doi: 10.20903/zpopov-2022-0014 UDK: 615.272:616.155.194]:616.61-008.64-78

Irena CAKALAROSKA[1], Lada TRAJCESKA[2], Ninoslav IVANOVSKI[2], Zivko POPOV[3,4], Koco CAKALAROSKI[2]

# INFLUENCE OF CALCITRIOL ON ANAEMIA IN HAEMODIALYSIS PATIENTS

## Abstract

36 haemodialysis patients were treated with oral calcitriol and were then analysed. The patients received from 23 to cca 518 μg calcitriol (total dose) with a daily regimen of 0.25 to 0.50 μg every second day. This was done to prevent metabolic renal osteopathy (known as a "renal osteodystrophy", ROD). In 30 of 36 (83.3%) patients, we found a significant increase in blood haemoglobin (Hgb) concentration (more than 10%) after treatment with calcitriol. All haemodialysis procedures, dietetic measures, and other medical treatments remained unchanged when compared to the pre-treatment period.

We have found that orally administered calcitriol is also effective in the therapy of uremic anaemia. More detailed studies on larger numbers of patients are needed to make a final conclusion.

**Keywords:** uremic anaemia, PTH, calcitriol

## Introduction

Calcitriol [$1,25(OH)_2D_3$] is a hormonally active metabolite of vitamin $D_3$ and the basic regulator of bivalent ions homeostasis, in addition to

[1] Department of pediatrics, Faculty of Medicine, University "St. Cyrill and Methodius", Skopje,

[2] Department of nephrology, Faculty of Medicine, University "SS. Cyrill and Methodius", Skopje, North Macedonia

[3] Department of urology, Faculty of Medicine, University "SS. Cyrill and Methodius", Skopje

[4] Macedonian Academy of Sciences and Arts, Skopje, North Macedonia

having the effect of a parathyroid hormone (PTH) and calcitonin (CT). Calcitriol (C) is created in the mitochondria of epithelial cells in the upper renal tubules under the action of the complex enzyme 25(OH)-C 1α hydroxylase (a complex of reductase, rhenodoxine and cytochrome P-450 oxide/reductase). Calcitriolemia controls the creation of 1,25 $(OH)_2D_3$ by a feed-back mechanism, while PTH, CT and hypophosphatemia directly stimulate its synthesis. On the other hand, C is a very active substance which modulates the activity of some other hormones and affects the differentiation of certain tissues through specific receptors (known as the pleiomorphic effects of calcitriol).

C is also known that it supresses indirectly (through calcemia) and directly. It suppresses the synthesis of PTH, thus leading to a reduction of the serum alkaline phosphatase concentration (SAP) and the bone GLA-protein (BGP).

The suppression of secondary hyperparathyroidism (sHPT) would improve erythropoiesis, reduce haemolysis and correct myelofibrosis associated with renal osteodystrophy.

The aim of the study is to evaluate the influence of C on sc "uremic anaemia" in our patients and to rouse the interest for further study of the stated problem on a larger group of patients

## Patients and methods

Out of 214 patients treated with chronically repeated haemodialysis (Department of Nephrology, Faculty of Medicine, Skopje) only 36 (16.8%) patients (19 males and 17 females) with chronic kidney disease who were undergoing haemodialysis (mean age of 45.5±13.1 years) were treated with calcitriol (0.25μg every day or 0.50-1.50 μg every second day at a minimum of three months before testing). The duration of the haemodialysis treatment was from 9 to 157 months (X = 54.2 ± 38.1, CV = 70.3%). Two female patients have an artificial hip implant due to the pathologic intracapsular fracture of the femoral neck, and three more patients had been previously parathyroidectomized and received a transplant. The data of the studied patients are shown in Table 1.

