Patients with Rapidly (Crescentic) Progressive Glomerulonephritis after Strepococcal Infection: A Case Report

Hengki Wijaya[1,2]*, Nunuk Mardiana[1,2]

[1]*Division of Nephrology, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya*
[2]*Division of Nephrology, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya*

*Corresponding author: primush2001@yahoo.com

KEYWORDS

RPGN, Kidney injury, Methylprednisolone, Crescentic glomerulonephritis

ABSTRACT

Rapidly Progressive Glomerulonephritis (RPGN) is a rare and prevalent condition worldwide, with an incidence of approximately 7 cases per 1 million people in the United States each year. It is the most common cause of kidney injury at a young age in the Middle East, Africa, Australia, and worldwide. The cause of death in RPGN is usually pulmonary involvement. The approach to diagnosis and therapy of RPGN is challenging for clinicians. A 24-year-old woman presented with symptoms such as shortness of breath, edema, subconjunctival hemorrhage, and red and foamy urine. The patient had a history of fever and swallowing pain. Initial therapy included ampicillin sulbactam, metronidazole, furosemide, candesartan, and nifedipine GITS. On day 8, the patient experienced worsening shortness of breath and anuria. Hemodialysis and methylprednisolone pulse therapy were initiated, and the patient improved clinically. Outpatient therapy included methylprednisolone, azathioprine, nifedipine GITS, ramipril, and bisoprolol. The patient's kidney biopsy results showed a 61% glomerular crescentic condition with negative IF IgG, IgA, IgM, C1q, and C. Untreated RPGN can lead to rapid loss of renal function.

Introduction

Rapid progressive glomerulonephritis (RPGN) is a clinical and pathological syndrome characterized by a rapid decline in renal function over a brief duration (days to weeks). Histopathological examination reveals cellular crescentic formations in the glomeruli, indicative of a proliferative cellular response occurring outside the glomerulus within Bowman's capsule; due to its crescentic morphology, it is also termed crescentic glomerulonephritis [(1)]. Rapidly progressing glomerulonephritis is exceedingly uncommon globally. The incidence in the United States is roughly 7 cases per 1 million individuals per year, while in the United Kingdom, it is about 2 cases per 1 million individuals annually. RPGN is comparatively uncommon among African Americans. The male-to-female ratio in the majority of research is approximately 1:1. In the majority of studies, the mean age is approximately 30 years, with a secondary peak occurring in the late sixties to seventies [(2,3)].

Post-streptococcal glomerulonephritis (PSGN) is marked by a fast deterioration in renal function resulting from an inflammatory response (type III hypersensitivity reaction) subsequent to streptococcal infection. PSGN typically manifests in youngsters approximately 1 to 2 weeks following a sore throat or 6 weeks subsequent to a skin illness (impetigo) [(1,4)]. The elevated prevalence of PSGN in developing nations is attributable to a rise in skin diseases (pyoderma). Despite a decline in industrialized nations, it remains the predominant cause of

glomerulonephritis (GN) among children in the United States [5].

PSGN is the predominant cause of renal damage in youth across the Middle East, Africa, Australia, and globally. The yearly occurrence of new PSGN cases in poor nations varies from 8.5 to 28.5 per 100,000 individuals. Approximately 97% of documented instances of PSGN occur in disadvantaged nations. The condition predominantly impacts children aged 3 to 12 years, with a peak incidence occurring between 5 and 6 years, as well as elders over 60 years of age. Incident edema occurs in around 65-90% of instances. Periorbital edema is characteristic of nephritic syndrome [1].

Based on this explanation, the approach to diagnosis and therapy of post-streptococcal RPGN is a big challenge for clinicians. In this case report, we report a case of rapidly progressive glomerulonephritis after streptococcal infection.

Case

A patient, Mrs. RF, 24 years old, female gender, Javanese ethnicity, unmarried, works as a mall cashier, resides in Surabaya, came to the emergency room of Dr. Soetomo Hospital with complaints of swelling since 5 days before hospitatalized. The swelling appeared initially on both legs and was followed by swelling of the whole body 3 days after. The patient had an increase in weight of 5 kg since 5 days before hospitalized (last weight before hospitalization 56 kg). The patient also complained of shortness of breath, which was felt for 2 days before hospitalized. Shortness is felt continuously, not affected by activity. The patient said he had a fever for 3 days at 7 days before hospitalized, without coughing or runny nose. There were also complaints of redness of both eyes, without complaints of itching or decreased sharp vision, which was felt since 5 days before hospitalized. There were no complaints of nausea or vomiting. The patient felt that his urine was cloudy, thick, and foamy, but the patient did not feel that the amount of urine had decreased, and there were no complaints of defecation. History of frequent hair loss was denied; joint pain was denied. The patient also claimed to have no history of type 2 diabetes mellitus, hypertension, heart disease, liver disease, or kidney disease. The patient has also never been sick like this before.

