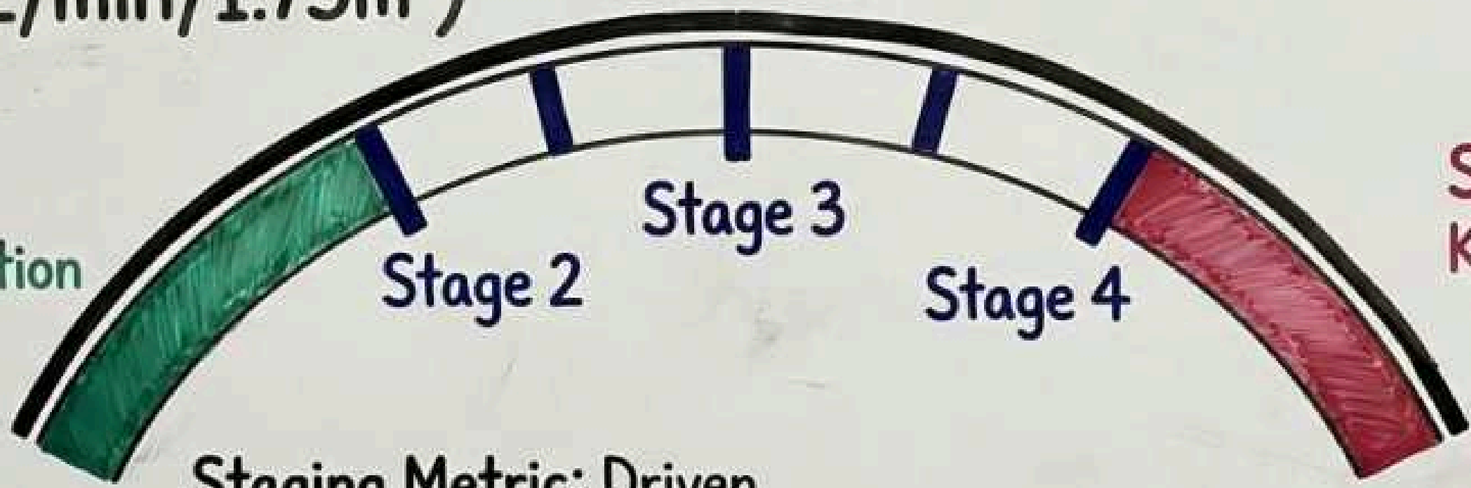
The Definition & Staging of CKD

¶ 5.1

eGFR (mL/min/1.73m²)

Diagnostic Criteria: Kidney damage or reduced function persisting for > 3 months.

Stage 1:
Normal filtration but damaged



Stage 5:
Kidney Failure (eGFR < 15)

Staging Metric: Driven primarily by eGFR.



Albumin ↑ = CV Risk ↑

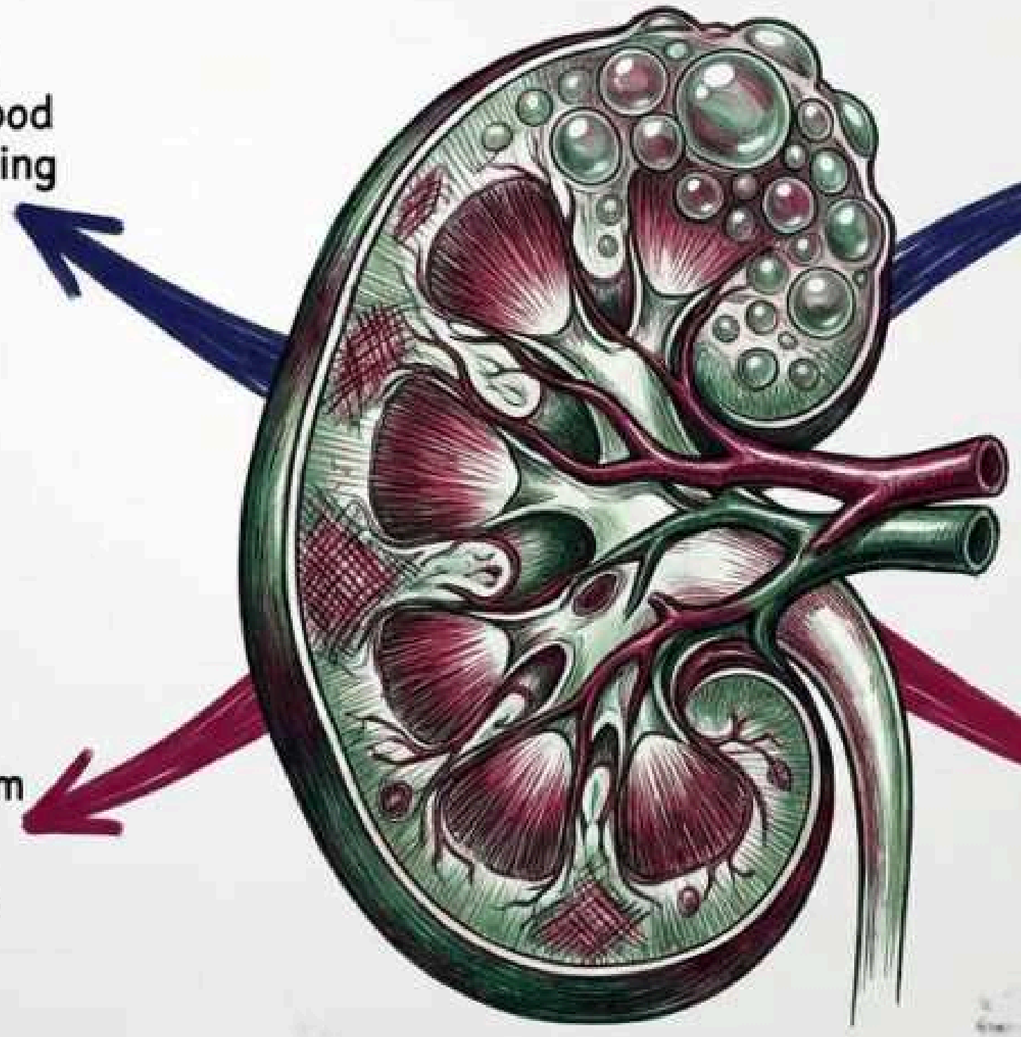
Key Marker: Urine albumin leakage is a critical secondary metric—higher protein loss predicts faster disease progression and elevated cardiovascular risk.