Table 1

Basic data for analysed patients

| *Patient* | *Age (years)* | *Sex* | *Disease* | *HD-months* | *Total dose of Calcitriol (μg)* | *Comments* |
|---|---|---|---|---|---|---|
| ***AR*** | 33 | m | HTN/NaS | 40 | 305 | - |
| ***DN*** | 66 | f | PKD | 52 | 350 | - |
| ***KR*** | 25 | m | GN chr | 22 | 175 | |
| ***AA*** | 22 | m | GN RP | 72 | 215 | - |
| ***VV*** | 40 | m | T2DM | 36 | 135 | - |
| ***KS*** | 46 | m | IPN chr | **157** | **510** | - |
| ***AS*** | 55 | f | HTN/NaS | 24 | 190 | - |
| ***RD*** | 55 | m | PKD | 30 | 83 | - |
| ***JS*** | 23 | m | GN chr | 15 | 56 | - |
| ***NLj*** | 44 | f | TIN chr | 18 | 135 | - |
| ***IV*** | 75 | f | T2DM | 10 | 38 | - |
| ***RLj*** | 61 | f | T2DM | 30 | 135 | - |
| ***AV*** | 58 | f | HTN/NaS | 22 | 112 | - |
| ***MM*** | 37 | f | GN MP | 122 | 45 | PTHX/TR |
| ***EM*** | 49 | m | HTN/Nas | 37 | 215 | - |
| ***OV*** | 40 | m | ON | 48 | 255 | - |
| ***GM*** | 56 | f | ON | 37 | 66 | - |
| ***PN*** | 61 | f | TIN chr | 90 | 105 | F-ra coli fem. |
| ***PR*** | 57 | f | PKD | 84 | 315 | - |
| ***CA*** | 50 | f | GN chr | 88 | 45 | - |
| ***DD*** | 55 | m | HTN/Nas | 28 | 147 | - |
| ***NM*** | 45 | f | ON | 21 | 158 | - |
| ***MR*** | 51 | m | T2DM | 42 | 98 | - |
| ***LjF*** | 29 | f | GN chr | 30 | 115 | - |
| ***RD*** | 49 | m | PKD | 30 | 83 | - |
| ***SM*** | 46 | f | PKD | 131 | 38 | - |
| ***KS*** | 40 | m | IPN chr | 147 | 410 | PTHX/TR |
| ***SD*** | 30 | m | T2DM | 45 | 45 | - |
| ***ML*** | 52 | f | IPN chr | 59 | 30 | F-ra coli fem. |
| ***TS*** | 51 | f | HTN/NaS | 65 | 45 | - |
| ***RD*** | 46 | f | BEN | 59 | 90 | - |
| ***MV*** | 50 | m | BEN | 83 | 255 | - |
| ***GG*** | 24 | m | GN RP | **9** | 31 | - |
| ***BA*** | 52 | m | Gn RP | 36 | 46 | - |
| ***CE*** | 43 | m | GN chr | 48 | **16** | - |
| **BJ** | 21 | m | VUR bill | 84 | 45 | PTHX/TR |

Table 1 shows that the duration of haemodialysis, as well as the total dose of consumed medicine (calcitriol) in the analysed study period, varies considerably (from 16 to 510 μg; X = 142.7± 118.7 μg, CV = 83.2%). C has been administered in doses which should provide the inorganic serum phosphorous (Pi) below 2 mmol/L (0.25-0.50 μg/daily or 1.0-1.5 μg every second day orally). Analysing the files of the aforementioned subjects, it was observed that the patients had improved their blood count tests after treatment with C. We have compared the mean values of haemoglobin (Hgb g/L), red blood cells (Er, n $x10^{12}$), haematocrit (Htc, percentage), calcium (Ca, mmol/L), Pi (mmol/L), and serum alkaline phosphatase (SAP,U/L) during the last three months prior the onset of treatment with C – with the respective values (average for the last three months) in the course of calcitriol therapy.

Blood samples were taken as described immediately before haemodialysis, and the measurements were made by routine laboratory methods (Clinical Biochemistry, Faculty of Medicine, Skopje). The haemodialysis procedure (including time of dialysis and type of membrane), dietetic regimen, and the other medicines were not changed in comparison with the pretreatment period. All patients were haemodialyzed on the same membrane three times weekly. Blood transfusions were not used during the follow-up.