The patient presented with moderate general condition and compos mentis consciousness (GCS 456). Blood pressure was 161/69 mmHg; pulse 92×/min, strong lift, regular; respiratory rate 24×/min; axilla temperature 36.8°C. Peripheral oxygen saturation (SpO_2) was 96% with a nasal cannula at 3 liters per minute. Body weight was found to be 70 kg. On head and neck examination, we found slightly anemic conjunctiva, non-icteric sclera, no dyspnea, and no cyanosis. Thoracic movements appear symmetrical and without retraction. There is no dilation of the heart borders. The first (S1) and second (S2) heart sounds are single and are not accompanied by a murmur or gallop. Vesicular sounds were heard in all lung fields, but there were fine wet rales in both lung fields without wheezing. On abdominal examination, we found a flat abdominal contour, palpation of a supple abdomen, and normal bowel sounds with a frequency of 12 ×/min. Meanwhile, on the superior and inferior extremities, we found minimal edema in both superior and inferior extremities; the acral was warm, dry, and red. Capillary refill time (CRT) was <2 seconds. From the fluid balance examination, we found the patient's total input was 806 ml and urine output was 1000 ml along with an estimated insensible water loss (IWL) of 500 ml, resulting in a fluid balance of -694 ml.

The routine laboratory panel when the patient first arrived showed the following results: Hb 9.5 g/dL; HCT 28.4%; MCV 81.4 fL; MCH 26.9 pg; Leucocyte 19,850/µL; Neutrophil 76%; Lymphocyte 13%; Platelet 385.000 µL; Reticulocyte 2.26%; Immature Reticulocyte Fraction (IRF) 7.8%; PPT 14.8 sec (control 11.6-14.5 sec); APTT 30.9 sec (control 28.6-42.2 sec); BUN 21 mg/dL; Serum Creatinine 0.94 mg/dL; Na 121 mmol/L; K 4.7 mmol/L; Cl 87 mmol/L; AST 27 U/L; ALT 20 U/L; Bilirubin-Direct 0.16 mg/dL; Bilirubin-Total 0.55 mg/dL; Albumin 2.3 g/dL; Random Blood Glucose 99 mg/dL; CRP 0.3 mg/dL; HBsAg non-reactive; Rapid HIV non-reactive. From the results of blood gas analysis with

nasal cannula oxygen 3 lpm, pH 7.43; pCO2 26 mmHg, pO2 115 mmHg, HCO3 17.3 mmol/L, BE -7.0 mmol/L, and SaO2 99%. Urinalysis examination as follows: Color red; clarity clear; pH 5.5; nitrite (-); leukocytes (2+), ketones (-); glucose (-); erythrocytes (3+); bilirubin (-); urobilinogen normal; protein 4+); P:C ratio ≥ 0.5 mg/g; AC ratio ≥ 300 g/g. Based on these results, we found normocytic normochromic anemia, reticulocytosis, increased IRF, hypervolemic hypotonic hyponatremia, hypoalbuminemia, and hematuria.

On thoracic x-ray examination, there was a reticular pattern in the right and left parahilar-paracardial ridges, which could be interstitial lung edema, and there was loss of the costophrenic angle at the level of costa 9-10 bilaterally, indicating minimal bilateral pleural effusion. In the BOF photo, there was normal bowel contour, and no free fluid in the abdominal cavity, indicating no ascites. Ultrasound examination showed bilateral parenchymal kidney disease.

Based on these data, the initial diagnosis was acute pulmonary edema, suspected nephrotic syndrome differential diagnosis with nephritic, hypoalbuminemia, hypervolemic hypotonic hyponatremia, normocytic normochromic anemia, stage 1 hypertension, bilateral subconjunctival hemorrhage. Further work-up included lipid profile, procalcitonine, ANA test, anti ds-DNA, C3, C4, ASTO titer, Esbach urine protein, and urine sediment. Initial therapy in this patient was nasal cannula oxygen 4 liters per minute (lpm), high calory-enough protein-low salt diet 1900 kcal/day, Ampicillin Sulbactam 1.5 g/6 hours, Metronidazole 500 mg/8 hours, Furosemide 20 mg/8 h, Candesartan 16 mg/24 h, nifedipine GITS 30 mg/24 h, maximum drink 1000 ml/24 h, and Naphazoline HCl eye drops 1 mg 6 drops/24 h. The patient was also referred to the cardiology department. The patient was also referred to cardiology, and echocardiography was planned for the patient.

