Case Report

An Interesting Acute Renal Failure Case which Developed in a Patient with Chronic Kidney Disease

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Abstract: Chronic Kidney Disease (CKD) is defined as a chronic and progressive deterioration in kidney functions such as, balancing fluid-solute equilibrium and metabolic-endocrine effects as a result of a decrease in glomerular filtration rate. In present case, we aimed to report an acute renal failure case in an elderly man with CKD. A sixty two year old man presented with dyspnea, leg swelling and loss of appetite for two weeks. He is diagnosed with acute exacerbation of CKD caused by infected diabetic foot ulcer. He responded well to antibiotic and diuretic treatment. Management of acute exacerbation of kidney failure in patients with CKD requires a systematic approach and elimination of underlying causes. Careful treatment usually results in restoration of kidney function to the pre-exacerbation stage.

Keywords: Chronic kidney disease, Diabetic foot ulcer, Diuretic, Antibiotic, Kidney function, Glomerular filtration rate.

INTRODUCTION

Chronic Kidney Disease (CKD) is defined as a chronic and progressive deterioration in kidney functions such as, balancing fluid-solute equilibrium and metabolic-endocrine effects as a result of a decrease in glomerular filtration rate [1]. CKD can be aggravated by infection, interventional procedures with radiocontrast agents and certain medications including non-steroidal anti-inflammatory drug analgesics [2]. Patients may present with a variety of symptoms including edema, decreased urine, and dyspnea. In present case, we aimed to report an acute renal failure case in an elderly man with CKD.

CASE REPORT

A sixty two year old man presented with dyspnea, leg swelling and loss of appetite for two weeks. He had swelling on his both legs for two years but his complaints worsened in last 15 days. He did not report cough, fever or chest pain. He figured that the amount of urine during urination was also decreased. His medical history was positive for type 2 diabetes mellitus for 10 years, hypertension for 8 years, and CKD for 4 years. His daily medications include amlodipine 10 mg once a day, doxazocin 4mg once a day, furosemide 40mg every other day, insulin aspart 12 IU before meals and insulin detemir 25IU once a day.

On physical examination, he was ill appeared. Bilateral 3+ pretibial pitting edema in lower extremities, diabetic foot ulcers in plantar face of both foots and basilar crackles in both lungs were noted. Funduscopic examination revealed findings associated with diabetic and hypertensive retinopathy. Other findings in examination were unremarkable.

Laboratory studies revealed elevated blood urea (90mg/dL) and creatinine (3,15mg/dL) levels. Glomerular Filtration Rate (GFR) was calculated as 20%. Other laboratory tests were as follows: C-Reactive Protein (CRP): 2.5mg/L, serum Na: 140meq/L, K: 4,7meq/L, Ca:8.3mg/dL, leukocyte count: 5500/mm^3, Hemoglobin: 10,7g/dL, platelet count: 290000/mm^3, and glycated Hb (HbA1c): 10,7%. Arterial blood gas analysis showed a pH: 7,37, a PCO2: 36, and HCO3: 21,2. There was 3+ protein in spot urine dipstick test. A PA thorax radiography revealed bilateral basilar pleural effusion.

1g of ampicillin sulbactam three times a day initiated for infected foot ulcers. Furosemide infusion at a 10mg/hour rate was prescribed. As edema resolves, furosemide infusion rate reduced to 5mg/hour and finally to 40mg twice a day tablets P.O. Granulation tissue developed over infected ulcers during treatment. His CRP level reduced to 1.1mg/L. Control thorax radiography revealed diminished pleural effusion. His body weight reduced from 87 to 75 kg. Serum creatinine levels reduced to the basal value, 1,2 mg/dL (Fig. **1**). Since clinical and laboratory improvement achieved, the patient discharged from hospital on 8th day of his hospitalization.

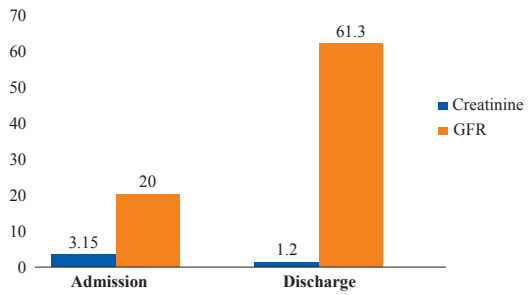

Fig. (1). Change in the Serum Creatinine and GFR Levels on Admission and Discharge of the Patient.

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DISCUSSION

An acute exacerbation of kidney failure in a patient with CKD was presented in this case report.

Renal functions in patients with CKD are vulnerable to any threat. Nephrotoxic agents, such as non-steroidal anti-inflammatory drugs, radiocontrast agents, infections (especially urinary tract infections), hypovolemia, and obstructive uropathy may cause sudden and further deterioration in kidney functions in subjects with CKD [3]. We carefully investigated the possible cause of acute deterioration in kidney functions in present patient. Present case had infected diabetic foot ulcers which may cause a reduction in renal functions. Moreover, he lost his appetite which cause reduced fluid intake and thus call forth prerenal azotemia. However, this cause was less likely since the patient had pleural effusion, a sign of hypervolemia.

Treatment of acute exacerbation of CKD depends on the treatment of underlying cause [4]. Antibiotics initiated for infected diabetic foot ulcers in present case. Adequate diuretic treatment should also be ordered in sake of reducing pleural effusion and peripheral edema in hypervolemic subjects [5, 6]. Furosemide infusion followed by oral furosemide tablets was initiated to overcome hypervolemic state in present case. Pretibial edema diminished and pleural effusion reduced with diuretic treatment during clinical follow up.

Measuring body weight is critical during treatment with diuretics in hypervolemic subjects [7]. We figured that, as edema diminished, patient's weight reduced by 12 kg, which suggests the treatment was effective.

Another issue during diuretic therapy is monitoring serum electrolytes. This is because furosemide may cause reduction in blood sodium and potassium levels [8, 9]. We regularly monitored serum electrolyte levels and did not notice any reduction below the reference range during diuretic treatment.

CONCLUSION

In conclusion, management of acute exacerbation of kidney failure in patients with CKD requires a systematic approach and elimination of underlying causes. Careful treatment usually results in restoration of kidney function to the pre-exacerbation stage.

AUTHORS' CONTRIBUTION

Elif Basaran, Burcin Meryem Atak Tel, Ozge Kurtkulagi and **Gulali Aktas**: Followed the patient.
Elif Boduc Bulunmaz, Muhammed Emin Demirkol and **Satilmis Bilgin**: Performed literature analysis.
Elif Basaran, Gulali Aktas, Tuba Taslamacioglu Duman and **Gizem Kahveci:** Wrote the first draft.
All authors approved the final version.

CONFLICT OF INTEREST

Declared none.

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