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The Role of Calcitonin and Vitamin D in patients with Chronic Renal Failure and Arthritis

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Abstract
Background: Chronic kidney disease is an end stage leads to problems in bone as rheumatoid arthritis (RA) with both glomerular and tubular damage. It is often difficult to differentiate between damage due to disease activity and that due to drugs used to treat Rheumatoid Arthritis.
The aim: To estimate the levels of Calcitonin and Vitamin D and their relationship with renal Osteodystrophy
Methods: Calcitonin and Vitamin D were obtained from 135samples: 85 samples from patients with chronic renal failure, 25 samples with (CRF with arthritis) renal Osteodystrophy, and 25 samples apparently healthy.
Results: A highly significant increase ($p<0.001$) in the level of calcitonin in serum of patients with Chronic renal failure and renal osteodystrophy disease compared with healthy individuals. Highly significant decrease ($p<0.001$) in the level of vitamin D in serum of patients with chronic renal failure and renal osteodystrophy disease compared with healthy individuals.
Keywords: Chronic renal failure & Arthritis biomarkers, Calcitonin, Vitamin D
List of Abbreviation:- CRF= Chronic renal failure, Vit. D=Vitamin D, GFR=glomerular filtration rate, CT=Calcitonin, CGRP= calcitonin gene-related peptide, HCT=Human calcitonin, BMD= bone mineral density, CTRs= calcitonin receptors, PL-C= phosphor lipase-C, CKD=Chronic kidney disease, KDIGO=Kidney Diseases: Improving Global Outcomes, ESRD= end stage renal disease VDR=Vitamin D Receptor, cAMP= Cyclic adenosine mono phosphate.

Introduction
Chronic kidney disease (CKD) describes abnormal kidney function and/or structure. It is common, frequently unrecognised and often exists together with other conditions (such as cardiovascular disease and diabetes). Moderate to severe CKD is also associated with an increased risk of other significant adverse outcomes such as acute kidney injury, falls, frailty and mortality. The risk of developing CKD increases with age**[1].**
Rheumatoid Arthritis (RA) is an autoimmune disorder that can cause aching and swelling in joints. When the body's immune system wrongly identifies body cells as invaders, it fights and attacks good tissues, causing inflammation**[2] .**
Bone mineral disease is basically a multifactorial disorder of bone remodeling. The most important factor which is responsible for the development of secondary hyperparathyroidism is a deficit of active vitamin D (calcitriol). Diseased kidneys cannot sufficiently hydroxylate 25-hydroxycholecalciferol, which is a precursor of calcitriol (1,25-dihydroxycholecalciferol)[**3]**. In a patient with kidney failure, the kidneys stop making calcitriol, a form of vitamin D. The body then can't absorb calcium from food and starts removing it from the bones**[4]**.

Calcitonin
The calcitonin (CT) is a polypeptide hormone. CT is able to decrease blood calcium levels by direct inhibition of mediated bone re absorption and by enhancing calcium excretion by the kidney. Human CT is a single-chain peptide of 32- amino acid residues**[5].**
The activities of CT are mediated by high affinity calcitonin receptors (CTRs). The CTR is a member of a subfamily of the seven-transmembrane domain G-protein coupled receptor super family that includes several peptides. Members of this family have a similar structure with other seven-membrane-spanning domain G-protein coupled receptors. This receptor was characterized by a long NH2-terminal domain that was extracellular. It was similar to parathyroid/parathyroid hormone- related peptide receptor and the secreting receptor**[6**].
The principal mechanism of action of CT it due to the ability of its receptor to couple at least two signal transduction pathways. One of the most important pathways is coupled with the cAMP signal transduction. However, CTRs can also couple to the phospholipase C (PLC) enzyme pathway. The PLC pathway, as with the cAMP pathway, can be initiated by the coupling of receptors to multiple G-proteins. Activation of the PLC causes the release of Ca^{2+} from intracellular stores and promotes an influx of external calcium . In addition, CTRs are able to activate the phospholipase D (PLD) **[7**] .
At the renal level, calcitonin tends to inhibit tubular reabsorption of calcium. At bone, calcitonin inhibits osteoclastic bone resorption. This acute inhibition of osteoclastic activity allows the continuing osteoblastic activity to increase net uptake of calcium by bone[8,9].

