**Glomerulonephritis: Timestamp Sheet**

Kelsey Crowley, Anjali Duke, Taiya Duke & Jessye Large

Thompson Rivers University

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Anila RN, BScN, MN, PhD

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**Script**

**[00:00-00:03]:** \*Music\*

**[00:04-00:07] All:** “Patho Pod, tune right in, nursing knowledge, let’s begin” (OpenAI, 2023)**.**

**[00:12-00:24] Kelsey:** Hello, thank you for tuning in today to the Patho pod, where each week we discuss different interesting pathophysiology topics through case studies! This podcast can be enjoyed by anyone but is targeted at nursing students. My name is Kelsey

**[00:25-00:26] Jessye:** I’m Jessye.

**[00:26-00:27] Anjali:** I am Anjali.

**[00:27-00:28] Taiya:** And I am Taiya.

**[00:29-00:40] Kelsey:** Our topic today is on glomerulonephritis, which we will refer to as GN for the duration of this podcast (Kazi & Hashmi, 2023). To help better explain this topic, we will be using a case study to bring GN to life and provide clarification.

**[00:41-01:10] Taiya**: Oh my goodness I love case studies. Let me tell you about our patient, Ren. L. Failyer. He is a 76-year-old male with a history of diabetes and high cholesterol. He is admitted with a lower skin ulcer infection to his right extremity, which is associated with Methicillin-Resistant Staphylococcus Aureus or also known as MRSA (Nasr et al., 2024). He presents with hematuria, edema to the extremities, and high blood pressure (Kazi & Hashmi, 2023). Although his physicians don’t know it yet, Ren has developed glomerulonephritis related to MRSA (Nasr et al., 2024).

**[01:13- 01:35] Anjali**: GN is a leading cause of renal impairment (Kazi & Hashmi, 2023). There are many underlying causes of GN, however, today we will be discussing staphylococcus-associated GN and exploring the immune-mediated pathogenic process, involving both humoral and cell-mediated mechanisms (Kazi & Hashmi, 2023). Jessye, can you give me a run down on a staphylococcus mediated GN case?

**[01:36- 02:06] Jessye:** Sure can. Now although there are many triggers to GN, in Ren's case, it begins with a staphylococcus infection (Nasr et al., 2024). Staphylococcus-associated GN is an immune complex-mediated disease (Nasr et al., 2024). The antigen component of the immune complex is derived from the infective agent (Usman& Annamaraju, 2023). Staphylococcus antigens activate the T cells to produce B cells (Takayasu et al., 2022). These B cells then create and bind antibodies, IgA, IgG and IgM which create the immune complex (Liang et al., 2016).

**[02:07-02:14] Taiya:** Wait, doesn’t staphylococcus antigens produce way higher amounts of IgA antibodies than the others? (Liang et al., 2016).

**[02:15 - 02:36] Jessye:** Yes Taiya, correct. These immune complexes then circulate through our bloodstream (Usman & Annamaraju, 2023). Problems arise when the antigen-to-antibody ratio favours the antigen, creating soluble immune complexes (Usman & Annamaraju, 2023). The soluble immune complexes are small enough that they filter out of the bloodstream and into our organs, and in Ren's case, they have deposited in his kidneys (Usman & Annamaraju, 2023).

**[02:37-02:39] Anjali:** Oh no!! What happens next?

**[02:40-03:08] Kelsey:** Well, let me tell you Anj, now the classic pathway of the complement system is then activated due to the accumulation of immune complexes in the glomerulus (Jain et al., 2021). This pathway causes inflammation through opsonization, chemotaxis, mast cell degranulation and the membrane attack complex (Kareem et al., 2023). Pro-inflammatory cytokines and complement proteins arrive at the site of injury causing the bowman's capsule membrane cells to proliferate and then the membrane walls therefore thicken (Kareem et al., 2023).

**[03:09-03:20] Taiya:** Alright, now that we have reviewed how the inflammation begins, let’s discuss what symptoms we will see manifest as a result, and why. Ren’s symptoms include hematuria, edema to the extremities and hypertension (Kazi & Hashmi, 2023).

**[03:21-03:58] Anjali:** Because Ren has had thickening of the cell membranes in the glomerulus, it will limit his body's ability to clear creatinine and other wastes, which is why we would also see active urine sediments such as red blood cells form (Kazi & Hashmi, 2023). Hematuria (blood in the urine) and proteinuria (high levels of protein in the urine) are caused by the kidney's inability to adequately filter the blood when it becomes inflamed (Riley Children’s Health, n.d.). It can also lead to oliguria or anuria (Kazi & Hashmi, 2023). Eventually, the build-up of unfiltered waste products in the blood results in increased volume intravascularly, causing edema, and systemic hypertension (Kazi & Hashmi, 2023).

**[03:59-04:51] Jessye**: Now using our nursing brains, we can create a nursing diagnosis of excess fluid volume related to impaired renal function. To gain further clarification of what is really going on with Ren, further diagnostics will need to be run. In the emergency department, we know a culture swab of his wound was done and found to be MRSA (Nasr et al., 2024). Because we know MRSA can lead to GN, we can assume that this was the cause, however, we should rule out other possible causes (Nasr et al., 2024). The other testing could include hepatitis serology tests to rule out Hepatitis C associated GN, and autoantibody tests to determine whether to rule out autoimmune diseases (Kazi & Hashmi, 2023; Mastroianni-Kirsztajn, G et., 2015). There are many more diagnostics to rule out specific underlying causes such as immunoglobulin levels, complement protein levels, liver function tests, CBC levels, and blood cultures (Kazi & Hashmi, 2023). How do we specifically diagnose GN though?

