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Case Report *Medical research*

A case report on management of chronic renal failure

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ABSTRACT

Chronic kidney disease may result from a primary intrinsic kidney disease, from anatomic or obstructive abnormalities, as a secondary complication of another systemic disease, or from acute kidney failure that never fully resolves. The major diseases that lead to chronic kidney failure and new cases of ESKD are diabetes, hypertension, and glomerulonephritis. Notably, diabetes mellitus (DM) now accounts for nearly 45% of new ESKD cases.[3] Approximately 20% to 40% of patients with diabetes develop kidney disease.[4] Patients who have received kidney transplants account for an expanding fraction of the CKD population. Although kidney transplant may cure ESKD, the transplanted kidney may eventually fail for a number of reasons, including recurrent damage from the original systemic disorder, acute or chronic rejection, and drug-related nephrotoxicity associated with the use of certain immunosuppressants (e.g., cyclosporine, tacrolimus). Kidney transplant recipients are a very complex subset of patients; however, many of the issues managed in CKD remain pertinent to this population. [11]

We report one such case of chronic kidney failure in a 13 years old male who was diagnosed with CKD due to Aprot's syndrome 6 years back. Patient developed malignant hypertension and pulmonary edema and on maintenance hemodialysis who was treated by Dr. Appa Rao with the protocol involving immunonutritive therapy.

Keywords: Chronic Kidney disease, Hemodialysis, Immunonutritive therapy

INTRODUCTION

The term chronic kidney disease was proposed by the National Kidney Foundation–Kidney Disease Quality Outcome Initiative (K/DOQI) as a way of simplifying and codifying the language used to communicate about the disease. CKD is defined as either of the following conditions for a minimum of 3 months: glomerular filtration rate (GFR) less than 60 mL/min/1.73 m^2, or old damage to the kidney(s) with or without a decrease in GFR. This damage may be evidenced by abnormalities in the composition of blood or urine, or by changes seen in imaging studies. [1] The K/DOQI2 Working Group further categorizes the extent of kidney disease according to the presence of kidney damage and the GFR. Stage 1 CKD is defined as the presence of kidney damage even though GFR may be normal or even increased (≥90 mL/min/1.73 m^2). Stage 2 CKD is evidenced by a GFR between 60 and 89 mL/min/1.73 m^2. Patients with stage 3 CKD may or may not have kidney damage, while their

GFR is reduced to between 30 and 59 mL/min/1.73 m^2. CKD patients with stage 4 disease also may not have intrinsic kidney damage, but their GFR is severely reduced to between 15 and 29 mL/min/1.73 m^2. The last stage, stage 5, is also known as end-stage kidney disease (ESKD). This condition, formerly known as end-stage renal disease (ESRD), is defined as a GFR less than 15 mL/min/1.73 m^2 or the need for renal replacement therapy (RRT) for survival. Most forms of kidney disease will cause irreversible, progressive deterioration of kidney function if not identified and treated properly. Depending on the cause, the disease may progress to complete loss of function over months to years. As the extent of deterioration increases, the kidney is unable to perform normal homeostatic functions. This leads to fluid and electrolyte abnormalities, acid-base disturbances, hormonal dysregulation, and other systemic disturbances. When the GFR falls to below 15 mL/min/1.73 m^2, patients generally require some form of RRT for survival. Options for RRT include hemodialysis, peritoneal dialysis, and kidney transplantation. Although patients may be maintained on dialysis or may receive a kidney transplant, they have increased risks of morbidity and mortality. [2]

Chronic kidney disease may result from a primary intrinsic kidney disease, from anatomic or obstructive abnormalities, as a secondary complication of another systemic disease, or from acute kidney failure that never fully resolves. As was mentioned previously, the major diseases that lead to chronic kidney failure and new cases of ESKD are diabetes, hypertension, and glomerulonephritis. Notably, diabetes mellitus (DM) now accounts for nearly 45% of new ESKD cases. [3] Approximately 20% to 40% of patients with diabetes develop kidney disease. [4]

SIGNS AND SYMPTOMS

Most patients experience few symptoms of CKD until less than 25% of normal renal function remains. CKD can therefore progress insidiously over months to years, evident only through abnormal biochemical parameters, such as gradually rising levels of BUN and serum creatinine values. Nonspecific complaints such as malaise, fatigue, and nocturia may be noted. Urine output may or may not be diminished. Hypertension may develop and, if discovered, presents a critical opportunity for investigation of renal implications. Unless patients are recognized to be at risk of and are monitored for kidney disease, they usually do not seek medical attention until the onset of uremic symptoms. At this point, interventions to forestall progression to ESKD are largely unfruitful. [5]

