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Study of Parathyroid Hormone and Vitamin D Levels in Women with Renal Failure

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ABSTRACT
Objective: Because chronic kidney disease (CKD) is widely linked to a number of comorbidities, elevated cardiovascular issues, mortality events, and high medical costs, it is a major global health concern. Vitamin D regulates the balance of calcium and phosphate and has a number of other skeletal impacts on human systems, including the neurological, cardiovascular, immunological, cell differentiation and growth, and hormonal systems. As CKD worsens, the prevalence of 25-vitamin D insufficiency increases.
The aim of this study was to investigate the relationship between parathyroid hormone and Vit D levels and to study the variables resulting from their imbalance in patients with (CKD).
Methods: Participants who were between the ages of 30 and 65 and not pregnant were recruited after obtaining informed consent from 44 individuals who visited government clinics at Yarmouk Teaching Hospital. Assessments should be made of the complete blood count (CBC), lipid profiles, fasting sugar, liver and kidney biochemistry indicators, intact parathormone (PTH), and a total of 25 (OH) D-vitamin levels.
Results: Compared to controls, patients' blood calcium levels were noticeably lower. While blood PTH levels in patients are much higher than in controls when compared to controls, patients' blood Vit D levels are significantly lower and there was a notable rise in blood urea, creatinine, and PHO levels in patients compared to controls.

Keywords: Vitamin, blood urea, creatinine, biochemistry

INTRODUCTION

Because chronic kidney disease (CKD) is widely linked to a number of comorbidities, an increased risk of cardiovascular issues, mortality events, and high medical costs, it is a major global health concern. The worldwide prevalence of chronic kidney disease (CKD) ranged from 8 to 16%, depending on the illness criteria, research methodology, and racial classifications.With a nationwide prevalence of 11.9%, Taiwan has the highest incidence and frequency of end-stage renal disease (ESRD) worldwide [2], [3]. For early detection and intervention to lessen the burden of the disease, accurate identification of CKD risk factors is necessary. Numerous risk factors, including metabolic syndrome, smoking, obesity, hypertension, diabetes, and dyslipidemia, have been linked to chronic kidney disease (CKD) in well-established studies [4].
The correlation between blood vitamin D levels and nonskeletal conditions such cardiovascular events [5-7], metabolic syndrome [8, 9], cancer [9, 10], autoimmune disease [11], and critical illness [12] has been emphasized by the observational research. Lower glomerular filtration rates in CKD patients limit the substrate supply to 1-alpha-hydroxylase, which prevents the kidneys from producing 1,25 (OH) vitamin D. The relationship between vitamin D levels, fibroblast growth factor-23 (FGF-23), and renal function deteriorates when renal function declines, parathyroid hormone (PTH) stays constant, and calcium-phosphorus-bone metabolism is disrupted. [13–15]. The relationship between PTH, vitamin D, and metabolic calcium phosphate and the onset of chronic kidney disease is still up for debate, though.

Role of vitamin D and parathyroid

In addition to having multiple extraskeletal effects on the neurological, immunological, cardiovascular, cell differentiation and development, and hormonal systems, vitamin D controls the balance of phosphate and calcium [16].

As CKD worsens, the prevalence of 25-vitamin D insufficiency increases; in stage 5 cases, it approaches 80%.

Reduced blood calcium and vitamin D availability are biological markers of PTH secretion enhancement. By enhancing intestinal calcium absorption, PTH encourages the manufacture of calcitriol, the most active metabolite of vitamin D, as well as the reabsorption of calcium in kidney tubules, phosphate and calcium absorption from bone tissue [17]. Restoring normal calcium and vitamin D levels requires increased PTH production [17]. Therefore, a compensatory syndrome that seeks to compensate for decreased calcium and/or vitamin D bioavailability is frequently referred to as secondary hyperparathyroidism. When renal failure occurs Early onset of secondary hyperparathyroidism and its quick progression without treatment might result in potentially extra-skeletal (soft tissue calciumifications, cardiac arteries, and valves) and severe skeletal (renal osteodystrophy) problems in the majority of renal failure patients[18].

