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Overview on Management of Acute Glomerulonephritis in the ED

Bedor Alghofaili Ayesh Alotaiby[1*], Nesreen Faiz Falemban[2]

[1]East Jeddah Hospital, Saudi Arabia
[2]Yanbu General Hospital MOH, Saudi Arabia

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***Corresponding author:** Bedor Alghofaili Ayesh Alotaiby
East Jeddah Hospital, Saudi Arabia

Abstract

The word "glomerulonephritis" refers to a group of kidney illnesses marked by immune-mediated destruction to the basement membrane, mesangium, or capillary endothelium, resulting in hematuria, proteinuria, and azotemia. Acute Kidney Injury episodes in glomerular disease are typically caused by rapidly progressive glomerulonephritis (RPGN). acute glomerulonephritis is caused by immunologically mediated damage caused by numerous infectious agents such as viruses, bacteria, or protozoa, as well as non-infectious causes such as Henoch–Schonlein purpura (HSP). The most prevalent infectious cause is post-streptococcal glomerulonephritis (PSGN). The emergency physician must conduct a thorough physical examination and obtain a complete medical history, including herbal agents, sports supplements, non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and calcineurin inhibitors, among other medications. In addition, the patient's blood pressure, weight, hydration status, edoema, skin manifestations, pulmonary and cardiac examinations must all be correctly monitored. Because there is no particular medication for renal illness, the treatment for acute poststreptococcal glomerulonephritis (PSGN) is mostly supportive. The underlying infections must be addressed when acute glomerulonephritis (GN) is accompanied with chronic infections. The critical care unit's expertise may be required for the treatment of individuals with hypertensive encephalopathy or pulmonary edoema. A nephrologist's consultation may be necessary. Renal function, blood pressure, edoema, serum albumin, and urine protein excretion rate should all be evaluated on an outpatient basis. In this article, we will be reviewing Acute glomerulonephritis, its evaluation as well as management.

Keywords: Kidney, glomerunephritis, nephritis, renal failure.

INTRODUCTION

The 'nephron' is the anatomical and functional unit of the kidney, consisting of a renal corpuscle (glomerulus surrounded by a Bowman capsule) and a renal tubule. Mature human kidney includes around 1 million nephrons. The inner glomerular layer is formed by a fenestrated endothelium, which is followed by a layer made of diverse extracellular proteins that create a meshwork known as the glomerular basement membrane (GBM). Visceral epithelial cells or podocytes, as well as mesangial cells, make up the outer layer. The complicated structure serves as the foundation for continuous plasma volume filtration at the glomerular level. The word "glomerulonephritis" refers to a group of kidney illnesses marked by immune-mediated destruction to the basement membrane, mesangium, or capillary endothelium, resulting in hematuria, proteinuria, and azotemia [1].

Glomerulonephritis accounts for around 10% of AKI in adults [2, 3]. AKI episodes in glomerular disease are typically caused by rapidly progressive glomerulonephritis (RPGN), a condition in which renal function gradually diminishes over days or weeks. Small-vessel vasculitis and anti-glomerular basement membrane (GBM) disease are the most prevalent causes, although other glomerular disorders, such as IgA nephropathy (IgAN), thrombotic microangiopathy (TMA), lupus nephritis, and post-streptococcal glomerulonephritis, can induce acute renal impairment. Acute renal failure in glomerulonephritis can also be caused by non-glomerular diseases such as acute tubular necrosis (ATN) caused by renal hypoperfusion or the nephrotic syndrome, as well as drug- or radiocontrast agent-induced tubular epithelial cell damage [2, 4].

acute glomerulonephritis is caused by immunologically mediated damage caused by numerous

infectious agents such as viruses, bacteria, or protozoa, as well as non-infectious causes such as Henoch–Schonlein purpura (HSP). The most prevalent infectious cause is post-streptococcal glomerulonephritis (PSGN), which is a non-suppurative sequel to Group A - hemolytic streptococci. Tropical places are more likely to have pyoderma-related PSGN, whereas temperate climates are more likely to have pharyngitis linked with PSGN [5-8].

Impaired tubular sodium reabsorption due to decreased perfusion induces afferent arteriole constriction and a further decrease in glomerular filtration rate (GFR). This compensatory mechanism (tubuloglomerular feedback), which is intended to protect the downstream nephron, may cause injury if it is sustained or if normal regulation of arterial tone is disrupted (for example, by nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin converting enzyme inhibitors (ACEIs), or angiotensin receptor blockers (ARBs)). Reduced blood flow in the peritubular capillaries causes ischemic damage in vascular endothelial cells, resulting in cell swelling and the production of cell adhesion molecules, which further reduces flow and activates leucocytes. Adherent leucocytes obstruct blood flow even further by producing cytokines and reactive oxygen species that harm endothelium and tubular epithelial cells [9].

