + blood pressure lowering
   - The current recommendations →to target a blood pressure of 130/80 mmHg in diabetic pt
   - Slow down kidney damage even beyond BP lowering.
4. **SGLT2 Inhibitors**:
   - New drugs that protect kidneys and heart (e.g., empagliflozin).

2 inhibition 2 Control

🧠**Memory Tip for Diabetic Nephropathy Management**:

**G-BASICS**
**G**lycemic control
**B**lood pressure control
**A**CEi/ARB
**S**GLT2 inhibitors
**I**nitiate low-sodium diet ✓

it reabsorbs Glucose into blood so we inhibit that.

**C**ontrol cholesterol
**S**top smoking

---

### 🔷 Other Important Treatments

- **Low sodium diet**
- Smoking Cessation
- **Use of diuretics**:
  - may also enhance the antiproteinuric effects of RAAS inhibition while simultaneously decreasing fluid overload and HTN
  - **Loop diuretics if GFR <40** (at least twice daily)
  - **Thiazides if GFR >40**. (limited)
- **Additional antihypertensive drugs** depending on patient's comorbidities:
  - Beta-blockers for CAD or arrhythmias or congestive HF
  - Calcium channel blockers if no cardiac problems

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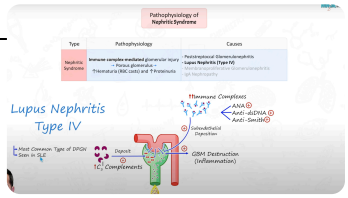
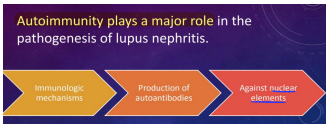



## 🧯 Part 2: Lupus Nephritis (LN)

### 🔷 Definition

- **Lupus nephritis** =one of the most serious manifestation of SLE
- Most patients with SLE →show **histological evidence** of lupus nephritis, even if clinically silent.
- It's considered **one of the most serious complications** of SLE.
- **Timing:** Usually develops **within 5 years** after being diagnosed with SLE.

### 🔷 Pathophysiology (Disease Mechanism)

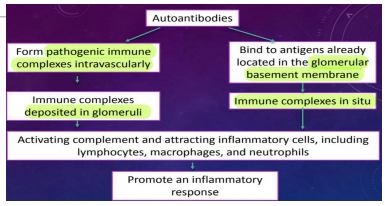



- What causes lupus nephritis? 👉 **Nephritogenic autoantibodies**.

✅ Autoantibodies specifics:

1. **Directed against nucleosomes or dsDNA (double-stranded DNA).**
   - Some of these antibodies *cross-react* with the (GBM).
2. **High-affinity antibodies**:
   - Form immune complexes inside blood vessels→These complexes deposit inside glomeruli.
3. **Cationic antibodies**: (higher affinity)
   - They stick more easily to the **anionic (negatively charged)** GBM.
4. **Isotype matters**:
   - IgG1 and IgG3 are the (worst)— they **activate complement** strongly → more inflammation.

### 🔷 Factors Influencing the Type of Nephritis




- Depends on:
  - Autoantibody type
  - Immune response
  - Genetic and environmental factors

### 🔷 How Common?

- **35%** of adults with SLE have nephritis at the time of diagnosis.
- **50-60%** develop nephritis during first 10 years.
- More common in **females**.
- **Peak age:** 21–40 years old.
- **100%** have positive **Anti-dsDNA antibodies**.
- **40% common histo type → class 4**
- Among secondary glomerulonephritis causes, lupus nephritis is **the most prevalent**.

Type 4: Diffused proliferative type:
Diffused: 50% of Glomeruli is affected
Proliferative: lots of immune cell attach and damage the glomeruli

### 🧠 Memory Tip:

Lupus nephritis = Young women + positive Anti-dsDNA + Class IV mostly.

---

🔹 **Clinical Features (Symptoms)**

- **May be asymptomatic** at first (silent damage).
- **Systemic SLE symptoms**: active
  - Fatigue
  - Fever
  - Skin rash
  - Arthritis
  - Serositis (inflammation of body linings)
  - CNS symptoms (confusion, psychosis)
- **Renal (kidney) symptoms**:
  - Peripheral edema (swollen legs, face) secondary to HTN or hypoalbuminuria
  - Symptoms of hypertension -diffuse lupus- (headache, dizziness , blurred vision, signs of cardiac decompensation).