Vitamin D
Vitamin D, is steroid hormone also known as calciferol, is a fat soluble vitamin. It is different from all other fat soluble vitamins, in that the body can synthesis it with the help of sunlight, from a precursor that the body makes from cholesterol**[10]**.
In its native form vitamin D is not biologically active, the active form is 1, 25(OH) 2D. The conversion of

vitamin D to 1, 25(OH) 2D requires two hydroxylation in tandem. Vitamin D is first hydroxylated by the liver to form 25(OH) D, which is then hydroxylated by the kidney to form 1, 25(OH) 2D. 25(OH) D has low biological activity, but it is the major form of vitamin D that circulates in the blood stream. Serum 25(OH)D concentrations are generally thought to reflect nutritional status **[11]**.When adequate amounts of vitamin D are available, the kidney, the major site of 1,25(OH)2D production converts some of the 25(OH)D to alternate hydroxylated metabolites, which have low biological activity (e.g., 24,25 (OH)2D or 1,24,25(OH)3D). Renal synthesis of 1,25(OH)2D is tightly regulated by plasma parathyroid hormone, together with serum calcium and phosphorus concentrations. Additional tissues that express the enzyme that catalyses the conversion of 25(OH) D to 1, 25(OH) 2D, 25-hydroxyvitamin D3-1-α-hydroxylase, include colon, prostate, mammary gland, macrophages, antigen-presenting cells, osteoblasts and keratinocytes[12].

Major sites of action include intestine, bone, parathyroid, liver and pancreatic beta cells. Biological actions include increases in intestinal calcium absorption, transcellular calcium flux and opening gated calcium channels allowing calcium uptake into cells such as osteoblasts and skeletal muscle[13].

One of the major biological functions of vitamin D is to maintain calcium homeostasis which impacts on cellular metabolic processes and neuromuscular functions. Vitamin D affects intestinal calcium absorption by increasing the expression of the epithelial calcium channel protein, which in turn enhances the transport of calcium through the cytosol. Vitamin D also facilitates the absorption of intestinal phosphate. 1,25(OH)2D indirectly affects bone mineralization by maintaining plasma calcium and phosphorus concentrations, and subsequently extracellular calcium and phosphorus concentrations at the supersaturating range necessary for mineralization. 1,25 (OH) 2D, in concert with parathyroid hormone, also causes demineralization of bone when calcium concentrations fall to maintain plasma concentrations within a narrow range. It has yet to be determined whether 1, 25(OH) 2D directly influences bone mineralization.

In addition to intestine and bone, a wide range of other tissues and cells that are influenced by vitamin D. Five biological systems have vitamin D receptors and are responsive to 1,25(OH)2D., These systems include immune, pancreas, cardiovascular, muscle and brain; and control of cell cycle. The biological effects of 1, 25(OH) 2D are diverse. For example, as recently noted, 1, 25(OH) 2D inhibits PTH secretion and promotes insulin secretion, inhibits adaptive immunity and promotes innate immunity, and inhibits cell proliferation and stimulates their differentiation, A number of recent reviews have appeared on these topics **[14, ,15,16]**.

The vitamin D endocrine system is a potent modulator of parathyroid function. Whereas vitamin D deficiency results in parathyroid hyperplasia and increased PTH synthesis and secretion, $1,25(OH)_2D_3$ administration inhibits PTH synthesis and parathyroid cell growth, thus rendering 1,25 $(OH)_2$ D_3 therapy effective in treating the secondary hyperparathyroidism of chronic kidney disease [**17]**.The most important endocrine effect of 1,25 $(OH)_2D_3$ in the kidney is a tight control of its own homeostasis through simultaneous suppression of 1α-hydroxylase and stimulation of 24-hydroxylase and very likely through its ability to induce megalin expression in the proximal tubule[**18]**.1,25 $(OH)_2$ D_3 involvement in the renal handling of calcium and phosphate continues to be controversial due to the simultaneous effects of $1,25(OH)_2D_3$ on serum PTH and on intestinal calcium and phosphate absorption, which affect the filter load of both ions. 1, 25(OH) $_{2D3}$ enhances renal calcium reabsorption and calbindin expression and accelerates PTH-dependent calcium transport in the distal tubule, the main determinant of the final excretion of calcium into the urine and the site with the highest Vitamin D Receptor (VDR) content [**19**].

