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A Case Study on Hyperparathyroidism Patient with Complication of Chronic Kidney Disease in Clinical Based Tertiary Hospital

Gundeti Sreeja *, Kunamalla Sai kiran, Manne Anudeep
H.no: -113, Rompikunta, Kamanpur, Karimnagar dist., Telangana – 505188

***Corresponding Author:**

Gundeti Sreeja
H.no: -113, Rompikunta, Kamanpur, Karimnagar dist., Telangana – 505188

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ABSTRACT

Chronic kidney disease is characterized by a progressive deterioration in kidney function with time characterized by irreversible structural damage to existing nephrons. A variety of risk factors associated with development, initiation, and progression of CKD have been identified. Initiation factors are medical conditions that directly cause kidney damage. Risk factors for the progression of CKD exacerbate kidney damage and are related to an accelerated decline in kidney function with time. The majority of susceptibility factors is not modifiable, but may identify people who are at high risk for developing CKD. Mineral and bone disorder of CKD (CKD-MBD) is the term used to collectively describe the mineral (e.g., phosphorus, calcium, parathyroid hormone), bone (osteodystrophy), and soft-tissue calcification abnormalities that develop as a complication of CKD.

Keywords: Hyperparathyroidism, Calcification, Chronic kidney disease, Anemia

INTRODUCTION

Mineral and bone disorder of CKD (CKD-MBD) is the term used to collectively describe the mineral (e.g., phosphorus, calcium, parathyroid hormone), bone (osteodystrophy), and soft-tissue calcification abnormalities that develop as a complication of CKD. The older collective term of kidney osteodystrophy failed to adequately illustrate the broader clinical complications associated with the biomarker abnormalities and calcification, and is now only used to describe, specifically, the bone pathology. The 2003K/DOQImineral and bone disease guidelines have traditionally set the target goals for management of mineral and bone disease (i.e., phosphorous, calcium, and parathyroid hormone).144 In 2009, KDIGO published guidelines for the management of CKD-MBD.

Although KDIGO are the new guidelines, many clinicians have not totally adopted them into practice, and current clinical performance measurements that dictate reimbursement schemes are still determined by the K/DOQI CKD-MBD guidelines.145 Until reimbursement schemes are consistent with the KDIGO guidelines, most clinicians will continue to manage patients according to the K/DOQI guidelines.

Hyperphosphatemia, hypocalcemia, hyperparathyroidism, decreased production of active vitamin D, and resistance to vitamin D therapy are all frequent problems in CKD that can lead to the secondary complications of CKD-MBD. Although the interrelationships among phosphorus, calcium, vitamin D, and PTH have been reviewed extensively, fibroblast growth factor, a phosphaturic hormone discovered within the last decade, has added some new insight. Increased dietary phosphorus intake stimulates FGF23 secretion. FGF23 increases phosphorus excretion via the proximal tubules,

inhibits vitamin D activation, increases activated vitamin D catabolism, and is associated with kidney disease progression. The "trade-off" hypothesis best describes the events leading to changes in bone metabolism. As eGFR decreases, phosphorus excretion by the kidney decreases, resulting in hyperphosphatemia. Hyperphosphatemic conditions lead to a corresponding decrease in ionized calcium concentration, a primary stimulus for release of PTH from the parathyroid gland. Higher concentrations of PTH decrease kidney tubular reabsorption of phosphorus and promote its excretion. Both serum phosphorus and calcium concentrations are corrected depending on the degree of remaining kidney function, but this occurs at the expense of an elevated PTH concentration . As kidney disease becomes more severe (eGFR <30 mL/minute/1.73m2), the phosphaturic response to PTH diminishes, andsustained hyperphosphatemia, elevatedFGF23, andhypocalcemia develop. In response to hypocalcemia, calcium is mobilized from the bone, a mechanism largely controlled by PTH. Retention of phosphorus and secondary hyperparathyroidism (sHPT) play a major role in the development of osteitis fibrosa or high-turnover bone disease.

Virtually all patients with kidney failure develop sHPT.Decreased PTH degradation by the kidney may also contribute to the hyperparathyroid state in patients with kidney disease. The kidney is the principal organ responsible for vitamin D production, and, as such, vitamin D metabolism is altered in the presence of uremia. The discovery of FGF23 has required an update in this trade-off hypothesis, providing a mechanism for the declining vitamin D level that develops as CKD progresses. Persistent hyperphosphatemia stimulates the release of excessive FGF23, which inhibits the normal conversion of 25- hydroxyvitamin D3 to its biologically active metabolite, 1,25-dihydroxyvitaminD3, by the enzyme 1-*α*-hydroxylase (Fig. 31-2). This enzyme is present in proximal tubular cells of the kidney and is necessary for conversion of vitamin D to the active form. This active form of vitamin D, also known as *calcitriol,* increases gut absorption of calcium and interacts with vitaminD receptors on the parathyroid gland to suppress PTH release. As a result of decreased calcitriol production, the absorption of dietary calcium in the gut is diminished. Decreased suppression of PTH release by vitamin D in conjunction with hypocalcemia promotes continued stimulus for mobilization of calcium from bone. Furthermore, uremic patients require a higher extracellular calcium concentration to suppress secretion of PTH. This is also described as an increase in the calcium "set point" or the concentration of calcium required inhibiting 50% of maximal PTH secretion.