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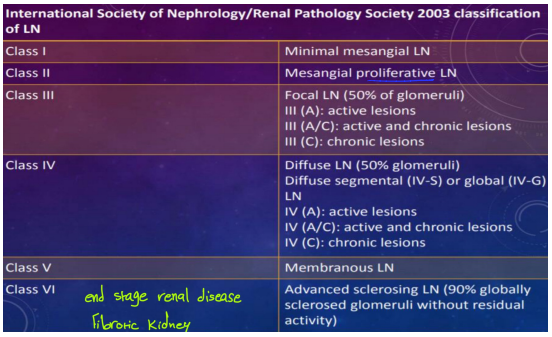
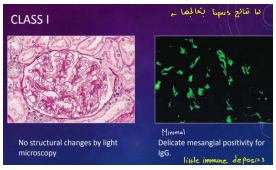
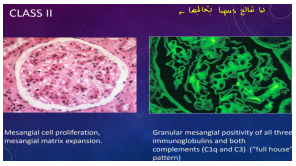
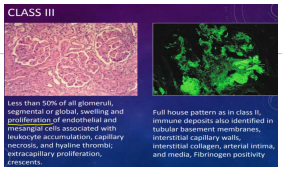
🔹 **Important Table: Clinical Features (with their frequencies)**

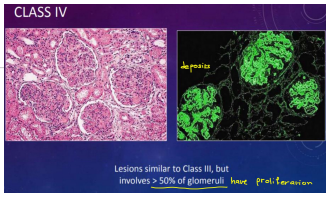
| Feature | % of Patients |
|---|---|
| **Proteinuria** | 100% |
| **Nephrotic syndrome** | 45-65% |
| **Granular casts** | 30% |
| **Red cell casts** | 10% |
| **Microscopic hematuria** | 80% |
| **Macroscopic hematuria** | 1-2% |
| **Reduced renal function** | 40-80% |
| **Rapid decline in renal function** | 30% |
| **Acute renal failure** | 1-2% |
| **Hypertension** | 15-50% |
| **Hyperkalemia** | 15% |
| **Tubular abnormalities** | 60-80% |

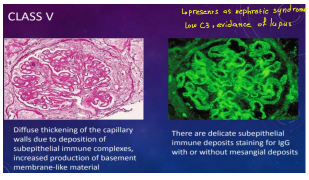


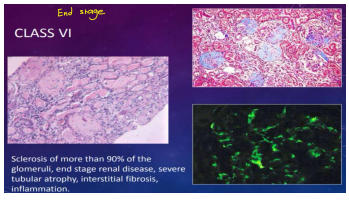




🧠 **Memory Tip (for proteinuria):**
**In Lupus Nephritis, proteinuria = always 100%.**
**If no proteinuria → think again!**

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🔹 **Laboratory Tests**

- **Blood Urea Nitrogen (BUN)**
- **Serum Creatinine** (check kidney function)
- **Urinalysis** (protein, RBCs, cellular casts)
- **Spot urine creatinine and protein**

✅ Normal urine values:

- Creatinine excretion: 1000 mg/day/1.73 $m^2$
- Protein excretion: 150–200 mg/day
- Protein/creatinine ratio: <0.2

---

🔹 **Serology Specific for Lupus Nephritis**

| Test | Result |
|---|---|
| ANA (dx=SLE)(antinuclear antibody) | Positive |
| Anti-dsDNA | Elevated |
| Complement (C3, C4, CH50) | Decreased |
| ESR | Elevated |
| CRP | Normal or slightly raised |

| | |
|---|---|
| Anti-C1q antibody | Elevated (less sensitive than Anti-dsDNA, but more specific) |

🧠 **Memory Tip:**
**↓ C3 + ↑ Anti-dsDNA = active lupus nephritis!**

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🔷 **Indications for Renal Biopsy (When to do it?)**
✅ Do a biopsy if:

- **Creatinine rising** without clear reason (not dehydration, not drugs)
- **Proteinuria ≥ 1 g/day**
- **Proteinuria ≥ 0.5 g/day plus**:
  - Hematuria (≥5 RBCs/high power field)
  - OR cellular casts.