Vitamin D is a hormone essential for bone and mineral homeostasis and is also involved in the regulation of cells in the innate and adaptive immune system through the Vitamin D Receptor (VDR) as a suppressor of proinflammatory responses (Mathieu et al., 2001). Although vitamin D has been implicated in a decreased risk of autoimmune diseases such as type 1 diabetes and multiple sclerosis, its role in decreasing the risk of RA remains equivocal[**20]**. An inverse association between vitamin D intake and RA was found in the prospective cohort study done by the Iowa Women's Health Study**[21]**.

Materials and Methods

Patients and control were divided into Three groups:

Group 1: It included a total of 85 patients (50 males and 35 females); their ages ranged from 20-70 years. All of them are suffering from chronic kidney disease.

Group 2: It included a total of 25 patients (10 males and 15 females); their ages ranged from 20-70 years .They were suffering from chronic kidney disease with Arthritis disease.

Group3 : This group was include 25 apparently healthy subjects as control group (12 males and 13 females), and their ages ranged from 20to 70 years .

The calcitoin assay employs the quantitative sandwich enzyme immunoassay technique suppliers by cusabio companies **[22].**

The LIAISON ®25OHVitamin D assay is a direct competitive chemiluminescence immunoassay (CLIA) quantitative determination of total 25 OH vitamin D in Serum. Suppliers by Dia Sorin companies**[23]**.

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Results and discussion

Estimation of Serum Calcitonin& Vitamin D level

Results showed that there was a highly significant increase ($p<0.001$) in the serum levels of calcitonin in CRF and CRF with arthritis patient (62.3 ± 10.0) and (78.5± 11.6) respectively compared to control group (30.0 ± 6.2). while there was a highly significant decrease in the serum levels of Vit.D in G1 and G2 (15.15±5.36) and (11.59±5.51) respectively for a group of patients compared to control group (27.6 ± 6.6) as shown in the table and figure 1.

Table 1: The concentration of Calcitonin & Vitamin D in the blood of patients with CRF& CRF with Arthritis compared to control

Parameter	Mean± SD		
	G1(n=85) CRF	G2(n=25) (CRF+Arthritis)	G3(n=25) Control
Calcitonin (pg/ml)	62.3 ± 10.0*	78.5± 11.6*	30.0± 6.2
Vit.D (ng/ml)	15.15± 5.36*	11.59± 5.51*	27.6±6.6

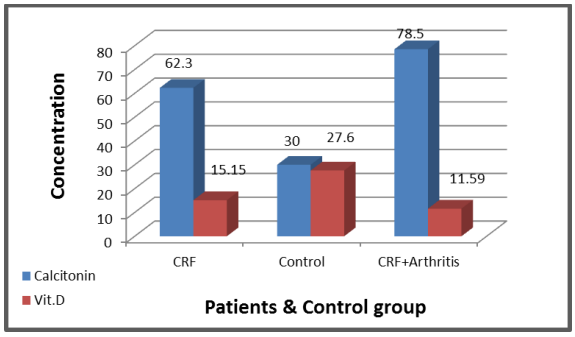

Figure 1: The concentration of calcitonin and vtamin D in the blood of patients with chronic renal failure compared control group.

The table and figure 2 Showed that there was no significant difference in calcitonin levels in age groups (55.27 ± 30.67),(59.7 ± 34.7), (63.90 ± 31.71), (69.50 ± 25.75), (60.74 ± 31.81) respectively, and no significant difference in vit.D Concentration for age groups (15.63 ± 5.17), (12.93 ± 3.60), (15.57 ± 6.77), (15.82 ± 4.61), (14.51 ± 5.56) respectively.

Table 2: The concentration of Serum Calcitonin & Vitamin D in the blood of patients with chronic Renal failure depending on age.

