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Assessing Clinical Outcomes and Treatment Efficacy in A Patient with Crescentic Glomerulonephritis (CGN): A Case Report

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Abstract

Crescentic glomerulonephritis (CGN) is a rapidly progressive renal condition marked by crescent formation in the glomeruli, often resulting in end-stage renal disease (ESRD) if not promptly addressed. This case report examines a 54-year-old female with a sudden onset of generalized edema and reduced urine output, which led to her hospitalization. Laboratory evaluations revealed acute renal failure with markedly elevated serum creatinine levels. Despite a background of Antineutrophil Cytoplasmic Antibodies (ANCA)-associated vasculitis, her management was complicated by inconsistent adherence to immunosuppressive therapy. Notably, severe hypertension exacerbated her renal impairment, necessitating immediate intervention. The clinical progression of CGN highlights the critical importance of early diagnosis, continuous monitoring, and a multidisciplinary approach for effective management. Emphasizing patient education and lifestyle modifications, particularly in dietary habits and blood pressure control, can significantly influence treatment outcomes. This case illustrates the urgency of addressing CGN and the necessity of a collaborative healthcare strategy to improve prognosis and enhance the patient's quality of life.

Keywords: Crescentic glomerulonephritis (CGN), Antineutrophil Cytoplasmic Antibodies (ANCA), ANCA-associated vasculitis (AAV).

Introduction

Crescentic glomerulonephritis (CGN) is a rare but rapidly progressive form of glomerulonephritis characterized by the formation of crescents in the Bowman's space of the glomeruli. These crescent formations result from the proliferation of parietal epithelial cells and the infiltration of monocytes and macrophages, which are associated with severe glomerular injury. Left untreated, the CGN will rapidly and often irreversibly lead to deterioration into chronic kidney disease (CKD) and eventually ESRD[1,2].

The term "crescentic" refers to the crescent-shaped structure seen on biopsy, which is the hallmark of the disease. CGN can be caused by various etiologies, including immune-mediated disorders, infections, and systemic diseases, although it may also occur as a primary renal condition without systemic involvement[3]. Clinically, CGN manifests with symptoms of acute nephritic syndrome, such as hematuria, proteinuria, oliguria, and hypertension. In some cases, CGN may present as a medical emergency due to rapidly progressing renal failure[4].

Among the various underlying causes of CGN, ANCA-associated vasculitis (AAV) plays a significant role. AAV is characterized by anti-neutrophil cytoplasmic antibodies that induce inflammation of small to medium-sized blood vessels, leading to renal injury and subsequent crescent formation[5,6]. While CGN can arise from multiple triggers, AAV is a notable contributor, resulting in a significant risk of rapid progression to renal failure. The interplay between ANCA-mediated inflammation and glomerular injury is one of the most prominent and requires prompt diagnosis and intervention to reduce the incidence of CKD and ESRD[7].

The renal biopsy was confirmed for CGN with crescents in more than 50% of the glomeruli, with variable degrees of glomerular necrosis and fibrosis[8]. Thus, Intensive treatment should be initiated much earlier, pre-ESRD, as the disease may progress rapidly in weeks to months if untreated. In CGN, immunosuppressive treatment with corticosteroids and cytotoxic agents is the treatment of choice, while renal replacement therapy would be required in advanced cases of renal failure[9].

Case Presentation

Patient Information:

A 54-year-old female patient presented to the nephrology department with complaints of anasarca (generalized edema) and oliguria (reduced urine output). She has no significant past medical history of renal disease but reported a hysterectomy in her surgical history. The patient has a history of hypertension, currently managed with Amlodipine (5 mg) and Atenolol (50 mg).

Chief Complaints:

- Generalized swelling (anasarca)
- Reduced urine output (oliguria)

History of Present Illness:

The patient started noticing, over the past couple of weeks, a gradual worsening of her symptoms; progressive edema and decreased urine output had been noted, along with fatigue and a loss of appetite. The patient denies any significant family history of kidney disease or recent infections or environmental exposures. Exacerbation of symptoms of renal disease; admitted for further evaluation and management.

Physical Examination: On physical examination, the patient exhibited the following vital signs and general findings:

- Pulse rate: 80 bpm
- Blood pressure: 170/100 mmHg
- Blood glucose random (GRBS): 140 mg/dL
- Oxygen saturation (SPO2): 98%
- Temperature: 98°F

Clinical Findings:

Complete Blood Picture:

Category	26/08/2024	27/08/2024	28/08/2024
WBC	7,200	7,600	7,100
RBC	8.9	4.2	4.4
Hemoglobin	14 g/dL	10.9 g/dL	10.1 g/dL
Platelets	159,000	164,000	174,000
Neutrophils	50-70%	87%	80%

Lymphocytes	25-40%	10%	13%
Eosinophils	1-4%	1%	2%
Monocytes	9%	2%	5%

Renal Function Tests (RFT):

