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The State of Calcium Metabolism in Patients with Renal Syndrome

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Abstract: The paper is a Master's thesis in the field of biomedical science and medical biochemistry, focusing on the state of calcium and phosphorus metabolism in patients with renal syndrome.

Contributions of the paper "The state of calcium and phosphorus metabolism in patients with renal syndrome":

The paper contributes to the field of biomedical science and medical biochemistry by studying the state of calcium and phosphorus metabolism in patients with renal syndrome.

It aims to provide insights into the alterations in calcium and phosphorus metabolism observed in chronic kidney disease (CKD) patients, which can help in understanding the pathophysiology of CKD-related complications.

The study focuses on the role of the kidneys in maintaining the balance of calcium and phosphorus in the body and highlights the importance of restricting phosphorus intake and absorption to manage CKD-MBD (chronic kidney disease-mineral and bone disorder)

Results of the paper "The state of calcium and phosphorus metabolism in patients with renal syndrome":

The paper likely presents the results of a study investigating the state of calcium and phosphorus metabolism in patients with renal syndrome, focusing on chronic kidney disease (CKD) patients.

The results may include findings related to the alterations in calcium and phosphorus levels observed in CKD patients, as well as the impact of these alterations on the development of CKD-related complications.

The study might provide insights into the relationship between calcium and phosphorus in renal failure, phosphate kinetics during hemodialysis, and calcium balance in CKD.

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Key words: CKD, pathophysiology, calcium and phosphorus

Introduction

The paper highlights the importance of monitoring calcium and phosphorus metabolism in patients with renal syndrome, particularly those with chronic kidney disease (CKD)[1,2,3].

Understanding the state of calcium and phosphorus metabolism can help in assessing the prognosis and long-term outlook of CKD patients, both before and during hemodialysis.

Maintaining serum calcium and phosphorus levels within the target control range is associated with better long-term prognosis in CKD patients, as deviations from the control target can have negative implications[4,5].

The findings of this study can inform healthcare professionals in managing calcium and phosphorus levels in CKD patients, potentially improving their overall outcomes and reducing the risk of complications.

Further research in this area is needed to better understand the relationship between calcium and phosphorus metabolism and its impact on patients' outlook in different stages of CKD

Introduction of the paper "The state of calcium and phosphorus metabolism in patients with renal syndrome"[6,7]:

Nephrotic syndrome, characterized by excessive protein excretion in urine, is often caused by damage to the kidneys' blood vessels, leading to various health issues and swelling in the feet.

Chronic kidney disease (CKD) is commonly associated with disorders in calcium and phosphorus metabolism, as the kidneys play a crucial role in maintaining the balance of these minerals in the body. [7,8]

Insufficient renal calcitriol synthesis and abnormal vitamin D metabolism in CKD patients can lead to alterations in osteoblast metabolism and impaired intestinal calcium absorption .

CKD patients with low blood calcium levels, even below the normal range, are at a higher risk of kidney failure compared to those with higher calcium levels. [9]

Patients with ongoing kidney sickness (CKD) have checked disturbance in bone and mineral digestion bringing about a complicated issue that has been named CKD-mineral bone problem (CKD-MBD). Annoyances start in the earliest phases of the CKD and deteriorate with moderate kidney illness [10]. The biochemical adjustments of CKD-MBD incorporate raised fibroblast development factor-23 (FGF23) and parathyroid chemical (PTH), diminished 1,25-dihydroxyvitamin D (1,25D), raised serum phosphate, and diminished serum calcium. Furthermore, diminished calcium retention and diminished urinary calcium discharge are noticed, as well as heterogeneous bone sickness and extreme vascular and delicate tissue calcification. CKD-MBD is related with an expanded crack gamble and higher pace of cardiovascular occasions and cardiovascular-related passings [11,12,13]. Nonetheless, the fundamental infection process isn't totally perceived, the initiators of the noticed irregularities are indistinct, and conclusive treatments are inadequate. Both negative and positive calcium balance present potential wellbeing dangers in CKD-MBD: negative equilibrium might increment risk for osteoporosis and break, and positive equilibrium might increment risk for extraskeletal calcification and cardiovascular occasions. In any case, it is impossible that negative or positive calcium balance alone is the starting element and it is problematic, albeit clinically conceivable, that negative or positive calcium balance adds to CKD-MBD illness movement in grown-ups. The motivation behind this survey is to analyze the accessible writing on calcium balance in CKD, examine information holes and the future examination needs around here, and propose useful proposals in light of current accessible proof. [14,15]

Method

A sum of 130 patients determined somewhere in the range of 2022 and 2023 to have renal condition in the Emergency clinic and The conclusion of renal disorder depended on the prevailing testimony of IgA in the mesangial region as seen with immunofluorescence; patients with auxiliary IgA nephropathy, for example, IgA vasculitis, fundamental lupus erythematosis and rheumatic illness, were prohibited. All patients were followed up routinely every 3−12 months.