Age / Parameter	Mean± SD	
	Calcitonin (pg/ml)	Vit.D (ng/ml)
20-30 (n=13)	55.27± 30.67	15.63± 5.17
30-40 (n=8)	59.7± 34.7	12.93± 3.60
40-50 (n=18)	63.90 ± 31.71	15.57± 6.77
50-60 (n=21)	69.50± 25.75	15.82± 4.61
60-70 (n=25)	60.74± 31.81	14.51± 5.56

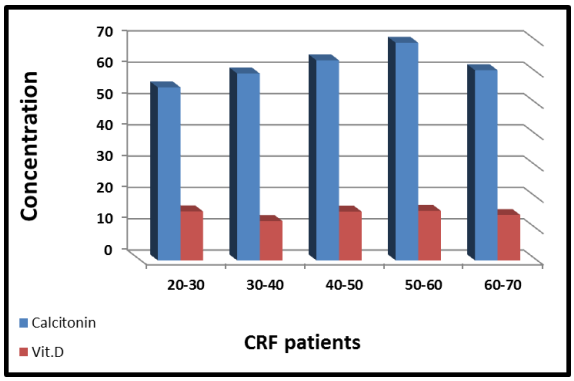

Figure (2) The concentration of Serum Calcitonin & Vitamin D in the blood of patients with chronic Renal failure depending to Aon age.

Table and figure 3 showed that the patient levels of calcitonin have no significant differences among sex groups, (57.2 ± 10.9) (68.1 ± 18.3) for males and females respectively.

There was no significant differences among sex groups for Vitamin D level in both males and females (15.36± 5.51) (14.86 ± 5..21) respectively.

Table 3: The concentration of Serum Calcitonin & Vitamin D in the blood of patients with chronic Renal failure depending on Sex.

Sex / Parameter	Mean± SD	
	Calcitonin(pg/ml)	Vit.D (ng/ml)
Males(n=50)	57.2± 10.9	15.36± 5.51
Females(n=35)	68.1± 18.3	14.86± 5..21

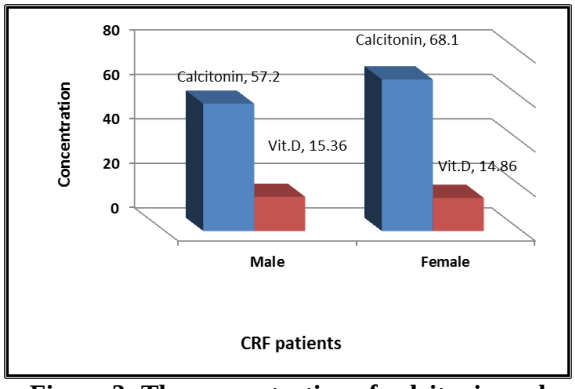

Figure 3: The concentration of calcitonin and vitamin D in the blood of patients with chronic renal failure depending on Sex.

The kidney plays an essential role in vitamin D metabolism in circulation. Chronic kidney disease (CKD) is a condition characterized by a gradual loss of kidney function over time. The abnormalities in vitamin D metabolism could contribute to the development of mineral and skeletal disorders, increases in PTH, hypertension, systemic inflammation, and finally result in renal damage. The KDIGO (Kidney Diseases: Improving Global Outcomes) clinical practice guidelines recommended correcting 25(OH)D deficiency and insufficiencies for the general population**[24] .**

The reasons for this marked vitamin D deficiency in CKD are multi-factorial. CKD can induce a progressive loss of the capacity of the kidney not only to convert 25(OH)D to circulating calcitriol (the vitamin D hormone), but also to maintain serum 25(OH)D levels for non-renal calcitriol synthesis. The resulting calcitriol and 25(OH)D deficiency associates directly with accelerated disease progression and death Another interesting hypothesis is that urinary loss of 25(OH)D-VDBP (the main plasma carrier of vitamin D in circulation) associated with proteinuria and reduced medaling-mediated uptake might result in vitamin D deficiency. Alternatively, reduced levels of 25(OH)D might be a result of compromised endogenous pre-vitamin D production in the skin due to severe renal dysfunction or simply lack of outdoor sunlight exposure due to morbidity. Most, though not all, the observational studies have demonstrated 25(OH)D deficiency is independently associated with impaired renal function **[25,26]**

Cohort studies among patients with end stage renal disease (ESRD) also indicated higher 25(OH)D or 1,25(OH)2D levels were associated with decreased overall mortality [101–103]. Most of the current observational findings are from patients with CKD or severe kidney dysfunction; studies among individuals with mildly or moderately declined renal function especially among postmenopausal women are few**[27,28**]

Low vitamin D level has been implicated as a risk factor in RA development. vitamin D deficiency or insufficiency was more common in patients of RA than in healthy controls and even mean serum vitamin D level of patients of RA was found to be significantly low compared to healthy subject. Vitamin D has regulatory activity and vitamin D receptors are present in a number of cells of the immune system. Immuno modulation is mediated via activated T lymphocyte and activated B lymphocyte**[29]**.