In stage 1 and stage 2 CKD, reduced GFR alone does not clinch the diagnosis, because the GFR may in fact be normal or borderline normal. In such cases, the presence of one or more of the following markers of kidney damage can establish the diagnosis [6]:

Albuminuria (albumin excretion >30 mg/24 hr or albumin:creatinine ratio >30 mg/g [>3 mg/mmol]), Urine sediment abnormalities, Electrolyte and other abnormalities due to tubular disorders, Histologic abnormalities, Structural abnormalities detected by imaging, History of kidney transplantation in such cases Hypertension is a frequent sign of CKD but should not by itself be considered a marker of it, because elevated blood pressure is also common among people without CKD.

In an update of its CKD classification system, the NKF advised that GFR and albuminuria levels be used together, rather than separately, to improve prognostic accuracy in the assessment of CKD. [7,8] More specifically, the guidelines recommended the inclusion of estimated GFR and albuminuria levels when evaluating risks for overall mortality, cardiovascular disease, end-stage kidney failure, acute kidney injury, and the progression of CKD. Referral to a kidney specialist was recommended for patients with a very low GFR (<15 mL/min/1.73 m^2) or very high albuminuria (>300 mg/24 h). [6, 7]

Patients with stages 1-3 CKD are frequently asymptomatic. Clinical manifestations resulting from low kidney function typically appear in stages 4-5.

Patients with CKD stages 1-3 are generally asymptomatic. Typically, it is not until stages 4-5 (GFR <30 mL/min/1.73 m^2) that endocrine/metabolic derangements or disturbances in water or electrolyte balance become clinically manifest.

Signs of metabolic acidosis in stage 5 CKD include the following: Protein-energy malnutrition, Loss of lean body mass, Muscle weakness

Signs of alterations in the way the kidneys are handling salt and water in stage 5 include the following: Peripheral edema, Pulmonary edema, Hypertension

Anemia in CKD is associated with the following: Fatigue, Reduced exercise capacity, Impaired cognitive and immune function, Reduced quality of life, Development of cardiovascular disease, New onset of heart failure or the development of more severe heart failure, Increased cardiovascular mortality

Other manifestations of uremia in end-stage renal disease (ESRD), many of which are more likely in patients who are being inadequately dialyzed, include the following: Pericarditis: Can be complicated by cardiac tamponade, possibly resulting in death if unrecognized, Encephalopathy: Can progress to coma and death, Peripheral neuropathy, usually asymptomatic, Restless leg syndrome, Gastrointestinal symptoms: Anorexia, nausea, vomiting, diarrhea, Skin manifestations: Dry skin, pruritus, ecchymosis, Fatigue, increased somnolence, failure to thrive, Malnutrition, Erectile dysfunction, decreased libido, amenorrhea, Platelet dysfunction with tendency to bleed

Screen adult patients with CKD for depressive symptoms; self-report scales at initiation of dialysis therapy reveal that 45% of these patients have such symptoms, albeit with a somatic emphasis. [8]

Treat these pathologic manifestations of chronic kidney disease (CKD) as follows

Anemia

When the hemoglobin level is below 10 g/dL, treat with an erythropoiesis-stimulating agent (ESA) such as epoetin alfa or darbepoetin alfa (previously, peginesatide was also considered an option for anemia in CKD, but this agent was withdrawn from the market in February 2013 due to serious hypersensitivity reactions [9]; caution should be exercised in patients with malignancy

Hyperphosphatemia: Treat with dietary phosphate binders and dietary phosphate restriction

Hypocalcemia: Treat with calcium supplements with or without calcitriol

Hyperparathyroidism: Treat with calcitriol, vitamin D analogues, or calcimimetics

Volume overload: Treat with loop diuretics or ultrafiltration

Metabolic acidosis: Treat with oral alkali supplementation

Uremic manifestations: Treat with long-term renal replacement therapy (hemodialysis, peritoneal dialysis, or renal transplantation)

Cardiovascular complications: Treat as appropriate

Growth failure in children: Treat with growth hormone [10]

CASE PRESENTATION

A 13 years old male was diagnosed with CKD due to Alport's syndrome 6 years back. The patient was not followed up since 6years and past medical reports are not available. He was on supportive medication for the past 6 years.