We calculated the mean value of the analysed series of data before and after treatment with C and have determined the relationship between them as well as the statistical significance of the differences related to these values. The blood count values changed significantly after therapy with C, namely an increase in the red blood cells number (more than $0.5x10^{12}$/L), concentration of Hgb (about 10 g/L or change for more than 10% ), and Htc (increase for about 0.05%). The values for total Ca (tCa; RV – 2.1-1.6 mmol/L), inorganic phosphorous (Pi; RV – 0.81-1.45 mmol/L) and alkaline phosphatase (RV-to 90 U/L) were measured as well.

The analysed group of patients also serves as a control. The patients have not been treated with despheroxamine, androgens, ultraviolet rays, different time of sun expositions, nor have additional ultrasonic investigations been made to find secondary (degenerative) cysts in the residual kidney parenchyma.

1,25 $(OH)_2D_3$ inhibits, indirectly (through ionic calcemia) and directly (through the cytosolic and nuclear receptors at the main parathyroid cells),

the production of PTH (pre/pro PTH sequence) and reduces its unfavourable effects on haematopoiesis (especially if the administration of calcitriol is done intravenously; for example, amp. Calcijex® (Abbot) à 1 μg/mL).

Calcitriol would influence the control of intra- and extramedullar haematopoiesis by the elimination of the peritrabecular fibrocystic myelofibrosis (blocking the PTH profibrotic effects), by reduction of the secondary hypersplenism and by stimulation of testosterone secretion, which stimulates the production of erythropoietin (EPO).

## Results

Table 2 presents the findings of the study:

Table 2

Values of Er, Hgb, Htc, Ca, Pi, and SAP in the studied patients before and after treatment with Calcitriol

| *Patient* | *RBC ($x10^{12}$/L) b/a* | *Hgb (g/L) b/a* | *Htc b/a* | *tCa (mmol/L) b/a* | *Pi (mmol/L) b/a* | *SAP (U/L) b/a* |
|---|---|---|---|---|---|---|
| *AR* | 2.3/2.4 | 80/79 | 0.21/0.22 | 1.9/2.0 | 1.4/1.1 | 68/66 |
| *DN* | 2.4/3.2 | 88/101 | 0.24/0.31 | 1.8/1.9 | 2.1/1.8 | 116/120 |
| *KR* | **1.6**/**2.0** | **51**/79 | **0.07**/0.19 | 2.2/1.9 | 1.6/1.8 | 698/741 |
| *AA* | 2.6/2.9 | 91/87 | 0.26/0.26 | **1.6**/**1.5** | 2.4/2.1 | **1000/1270** |
| *VV* | 2.6/2.3 | 85/**74** | 0.23/0.22 | 2.2/2.3 | 1.4/1.2 | 343/108 |
| *KS* | 2.5/2.9 | 89/89 | 0.26/0.28 | 2.3/2.5 | 1.3/1.2 | 233/257 |
| *AS* | 2.6/3.0 | 77/82 | 0.26/0.28 | 1.8/2.0 | 0.9/**0.7** | 120/110 |
| *RD* | 2.9/3.4 | 96/89 | 0.29/0.31 | 2.3/2.5 | 1.3/1.2 | 261/223 |
| *JS* | 2.2/2.8 | 78/94 | 0.21/0.28 | 1.7/1.9 | **3.1**/2.9 | 172/129 |
| *NLj* | 2.6/3.1 | 88/102 | 0.24/0.32 | 1.8/1.9 | 1.6/1.8 | 425/341 |
| *IV* | 2.6/2.2 | 88/102 | 0.24/0.32 | 2.3/2.2 | 1.4/1.2 | 101/87 |
| *RLj* | 2.9/3.8 | 77/99 | 0.22/0.31 | 2.4/2.1 | 1.5/1.4 | 235/190 |
| *AV* | 2.8/3.0 | 95/104 | 0.25/0.28 | 2.0/1.7 | 1.6/1.6 | 369/328 |
| *MM* | 3.0/**4.5** | 68/**127** | 0.29/**0.44** | 2.3/2.2 | 2.3/2.5 | 93/99 |
| *EM* | 3.4/3.2 | 95/92 | 0.29/0.29 | 2.2/2.0 | 1.7/1.8 | 205/174 |