This may be the result of lower vitamin D levels in patients with high disease activity levels because of poor exposure to direct sun light. There was no information about sun exposure in this study[**30]**. However, this association remains despite adjusting for age, gender and medication use, demonstrating a strong relationship. Results of this agreement with Diaz (2009) et al who studied a group of patients with lupus nephritis[**31]**. The kidney is the principal site of Calcitonin (CT) degradation by neutral endopeptidase (NEP). In normal men the effect of Calcitonin (CT) on the kidney is to stimulate diuresis and increases the fractional excretion rate of sodium and chloride. In addition, in urine a calcium and phosphate excretion increases. In patients with metastatic bone disease, the administration of Calcitonin (CT)induces a rapid fall in serum calcium due primarily to inhibition of renal tubular reabsorption**[32]**.

Osteoclasts are the major target for the action of CT. It is able to interfere with osteoclast differentiation from precursor cells and fusion of mononucleated precursors to form multinucleated cells in bone marrow cultures. CT plays an important role in skeletal homeostasis, being a key modulator on bone resorption. It acts directly on calcitonin receptors (CTRs) to inhibit bone resorption by inducing contraction and inhibits osteoclast motility (Q effect) occurring within 1 min and this is followed by a more gradual retraction of the osteoclasts (R effect). Both cAMP and intracellular calcium (Ca2+) are second messengers for the Q and R effects, and both are G-protein mediated. CT inhibits also other components of the osteoclast, such as the release of acid phosphatase and the expression of carbonic anhydrase II[**33**].

Conclusion

Hormones levels measurement showed a significant of variety hormones such as (calcitonin and Vitamin D in the renal osteodystrophy, and kidney disease patients while more sensitive in osteodystrophy which give: valuable information for diagnosis, good monitoring disease status and progression of the disease.

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دور هرمون الكالسيتونين وفيتامين د في مرضى الفشل الكلوي المزمن والتهاب المفاصل

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الملخص

أظهرت هذه الدراسة أن أمراض الكلى المزمنة وفي المرحلة الأخيرة من المرض يؤدي إلى مشاكل في العظام تعرف بالمرض العظمي الكلوي كالتهاب المفاصل الروماتيزمي (RA) مع كل من التلف الكبيبي والنبيبي. وغالبا ما يكون من الصعب التفريق بين التلف الناتج من نشاط المرض أو بسبب الأدوية المستخدمة لعلاج التهاب المفاصل الروماتيزمي.

أظهرت النتائج التي تم الحصول عليها من هذه الدراسة :

1- وجود ارتفاع معنوي عالي ($p<0.001$) في تركيز هرمون الكالسيتونين في مصل دم مرضى الفشل الكلوي المزمن ومرض التهاب المفاصل مقارنة مع مجموعة السيطرة (الأصحاء) , لا توجد فروقات بين الذكور والإناث , كما لا توجد فروقات معنوية بين الفئات العمرية , يوجد ارتفاع معنوي في تركيز الكالسيتونين في مصل دم مرضى العظمي الكلوي مقارنة مع مرضى الكلى .

2- وجود انخفاض معنوي عالي ($p<0.001$) في تركيز فيتامين د3 في مصل دم مرضى مرضى الفشل الكلوي المزمن ومرضى مع التهاب المفاصل, مقارنة مع مجموعة السيطرة (الأصحاء) , لا يوجد فروقات معنوية بين الذكور والإناث و لا بين الفئات العمرية , كما يوجد انخفاض معنوي في فيتامين د3 في مصل دم مرضى العظمي الكلوي مقارنة مع مرضى الكلى.