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The Global Drivers of CKD

¶ 5.2

The Twin Pillars: Diabetes and High Blood Pressure are the leading worldwide causes.



Secondary Causes: Glomerular diseases, inherited conditions (e.g., Polycystic Kidney Disease characterized by fluid-filled cysts).

Structural: Long-term obstruction and repeated infections.

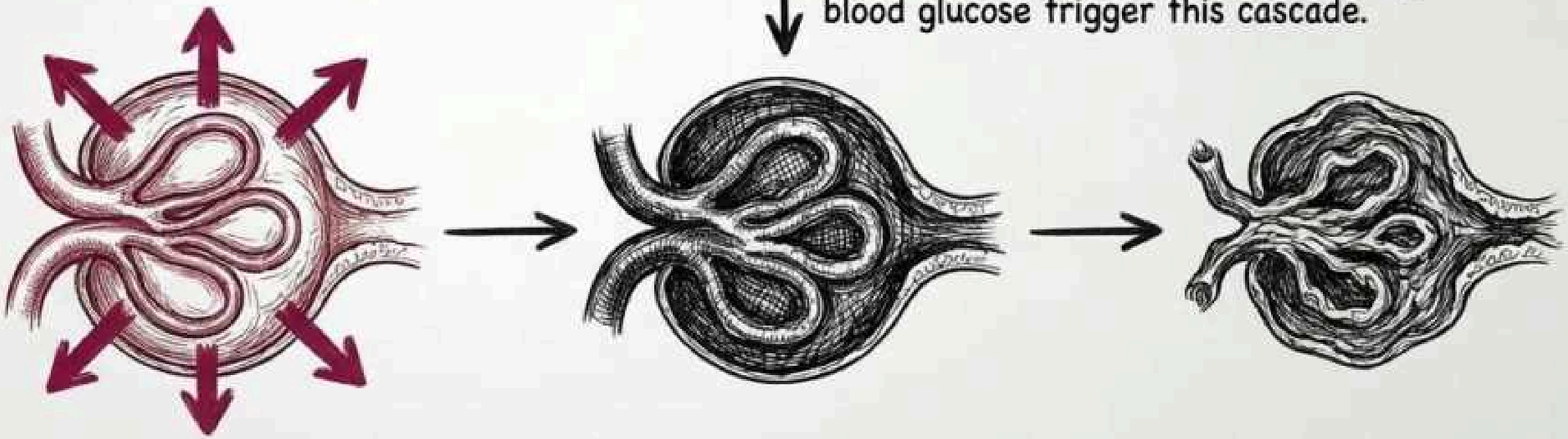
Emerging Vectors: Chronic Kidney Disease of uncertain cause (CKDu), heavily affecting young agricultural workers in specific regions, with causes still under active scientific study.

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The Pathogenesis of Diabetic Kidney Disease ¶ 5.3

$$C_6H_{12}O_6$$

The Catalyst: Years of unmanaged high blood glucose trigger this cascade.



Hyperfiltration → **Thickening & Scarring** → **GFR Decline**

Clinical Progression: Microalbuminuria (small leaks) → Macroalbuminuria (large leaks) → Declining GFR.