Category	27/08/2024	28/08/2024	29/08/2024
BUN	13.0 mg/dL	18 mg/dL	171 mg/dL
Serum Creatinine	1.3 mg/dL	1.3 mg/dL	18 mg/dL
Serum Uric Acid	6.1 mg/dL	6.6 mg/dL	6.6 mg/dL

Liver Function Tests (LFT):

Category	26/08/2024	27/08/2024	28/08/2024
Total Bilirubin	0.4 mg/dL	0.3 mg/dL	0.3 mg/dL
Direct Bilirubin	0.1 mg/dL	0.1 mg/dL	0.1 mg/dL
SGOT (AST)	10 IU/L	10 IU/L	12 IU/L
SGPT (ALT)	18 IU/L	12 IU/L	12 IU/L
Alkaline Phosphatase	320 IU/L	284 IU/L	284 IU/L

Electrolyte Tests:

Category	28/08/2024	26/08/2024	27/08/2024
Sodium (Na+)	133 mmol/L	134 mmol/L	132 mmol/L
Potassium (K+)	4.2 mmol/L	4.3 mmol/L	4.2 mmol/L
Chlorides (Cl-)	102 mmol/L	103 mmol/L	102 mmol/L

Ultrasound Scan of Abdomen:

Finding	Impression
Liver	Normal in size, shape, and echogenicity, with no focal mass lesions.
Gall Bladder	Multiple calculi measuring 7-8 mm.
Kidneys	Bilateral mild pleural effusion, increased echogenicity.
Peritoneal cavity	Minimal ascites present.
Impression for Ultrasound:	Mild pleural effusion with gallstones; requires monitoring.

Echocardiogram:

Finding	Impression
Ejection Fraction	50-55%
Concentric left ventricular hypertrophy	Normal right ventricle, trivial tricuspid regurgitation.
Impression for Echocardiogram:	Mildly reduced ejection fraction with concentric hypertrophy; further cardiac evaluation is recommended.

Doppler Study of Both Upper Limbs:

Finding	Impression
Phlebitis	Right basilic vein and bilateral cephalic veins.
Atherosclerotic changes	Intimal media thickening in bilateral upper limb arteries.
Impression for Doppler Study:	Presence of phlebitis in upper limbs; monitor for complications.

Discussion

Crescentic glomerulonephritis is one of the faster-progressive forms of renal disease, with significant damage to the glomeruli resulting in crescents. This patient, a 54-year-old female, presented with the classical presentation of this condition: anasarca and oliguria. Classic signs of severe renal impairment. This is proved by laboratory findings of an acute increase in serum creatinine from 1.3 mg/dL to 18 mg/dL and elevated BUN levels, at up to 171 mg/dL, that confirm the rapid loss of renal function leading to ESRD. This acute deterioration puts a stamp of urgency on the need for early diagnosis and intervention in CGN.

This is highly relevant as ANCA antibodies may activate intense renal inflammation, and glomerular damage may have been in association with her history of ANCA vasculitis. Variable treatment of her disease complicated this patient's clinical picture because it likely worsened her renal function. Crescent formation in CGN indicates that severe inflammatory processes result in irreversible damage if left untreated. Regarding this, the patient's lack of adherence to immunosuppressive therapy, so crucial in inflammation control by ANCA, worsened her condition.

Hypertension is one of the most common findings in patients with CGN. This patient had a blood pressure of 170/100 mmHg; hence, controlling the blood pressure is essential to avoid further glomerular injury. The well-known potential effect of uncontrolled hypertension on renal function makes it very important in management.

Gradual impairment of renal function, manifested by abrupt elevations of creatinine and BUN, points to emergent treatment and, more than likely, renal replacement therapy. The acute impairment of renal function further suggests that her illness required emergent care or else dire consequences would have fallen upon her. Research illustrates that early diagnosis and intensification of treatment of CGN may likely result in a better outcome, just as did in this case due to the abrupt progression in the clinical course in this patient.

Moreover, multidisciplinary management should be consulted. Patients with CGN require the interdisciplinary coordination of doctors specialized in nephrology, dietitians, and primary care providers to optimize their outcomes. Arrange for patient follow-up visits to assess renal function and all the therapies as part of care. Lifestyle modification alone includes restriction of dietary sodium and hypertension, which is effective in reducing complications related to CGN.

Conclusion

This case points out the very urgent need for an early diagnosis and then prompt intervention in cases of crescentic glomerulonephritis to avert the progression to end-stage renal disease. Her continually rising serum creatinine and blood urea nitrogen levels indicate the patient's steady decline in kidney function. Irregular drug adherence was the definite key to worsening the situation, and it underlines the fact that the management of immunosuppressive therapy needs strict adherence. This requires a multidisciplinary approach that typically includes specialists and other health care providers in providing comprehensive care, from careful follow-up of renal function to educating the patient about the necessity of constant treatment. Lifestyle changes, especially diet therapy with adequate blood pressure management, complement patient care. Aggressive and cooperative approaches have been able to prevent any further damage caused to the kidneys, and the quality of life for those suffering can dramatically be improved by crescentic glomerulonephritis.

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