On the 2nd day of treatment, the patient was examined for lipid profile, procalcitonine, ANA test, anti ds-DNA, C3, C4, Esbach urine protein, and urine sediment, with the following results: triglycerides 93 mg/dL; total cholesterol 184 mg/dL; calcium 7.43 mg/dL (corrected calcium 8.6); phosphate 4.65 mg/dL; procalcitonin 0.37 ng/mL; ANA test 14.67 AU/mL; anti ds-DNA 10.06 IU/mL; C3 11 mg/dL; C4 14 mg/dL; ASTO titer 400 IU/mL. For protein examination, Escbach was found to be 0.02 grams/24 hours with a urine amount of 200 ml/24 hours (urine protein level of 0.1 grams/liter). In the urine sediment examination, we found erythrocytes 1279/microL, leucocytes 1204/microL, EC (Epithelial Cast) 14.6/microL, and Cast 28.4/microL. From these results, we found decreased C3 and C4, proteinuria, and hematuria. Based on these data, our provisional diagnosis was acute post-streptococcal glomerulonephritis. Then, during the next treatment, we closely observed the patient regarding complaints of shortness of breath, fluid balance, and daily weight.

On the 7th day of treatment, there was a decrease in condition with complaints of tightness still present. The patient's weight was still 69 kilograms. On thoracic examination, we found abnormalities in the form of fine wet rhonchi sounds in both lung fields without wheezing. On the inferior extremities, we found edema; the acral was warm, dry, and red. From the fluid balance examination, we found the patient's total input was 620 ml and the output of urine production was low, 150 ml, along with an estimated insensible water loss (IWL) of 500 ml, resulting in a total fluid balance of +30 ml.

The results of a complete blood examination showed a decrease in Hb (9.8 g/dL), a decrease in leucocytes (13,930/µL), an increase in BUN (75 mg/dL), an increase in serum creatinine (7.59 mg/dL), an increase in potassium (7.5 mmol/L), a decrease in albumin (2.3 g/dL), and mixed type acidosis (pH 7.26; pCO_2 40 mmHg; pO_2 41 mmHg; HCO_3 17.9 mmol/L; BE -9.2 mmol/L; SaO_2 98%). In response to these results, we provided therapy in the form of a furosemide pump 5 mg/hour, correction of hyperkalemia in the form of an injection of dextrose 40% 25 ml and 2 units of rapid acting insulin for 4 cycles with an interval of 30 minutes accompanied by an injection of Ca Gluconas 10% 10 ml bolus, and infusion of 20%

albumin 100 ml consumed in 4 hours. We also conducted a supporting examination of the patient's repeat thoracic photos and obtained a picture of bilateral pleural effusion and cardiomegaly.

On the 8th day of treatment, there was a decrease in condition with complaints of tightness that remained. The patient's weight was 69 kilograms. On thoracic examination, we still found decreased breath sounds in the right lower lung field, accompanied by fine wet rales sounds in both lung fields without wheezing. On the inferior extremities, edema was still found on both inferior extremities. From the fluid balance examination, the patient's total input was 500 ml and urine output decreased by 70 ml (with furosemide pump 5 mg/hour) along with an estimated insensible water loss (IWL) of 500 ml, resulting in a total fluid balance of +50 ml. In response to these results, we considered cito dialysis with a prescribed duration of 2.5-3 hours, Qb (quick blood volume) 125-150 ml/min, Qd (quick dialysate volume) 500 ml/min, with minimal heparin, and ultrafiltration of 1000 ml + fluid input.

On the 9th day of treatment, which was 1 day after dialysis, the complaint of shortness of breath was found to be reduced but still present. From the fluid balance examination, we found that the patient's total input was 620 ml and the urine production output was still small, namely 50 ml, along with an estimated insensible water loss (IWL) of 500 ml, with a total fluid balance of +30 ml. The complete blood test results showed decreased Hb (9.4 g/dL), increased BUN (77 mg/dL), increased serum creatinine (9.02 mg/dL), increased potassium (8.1 mmol/L), decreased sodium (116 mmol/L), decreased albumin (2.3 g/dL), mixed type acidosis (pH 7.32; pCO2 38 mmHg; pO2 113 mmHg; HCO3 19.6 mmol/L; BE -6.5 mmol/L; SaO2 95%). In response to these results, we increased the dose of the furosemide pump to 10 mg/hour, corrected hyperkalemia in the form of an injection of dextrose 40% 25 ml and 2 units of rapid acting insulin for 4 cycles with an interval of 30 minutes accompanied by an injection of Ca Gluconas 10% 10 ml bolus, and administered a methylprednisolone pulse dose of 500 mg/day. We also planned a repeat thoracic examination for post hemodialysis evaluation and found bilateral pleural effusions with an improved impression.