Intervention Window: Tight control of both blood glucose and blood pressure actively slows this anatomical timeline.

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The Vicious Circle of Hypertensive Kidney Disease

¶ 5.4

High Blood Pressure

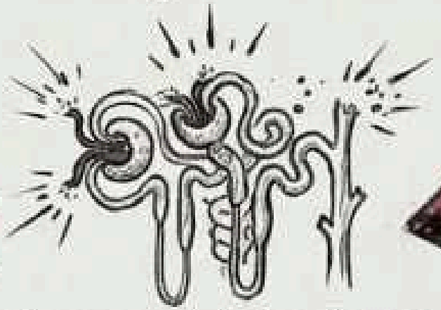
The Cycle:
Systemic hypertension directly damages the small arteries of the kidney. In response, the damaged kidneys inherently raise blood pressure further.



Kidneys Release Signals to Raise BP



The Overwork Hypothesis:
As nephrons die off, the remaining healthy nephrons are forced to filter at dangerously high pressures to compensate. This mechanical overwork eventually injures them too, driving inevitable disease progression regardless of the original cause.



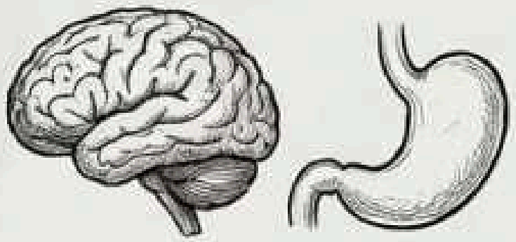
Stressed Nephrons (Overworked)

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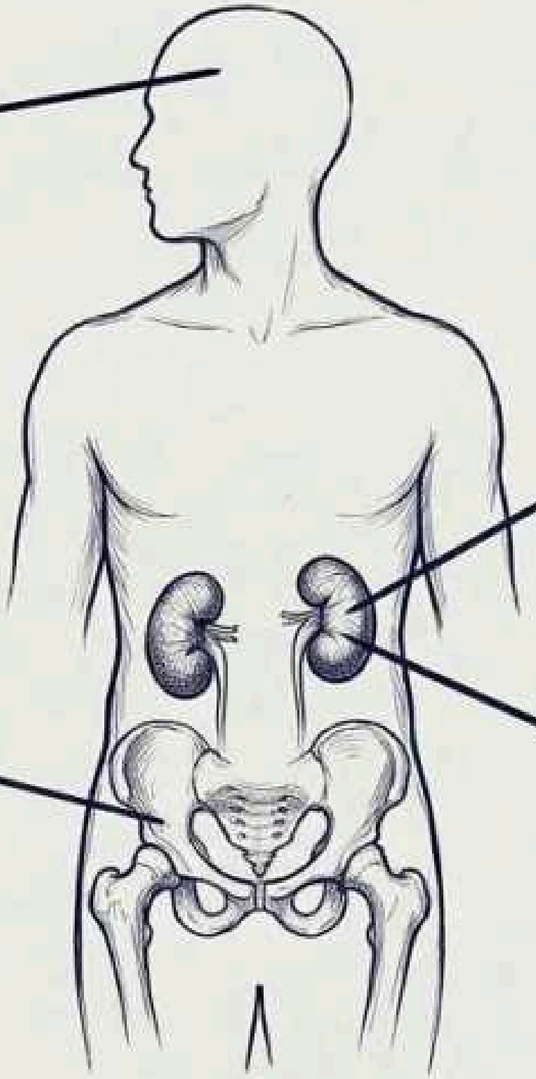
Warning: Usually silent until far advanced.

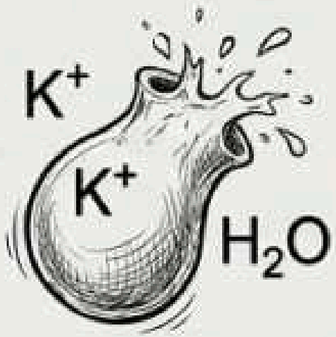
¶ 5.5

Systemic Manifestations of Advanced CKD

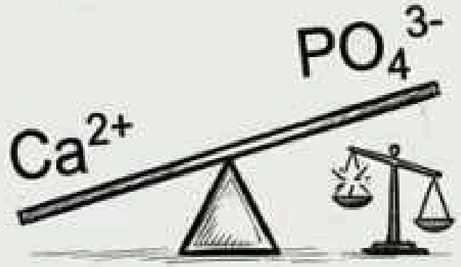


Brain & Stomach: Uremia buildup pathway → Results in tiredness, poor appetite, nausea, itching, and eventually confusion.





General Homeostasis: Indicates loss of fluid and potassium control.



Bones: Calcium/Phosphate out of balance linked to a lack of active Vitamin D → Weakens bone structure.