The patient was presented to a physician at Vijayawada 2 months ago with malignant hypertension and pulmonary edema. He was initiated on Hemodialysis and managed with medical measures. He developed Malignant hypertension with seizures, then Peritoneal Dialysis catheter was placement on 25 March 2014 which was a failure within a month due to catheter tip malfunction. Hemodialysis catheter line was infected then femoral line was placed. Femoral vein thrombosis was treated with heparin. Patient developed Gastrointestinal bleeding following heparin, which was managed conservatively with PRBC Transfusion. Right IJV HD catheter was placement on 17 April 2014 and the patient has received PRBC transfusions for 5 times over the past 2 months. Then the patient visited Apollo Hospital for the first time on 25 April 2014. He presented with complaints of body swellings since 7 days, headaches, abdominal pain since 5days and vomiting since 3 days. He also had intermittent fever since 7 days. He complained of photophobia and decreased vision since 2 months. There is no family history of kidney disease. He has no known drug allergies.

On examination the patient was found to be malnourished weighing 24.5 kgs with height 124cm, where his BMI was found to be 15.9kg/m^2. His blood pressure was found to be 140-150/80-100mmHg, Anasarca ++, Mild pallor, No icterus, Chest – scattered crackles, Heart sound regular, no gallop, Abdomen was softly distended with tender hepatomegaly of 5cm.

PD catheter exit site and tunnel were healthy. CNS on examination was nonfocal. Spine and joints were normal. No rickets observed.

He underwent permanent PD catheter placement under GA on 22 May 2014. He was transfused with 1 unit of leukoreduced PRCB pre-procedure as his Hgb was 6.2 gm/dl. Post procedure he was stable and PD catheter was flushed the next day.

Laboratory Reports

Test	Results	Normal Range
Urea-Serum/Plasma	120	15-36mg/dl
Urea Nitrogen-Serum/plasma Calculated	56	7-18mg/dl
Creatinine	8.1	0.8-1.3mg/dl
Se. Sodium	140	135-145mmol/l
Se. Potassium	5.7	3.5-5.1mmol/l
Se. Chloride	107	98-107mmol/l
Carbon dioxide serum	10.9	21-32mmol/l
Urine Routine		
Proteins	4+	
Glucose	Nil	
RBC	2-3	
WBC	10-12	

USG Abdomen Showed bilateral renal parenchymal disease and Grade II prostatomegaly.
2D Echo Report suggested concentric LVH and Grade II diastolic dysfunction.

Then he visited Dr.Appa Rao's clinic and started his treatment. At his first visit he was very weak, unable to sleep, and had low appetite. But with Dr.AppaRao's immunonutritive therapy, his general condition was improved. At the same time he is continuing with his Heamodialysis 6 cycles / day at Apollo Hospital.

DISCUSSION

CKD carries a poor prognosis. If it is treated in the early stages, progression to ESKD may be delayed, and some complications of the disease may be avoided. For these goals to be achieved, it is imperative that patients at risk for CKD or those in the early stages of the disease be identified. This task is made even more difficult by the fact that many patients do not even realize that they have CKD. [12] Timely identification and treatments are paramount. Without treatment, progressive, irreversible damage is likely and will result in ESKD. Chronic renal disease is costly. [3] Various newer therapies are still under study. The protocol designed by Dr. Appa Rao is beneficial to many.

CONCLUSION

A 13 years old male who was diagnosed with CKD due to Aprot's syndrome 6 years back. Patient developed malignant hypertension and pulmonary edema and is on maintenance hemodialysis. The patient found no improvement in his condition with the previous treatment and then he was treated by Dr. Appa Rao with the protocol involving immunonutritive therapy.

Treatment schedule and follow-up

Injection Human normal immunoglobulin (12 mg) and histamine dihydrochloride (0.15 mcg), (Belongs to any manufacturer). Two vials once in three days (3 doses) followed by two vials once in a week until 8 weeks. Aceclofenac 100mg twice a day for one month. Prednisolone tapered and maintained 5 mg per day. Ranitidine 150 mg once a day in the morning.
Tomato, Banana fruit, Prawns and milk were restricted in nutrition.

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