The review was supported by the nearby morals boards and led as per the standards of the Announcement of Helsinki.

Clinical information, including age, sex, 24-h pee protein discharge, systolic and diastolic pulse, serum creatine level, serum egg whites level at the hour of kidney biopsy and serum phosphorus and calcium levels at the hour of kidney biopsy and at each visit were gathered. Histopathologic components were assessed by the Oxford grouping. The assessed glomerular filtration rate (eGFR) was determined utilizing the Constant Kidney Illness The study of disease transmission Joint effort condition and Mean blood vessel pressure (Guide) was determined as the amount of 33% of heartbeat pressure and the diastolic pulse.

Results

Table 1-distribution of patients according to age

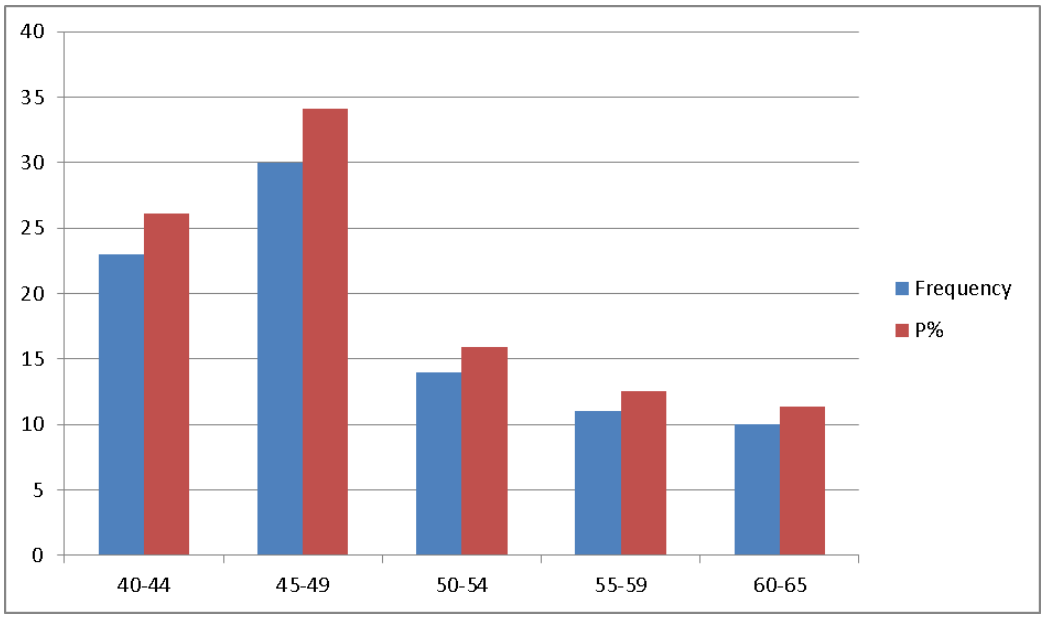
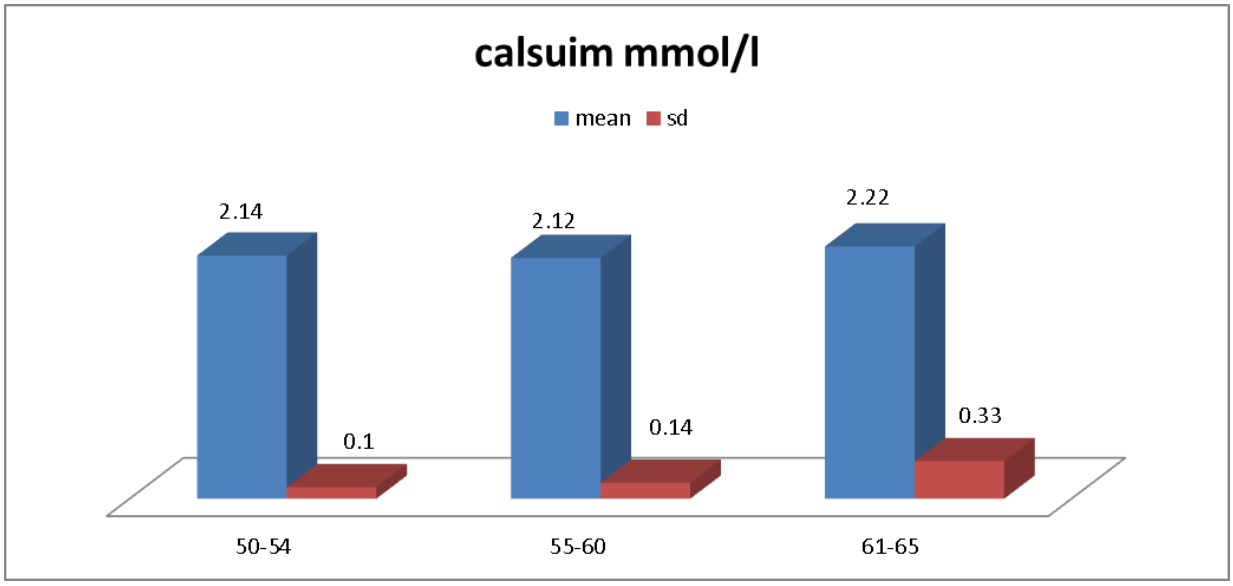