Erythropoietin (EPO)

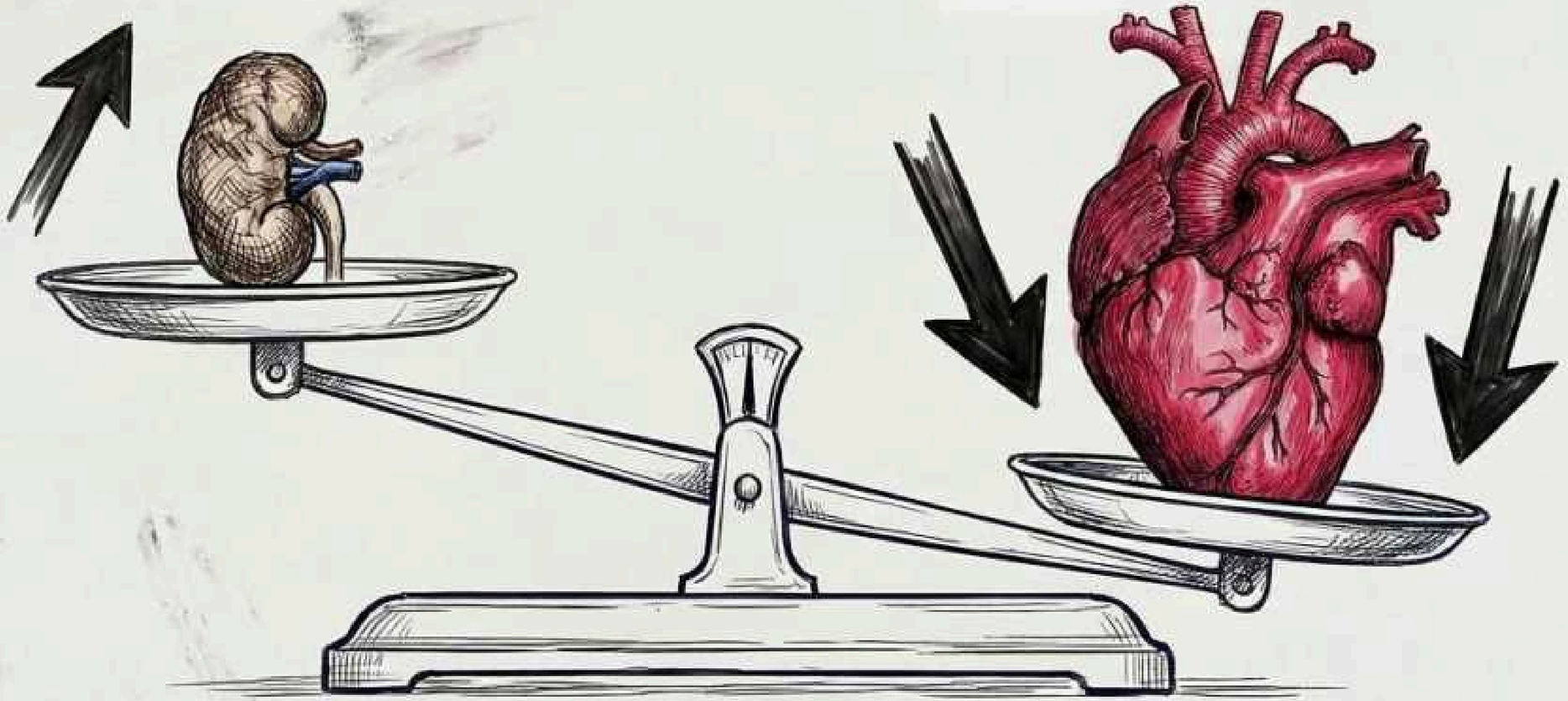


Blood Vessels: Loss of EPO → Causes severe anaemia.

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The True Danger: Cardiovascular Risk

¶ 5.6



The Statistical Reality:
CKD massively amplifies the risk of heart attacks, strokes, and heart failure.