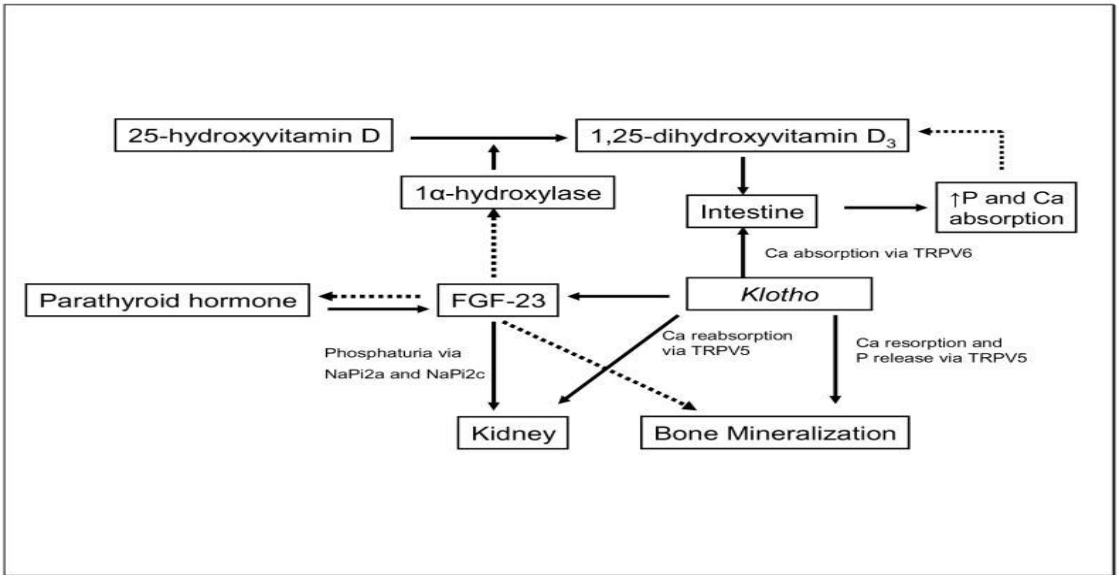


Figure 1: pathway of synthesis of vitamin D and its role in the bone synthesis

Vit. D and its Metabolites Changes

In the early stages of chronic kidney disease, alterations in vitamin D metabolism are noted when an individual's glomerular filtration rate is less than 60 ml/min/1.73 m2 [19]. Additionally, people with CKD have a decreased development of the vitamin D receptor [20]. Serum calcium and phosphate levels, bone alkaline phosphatase, and PTH variations are also observed. These abnormalities are a component of the metabolic aspect of CKD-related mineral bone damage.

Method Patient Setting and Data Description

From March 2023 to February , Yarmouk Teaching Hospital hosted community health events in which 44 patients and 44 healthy individuals were chosen for the study. Participants who were between the ages of 30 and 65 and were not pregnant were added with their informed consent. The study excluded participants with inadequate baseline biochemical data and those who took any over-the-counter vitamin supplements, including vitamin D drugs.

Demographic data was gathered through questionnaires. Blood samples were collected during a 10-hour or overnight fast in order to assess the complete blood count (CBC), lipid profiles, intact parathormone (PTH), fasting sugar, liver and kidney biochemistry indicators, and a total of 25 (OH) D-vitamin levels.

The aim of this study was to investigate the relationship between parathyroid hormone and vitamin D levels and to study the variables resulting from their imbalance in patients with chronic renal failure, such as calcium, creatinine, glucose, phosphorus and urea levels.

Determination of Biochemical Parameters

The Hitachi Cobas C311 device from Roche Diagnostics was utilized to measure the elements calcium, phosphate, and magnesium as well as the serum components of urea and creatinine. The Tridenter-reaction, an enzymatic and colorimetric method that employs a chromogen, is the fundamental idea underlying the

methodology. The substance of the quantities under assay determines the relevance of the coloration or turbidity that is created. To measure PTH and 25(OH)D 5 hormones, the Enzyme Linked Immuno Assay (ELISA) method is employed. This two-stage immuno-enzymological enzyme detection technique allows for the imaging of a colored antigen-antibody reaction that arises from an enzyme that has previously been coupled to the antibody on a substrate. This technique acts as an analytical indicator for the detection test since the intensity of the resulting light is proportionate to the amount of the chemical that was detected in the sample.

RESULTS

Table 1: Statistical parameters (Meanand SD) for the concentrations of biochemical markers.