2. CASE REPORT

A male patient of 62 years brought to the emergency department with the noticeable chief complaints of Presence of oliguria since 1 week , Breathlessness , presence of edema in both legs , itching on the skin . past medical history includes Hypertension, benign prostatic hyperplasia, hyperpara thyroidism ,angina . Social habits alcoholic since 10 years. In vitals his blood pressure was elevated 180/100 mmHg. In laboratory investigations HB is decreased. All renal biomarkers like BUN Sr creatitine increased. Calcium and phosphorus are elevated. During the overall stay in hospital the patient has developed tarchycardia revealed by ECG. The treatment was initiated immediately in the under mentioned table

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Drug name	Prescribed dose	Duration
Tab.Metoprolol succinate	50mg	1 month
prazosin	5mg	7 days

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Tab.Calcitriol	0.25mcg	1 month
Nitroglycerin	0.6mg	SOS
Iv .Furosemide	20mg	Day 1 and 2
Tab.Folic acid & ferrous fumerate	1 tablet	1 month
Tab.Sodium bicarbonate	3g	1 week

3. DISSCUSSION:

Chronic kidney disease is characterized by a progressive deterioration in kidney function with time characterized by irreversible structural damage to existing nephrons. In the present case patient developed hyperparathyroidism due to CKD. Most of the patients with hyperparathyroidism will have a past history of CKD 5-10 cases. In the lab evidence patient has high phosphorus level because of nephron damage , so it leads to itcching of the skin . In general, serum phosphorus should be lowered toward near normal levels. Laboratory parameters and indices used in the clinical setting to evaluate kidney function and to monitor disease progression time include SCr, CrCl, and eGFR. Specifics regarding the equations and methods used to calculate CrCl and eGFR Estimated.

The Modification of Diet in Renal Disease (MDRD) equation is used to calculate eGFR to stage CKD, and the Cockcroft-Gault equation is used to determine drug dosing of medications cleared by the kidneys in patients with impaired kidney function. Appropriate management of CKD includes measures to slow progression of the disease and regular evaluation of kidney function to assess changes in disease severity and to monitor therapy. This includes aggressive strategies to manage the disorders that cause kidney disease or are known to accelerate the disease process, such as diabetes mellitus, hypertension, high protein intake, and dyslipidemias Complications specific to CKD begin to develop as kidney disease progresses, most often when patients reach stage 3 disease (eGFR <60 mL/minute/1.73 m2). These complications include fluid and electrolyte abnormalities, metabolic acidosis, anemia, mineral and bone disorder, cardiovascular complications, and poor nutritional status. Often, these complications go unrecognized or are inadequately managed during the earlier stages of CKD, leading to poor outcomes by the time a patient is in need of dialysis therapy. Hypoalbuminemia and anemia were identified

in more than 50% of a population of patients newto dialysis therapy, and these findings were associated with a decreased quality of life.33 Late referral to a nephrologist to manage CKD and its associated complications has also been associated with increased mortality in the ESRD population.34 These and similar reports underscore the need for early and aggressive therapy to manage complications of CKD.

4. CONCLUSION

It is concluded that prazosin is essential to manage benign prostate hyperplasia . Ferrous supplements are mandatory to maintain HB level in CKD patients Because the kidneys secrete 90% of the endogenous hormone erythropoietin, a hormone necessary for erythropoiesis, declining kidney function can lead to erythropoietin deficiency and anemia. Calcitrail is important for vitamin D3 . Furosimide helps to treat the pedal edema .

Asignificant reduction of serumphosphorus is difficult to achieve with dietary intervention alone, particularly in patients with more advanced kidney disease (eGFR<30 mL/minute/1.73m2). For these patients, phosphate-binding agents used in conjunction with dietary restriction are necessary.

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Phosphate-binding agents limit phosphorus absorption from the GI tract by binding with the phosphorus present from dietary sources Patient should improve the quality of life. To acieve increased effectivenss of the drugs, the treatment should initiate immediately, each day counts

5. KEY REFERENCES AND WEBSITES

A full list of references for this chapter can be found at **http://thepoint.lww.com/AT10e.** Beloware the key references and websites for this chapter, with the corresponding reference number in this chapter found in parentheses after the reference.

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