RN in Training Script & Timestamp of Podcast

Manjot Monica K Dhami, Amrik S. Faloure, Mitchell Blusson, & Smita Mainali

Department of Nursing, Thompson River University

HLSC 4650 Health Sciences: Pathophysiology 3

Anila Virani, Ph.D. RN, BScN, MS

November 14th, 2025

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TIMESTAMP:	SCRIPT:
0:00-0:00	**Podcast Artwork:** Podcast artwork featuring the PSGN diagram is taken from The Paediatric Society of New Zealand (n.d.).
0:00-0:11	**Intro (Monica):** Hey everyone! It's Monica here — and I'm joined by Mitchell, Smita, and Amrik! Welcome to RN in TRAINING, where we turn textbook patho into real-life nursing sense.
0:11-0:20	**Case Study Intro (Amrik):** Yeah! So today we dive into the story of *P. Smith*, a 12-year-old from Abbotsford, B.C., who showed up with swollen eyes, puffy ankles, and cola-colored urine.
0:20-0:29	**Case Study Background (Mitchell):** Yeah — and the wild part? Just two weeks prior to that, he had a mild sore throat. No antibiotics,didn't take anything. He just shrugged it off.
0:29-0:40	**Case Study Presentation (Smita):** And now he's here with high blood pressure, fatigue, and low urine output. So let's break down how a simple sore throat snowballed into his kidneys going rogue — and what's actually happening inside.
0:40-0:58	**Patho of Kidneys (Monica):** So, to figure out what's going on with P. Smith, we need to zoom inside his kidneys — the body's natural filtration system. Each kidney filters about 150 quarts of blood daily, using around one million nephrons (National Institute of Diabetes and Digestive and Kidney Diseases, 2020). Inside each nephron is the glomerulus, that tuft of capillaries sitting in Bowman's capsule (National Institute of Diabetes and Digestive and Kidney Diseases, 2022).

0:58-1:24	**Patho of Glomerulus (Amrik):** And that filtration barrier it has three main layers: the fenestrated endothelium, its full of pores that let plasma pass but it blocks larger molecules; and the GBM, the glomerular basement membrane, it's a protein mesh that works like a selective filter; and there's the outer layer it's made up of the visceral epithelial cells, podocytes, and mesangial cells, which together keep filtration continuous and controlled (Kazi & Hashimi, 2023).
1:24-2:08	**Pathophysiology of Glomerulonephritis (Mitchell):** Under normal conditions, this whole system keeps proteins and red blood cells in the bloodstream while letting water and waste move through to make urine. But in glomerulonephritis, that protective barrier comes under attack (National Institute of Diabetes and Digestive and Kidney Diseases, 2022; Kazi & Hashimi, 2023). Glomerulonephritis, or GN,as we will call it, is a group of kidney disorders caused by immune-mediated injury to the basement membrane, mesangium, or capillary endothelium disrupting filtration. Acute GN may result from a primary renal disorder or a secondary illness. If acute GN isn't managed, it can progress to chronic disease, leading to fibrosis, reduced glomerular filtration rate or GFR, chronic kidney disease,and end stage renal disease, or even lead to cardiovascular complications (Kazi & Hashimi, 2023).
2:08-2:30	**Pathophysiology of Glomerulonephritis Part 2 (Smita):** And do you guys remember that sore throat he had? That was Strep A. His immune system produced IgG antibodies, but some of those antibodies formed complexes with bacterial antigens (Rawla et al., 2022). Those immune complexes circulated around and got stuck in the GBM and mesangium, activating C3 complement and triggering inflammation (Kazi & Hashimi, 2023).
2:30-3:09	**Pathophysiology of Glomerulonephritis Part 3 (Monica):** Exactly — that infection activated the complement cascade, especially C3, which essentially pulls the immune system's fire alarm (Kazi & Hashimi, 2023). GN can develop through four main immune-mediated injury patterns: Post-streptococcal GN, where circulating antigen–antibody complexes become trapped along the GBM; Staphylococcal-associated GN, which typically shows IgA immune deposits with C3; Systemic GN, seen in conditions such as systemic lupus erythematosus or IgA nephropathy; and small-vessel vasculitis, where T-cell and macrophage–driven inflammation causes direct, cell-mediated injury to the glomerulus (Kazi & Hashimi, 2023).

