

A DESCRIPTIVE STUDY TO KNOWING HOW TO ANALYSE CALCIUM BALANCE IN PATIENTS SUFFERING FROM KIDNEY FAILURE PROBLEMS

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Abstract:

There are many causes of hypercalcemia, such as primary hyperparathyroidism, malignancy, and vitamin D toxicity. Immobility is one of the rare causes and is often ignored. Hypercalcemia on immobilization is common in patients with renal failure

Serum calcium can be lowered by resuming weight-bearing activities, active volume expansion, diuresis, inhibition of bone resorption and other measures. This article reviews the incidence of hypercalcemia, common populations, and mechanisms and proven treatments for hypercalcemia.

The main causes include primary glomerulonephritis, chronic pyelonephritis, hypertension, atherosclerosis, diabetic nephropathy, secondary glomerulonephritis, tubulointerstitial disease, hereditary kidney disease, long-term use of antipyretics and analgesics, exposure to heavy metals, etc. .

1. Efforts should be made to clarify the cause of chronic renal failure. It should be clarified whether the renal damage is primarily glomerular damage, renal tubulointerstitial lesions, or renal vascular lesions, so that targeted treatment can be based on clinical characteristics. .

2. Reversible factors that promote the progressive deterioration of renal function in chronic renal failure should be identified, such as infection, drug-induced kidney damage, metabolic acidosis, dehydration, heart failure, blood pressure falling too quickly or too low, etc.

3. Attention should be paid to looking for certain factors that aggravate the gradual deterioration and decline of kidney function in chronic renal failure, such as hypertension, hyperlipidaemia, hypercoagulability, high-protein food intake, massive proteinuria

Introduction

Introduction

In patients with chronic renal failure, due to abnormal metabolism of calcium and phosphorus, pathological changes such as hyperphosphatemia and hypocalcemia occur,[1,2] which stimulate the parathyroid glands to proliferate and even form adenomas, resulting in the production of large amounts of parathyroid hormone. Hypothyroidism, which in turn enhances the intestinal response to the disease, as calcium and phosphorus are absorbed, and at the same time, enhances osteoclast movement, calcium and phosphorus are released in large quantities, and mineral salts are lost in the bones, [3,4,5] which in turn leads to low bone density and osteoporosis. Fibrous cystic periostitis throughout the body, as well as metastatic calcification of the skin and cardiovascular system, leading to serious consequences for the patient's quality of life survival rate. In recent years, with the widespread development of dialysis treatment and progress in dialysis technology, the survival period of patients with chronic renal failure has been greatly prolonged, and the incidence of SHPT has continued to increase. Most patients can control the progression of the disease in the early stage by controlling phosphorus intake and drug therapy[6,7,8]. Currently, PTX is considered a safe and effective method for treating chronic renal failure associated with severe SHPT. PTX can quickly reduce the blood levels of iPTH and blood phosphorus in SHPT patients, maintain the balance of calcium and phosphorus in the body for a long time, and reduce the occurrence of complications such as cardiovascular disease and fractures, thus improving the patient's quality of life. [9]

The frequency of chronic kidney failure (CKD) increases with the aging of the population, it affects nearly 3 million CKD is a nephron reduction, most often due to chronic vascular nephropathy or diabetes. Complications are anemia, disorders of phosphocalcic metabolism and an increase in cardiovascular mortality.

Patients with CKD may have 1, 2 or 3 of the abnormalities listed. IR hypocalcemia is multifactorial; hypocalcemia/hyperphosphatemia leads to continuous stimulation of the parathyroids and an increase in PTH secretion; progressive appearance of secondary hyperparathyroidism. The elevation of PTH leads to an increase in bone resorption responsible for the abnormalities observed in CKD. The calcium receptor (CaSR) plays a central role in the regulation of PTH secretion, activated by extracellular Ca^{++} . It allows rapid adaptation of PTH secretion. [10]

Main hypothesis:

Patients with late stage 3/stage 4 CKD (estimated glomerular filtration rate [eGFR] 15-40 mL/min/m²) will excrete 20% of the absorbed calcium in a high-calcium diet (2000 mg). On a low-calcium diet (800 mg), patients with stage IV CKD excrete 50% of absorbed calcium. Gastrointestinal absorption will be 20% for both diets, resulting in a net positive calcium balance of 320 mg at a high calcium intake and 80 mg at a low calcium intake.

People with normal renal function (eGRF > 60 mL/min/m²) will excrete >90% of absorbed calcium with a high-calcium diet and >90% of absorbed calcium with a low-calcium diet. Gastrointestinal absorption will be 20% in both diets, resulting in no net positive calcium balance with either a higher calcium intake or a lower calcium intake.

Secondary hypothesis:

Patients with late stage 3/stage 4 CKD (eGFR 15-40 mL/min/m²) will excrete 60% of their absorbed phosphorus on a 1600 mg phosphate regimen. Normal subjects (eGFR > 60 mL/min/m²) are expected to excrete more than 90% of absorbed phosphorus with a 1600 mg phosphate regimen. Gastrointestinal phosphate absorption is expected to be 60% in both diets, resulting in a net positive phosphorus balance of 192 mg and 384 mg in late stage 3/stage 4 CKD, respectively. In addition, we hypothesize a decrease in fractional excretion of absorbed phosphorus with a decrease in glomerular filtration rate (eGFR).

Results

The study examines serum 25-OH vitamin D levels in patients with chronic kidney disease and vitamin deficiencies. Patients with levels below 30 ng/mL receive ergocalciferol 50,000 IU weekly for 4 weeks. If levels are replete, a second course is given for 4 weeks. Patients with levels below 30 ng/mL are excluded from further study. After satiation, patients will remain on 50,000 IU of ergocalciferol per month. Patients' calcium and phosphorus levels will be determined, and they will be excluded if they exceed 4.5 mg/dL or have normal calcium levels.

Patients will be placed on two diets in a randomized order for 9 days each. Randomization to regimens will be performed using interrupted blocks stratified by CKD stage. Patients will receive one of two diets for each 9-day period, then follow the second diet after at least one week of "washout period." This washout period can last up to 4 weeks to accommodate the patient's schedule or to study menstruating women in the same phase of their cycle.

On the last two days of each study period, patients will be admitted and two consecutive 24-hour urines for calcium, phosphorus, sodium, and creatinine will be collected, as well as a stool sample (see below). For calcium and phosphorus. Stool analysis for calcium and phosphorus will be used to determine the amount of calcium and phosphorus absorbed from the diet (fractionated absorption), while urine calcium and phosphorus will be used to determine the fraction of absorbed minerals that is excreted in the urine. Urinary sodium will be used to ensure consistent food intake while urinary creatinine will be used to ensure completeness of urine collections and to determine tubular phosphorus reabsorption.

Patients will also have a small saliva sample, about 3 ml, collected upon admission to the GCRC which will be used to measure the phosphorus concentration in saliva. These saliva samples will be stored at the GCRC until a laboratory is identified to perform these tests.

Patients will also have rapid serum calcium, phosphorus, creatinine, 1.25 vitamin D, 25 vitamin D, parathyroid hormone (PTH), and fibroblast growth factor (FGF) 23 measured at the end of each 24-hour urine collection. In addition, patients will have their blood phosphorus level measured before their evening meal on both evenings of their stay at the GCRC. After a "wash-out" period of at least one week, patients will begin the second week of the diet, at the end of which they will be admitted to the GCRC for two days. Patients can schedule their second diet period and remain on GCRC during one to four weeks after the previous diet and remain on GCRC at a time that suits them. Diets will be prepared by GCRC nutrition staff.

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