Parameters	Control		Patients		P-value
	Mean	**SD**	**Mean**	**SD**	
Age/year	24.83	2.14	53.88	15.27	<0.001*
Ca mg/dL	9.03	0.13	8.54	1.27	0.358
Crea mg/dL	0.67	0.10	7.56	2.20	<0.001*
Glu mg/dL	81.57	3.58	109.63	44.99	0.134
PHO mg/dL	3.03	0.15	4.77	1.59	0.009
Urea mg/dL	23.10	4.63	144.16	35.84	<0.001*
Vit D3 ng/mL	30.47	15.09	12.65	8.93	<0.001*
PTH pg/mL	26.82	4.47	475.38	134.09	<0.001*
*Significant at P≤ 0.05, NS: Non-Significant.					

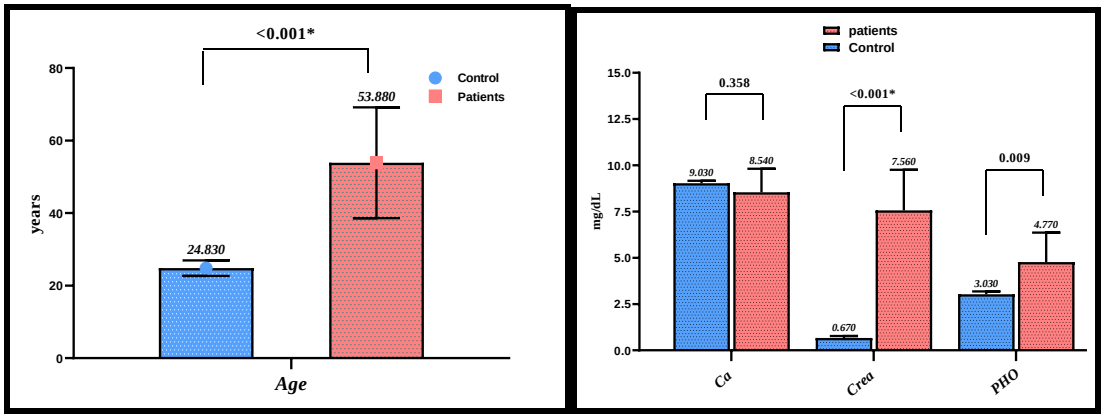
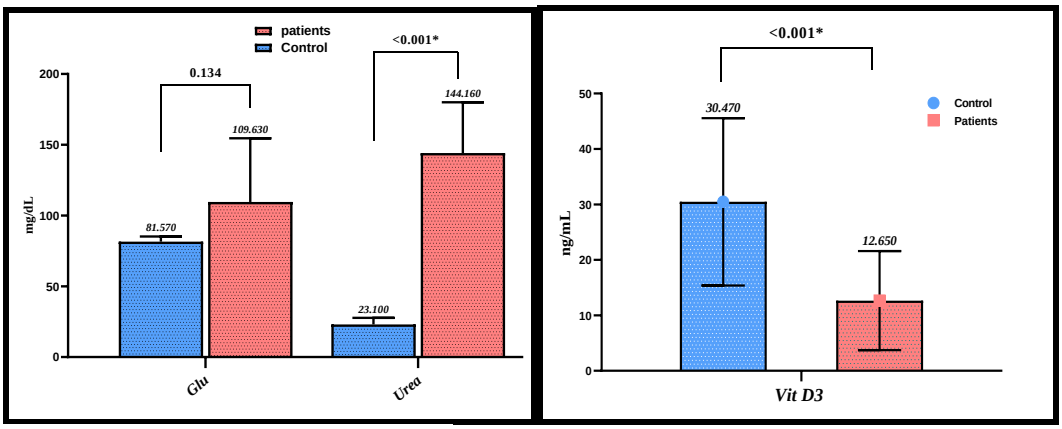