On the 11th day of treatment, we found the patient complaining of shortness of breath again. We found the patient's total input was 640 ml and urine output was 80 ml, along with an estimated insensible water loss (IWL) of 500 ml, with a total fluid balance of +60 ml (accumulative balance 2 days after dialysis was +90 ml). In response to this downward trend, we considered 2nd cito dialysis with the prescribed duration of 3 hours, Qb (quick blood volume) 185 ml/min, Qd (quick dialysate volume) 500 ml/min, with minimal heparin, and ultrafiltration of 1000 ml + infused fluid.

On the 14th day of treatment, it was found that complaints of shortness of breath had been much reduced and urine increased with a clearer color. From the fluid balance, we found that the patient's total input was 540 ml and urine production output increased sharply to 1500 ml along with an estimated insensible water loss (IWL) of 500 ml, with a total fluid balance of -1360 ml. The results of the complete blood examination showed a decrease in Hb (9.3 g/dL), an increase in BUN (102 mg/dL), an increase in serum creatinine (8.15 mg/dL), a decrease in potassium (5.2 mmol/L), an increase in sodium (128 mmol/L), mixed type acidosis (pH 7.21; pCO2 43 mmHg; pO2 79 mmHg; HCO3 17.6 mmol/L; BE -10.3 mmol/L; SaO2 67% venous impression). Based on these data, we considered that the patient had responded to steroid therapy and was now dehydrated due to significantly increased urine production. We gradually reduced the furosemide pump dose to 5 mg/h and then to 20 mg/8 h the following day and increased the patient's fluid input with a target of input = output + IWL.

On the 20th day of treatment, the complaint of tightness was found to be absent. From the fluid balance, the patient's total input was 3162 ml and urine output was 2000 ml, along with an estimated insensible water loss (IWL) of 500 ml, with a total fluid balance of +662 ml. The results of a complete blood examination

showed an increase in Hb (10 g/dL), a decrease in BUN (41 mg/dL), a decrease in serum creatinine (1.24 mg/dL), a decrease in potassium (3.4 mmol/L), an increase in sodium (140 mmol/L), and mixed type acidosis improved (pH 7.32; pCO2 41 mmHg; pO2 74 mmHg; HCO3 21.1 mmol/L; BE -5 mmol/L; SaO2 93%). For urine evaluation, there was significant improvement, as follows: Escbach protein 0.225 grams/24 hours with urine volume 2250 ml/24 hours (urine protein level 0.1 grams/liter); urine sediment examination obtained erythrocytes 3.9/microL; leucocytes 20.3/microL; EC (Epithelial Cast) 13.3/microL; and Cast 0.83/microL. Workup related to further therapy was planned via poly with plans for re-evaluation of laboratory panels and renal biopsy. The patient was discharged home with discharge medication: Methylprednisolone 32 mg/8 hours, Azathioprine 50 mg/12 hours, Nifedipine GITS 30 mg/24 hours, Ramipril 5 mg/24 hours, and Bisoprolol 2.5 mg/24 hours.

When the patient returned for follow-up, a kidney biopsy was performed, and the anatomical pathology result was Crescentic Glomerulonephritis (Rapidly Progressive Glomerulonephritis). We continued the therapy that had been given to the patient.

DISCUSSION

Rapid progressive glomerulonephritis (RPGN) is a clinical and pathological illness characterized by a swift decline in renal function over a brief period, typically ranging from days to weeks. Clinically, it is defined by nephritic urinalysis findings: proteinuria, micro- or macroscopic hematuria, dysmorphic red blood cells (RBCs), and RBC casts. Histopathological examination of kidney biopsy reveals cellular crescentic formation in the glomeruli, indicative of a proliferative cellular response occurring outside the glomerulus within Bowman's capsule. Due to its crescentic morphology, it is termed crescentic glomerulonephritis [(1)]. The severity of the disease correlates with the genesis of the immunologic process and the extent of crescent formation. Patients exhibiting 80% circumferential crescents in the glomerulus experience renal failure that may be refractory to treatment; conversely, those with crescents less than 50% typically demonstrate a slower development and a favorable response to therapy [(6)]. RPGN is categorized primarily by the mechanism of glomerular injury: Anti-GBM illness (Goodpasture syndrome), immune complex deposition, and pauci-immune (minimal or absent deposition) [(7)].