**[04:52-05:39] Anjali:** Well to specifically diagnose GN, some diagnostic tests include renal ultrasound to assess the size of the kidneys and visualize inflammation (John Hopkins Medicine, 2021). Renal Biopsy to help determine the precise diagnosis, evaluate the degree of disease activity and severity, and, as a result, support appropriate treatment and prognostic prediction (AlYousef et al,.2020; Nasr et al., 2024). An electron microscopy assists with the precise localization of deposits in the kidneys, ultrastructural and podocyte appearance as well (Nasr et al., 2024) Additionally, immunofluorescence microscopy involves microscopy to assess for glomerular disease by looking at the staining (Jain et al., 2021). It reveals C3 dominant or codominant if glomerular staining is present. C3 is the only immunoreaction detected by this test (Jain et al., 2021).

**[05:40-06:31] Kelsey**: Very cool Anj. To assess renal function and severity of impairment, the following lab tests should also be done. A renal function test can determine the glomerular filtration rate to assess how well the kidneys are filtering out wastes (Kazi & Hashmi, 2023). Urine tests such as urinalysis can measure urine content abnormalities (Kazi & Hashmi, 2023). Rens labs and urinalysis came back and were as follows: Blood urea nitrogen, C-reactive protein and lipids are all elevated (GN Clinic, 2024). The glomerular filtration rate is read as low and high protein levels are found in his urine (GN Clinic, 2024). These abnormalities show that the kidney's normal ability to filter the blood is impaired (Nasr et al., 2024) We also run labs to give us more insight into what immunoglobulins are present; IgG and IgA are elevated, and IgM is low (Takayasu et al., 2022). Complement levels, which included low serum levels of C3 and C4, and c-reactive protein elevation indicate the presence of inflammation (Takayasu et al., 2022).

**[06:32-06:38] Jessye:** Kelsey it is also important to notewe would expect these labs to change as the diagnosis progresses into chronic (Nasr et al., 2024).

**[06:39-06:44] Kelsey:** Oh good point Jessye! So now that we know Ren has GN, now what?

**[06:45-07:51] Taiya:** Now that Ren has been diagnosed with staphylococcus-associated GN, one treatment that should be considered is removing the antigen (staph infection) by using antibiotics like vancomycin (Nasr et al., 2024). Administering antihypertensive drugs, and diuretics, and considering a dietary salt restriction to assist with edema and hypertension (Nasr et al., 2024). These will help with fluid retention but can cause an increase in the renin-angiotensin system (Nasr et al., 2024). It's important to add angiotensin-converting enzyme inhibitors and angiotensin receptor blockers to help protect the kidneys (Nasr et al., 2024; National Kidney Foundation, n.d). They will assist with lowering blood pressure and slow the damage to the glomeruli (Nasr et al., 2024). Other medications that could assist with inflammation and immunosuppressive activities include glucocorticosteroids such as Prednisolone (Ponticelli & Locatelli, 2018; NHS, 2023) and Immunosuppressant medications would also be used such as Rituximab and cytotoxic agents such as cyclophosphamide (Nasr et al., 2024). These agents should be administered along with glucocorticoids (Nasr et al., 2024). Lastly, Plasma exchange can be used to remove potential harmful products in blood (Nasr et al., 2024)

**[07:53-08:15] Anjali:** If Ren left his glomerulonephritis untreated and it became chronic these are some of the potential conditions it could result in: Pulmonary edema, hypertension, generalized anasarca, hypoalbuminemia, hypertensive retinopathy, hypertensive encephalopathy, rapidly progressive glomerulonephritis, chronic renal disease, and lastly nephrotic syndrome (Nasr et al., 2024).

**[08:17-08:21] Taiya:** Hmmm… I wonder what happens if this acute flare of GN goes untreated?

**[08:22-09:07] Jessye:** Oh, Taiya it is not good**.** If the disease progresses into chronic GN, patients may have to undergo frequent routine lab draws to monitor renal function such as renal function tests, serum albumin, and urine protein excretion rate (Nasr et al., 2024). It’s important to monitor and control blood pressure by taking medications such as ACE, ARBs, and diuretics (Nasr et al., 2024). The complications of increasing chronic disease, such as anemia, acidosis, cardiovascular disease, restless legs, cramps, and bone mineral problems, can be managed by medical specialists (Nasr et al., 2024). A dietician referral could be beneficial to assist with a low-sodium diet and renal replacement therapy could be used for an acute or chronic kidney failure (Nasr et al., 2024).

**[09:08-9:45] Kelsey:** Well, I think we’ve said it all! Thank you so much for listening to our podcast on Glomerulonephritis. To recap, GN is a hypersensitivity III reaction of the immune system and can be caused by a variety of triggering factors. This disease causes inflammation in the kidneys leading to a variety of issues as discussed in this podcast. It can be difficult to diagnose but with the correct testing, imaging and treatment it can hopefully be dealt with early in the disease process, avoiding progression into the chronic problem. We appreciate all of our fans who have listened to our podcast, but we regret to inform you we will be taking a step back from podcasting to focus on finishing our nursing degrees. Thank you for understanding.

**[09:46-09:47] All:** Goodbye!

**[09:47-09:51]** \*Music\*