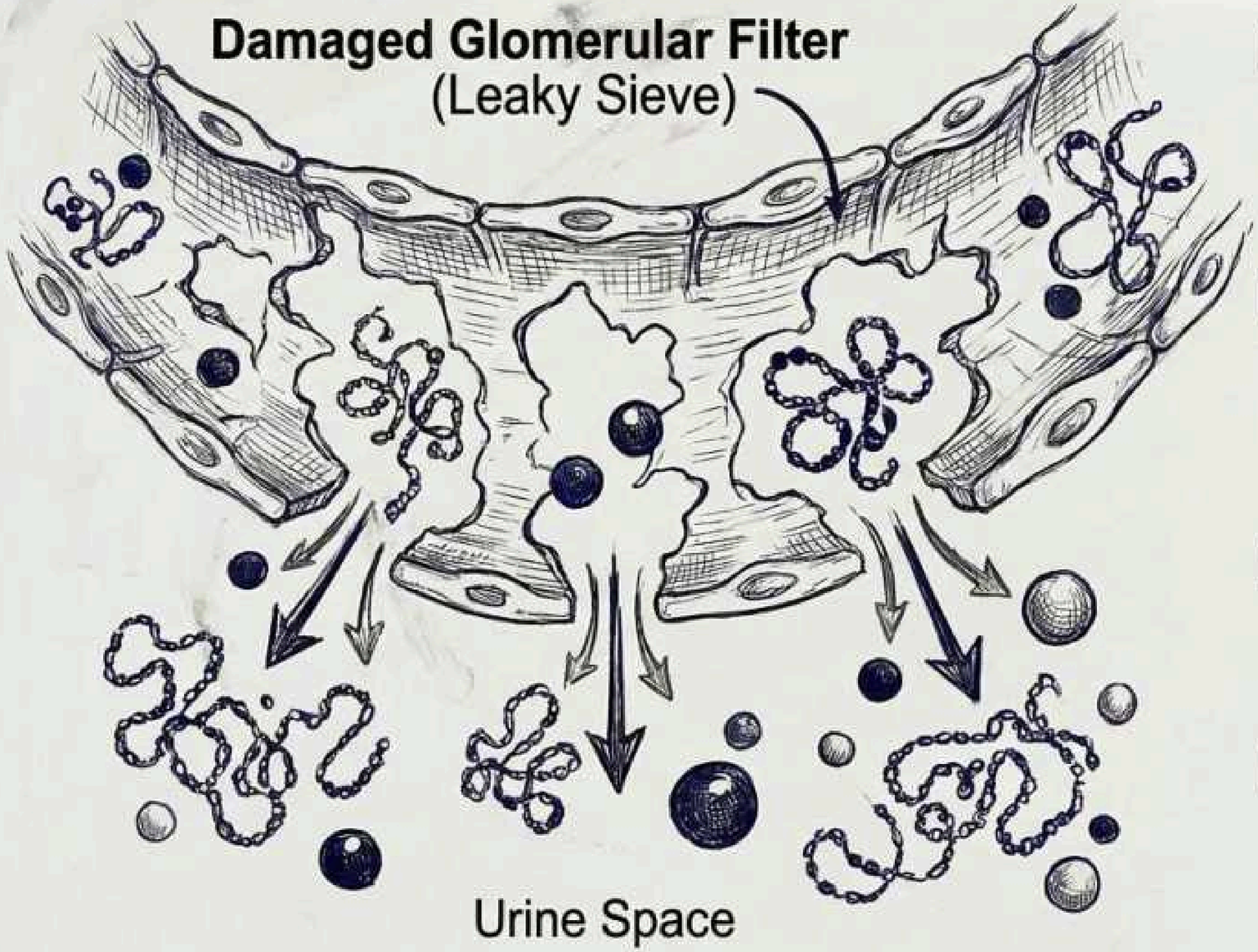
The Paradigm Shift:
Patients with moderate kidney disease are statistically more likely to die of cardiovascular disease than they are to ever reach end-stage kidney failure.

Clinical Directive:
Protecting the heart is a central, non-negotiable part of early CKD care.

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Glomerular Disease Pattern 1: Nephrotic Syndrome

¶ 5.7



- ☑ **Mechanism:** The filter becomes highly leaky to protein, notably without much inflammation.
- ☑ **Clinical Triad:** Heavy proteinuria, Hypoalbuminemia (low blood albumin), and Edema (swelling).
- ☑ **Visible Signs:** High cholesterol and frothy-appearing urine.
- ☑ **Common Causes:** Minimal change disease (in children), membranous nephropathy, and diabetes (in adults).