Crescentic glomerulonephritis linked to immune complex deposits is further categorized according to serological and histological characteristics of the underlying condition, including immunoglobulin A (IgA) deposits in IgA nephropathy and IgA vasculitis; antistreptococcal antibodies and subepithelial humps in post-streptococcal glomerulonephritis; anti-nuclear antibodies with immunofluorescent staining alongside IgG, IgA, IgM, C3, and C1q with mesangial-subendothelial deposits in lupus nephritis; and circulating cryoglobulin with intraluminal thrombus in cryoglobulinemia [(8,9)].

Post-streptococcal glomerulonephritis (PSGN) is marked by a fast deterioration in renal function resulting from an inflammatory response (type III hypersensitivity reaction) subsequent to streptococcal infection. The condition arises from a particular strain of group A beta-hemolytic streptococcus known as nephrogenic streptococcus. The ailment impacts the glomeruli and minor blood arteries of the kidney. PSGN typically manifests in youngsters 1 to 2 weeks following a sore throat or 6 weeks subsequent to a skin illness (impetigo) [(1,4)].

The precise mechanism underlying PSGN remains unknown. The body reacts to inflammation, namely nephrogenic streptococcal infection, by generating immune complexes that comprise streptococcal antigens and human antibodies. The antigens involved in immune complex development are diverse, comprising either exogenous (viral or bacterial) or autogenous (nuclear or tumor) antigens. Immune complex-mediated glomerulonephritis frequently results from multisystem diseases such as lupus or may develop as a consequence of other primary glomerulonephritis, including GN and C3 GN membranes [(10)]. Certain ideas

propose that these immunological complexes are deposited in the renal glomeruli via the bloodstream. The phenomenon of "in situ immune complex formation" arises from interactions with streptococcal antigens lodged in the glomerular basement membrane or, alternatively, from antibody responses to glomerular constituents that exhibit cross-reactivity with streptococcal antigens due to molecular mimicry [5,11].

Immune complexes activate the alternative complement pathway, resulting in leukocyte infiltration and mesangial cell proliferation in the glomerulus, which impairs capillary perfusion and decreases the glomerular filtration rate (GFR). Decreased GFR may result in renal failure (oliguria or anuria), acid-base disturbances, electrolyte imbalances, fluid overload, edema, and hypertension [1].

When symptomatic, post-streptococcal glomerulonephritis typically manifests with characteristics of nephritic syndrome, including hematuria, oliguria, hypertension, and edema. Uncommon presentations may resemble nephrotic syndrome with substantial proteinuria. Nephrogenic streptococcal infection precedes post-streptococcal glomerulonephritis, initially impacting the skin or oropharynx. Recently, PSGN has been more frequently linked to skin infections (impetigo) than to throat infections (pharyngitis) [1,12].

The predominant presenting symptom is macroscopic hematuria, observed in 30 to 50% of acute PSGN cases; patients frequently characterize their urine as cloudy, tea-colored, and resembling cola. Hematuria may be characterized as post-pharyngitic, occurring weeks subsequent to infection. Renal involvement is prevalent and temporary, with recovery occurring within 1 to 2 weeks. Fewer than fifty percent of the patients exhibit oliguria. Based on the degree of renal impairment, tdana, and indications of anuric renal failure or critical acid-base disturbances, electrolyte imbalances (notably hyperkalemia), and fluid overload will necessitate renal replacement therapy (RRT). Approximately 60-80% of patients exhibit hypertension, which typically resolves within 10 days [1,5].

Rapidly progressive GN is a common feature in all types of crescent GN. With the exception of pauciimmune GN, tdana and extra-renal symptoms may also be observed. Pulmonary hemorrhage and severe anemia are prevalent in Goodpasture syndrome. Arthralgia, skin rash, pleurisy, and pericarditis may be associated with lupus nephritis. Henoch-Schonlein purpura is characterized by purpura, arthralgia, and stomach colic. Purpura, fatigue, arthralgia, hepatosplenomegaly, and peripheral neuropathy are common manifestations of cryoglobulinemic nephritis. The clinical features of small vessel vasculitis vary widely [4].

In this case, a 25-year-old woman presented with complaints of swelling initially in both legs followed by swelling throughout the body. The patient was found to have an increase in body weight accompanied by continuous shortness of breath. The patient previously had a history of fever without a cold cough 7 days SMRS. The patient also complained of red eyes. The patient complained that his urine was cloudy, thick, and foamy but felt that his urine did not decrease. On physical examination, the patient was found to be hypertensive with a blood pressure of 161/69. The patient's body weight was 70 kg. The conjunctiva was slightly anemic. In the lungs, fine wet rales were found. The extremities were found to have minimal edema.