3:09-3:27	**Pathophysiology of Glomerulonephritis Part 4 (Amrik):** And with that alarm ringing, neutrophils and macrophages rush in and release interleukin-1, tumor necrosis factor-alpha, and platelet-derived growth factor. These chemicals make capillaries leaky and they cause glomerular cells to swell and multiply (Anton-Pampols et al., 2022; Kazi & Hashimi, 2023.
3:27-3:47	**Glomerulonephritis Symptoms (Mitchell):** Which brings us back to what we saw in his urine: The cola color is from hematuria —which are RBCs leaking through damaged capillaries (Saha et al., 2022). His 3+ protein lab result? That's proteinuria from widened podocyte slits letting albumin escape (Jiang et al., 2024). He's also showing azotemia, which means nitrogenous wastes are building up because filtration is impaired (Kazi & Hashmi, 2023).
3:47-3:57	**Glomerulonephritis Symptoms Part 2 (Smita):** And as albumin leaks out of his blood, oncotic pressure drops — so fluid moves into tissues (Gupta et al., 2019). That's the periorbital and pedal edema we saw (Kazi & Hashmi, 2023).
3:57-4:11	**Glomerulonephritis Symptoms Part 3 (Monica):** Meanwhile, the inflamed glomeruli narrow the capillary lumens, lowering GFR (Ihm, 2015). The kidneys sense poor perfusion and activate the RAAS system, leading to vasoconstriction and sodium retention (Ihm, 2015; Kanugula et al., 2023).
4:11-4:19	**Response (Amrik):** AHHHHH, that totally explains why his blood pressure shot up to 145 over 92 — thats the body's misguided way trying to compensate.
4:19-4:38	**Glomerulonephritis Symptoms Part 4 (Mitchell):** And with GFR dropping, uremic toxins like urea and creatinine accumulate as waste products in the blood (Kazi & Hashmi, 2023; Reyes-Rodriguez et al., 2020). Subsequently, cellular metabolism and ATP production decreases — leading to fatigue, malaise, and even muscle cramps (Rout et al., 2024). It's the early stage of uremia, when toxins accumulate because the kidneys can't clear them efficiently (Rout et al., 2024).

4:38-5:07	**Connection to Hypersensitivity Reaction Type (Smita):** This whole process is a Type III hypersensitivity reaction — meaning tissue damage happens when soluble immune complexes, like those antigen–antibody clusters, get stuck in the glomeruli and trigger inflammation (Power-Kean et al., 2022). In some chronic cases, T-cells and macrophages keep the attack going — a Type IV hypersensitivity reaction — which leads to fibrosis and glomerulosclerosis or hardening of kidney tissue over time (Kazi & Hashmi, 2023; Power-Kean et al., 2022).
5:07-5:30	**Diagnostics (Monica):** Conditions such as Goodpasture's disease can cause GN through Type II hypersensitivity mechanism, where antibodies directly target the GBM (Power-Kean et al., 2022). This immune injury leads to inflammation and increased glomerular cellularity due to the proliferation of endothelial, mesangial, and epithelial cells (Kazi & Hashmi, 2023).
5:30-5:40	**Response (Amrik):** Endocapillary proliferation happens inside the tufts, while extra capillary proliferation forms crescents in Bowman's capsule (Kazi & Hashmi, 2023).
5:40-5:56	**Glomerulonephritis Symptoms Part 5 (Mitchell):** The GBM may look thickened with immune deposits, and these structural changes block filtration, leading to oliguria —reduced, which is reduced urine output (Haider & Aslam, 2023; Kazi & Hashmi, 2023). Over time, this ongoing inflammation and scarring causes hyalinization and sclerosis, marking permanent kidney damage (Kazi & Hashmi, 2023).
5:56-6:05	**Response (Smita):** So basically, while his immune system was trying to protect him, it ended up hurting his kidneys — real friendly fire.
6:05-6:35	**Diagnostics Testing (Monica):** To confirm all this, we start with a urinalysis. P. Smith had protein, red blood cells, white blood cells, and several types of casts— those tiny clumps of cells or proteins formed inside inflamed tubules. His red blood cell and white blood cell casts point to glomerular injury, epithelial and tubular casts reflect inflammation, and granular, waxy, and fatty casts are all patterns seen in post-strep GN (Power-Kean et al., 2022).