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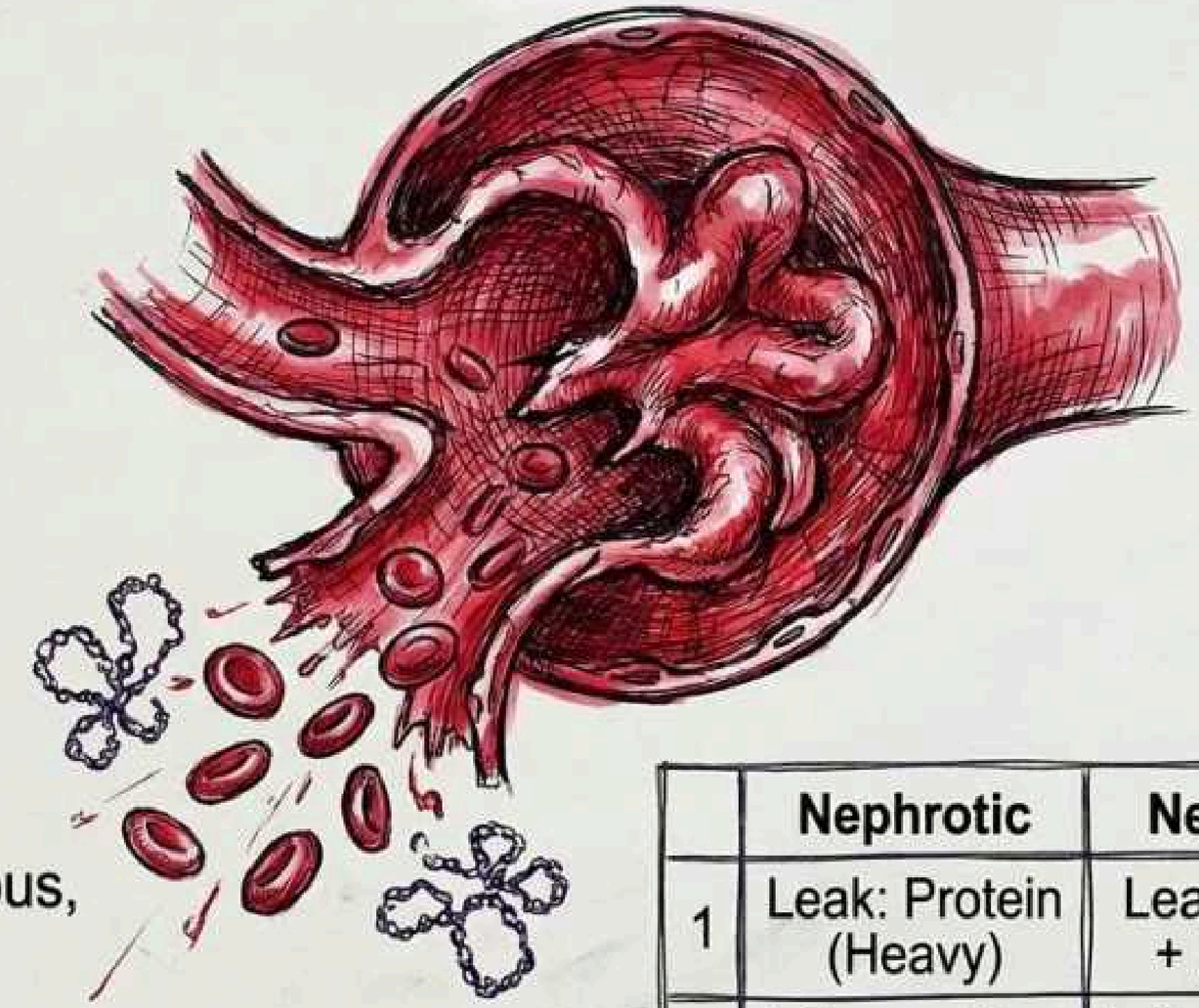
Glomerular Disease Pattern 2: Nephritic Syndrome

¶ 5.8

Mechanism: Driven by active, destructive inflammation of the glomeruli.

Clinical Picture: Hematuria (smoky or cola-coloured urine), accompanied by some protein loss, high blood pressure, fluid retention, and rapidly falling GFR.

Causes: Immune complex diseases like IgA nephropathy (the most common globally), lupus, and aggressive conditions that can destroy kidney function within weeks.