PSGN should be considered in persons with hypertension and heart failure, regardless of the presence of hematuria or prior episodes of sore throat or pyoderma. Evidence of prior streptococcal infection is assessed by quantifying anti-streptolysin titer (ASO) and anti-nicotinamide-adenine dinucleotidase (anti-NAD), both of which often elevate following pharyngitis. Additional antibodies, including anti-DNAse B and anti-hyaluronidase (AHase), are typically raised following pharyngitis and cutaneous infections. The ASO titer is the most commonly utilized test, although the streptozyme test, which assesses the titers of all aforementioned antibodies, is the most sensitive. ASO titers may be significantly diminished in patients undergoing antibiotic treatment for streptococcal infections [1].

Serum complement (C3) levels are often diminished due to their utilization in inflammatory responses. The reduction in C3 concentration typically precedes the rise in serum ASO levels. Complement levels typically normalize between 6 to 8 weeks. The urinalysis reveals macroscopic or microscopic hematuria, red blood cell casts, and mild proteinuria. Merely 5% of individuals with post-streptococcal glomerulonephritis have significant proteinuria indicative of nephrotic syndrome. White blood cell counts, hyaline casts, and cellular casts are typically observed in urinalysis. Tests for Renal Function: Blood urea nitrogen (BUN) and serum creatinine are often high during the acute period. These readings will typically revert to normal subsequently. Patients with heart failure with PSGN exhibit high NT-proBNP levels and evidence of pulmonary edema on chest X-ray.

Renal biopsy is warranted in cases with persistent renal function deterioration, anuria, renal failure, absence of a latent interval between acute glomerulonephritis and streptococcal infection, normal complement levels, and lack of elevation in antistreptococcal antibodies. Ultrasonography reveals that the kidneys are enlarged in a subset of patients. A chest X-ray may reveal pulmonary edema in patients exhibiting symptoms indicative of volume overload or heart failure. Light microscopy of glomerular preparations reveals nonspecific hypercellularity, comprising endothelial, mesangial, and inflammatory cells. Electron microscopy identified the predominant feature as sub-epithelial humps, which are electron-dense deposits located in the subepithelial region adjacent to the glomerular basement membrane. Immunofluorescence microscopy reveals the presence of IgG and C3 deposits within the initial 2 to 3 weeks of the disease [(1)].

Individuals with rapidly progressing glomerulonephritis may undergo serological testing for anti-GBM antibodies via ELISA or western blot techniques. The ANCA test, conducted using indirect immunofluorescence, along with its qualitative variants P-ANCA or C-ANCA, may also be evaluated. Serological assays for the identification of post-infectious conditions: antistreptolysin titer for post-streptococcal infection, and serological tests for HIV, Hepatitis B, and Hepatitis C. Complement C3 and C4 levels may be diminished in certain types of granular immune complex illnesses that lead to RPGN, including lupus, cryoglobulinemia, and primary MPGN. Serological tests for lupus, including anti-nuclear antibodies, double-stranded DNA, and anti-Smith antibodies, may be elevated for evaluation. Rheumatoid factor and cryoglobulin levels are assessed in suspected cases of cryoglobulinemia [(7,13,14)].

The lab examination of the patient showed Hb 9.5 g/dL; Immature Reticulocyte Fraction (IRF) 7.8%; Na 121 mmol/L; Alb 2.3 g/dL; Erythrocytes (3+); Protein 4+); P:C ratio $\geq$ 0.5 mg/g; A:C ratio $\geq$ 300 g/g. The lab work showed normocytic normochromic anemia, reticulocytosis, elevated IRF, hypervolemic hypotonic hyponatremia, hypoalbuminemia, hematuria, and proteinuria. Imaging showed minimal bilateral pleural effusion and bilateral parenchymal kidney disease. On the 2nd day of treatment, the results of the lab examination were total cholesterol 184, calcium 7.43 (corrected calcium 8.6), ANA-Test 14.67 AU/ml, anti ds-DNA 10.06 IU/mL, C3 11 mg/dL, C4 14 mg/dL, and ASTO titer 400 IU/mL. Urine sediment examination showed erythrocytes 1279/microL, leucocytes 1204/microL, EC (epithelial cast) 14.6/microL, and cast 28.4/microL. The examination showed decreased C3 and C4, proteinuria, and hematuria.

Untreated quickly progressing glomerulonephritis can lead to swift deterioration of renal function within weeks to months. Commencing treatment promptly is of paramount importance. Empirical treatment is recommended to commence prior to establishing a formal diagnosis, particularly in instances where serological tests and kidney biopsy are postponed for whatever reason. This empirical treatment comprises pulse intravenous administrations of methylprednisolone, either 500 mg or 1 g, for a minimum of three doses. Plasmapheresis may be contemplated particularly if the patient exhibits hemoptysis that raises suspicions of a severe variant of Goodpasture illness until a definitive diagnosis is established [(7)].