Evaluation

Patients may have a moderate prodromal phase before developing severe clinical characteristics such as macroscopic hematuria and/or AKI. The Goodpasture syndrome is marked by hemoptysis and dyspnea, which are common symptoms of lung involvement. Kidney and respiratory systems deteriorate at a faster rate than in any other kind of RPGN, and fatality is frequently caused by renal failure necessitating RRT or extensive alveolar haemorrhage. Clinical suspicion, based on the simultaneous involvement of the kidneys and lungs, is a crucial step in improving patient outcomes. Anti-GBM illness is distinguished from vasculitis and lupus nephritis by the fast deterioration in renal function, the development of a very active urine sediment, and the lack of systemic involvement [2, 10].

The emergency physician must conduct a thorough physical examination and obtain a complete medical history, including herbal agents, sports supplements, non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and calcineurin inhibitors, among other medications. In addition, the patient's blood pressure, weight, hydration status, edoema, skin manifestations, pulmonary and cardiac examinations must all be correctly monitored [11].

The following examinations assist to not only in determinate likely cause but also in determining the amount of the damage: [1]

- Complete blood count - A low hematocrit may indicate dilutional anaemia. Pleocytosis may be visible in the context of an infectious cause.
- Potassium levels in serum may be elevated in patients with severe renal impairment.
- Renal function tests reveal that BUN and creatinine levels are elevated, indicating a degree of renal impairment. GFR (glomerular filtration rate) might be low.
- Complement: Differentiation may help the operator to narrow the differentials (C3, C4 levels). Cryoglobulinemia, systemic lupus erythematosus, infective (bacterial) endocarditis, and shunt nephritis are all disorders with low complement levels. Certain renal disorders, such as membranoproliferative GN or poststreptococcal GN, may also be taken into account. An underlying abscess, polyarteritis nodosa, Henoch-Schönlein purpura, Goodpasture syndrome, idiopathic fast progressive GN, immunological complex illness, and immunoglobulin G or immunoglobulin A nephropathy are all possible causes of normal complement levels. Anti-factor B autoantibodies may assist identify new-onset poststreptococcal GN from hypocomplementemic C3 glomerulonephritis in individuals with new-onset nephritis and low C3 levels, according to Chauvet *et al.*, [1,12].
- Looking for microscopic hematuria and proteinuria with a urine analysis, urine protein, and creatinine ratio. The dysmorphic red blood cell features of glomerular hematuria can be detected via a microscopic inspection. Proteinuria might be in the sub-nephrotic or nephrotic spectrum [13].
- Due to renal function loss and decreased tubular secretion, cellular breakdown, dehydration, and possibly fluid overload, electrolyte imbalances are typical in AKI. Electrolyte abnormalities such as hyperkalemia, hyper or hyponatremia, and other electrolyte abnormalities necessitated immediate treatment, hence an ECG must be conducted first in all suspected AKI cases to rule out cardiac arrhythmias. Other laboratory tests are influenced by the clinical condition [11].
- In 60-80% of instances, the antistreptolysin O titer (ASOT) rises. In one to three weeks, the climb begins, peaks in three to five weeks, and then returns to baseline in six months. It has little to do with the degree, duration, or prognosis of renal failure [1].

Through a broad agreement of specialists, the Acute Dialysis Quality Initiative group created the Risk, Injury, Failure, Loss, and End-stage kidney disease (RIFLE) approach for diagnosis and categorization of a wide variety of acute impairment of kidney function. The RIFLE criteria were endorsed by the Acute Kidney Injury Network with a tweak to accommodate minor variations in serum creatinine (SCr) (more than 0.3 mg/dl or 26.5 mol/l) during a 48-hour period. These amended criteria were validated in two recent studies that looked at big datasets in the United States and Europe. Thakar and colleagues discovered that the severity of AKI was linked to a higher risk of mortality, regardless of comorbidities. Patients with stage 1 AKI (an increase in SCr of more than 0.3 mg/dl or 26.5 mol/l but less than a twofold rise) had an odds ratio of 2.2; patients with stage 2 AKI (equivalent to RIFLE-I) had an odds ratio of 6.1; and patients with stage 3 AKI (RIFLE-F) had an odds ratio of 8.6 [14-19].

Management:

Because there is no particular medication for renal illness, the treatment for acute poststreptococcal glomerulonephritis (PSGN) is mostly supportive. The underlying infections must be addressed when acute glomerulonephritis (GN) is accompanied with chronic infections. The critical care unit's expertise may be required for the treatment of individuals with hypertensive encephalopathy or pulmonary edoema. A nephrologist's consultation may be necessary. Renal function, blood pressure, edoema, serum albumin, and urine protein excretion rate should all be evaluated on an outpatient basis [20].

Wong *et al.*, evaluated the features and management of acute PSGN in 27 paediatric patients in a retrospective analysis from New Zealand, finding that the requirement for acute dialysis was more prevalent among the 11 children with crescentic GN in the study. These researchers also discovered that urinary sediment abnormalities persisted in crescentic GN patients even after a 3.2-year follow-up period, and that the advantages of immunosuppressive medication in these patients were uncertain [20, 21].