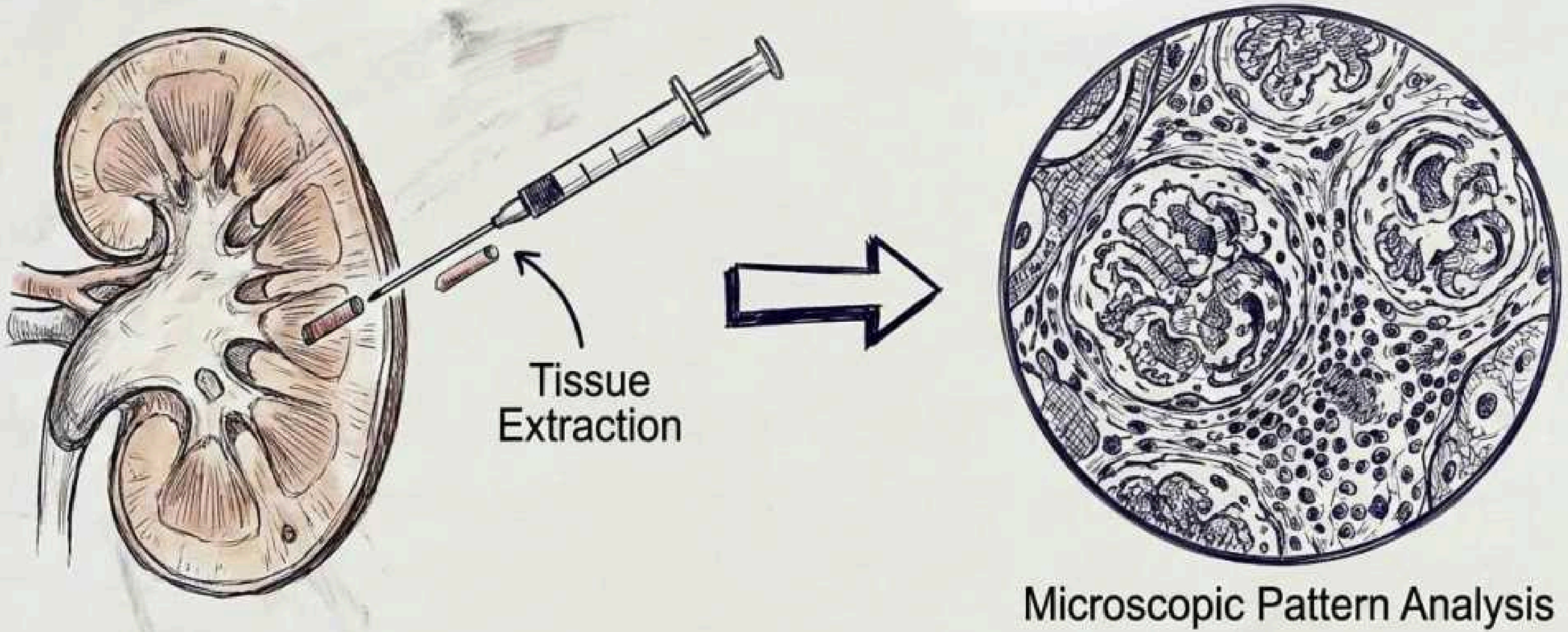
Urine Space

	Nephrotic	Nephritic
1	Leak: Protein (Heavy)	Leak: Blood + Protein
2	Inflammation: Low	Inflammation: High

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Diagnosing Glomerular Disease

¶ 5.9



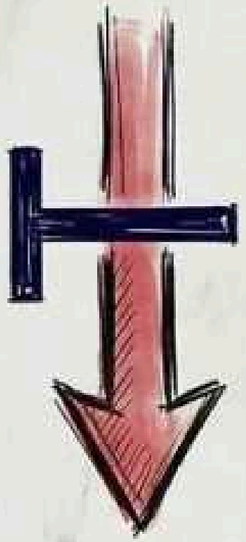
The Protocol: Accurate diagnosis strictly requires tissue-level confirmation via kidney biopsy.
Pathology to Treatment: The specific microscopic pattern of injury identified under the lens directly dictates the treatment plan.
Therapeutics: Because many of these diseases are immune-mediated, treatment heavily relies on targeted drugs that suppress the immune system.

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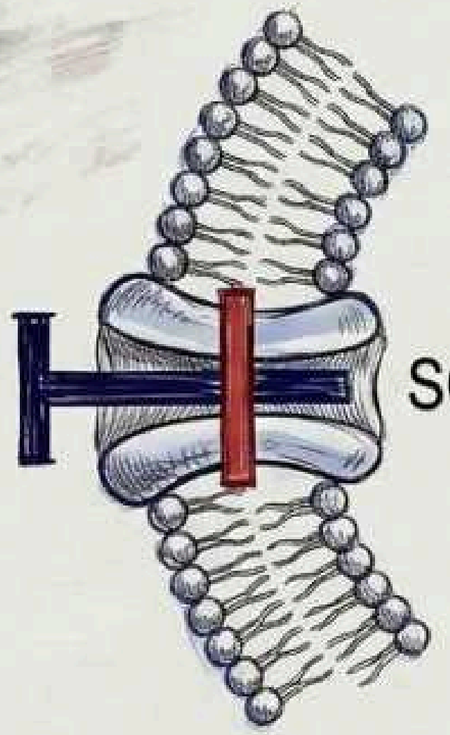
Pharmacological Interventions to Slow Progression

¶ 5.10

Renin-Angiotensin System (RAS)



BP & Proteinuria Control: Drugs blocking the renin-angiotensin system simultaneously lower blood pressure and reduce protein leakage.