Individuals with acute nephritic syndrome necessitate limitations on sodium and fluid consumption. For over thirty years, loop diuretics have been recognized for their ability to expedite the alleviation of edema and hypertension. Thiazide diuretics are ineffective, while aldosterone antagonists provide a danger of hyperkalemia. Nifedipine may be beneficial in instances of severe hypertension. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers provide a risk of hyperkalemia. Nitroprusside may be required to manage hypertensive encephalopathy, although solely in severe instances. Pulmonary edema may aggravate the clinical trajectory and necessitates treatment with oxygen and loop diuretics. Digitalis is ineffective and poses a risk of toxicity. Complications in Acute PSGN may include posterior reversible leukoencephalopathy and immune-mediated pneumonitis. Hemodialysis and peritoneal dialysis may be necessary to address severe azotemia, hyperkalemia, or circulatory stasis. Isolated findings indicate an increase in crescentic Acute PSGN with a quickly progressing clinical course associated with pulse intravenous methylprednisolone treatment [15,16,17].

In a case report by Yano, a patient with crescentic post-streptococcal acute glomerulonephritis (PSAGN) accompanied by small vessel vasculitis was treated with glucocorticoids, resulting in a successful reduction of disease activity and the cessation of hemodialysis. Immunosuppressive treatments, including steroids and calcineurin inhibitors, are typically contraindicated for post-streptococcal acute glomerulonephritis (PSAGN), even in pediatric patients. Nevertheless, certain data indicate that intensive treatment may be beneficial for severe instances of PSAGN characterized by cellular crescent formation and widespread interstitial infiltration of inflammatory cells. A study indicated that standard doses of oral prednisone may be administered alone or in conjunction with azathioprine, however the resultant benefit is typically minimal [8,9].

This patient received initial therapy in the form of nasal cannula oxygen 4 liters per minute (lpm); high calory-enough protein-low salt diet 1900 kcal/day; Ampicillin Sulbactam 1.5 g/6 hours intravenous; Metronidazole 500 mg/8 hours intravenous; Furosemide 20 mg/8 h intravenous; Candesartan 16 mg/24 h; nifedipine GITS 30 mg/24 h; maximum drink 1000 ml/24 h; and eye drops. Naphazoline HCl 1 mg 6 drops/24 h. The patient had hypercalcemia correction. The patient had hyperkalemia correction in the form of an injection of dextrose 40% 25 ml and 2 units of rapid acting insulin for 4 cycles with an interval of 30 minutes accompanied by an injection of Ca Gluconas 10% 10 ml bolus and an infusion of 20% albumin 100 ml consumed in 4 hours. In addition, the patient also performed cito hemodialysis and received a methylprednisolone pulse dose of 500 mg/day. The patient was discharged with medication: Methylprednisolone 32 mg/8 hours, Azathioprine 50 mg/12 hours, nifedipine GITS 30 mg/24 hours, Ramipril 5 mg/24 hours, and Bisoprolol 2.5 mg/24 hours.

PSGN is predominantly a self-limiting condition in the majority of instances, necessitating just symptomatic management. Supportive treatment seeks to manage volume overload consequences, including hypertension and edema, which are significant in the acute phase of the disease. Loop diuretics, such as furosemide, are favored over thiazide diuretics, like hydrochlorothiazide or chlorthalidone. Patients exhibiting increasing renal failure or crescents on renal biopsy may utilize corticosteroids. Dialysis is conducted solely to regulate acid-base equilibrium, rectify electrolyte imbalances (notably hyperkalemia), and manage fluid levels. [1,5,18].

SUMMARY

A 24-year-old woman with quickly crescentic post-streptococcus glomerulonephritis. fast progressive glomerulonephritis (RPGN) is a clinical and pathological illness that causes fast kidney function decrease in days to weeks. An inflammatory response (type III hypersensitivity reaction) after streptococcal infection causes post-streptococcal glomerulonephritis (PSGN), which rapidly

declines kidney function. The nephrogenic streptococcus strain causes this disease. An estimated 15% of RPGN cases involve immune complex accumulation in glomerular capillaries. Immune complexes are formed in circulation and deposited in glomerular capillary tufts or in situ in their walls. Urinalysis, ANCA serology, C3-C4, ASO titer, and ANA testing can exclude etiology in RAPGN patients, with biopsy to confirm histopathology. The patient received clinically indicated treatment. Patients can receive antibiotics, loop diuretics, antihypertensives, and hemodialysis. Crescent RPGN patients receive steroids.