| | | | | | | |
|---|---|---|---|---|---|---|
| ***OV*** | 3.4/3.5 | 85/86 | 0.26/0.27 | 1.9/2.3 | 1.2/1.1 | 432/432 |
| ***GM*** | 4.1/**4.5** | **113**/117 | **0.37**/0.41 | 2.0/2.0 | 1.3/1.5 | 132/147 |
| ***PN*** | 3.2/4.0 | 98/139 | 0.33/0.49 | **1.6**/2.1 | 1.6/2.2 | 81/74 |
| ***PR*** | 2.9/3.0 | 101/101 | 0.20/0.30 | 1.7/1.9 | 1.7/1.6 | 78/138 |
| ***CA*** | 2.1/2.8 | 79/96 | 0.23/0.28 | 2.1/2.0 | 1.6/1.6 | 251/269 |
| ***DD*** | 2.1/2.4 | 82/105 | 0.19/0.21 | 2.4/2.4 | 1.5/1.2 | 132/1304 |
| ***NM*** | 3.1/3.6 | 87/112 | 0.24/0.32 | 2.0/2.1 | 1.8/1.9 | 462/204 |
| ***MR*** | **4.3**/**4.5** | 109/114 | 0.36/0.39 | 2.2/2.2 | 1.6/1.7 | 608/543 |
| ***LjF*** | 2.8/4.2 | 68/118 | 0.19/0.35 | 2.0/1.9 | 2.4/1.5 | 970/254 |
| ***RD*** | 3.2/3.4 | 94/111 | 0.33/0.34 | 1.6/2.3 | 1.9/1.7 | 116/79 |
| ***SM*** | 2.6/2.5 | 68/67 | 0.23/**0.20** | 1.7/2.0 | 1.5/**3.1** | 91/99 |
| ***KS*** | 2.6/3.5 | 90/**127** | 0.28/0.42 | 1.7/1.9 | 3.0/2.7 | 141/408 |
| ***SD*** | 3.2/4.0 | 98/117 | 0.34/0.39 | 2.3/2.3 | 2.0/1.5 | 67/53 |
| ***ML*** | 2.0/2.2 | 69/76 | 0.21/0.22 | **1.6**/**2.6** | 2.5/2.1 | 114/191 |
| ***TS*** | 2.0/3.9 | 80/110 | 0.20/0.35 | 2.0/2.4 | 1.9/2.1 | 160/278 |
| ***RD*** | 1.8/3.7 | 61/125 | 0.15/0.40 | **2.5**/2.0 | **0.7**/0.9 | 179/144 |
| ***MV*** | 2.3/2.4 | 72/71 | 0.20/0.26 | 2.4/1.9 | 1.9/1.6 | 104/101 |
| ***GG*** | 2.2/2.4 | 80/67 | 0.20/0.20 | 2.3/2.0 | 2.8/2.5 | **35**/**50** |
| ***BA*** | 2.1/2.7 | 83/100 | 0.23/0.26 | 2.1/2.0 | 1.8/2.4 | 68/56 |
| ***CE*** | 2.2/2.3 | 84/87 | 021/0.22 | 1.7/1.8 | 2.8/2.7 | 68/112 |
| ***BJ*** | 1.8/3.5 | 57/98 | 0.16/0.29 | 1.8/2.0 | 1.6/1.3 | 812/113 |
| **X ± SD Coefficient of Variation (%)** | 2.6 ± 0.6 (23.2) / 3.2 ± 0.7 (22.5) | 83.4 ± 13.6 (16.3) / 98.4 ± 18.1 (18.3) | 0.24 ± 0.1 (25.0) / 0.30 ± 0.1 (23.3) | 2.0 ± 0.3 (13.9) / 2.1 ± 0.2 (11.5) | 1.8 ± 0.6 (31.1) / 1.8 ± 0.6 (33.0) | 265.0 ± 257.1 (97.0) / 258.1 ± 293.4 (113.7) |
| $\sigma_{X1-X2}$ **(p)** | < 0.03 | < 0.02 | < 0.03 | NS | NS | NS |

∗-the extremes are bolded

From the results presented in table 2, it is obvious that the statistical difference in RBC (red blood cells), Hgb (Haemoglobin) and Htc (Haematocrit) values before and after treatment with C is significant and clinically acceptable. This is not, however, also the case for tCa, Pi and SAP. The

distribution of the statistical units and data around the average value (X) ranges between 11.5% to 33.0% for RBC, Hgb, Htc, tCa, and Pi (values before and after treatment with C) and with much more scatter (CV = 97.0% - 113.7%) for SAP, the changes in the serum concentration of calcitriol and Pi as well as the activity of SAP.