The treatment of glomerulonephritis is divided into two categories: [1]

– Immunosuppression is one method of treatment. The following are some of the possibilities
  - Corticosteroids at high doses
  - Rituximab is a kind of antibody that is used to treat cancer (a monoclonal antibody that causes the lysis of B-lymphocytes)
  - Agents that are cytotoxic (e.g., cyclophosphamide, along with glucocorticoids are of value in severe cases of post-streptococcal glomerulonephritis)
  - Plasma exchange (glomerular proliferative nephritis, pauci-immune glomerulonephritis) which is considered a temporary treatment.

As the condition progresses into chronicity, overall care follows the same guidelines as chronic kidney disease: [1]

- Renal function tests (RFTs), serum albumin, and urine protein excretion rate are all monitored.
- Controlling blood pressure and blocking the renin-angiotensin axis using Loop diuretics, which have two functions: removing excess fluid and correcting hypertension.
- Vasodilators (e.g., nitroprusside, nifedipine) can be administered in patients with severe/refractory hypertension and/or encephalopathy.
- Anemia, bone mineral abnormalities, acidosis, cardiovascular disease, and restless legs/cramps are some of the problems linked with increasing chronic illness that can be managed.
- If necessary, preparation for renal replacement therapy (RRT).

Antibiotics (e.g., penicillin) are used to treat local symptoms and prevent infection from spreading to nearby people. Except if provided during the first 36 hours, antimicrobial medication does not appear to prevent the development of GN. Close contacts of the index patient may benefit from antibiotic therapy to help prevent the spread of PSGN [20].

For treating hypovolemia, hydroxyethylstarch (HES) is a frequently used and relatively affordable alternative to human albumin. There is little indication that resuscitation using colloids rather than crystalloids improves the risk of mortality in patients with trauma, burns, or after surgery, according to a recent Cochrane analysis. In addition to certain coagulation issues, especially with earlier versions of HES, the development of renal impairment has been linked to the use of mostly hypertonic HES. 11 randomised studies with a total of 1,220 patients were described in a meta-analysis. Hyperoncotic albumin reduced the risk of AKI by 76%, but hyperoncotic starch raised the risk by 92%. On the other hand, there were parallel impacts on mortality [14, 22, 23].

In edematous and hypertensive individuals, loop diuretics may be necessary to eliminate excess fluid and control hypertension. If severe hypertension or encephalopathy is present, vasodilator medications (e.g., nitroprusside, nifedipine, hydralazine, diazoxide) may be utilised. Except in extreme cases of PSGN, glucocorticoids and cytotoxic medicines are useless [20].

Loop diuretics (particularly furosemide) have long been recommended in the acute-care situation, based on numerous mechanistic studies and preclinical evidence, and a number of RCTs have evaluated whether furosemide is effective for the prevention or treatment of AKI. Prophylactic furosemide, in particular, was found to be useless or hazardous in preventing AKI following cardiac surgery, and to increase the risk of AKI when administered to avoid contrast-induced AKI. Epidemiologic evidence suggests that loop diuretics may increase mortality in individuals with critical illness and AKI, although contradictory evidence suggests that they have little effect on AKI. Finally, when utilised to treat AKI, furosemide treatment was unsuccessful and perhaps dangerous [14, 24-30].

Glomerulonephritis Nephrotic: ACE inhibitors/ARBs (3-6 months) are used to treat IgA nephropathy because they diminish proteinuria. Even after first medication, if proteinuria exceeds 1 gm (and GFR is more than 50), corticosteroids and fish oil might be administered. The same principles apply to Henoch Schonlein purpura (HSP). Steroids can also help with symptoms of the gastrointestinal tract (GIT) [1].

Renal blood flow is steady throughout a wide range of mean artery pressure (MAP) in healthy people due to renal autoregulation. Derangements in microcirculation and vasoreactivity, on the other hand, tend to raise the MAP threshold that ensures autoregulation in critically sick patients, particularly in septic shock. Current recommendations for preventing AKI in the ICU include achieving a MAP of over 65 mmHg, but emphasise that this goal pressure should be tailored to each patient's needs wherever feasible, especially in those with pre-existing chronic hypertension. In vasodilatory shock, there is excellent evidence that noradrenaline can enhance renal blood flow, restore urine production, and improve creatinine clearance. In critically sick patients, however, "renal-dose" dopamine has no impact in either preventing or ameliorating AKI [31].

CONCLUSION

There is no doubt that acute glomerulonephritis is serious case that require intensive care. The disease is caused mainly by immunologically mediated damage caused by numerous infectious agents such as viruses, bacteria, or protozoa, as well as non-infectious causes such as Henoch–Schonlein purpura (HSP). The most prevalent infectious cause is post-streptococcal glomerulonephritis (PSGN). The emergency physician must conduct a thorough physical examination and obtain a complete medical history. the treatment for acute poststreptococcal glomerulonephritis (PSGN) is mostly supportive. The underlying infections must be addressed when acute glomerulonephritis (GN) is accompanied with chronic infections. The critical care unit's expertise may be required for the treatment of individuals with hypertensive encephalopathy or pulmonary edoema.

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