Figure (3) : compared mean of study biomarkers between patients and controls



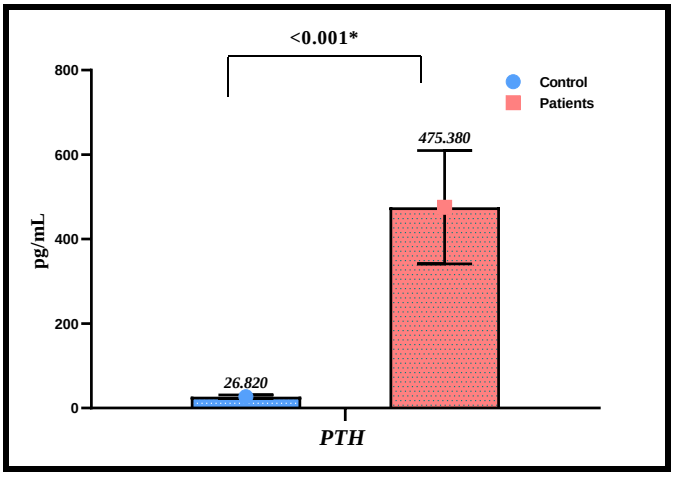


Figure (6) :compared mean of PTH between patients and controls

DISCUSSION

Compared to controls, patients' blood calcium levels were much lower.Vitamin D plays a role in calcium metabolism, as it stimulates gene expression to produce protein produced by the cells of the small intestine. This protein is a carrier of calcium that comes from food. Since most women, including Iraqi women, have a deficiency in vitamin D, this leads to a decrease in calcium levels that come from food. Then a process of sensing and responding to this deficiency will occur, as calcium has many important functions, including muscle contraction and relaxation, lung movement, mental ability, glycogen storage, and insulin secretion,due to this importance, three hormones control calcium levels: vitamin D, parathyroid hormone, and calcitonin.Although blood vitamin D levels in patients are significantly lower than in controls, there are two sources of vitamin D in blood circulation: exogenous (ergocalciferol, cholecalciferol, found in seafood or some plants) and endogenous. The second source of vitamin D is endogenous (cutaneous production)[20]. Sunlight-induced skin development is the primary source of vitamin D, with diet and supplements serving as supplementary sources [21]. Conversely, black people's skin color impacts how much vitamin D their skin produces, which has an impact on subsequent generations. Black people therefore had lower 25(OH)D levels, maybe as a result of melanin, the skin's pigment, acting as a natural sunscreen and reducing the skin's ability to synthesize vitamin D3. Reduced renal tubular reabsorption is the result of hypocalcemia. The patient's blood can transfer calcium since the dialysate's calcium level is greater than the quantity of ionized calcium in the blood (calcium bound to proteins can not diffuse).

Although patients' blood PTH levels are significantly higher than those of controls (hypocalcemia decreases renal tubular reabsorption and intestinal calcium absorption, which increases the synthesis of PTH by mRNA),

The body produces creatinine in the muscle as a byproduct of creatine phosphate, and patients' blood urea, creatinine, and PHO levels were significantly higher than those of controls. The majority of creatinine elimination from circulation occurs in the kidney. The level of blood creatinine rises when renal clearance declines. Serum creatinine and urea plasma concentrations increase when GFR falls.As for phosphorus, it forms crystals with calcium --- to mine and strengthen the bones. During kidney failure, the level of parathyroid hormone rises in response to the decrease in calcium. This causes these crystals to be consumed by the bones to compensate for the deficiency, and thus the level of phosphorus in the blood rises.

During renal failure, a number of metabolic problems arise, including secondary hyperparathyroidism. The latter is caused by a number of interconnected factors, such as disturbances in the decrease in 25-hydroxyvitamin D levels and phosphocalcic metabolism, both of which are connected to calcitriol levels. It is crucial to regulate those factors in order to lessen the detrimental effects of the illness.

Assessments of parathormone and 25-hydroxyvitamin D are relevant and necessary for better management of renal insufficiency.

CONCLUSION

This study relied on traditional routine measurements in evaluating medical conditions in addition to the presence of a medical consultation. We found an increase in creatinine and urea, which is an indication of weak kidney function. Vit D was measured in patients with kidney failure and we found a significant decrease in its level. This plays a major role in increasing the state of kidney failure due to its role in absorbing calcium from the digestive tract. Its decrease leads to a decrease in the level of calcium in the bloodstream. There was an increase in the level of PTH, and therefore we can rely on the level of vit D and PTH as biochemical indicators in diagnosing kidney failure.Both community sensitization and better care of the causes of renal insufficiency would aid in the early detection of the condition.

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