SGLT2

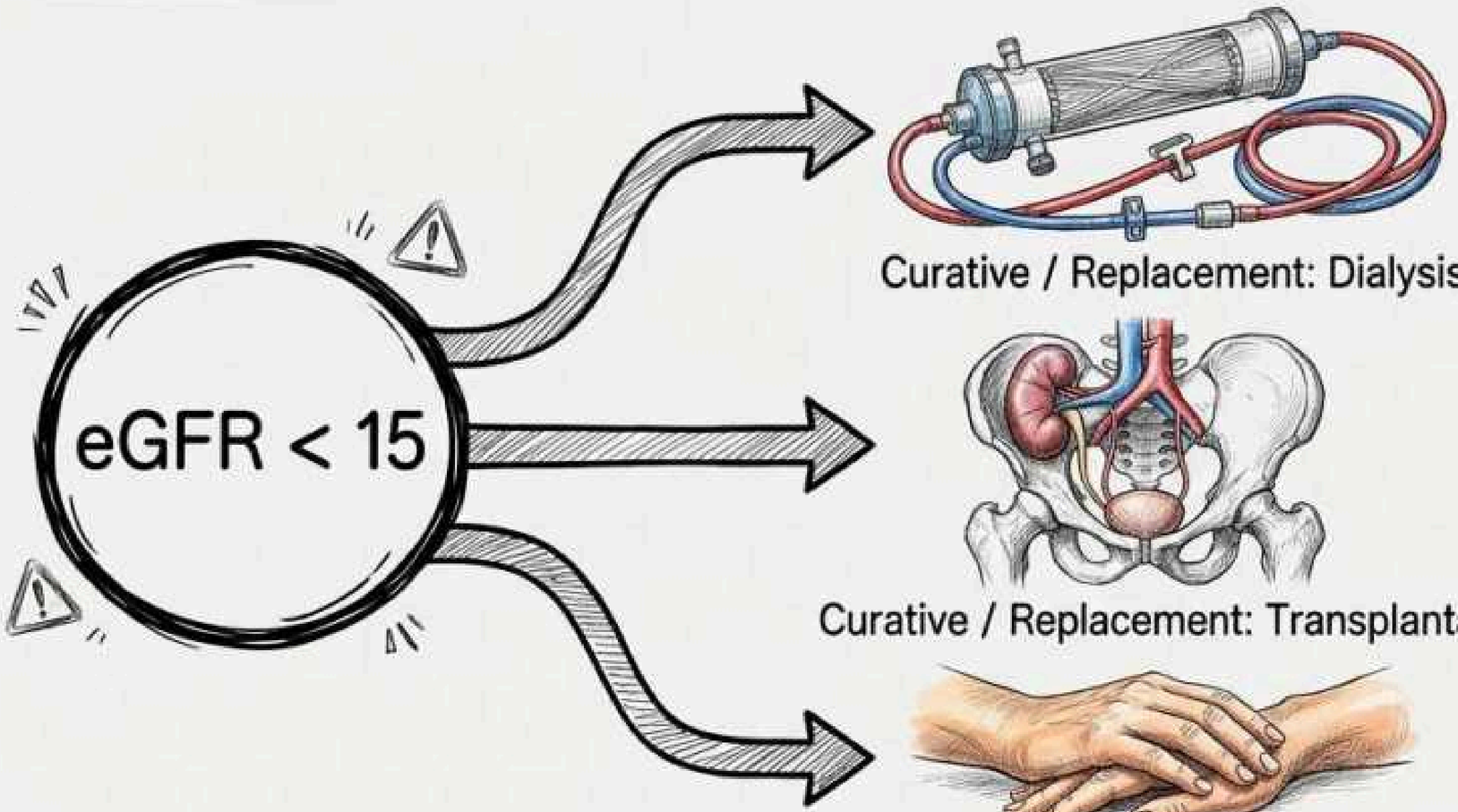
The Game Changer: SGLT2 inhibitors. Large trials prove they significantly slow kidney decline in patients both with and without diabetes.

- ☑ **Foundational Measures:** Treating diabetes tightly,
- ☑ aggressive smoking cessation,
- ☑ and strict avoidance of kidney-harming (nephrotoxic) drugs.

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Nearing Kidney Failure: The Crossroads

¶ 5.11



Curative / Replacement: Dialysis

Curative / Replacement: Transplantation

Conservative Care

The Imperative: As kidney failure approaches, proactive planning between patients and doctors is essential. Conservative Care Focus: Focused entirely on comfort without dialysis. This is a highly valid, crucial option for frail, older populations where the physical burden of dialysis may vastly outweigh its clinical benefit.

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Lecture Synthesis & Summary

¶ 5.12

Hypertension + Diabetes

Driven By

Chronic Kidney Disease
(Common & Silent Epidemic)

Filter Damage

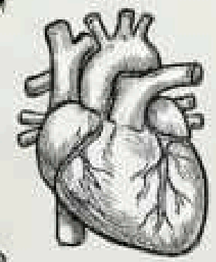
Nephrotic
(Protein leak, Low inflammation)

Nephritic
(Blood leak, High inflammation)

Intervention Goals

1. Slow Progression

2. Protect the Heart



(Next Lecture Preview: An exploration of kidney stones, the urinary tract, and renal replacement therapies).

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