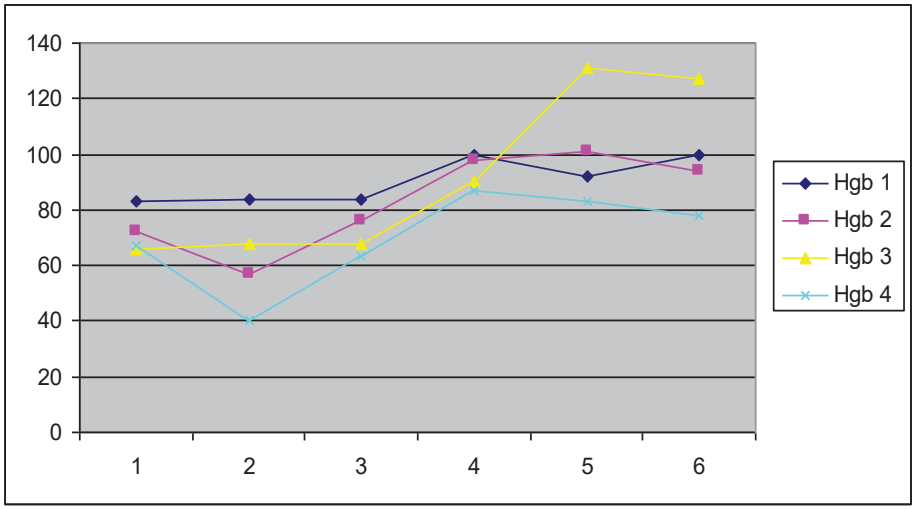


Figure 1 – Haemoglobin variations after three months treatment with calcitriol in four of our patients

From the fig 1 one could conclude that the concentrations of Hgb generally rise about three (3) months after the initiation of the C therapy.

In order to evaluate the direct association between the antianemic action of C and possible suppression of PTH secretion (indirectly, by the reduction of the total activity of SAP), we associated the percentage of change of SAP with the percentage variation of Hgb-emia in the group of analysed patients. We have found a weak but negative linear correlation ($r = -0.15$, $y = -0.25x + 20.1$). This finding leads to the conclusion that even a lesser degree of SAP activity diminution corresponds to a higher concentration of Hgb in the blood (mean percentage change for SAP = $11.6 \pm 59.6\%$; corresponding variation for Hgb = $33.6 \pm 34\%$).

## Discussion

Anaemia in patients with end-stage kidney disease is a nosocomial problem with multifactorial etiopathogenesis.(1),(2),(3)

Associating uremic anaemia with sHPT has been a subject of discussion over the past forty years, despite being very complex and controversial. PTH, the potential uremic toxin, is related with the inhibition of erythropoiesis (especially at the level of BFU-erythroid precursors).(4)

Because the failure of the function of $Na^+/K^+$-ATP-ase with reduced utilization of carbohydrates in the presence of PTH, the fragility of osmotic erythrocytes increases, and there is a tendency to haemolysis. With the reduction of the thrombocytes' function (mechanism dependent of calcium), there is an increase in the inclination of gastroduodenal haemorrhage, resulting in iron-deficiency anaemia. Considering the positive correlation between the serum PTH and stomach hyperchlorhydria, which is associated with ulcerations and/or bleeding of the upper digestive tract, microcytic iron-deficiency anaemia is quite acceptable. (5)

PTH stimulates general protein catabolism, thus reducing the globin synthesis in the haemoglobin structure, although there are a lot of contradictions when comparing the laboratory and clinical findings.