🧠 **Memory Tip:**
If **Protein > 0.5 g + hematuria or casts → biopsy NOW!**

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**Indications for renal biopsy in pt with SLE**
• ↑serum Cr without compelling alternative causes (such as sepsis, hypovolemia, or medication)
• Confirmed proteinuria of 1.0 gm per 24 hours
•pt with clinical evidence of active LN, previously untreated,
• Combinations of the following→ findings are confirmed in at least 2 tests done within a short period of time and in the absence of alternative causes:
- Proteinuria 0.5 gm per 24 hours + hematuria, defined as 5 RBCs per hpf ,
- Proteinuria 0.5 gm per 24 hours + cellular casts

🔷 **Importance of Biopsy**

- Confirm diagnosis.
- Identify additional or alternative causes of renal disease
- Classify according to **ISN/RPS system**
- Check **activity** (how much current inflammation) and **chronicity** (how much permanent damage).
- Plan correct treatment + determine prognosis

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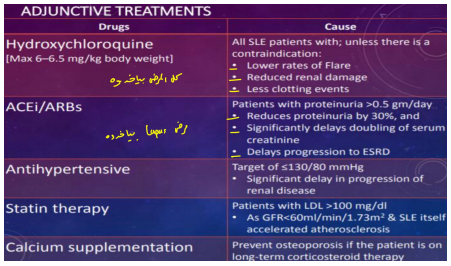
🔷 **Goals of Treatment**
🎯 What do we want?

- Save kidney function.
- Avoid progressive.
- Avoid side effects of drugs.
- Improve patient's life.




🔷 **Tx:**
• Adjunctive Treatments
• Primary → by immunosuppressive agents
1- Induction Therapy. 2- Maintenance Therapy
• Lifestyle Changes
immunosuppressive agents :
• Depends →on class of LN diagnosed on kidney biopsy with presence of extra-renal manifestations of SLE
• Goals of immunosuppressive treatment:

- Long-term preservation of renal function,
- Prevention of flares,
- Avoidance of treatment-related harms (side effects)
- Improved quality of life and survival

🔷 **Immunosuppressive Therapy**
**Two Phases:**

1. **Induction Phase** (to control active disease fast):

    - High-dose corticosteroids **PLUS**
    - Either **Cyclophosphamide** OR **Mycophenolate mofetil (MMF)**.
2. **Maintenance Phase** (keep the disease calm long-term):
    - **Azathioprine** OR **MMF** (lower doses) +/-low-dose steroids if needed.

🧠 **Memory Tip:**
Induction = fire extinguisher (high dose).
Maintenance = gentle water hose (low dose).

🔷 **Alternative Options**

- **Calcineurin inhibitors (e.g., Tacrolimus, Cyclosporine)** + low dose corticoids →For patients who can't tolerate MMF or Azathioprine. → may need dialysis and kidney transplant

🔷 **Lifestyle Changes**

- Hydrate properly.
- Low-salt diet.
- No smoking.
- No alcohol.
- Control cholesterol.
- Gentle exercise.
- Control blood pressure tightly.
- Avoid nephrotoxic drugs (e.g., NSAIDs).

🔷 **Relapse Risk**

- 25% relapse at 5 years.
- 46% relapse at 10 years.

✅ Predictors of flare:

- Low C3 and C4
- Rising anti-dsDNA
- RBC or WBC casts appearing in urine

🧠 **Memory Tip:**
**Low C + High Anti-dsDNA = Flare danger!**

🔷 **Types of renal flares:**
- Proteinuric (increase proteinuria)
- Nephritic (increase >30% of Scr and/or active urine sediment).
▪ Flares are highly predicted by RBC or WBC casts, low C3 and C4 and rise in ds DNA.

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🔷 **Special Situations**

✅ **Transplantation**:

- SLE patients = ~3% of kidney transplants.
- Wait 3 months on dialysis to make sure no spontaneous recovery.
- Ensure that the patient does not have active SLE disease at the time of transplantation.
- ~3.3% of patients on RRT have functional renal recovery and be off dialysis.
- Majority of patients has a decline in disease activity with ESRD treatment.
- Recurrence rate low (<2–4%).