Table 2- result of calcium of Iraqi patients



Discussion

Altered calcium and phosphorus metabolism in chronic renal failure is one of the areas of kidney disease that has undergone the most changes in terms of theoretical concepts, clinical impact, and treatment goals. Successive opinions, often quite different from previous opinions, and sometimes defended with great emphasis by the same authors, always coincide with the appearance of new therapeutic measures on the market.

While phosphorus retention and the importance of its control are concepts that have remained unchanged since their introduction more than four decades ago, opinions on calcium homeostasis in chronic renal failure have been less consistent and even controversial. In the initial publications it was highlighted that these patients had a negative calcium balance due to decreased intestinal absorption of vitamin D, and that calcium malabsorption was one of the causes of secondary hyperparathyroidism.. in D.S. David's classic book on the subject, published in 1977, it was pointed out that to prevent and treat secondary hyperparathyroidism, it was necessary to correct negative calcium balance by one of these three measures: Adding calcium supplements to the diet to promote its growth. Passive absorption (gradient-based), with vitamin D administration to correct calcium malabsorption, or in a patient dialyzed by calcium transfusion during hemodialysis with a bath calcium concentration equal to or greater than 6 mg/day. dL (3 mEq/L) 2. The author recommended starting treatment in the initial stages of chronic renal failure by giving alkaline calcium salts that provide this element and prevent phosphorus retention due to their chelating effect on it. The oral dose of calcium required for homeostasis increases with the progression of renal failure and depends on the adoption or non-adoption of the other two measures (use of vitamin D analogues and bath calcium concentration if the patient is already being treated with dialysis).

The hypothesis of a negative calcium balance and the need to correct it to prevent the development of secondary hyperparathyroidism has lost its importance in conjunction with the interest in the appearance and development of vascular calcifications. In existing reviews on the pathogenesis of mineral metabolism changes in chronic renal failure, calcium malabsorption has not been included among the factors causing the onset of secondary hyperparathyroidism 3,4 . There are even authors who consider that in chronic renal failure, calcium balance is not only negative, but tends to be positive because the decreased urinary calcium excretion compensates for its decreased

intestinal absorption.. According to this approach, calcium intake should be controlled because its retention can Contributes to the appearance of vascular calcifications[14,15,16,17].

The K/DOQI Guideline Working Group on Metabolism and Bone Disease in Chronic Renal Failure endorses the theory of positive calcium homeostasis and control of oral intake to prevent calcium retention. Recommendations 5.5 and 6.4, with the level of evidence only for opinion, recommend reducing oral calcium intake to a maximum of 2000 mg/day (500 mg/day as content in the diet and 1500 mg/day as content of chelated calcium). Phosphorus) 6. This same criterion has been adopted in the recently published guides of the Spanish Society of Nephrology7,8. In all of this clinical evidence, the indication for oral calcium salts is limited to their phosphorus-chelating effect, and at no time is their use as an oral calcium supplement considered. [18,19]

It should be noted that limiting oral calcium intake is not universal and is a source of controversy. Friedman and nephrologists from the Amiens School consider the evidence regarding oral calcium intake and vascular calcifications to be inconsistent and advocate the use of calcium chelators at doses higher than the limit recommended in guidelines. A recent experimental study showed that calcium carbonate supplementation not only increases but actually reduces vascular calcifications in a model of induced chronic renal failure in mice with lipoprotein E deficiency[20]

Conclusion

There have been many examinations on calcium and phosphorus digestion and its impact on anticipation in patients with stage 5 constant kidney sickness, especially those entered on long haul renal substitution treatment. For instance, an as of late distributed concentrate by Flug 2015 showed that those whose serum calcium and phosphorus levels were inside the objective control range had fundamentally preferable long haul visualization over those whose relating markers strayed from the control target. There are generally couple of concentrates on patients with CKD in stage 3-5 preceding hemodialysis, however the current information actually demonstrate that calcium and phosphorus digestion is fundamentally connected with patients' viewpoint.

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