When PTH and sHPT process the fibrocystic osteodysplasia the bone marrow undergoes myelofibrosis and calcification. It certainly reduces the erythroid potential of the bone marrow and precedes hypo or aregenerative anaemia. The favourable effect of PTHX-ia on uremic anaemia supports the previous position. The hypocalcemic /hypophosphatemic shape of sHPT (hypomineralizing hyperosteoidosis) develops a strong fibrous reaction in the bone marrow, which is especially suitable for C treatment.(6)

1,25 $(OH)_2$ $D_3$, indirectly (through ionic calcemia) and directly (through the cytosolic and nuclear receptors at the main parathyroid cells), inhibits production and incretion of PTH and reduces its unfavourable effects on haematopoiesis (especially if administered intravenously). (7)

C further influences uremic anaemia favourably by controlling the intramedullary haematopoiesis and spleen RBC sequestration. This is done by the elimination of fibrocystic myelofibrosis, stimulation of testosterone secretion in men (which in turn stimulated the EPO production), and the reduction of secondary hypersplenism. (8)

In addition to the classic effects of C (synthesis of the vitamin D-dependent calcium-binding protein s.c.Ca-BPs and stimulation of Ca-absorption), C increase the absorption of Pi in the distal kidney tubules (calcitriol receptors for the $C_1$-OH group).(9),(10).

Using the techniques of monoclonal antibodies, the presence of specific calcitriol receptors is described in various tissues and organs (fibroblast, hypophysis, beta-cells of the pancreas islets, kidneys, gonads, skeleton muscles, parathyroid glands, gastro-intestinal tract, heart, thymocytes, haematolymphoid, and malignant tissues).(11)

C stimulates the transformation of blood monocytes in tissue macrophages, inhibits the proliferation of renal epithelioma cells, modulates the reaction of target cells according to the activity of specific hormones (influence on cAMP synthesis), induces expression of prolactin gen, and stimulates the production of thrombocytic thromboxane ($TxA_2$).(12),(13). C reduces the sensitivity of osteoblasts to the influence of PTH and indirectly reduces the synthesis of BGP (bone GLa protein) and SAP.(14)

C influences insulin secretion by a feedback link increasing the concentration of hepatocyte cytosolic Ca and regulates, or rather, reduces the production of $25(OH)D_3$ (calcifediol/calcidiol, as a circulating reserve for calcitriol).(15) There is a great deal of information about the effect of C on the biotransformation of androstenedione in estron, possessing immunosuppressive properties. There is also data that show how it is also efficient in the treatment of idiopathic myelofibrosis (re-expansion of the bone marrow).(16),(17),(18)

Taking the above statements into consideration, for the present time still questionable, the participation of other, we can purport other PTH non-associated effects of C on uremic anaemia can be also supposed. (19), (20), (21)

## REFERENCES


1. Wills MR. Uremic Toxins, and Their Effect on Intermediary Metabolism Clin Chem. 1985; 31/1: 5-13.
2. Eschbach JW,Adamson JW. Anaemia of end-stage renal disease. Kidney Intern. 1985; 26: 1-5.