✅ **Pregnancy**:

- Avoid pregnancy if active lupus nephritis / nephrotic syndrome , severe HTN , inc serum Cr >2mg/dl ? aggravate renal disease
- Patients with well-controlled SLE who conceive after a 3- 6-month →period of remission have a 7-10% chance of renal flare.

- *Preexisting hypertension and antiphospholipid antibody syndrome →are the most 2 common predisposing factors to preeclampsia.
- Pregnancy risks: **Preeclampsia**, **renal flares(50-60%)** .

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📚 **Quick High-Yield Final Memorization Summary for You**

| **Diabetic Nephropathy** | **Lupus Nephritis** |
|---|---|
| Albuminuria >300 mg/day | Immune complexes (Anti-dsDNA) attack glomeruli |
| Progresses over 4 stages | Within 5 years of SLE diagnosis |
| Confirm if Retinopathy present | Biopsy mandatory if suspicion |
| Main management = ACEi/ARB | Main management = steroids + cyclophosphamide/MMF |
| Microalbuminuria precedes macroalbuminuria | Low C3 + High Anti-dsDNA = active disease |
| SGLT2 inhibitors are new promising drugs | High relapse risk, transplant possible |

# Revision

| | |
|---|---|
| Definition | - Persistent albuminuria >300 mg/day (hallmark).<br>- Clinical diagnosis if: albuminuria + retinopathy + no other kidney disease. |
| Epidemiology | - ~40% of diabetics develop nephropathy.<br>- More common in Type 2 diabetes (because Type 2 is more common overall). |
| Screening Recommendations | - Type 1 DM: Screen after 5 years of diagnosis, then yearly.<br>- Type 2 DM: Screen at diagnosis, then yearly. |
| Pathology Changes | - GBM thickening (sensitive early indicator).<br>- Loss of negative charge (heparan sulfate) → albumin leakage.<br>- Mesangial expansion → glomerulosclerosis.<br>- Kimmelstiel-Wilson nodules = diagnostic. |
| Progression Stages | 1. Hyperfiltration: High GFR, no proteinuria.<br>2. Microalbuminuria: 30-300 mg/day.<br>3. Overt Nephropathy: >300 mg/day proteinuria, nephrotic syndrome.<br>4. ESRD: Dialysis or transplant needed. |
| Clinical Signs/Symptoms | - First sign = albuminuria (even if GFR normal).<br>- Gradual GFR decline (avg 12 mL/min/year).<br>- Hypertension common.<br>- Peripheral edema early. |
| Mechanisms of Injury | - Loss of charge barrier.<br>- Podocyte injury.<br>- Glomerular hypertension. |
| Risk Factors for Progression | - Systemic hypertension.<br>- Poor glycemic control.<br>- Smoking.<br>- Dyslipidemia.<br>- Loss of autoregulation → susceptibility to damage. |
| Diabetic Retinopathy Link | - Type 1: 90% have retinopathy if nephropathy present.<br>- Type 2: 60% have retinopathy. |
| Diagnosis Confirmation | - Clinical mainly.<br>- Biopsy if atypical (e.g., hematuria, massive proteinuria at diagnosis, absence of retinopathy). |
| Treatment Goals | 🎯<br>1. Glycemic control (HbA1c target individualized). |

| | |
|---|---|
| | 2. BP control: <130/80 mmHg.<br>3. RAAS inhibition: ACEi or ARB.<br>4. SGLT2 inhibitors: (renal and CV protection).<br>5. Lifestyle: Low sodium, no smoking, statins if dyslipidemia. |
| Important Medications | - ACEi/ARB = first-line (protect kidneys beyond BP lowering).<br>- SGLT2 inhibitors (e.g., empagliflozin).<br>- Diuretics: Loop if GFR <40, Thiazides if GFR >40. |
| Screening Strategy (Memory Tip) | 🧠<br>- Type 1: 5 years → start.<br>- Type 2: Screen immediately.<br>- Puberty, poor glycemic/lipid control → screen earlier. |
| Memory Tip for Management | G-BASICS:<br>Glycemic control + Blood pressure control + ACEi/ARB + SGLT2 inhibitors + Initiate low-sodium diet + Control cholesterol + Stop smoking |