REFERENCES

1. Rawla, P., Padala, S.A. and Ludhwani, D. (2022) 'Poststreptococcal Glomerulonephritis', *Primary Care Update for Ob/Gyns*, 10(1), pp. 24–28. Available at: https://doi.org/10.1016/S1068-607X(02)00141-5.
2. Lingaraj, U. *et al.* (2017) 'of Kidney Diseases and Transplantation Original Article A " Mini - Epidemic " of Anti -Glomerular Basement Membrane Disease : Clinical and Epidemiological Study', *Saudi Journal of Kidney Diseases dne and Transplantation*, 28(5), pp. 1057–1063.
3. Berti, A. *et al.* (2019) 'Incidence, prevalence, mortality and chronic renal damage of anti-neutrophil cytoplasmic antibody-associated glomerulonephritis in a 20-year population-based cohort', *Nephrology Dialysis Transplantation*, 34(9), pp. 1508–1517. Available at: https://doi.org/10.1093/ndt/gfy250.
4. Moroni, G. and Ponticelli, C. (2014) 'Rapidly progressive crescentic glomerulonephritis: Early treatment is a must', *Autoimmunity reviews*, 13(7), pp. 723–729. Available at: https://doi.org/10.1016/J.AUTREV.2014.02.007.
5. Van De Voorde, R.G. (2015) 'Acute poststreptococcal glomerulonephritis: the most common acute glomerulonephritis', *Pediatrics in review*, 36(1), pp. 3–13. Available at: https://doi.org/10.1542/PIR.36-1-3.
6. Baldwin, D.S. *et al.* (1987) 'The existence of a protracted course in crescentic glomerulonephritis', *Kidney International*, 31(3), pp. 790–794. Available at: https://doi.org/10.1038/ki.1987.67.
7. Naik, R.H. and Shawar, S.H. (2022) 'Rapidly Progressive Glomerulonephritis', *StatPearls* [Preprint].
8. Yano, K. *et al.* (2019) 'Crescentic poststreptococcal acute glomerulonephritis accompanied by small vessel vasculitis: Case report of an elderly male', *BMC Nephrology*, 20(1), pp. 1–6. Available at: https://doi.org/10.1186/S12882-019-1663-9/TABLES/1.
9. Couser, W.G. (1988) 'Rapidly Progressive Glomerulonephritis: Classification, Pathogenetic Mechanisms, and Therapy', *American Journal of Kidney Diseases*, 11(6), pp. 449–464. Available at: https://doi.org/10.1016/S0272-6386(88)80079-9.
10. Ooi, J.D. *et al.* (2017) 'Dominant protection from HLA-linked autoimmunity by antigen-specific regulatory T cells', *Nature*, 545(7653), pp. 243–247. Available at: https://doi.org/10.1038/NATURE22329.
11. Balasubramanian, R. and Marks, S.D. (2017) 'Post-infectious glomerulonephritis', *Paediatrics and international child health*, 37(4), pp. 240–247. Available at: https://doi.org/10.1080/20469047.2017.1369642.
12. Hunt, E.A.K. and Somers, M.J.G. (2019) 'Infection-Related Glomerulonephritis', *Pediatric clinics of North America*, 66(1), pp. 59–72. Available at: https://doi.org/10.1016/J.PCL.2018.08.005.
13. Radice, A. and Sinico, R.A. (2005) 'Antineutrophil cytoplasmic antibodies (ANCA)', *Autoimmunity*, 38(1), pp. 93–103. Available at: https://doi.org/10.1080/08916930400022673.
14. McAdoo, S.P. and Pusey, C.D. (2017) 'Anti-Glomerular Basement Membrane Disease', *Clinical journal of the American Society of Nephrology : CJASN*, 12(7), pp. 1162–1172. Available at: https://doi.org/10.2215/CJN.01380217.
15. Zaffanello, M. *et al.* (2010) 'Evidence-based treatment limitations prevent any therapeutic recommendation for acute poststreptococcal glomerulonephritis in

children.', *Medical science monitor : international medical journal of experimental and clinical research*, 16(4), pp. RA79-84.

16. Wiles, K.S. *et al.* (2011) 'Rare immune-mediated pneumonitis in association with post-streptococcal glomerulonephritis', *Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association*, 26(12), pp. 4140–4142. Available at: https://doi.org/10.1093/NDT/GFR526.
17. Rodriguez-Iturbe, B. and Haas, M. (2016) 'Post-Streptococcal Glomerulonephritis', *Streptococcus pyogenes: Basic Biology to Clinical Manifestations* [Preprint].
18. Yang, T.J. *et al.* (2018) 'Role of Steroids in Post-streptococcal Glomerulonephritis Without Crescents on Renal Biopsy', *Cureus*, 10(8). Available at: https://doi.org/10.7759/CUREUS.3150.