6:35-7:03	**Diagnostics Lab Work (Amrik):** And then we look at blood work: · Blood urea nitrogen and creatinine those are elevated — meaning filtration has slowed (MacDonald, et al., 2022; Lewis et al.,2019). And creatinine clearance drops because less blood is passing through functioning glomeruli (MacDonald et al., 2022). ·And C3 complement is decreased because it's being used up in the inflammatory process (MacDonald, et al., 2022). We also check serum electrolytes — you know, potassium may rise with renal impairment — and albumin, which was also low from protein loss (Kazi & Hashmi, 2023).
7:03-7:18	**Diagnostics Testing Part 2 (Mitchell):** His ASO titer is positive — confirming recent Strep A (MacDonald, et al., 2022). Sometimes clinicians look at anti-DNase B or anti-GBM antibodies to differentiate autoimmune GN (MacDonald, et al., 2022). And if infection is still suspected, a throat culture helps detect lingering bacteria (Rawla et al., 2022).
7:18-7:56	**Diagnostic Imaging (Smita):** Imaging like a renal scan or IV pyelogram can show delayed contrast uptake when filtration is impaired (MacDonald, et al., 2022). But the gold standard is the renal biopsy (Power-Kean et al., 2022). Light microscopy would show endocapillary and extra capillary proliferation, electron microscopy shows thickened walls, and immunofluorescence reveals those bright IgG and C3 deposits (Power-Kean et al., 2022; Kazi & Hashmi, 2023). Together, all these pieces confirm the injury is immune-complex–mediated (Kazi & Hashmi, 2023). And as our doctor always reminds us — you never diagnose GN off one test. It's always the pattern that tells the story.
7:56-8:09	**Treatment (Monica):** P. Smith's treatment focuses on addressing both the triggers and the consequences. He was started on antibiotics to eliminate the strep antigen and prevent further immune activation (MacDonald et al., 2022).
8:09-8:15	**Treatment Part 2 (Amrik):** And to control his symptoms, he got diuretics to reduce edema and ACE inhibitors to lower blood pressure (Power-Kean et al., 2022; Kazi & Hashmi, 2023).

8:15-8:40	**Treatment Part 2 and Nursing Care (Mitchell):** He was also placed on sodium and fluid restriction and a low-protein diet to reduce kidney workload (Kazi & Hashmi, 2023; Ong, 2022; Ko et al., 2017). Nurses' tracked intake and outputs, daily weights, and monitored for changes in urine color and swelling (Koopmann, 2024; Kazi & Hashmi, 2023). Other possible treatments include corticosteroids, cytotoxic agents, and anticoagulants for controlling fibrin crescent formation in severe cases (Power-Kean et al., 2023). Dialysis can also be used to replace the loss of renal filtration in managing acute post-strep glomerulonephritis (Ong, 2022).
8:40-8:56	**Patient Education (Smita):** And education was huge — especially explaining why finishing antibiotics matters, and how to watch for swelling and changes in urine output (Rawla et al., 2022; Lewis et al.,2019). Within a week, his swelling improved, BP normalized, and urine output nearly doubled.
8:56-9:08	**Reflection (Monica):** P. Smith's case really shows how the immune, cardiovascular, and renal systems all intersect (Kazi & Hashmi, 2023). A simple sore throat became a cascade affecting his entire body.
9:08-9:16	**Reflection (Amrik):** And understanding the pathophysiology lets us connect every symptom, lab value, and treatment back to what's happening at the cellular level.
9:16-9:22	**Reflection and Wrap-up (Mitchell):** Exactly — our job isn't just to react to symptoms. It's to figure out what the body is trying to say.
9:22-9:28	**Wrap-up (Smita):** Right. Patho gives us the "why" behind the "what," and that's what guided his diagnosis, treatment, and recovery.
9:28-9:38	**Closing Remark (Monica):** Thanks for tuning in to RN in TRAINING! Keep learning, stay curious, and remember — even your kidneys deserve some lovein.