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🧠 Final Key High-Yield Points:

- First sign = Microalbuminuria → Confirm in 2 of 3 samples.
- Target BP = <130/80 mmHg always.
- Albuminuria + Retinopathy = strong clue for diabetic nephropathy.
- GFR loss is progressive but variable.
- ACEi/ARBs are gold even in normotensive diabetics with microalbuminuria.
- Control sugar early to prevent microalbuminuria progression!
- Biopsy only if atypical features (e.g., hematuria)

| **Section** | **Key Points** |
|---|---|
| **Definition** | - One of the most serious complications of SLE.<br>- Caused by nephritogenic autoantibodies (especially anti-dsDNA). |
| **Timing** | - Typically develops within **5 years** of SLE diagnosis. |
| **Pathophysiology** | - Immune complex deposition in glomeruli.<br>- Loss of GBM negative charge.<br>- IgG1 and IgG3 activate complement strongly → severe damage. |
| **Epidemiology** | - 35% have nephritis at diagnosis.<br>- 50–60% develop nephritis within 10 years.<br>- More common in females (peak 21–40 years). |
| **Clinical Features** | - Systemic SLE symptoms (fatigue, rash, arthritis).<br>- Renal symptoms: proteinuria, hematuria, hypertension, edema.<br>- 100% of patients have proteinuria! |
| **Laboratory Findings** | - ANA positive.<br>- Anti-dsDNA high.<br>- Low C3/C4 (hypocomplementemia).<br>- Urinalysis: RBC casts, proteinuria. |
| **Important Numbers (Clinical Features)** | - Proteinuria: 100%.<br>- Nephrotic syndrome: 45–65%.<br>- Microscopic hematuria: 80%.<br>- Granular casts: 30%.<br>- Rapid decline in renal function: 30%. |
| **Serological Markers** | - High anti-dsDNA.<br>- Low complement (C3, C4, CH50).<br>- Anti-C1q antibody (specific). |

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| **Indications for Renal Biopsy** | ✅Do a biopsy if:<br>- ↑Creatinine without clear cause.<br>- Proteinuria ≥ 1 g/day.<br>- Proteinuria ≥ 0.5 g/day + hematuria or cellular casts.<br>🧠Rule: ≥0.5g + hematuria/casts = Biopsy now! |
| **Goals of Treatment** | 🎯<br>- Preserve renal function.<br>- Prevent flares.<br>- Minimize treatment toxicity.<br>- Improve survival and quality of life. |
| **Immunosuppressive Therapy** | - **Induction Phase**: High-dose steroids + Cyclophosphamide or MMF.<br>- **Maintenance Phase**: Low-dose steroids + Azathioprine or MMF. |
| **Alternative Agents** | - Calcineurin inhibitors (Tacrolimus, Cyclosporine) for refractory cases.<br>- May eventually need dialysis or transplant. |
| **Lifestyle Measures** | - Control blood pressure.<br>- Low-salt diet.<br>- No smoking.<br>- Control cholesterol.<br>- Avoid nephrotoxic drugs (e.g., NSAIDs). |
| **Relapse Risk** | - ~25% at 5 years, ~46% at 10 years.<br>- Predictors of flare: low C3, low C4, rising anti-dsDNA. |
| **Types of Flares** | - **Proteinuric** (increased proteinuria).<br>- **Nephritic** (increased Scr >30% and/or active urine sediment). |
| **Special Situations** | - **Pregnancy**: Avoid if active nephritis.<br>- **Transplant**: 3-month dialysis period first; recurrence rate <4%. |
| **Memory Tips** | 🧠<br>- **↓C3 + ↑anti-dsDNA** = active flare.<br>- **Proteinuria 100%** in lupus nephritis.<br>- **Positive ANA** + **positive anti-dsDNA** = lupus nephritis strong clue. |

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🧠 **Final Key High-Yield Points:**

- Always **think biopsy** if unexpected kidney signs appear.
- **Induction** = attack the disease fast (high dose).
- **Maintenance** = keep disease calm (low dose).
- Target blood pressure in LN = strict control to prevent progression.
- Monitor **C3/C4 and anti-dsDNA** to catch early flares.