3. Zappacosta AR,Caro J,Erlev A. Normalization of haematocrit in patients with end-stage renal disease on CAPD. Am J Med. 1982; 72: 53-57.
4. Drüeke TB,Eckardt KU. Role of secondary hyperparathyroidism in erythropoietin resistance of chronic renal failure patients. Nephrol Dial Transplant. 2002; 17 Suppl 5: 28-31.
5. Bogin E,Massry SG,Levi j et al. Effects of PTH on osmotic fragility of human erythrocytes. J Clin Invest. 1982; 69: 1017.
6. Barbour GL. Effect of parathyroidectomy on anaemia in chronic renal failure. Arch Intern Med. 1979 Aug; 139(8): 889-91.
7. Hasnain M, Hauncher C, Pegoraro AA, Arruda JA, Dunea G. Suppression of hyperparathyroidism by calcitriol therapy. ASAIO J. 1999 Sep-Oct; 45(5): 424-7.
8. Nomura S, Ogawa Y, Osawa G, Katagiri M, Harada T, Nagahana H. Myelo-fibrosis secondary to renal osteodystrophy. Nephron. 1996; 72(4): 683-687.
9. Bikle DD. Vitamin D Metabolism, Mechanism of Action, and Clinical Applications. Chemistry&Biology. 2014; 21: 319-329.
10. Merke J,Ritz E,Roland R. Are recent findings on 1.25-dihydroxycholecalciferol metabolism relevant for the pathogenesis of uremia? Nephron. 1986; 42: 277-284.
11. Minghetti PP, Norman AW. 1,25(OH)2-vitamin D3 receptors: gene regulation and genetic circuitry. FASEB J. 1988 Dec; 2(15): 3043-53.
12. Sperschneider H, Humbsch K, Abendroth K.Oral calcitriol pulse therapy in hemodialysis patients. Effects on histomorphometry of bone in renal hyperparathyroidism. Med Klin (Munich). 1997; 92(10): 597-6.
13. Kassi E,Adamopoulos Ch,Basdra EK,Papavassiliou AG. Contemporary Reviews in Cardiovascular Medicine: Role of Vitamin D in Atherosclerosis. Circulation. 2013; 128: 2517-2531.
14. Motlagh BM, Ahangaran NA, Froushani SMA. Calcitriol modulates the effects of bone marrow-derived mesenchymal stem cells on macrophage functions. Iran J Basic Med Sci. 2015; 18: 672–676.
15. Inoue D, Matsumoto T, Ogata E, Ikeda K.22-Oxacalcitriol, a noncalcemic analogue of calcitriol, suppresses both cell proliferation and parathyroid hormone-related peptide gene expression in human T cell lymphotropic virus, type I-infected T cells. J Biol Chem. 1993 Aug 5; 268(22): 16730-6.
16. Patel S, Simpson RU, Hsu CH. Effect of vitamin D metabolites on calcitriol metabolism in experimental renal failure. Kidney Int. 1989; 36(2): 234-9.

17. Kerr DNS. Hypercalcemia and metastatic calcification. Cardiovascular Res-earch. 1997; 36: 293-297.
18. Andress DL, Norris KC, Coburn JW, Slatopolsky EA, Sherrard DJ. Intravenous calcitriol in the treatment of refractory osteitis fibrosa of chronic renal failure. N Engl J Med. 1989 Aug 3; 321(5): 274-279.
19. Muscogiuri G. et al. Mechanisms in endocrinology: Vitamin D as a potential-al contributor in endocrine health and disease. European Journal of Endo-crinology. 2014; 171: 101–110.
20. Erman A, Raz A. Effects of bivalent cations on prostaglandin biosynthesis and phospholipase A2 activation in rabbit kidney medulla slices. Biochem J. 1979; 182: 821-825.
21. Panichi V, Andreini B, De Pietro S, Migliori M, Taccola D, Giovannini L, Ferdeghini M, Palla R. Calcitriol oral therapy for the prevention of secondary hyperparathyroidism in patients with predialytic renal failure. Clin Nephrol. 1998; 49: 245-250.

Ирена ЧАКАЛАРОСКА, Лада ТРАЈЧЕСКА, Нинослав ИВАНОВСКИ, Живко ПОПОВ, Кочо ЧАКАЛАРОСКИ

## ВЛИЈАНИЕ НА КАЛЦИТРИОЛ ВРЗ АНЕМИЈА КАЈ ХЕМОДИЈАЛИЗНИ ПАЦИЕНТИ

### Резиме

Анализирани се 36 хемодијализни пациенти третирани со орален калцитриол. Пациентите примале од 23 до сса 518 μg калцитриол (вкупна доза) со дневна nozologija од 0,25 до 0,50 μg секој втор ден, со цел да се спречи метаболичката бубрежна остеопатија (porano позната како „бубрежна остеодистрофија“, ROD). Во 30 / 36 (83,3%) пациенти, откривме значително зголемување на концентрацијата на хемоглобин во крвта (Hgb) (повеќе од 10%) по третманот со калцитриол. Сите постапки asocirani so хемодијализа, диететски мерки и други медицински третмани останаа непроменети во споредба со периодот на пред-третман.

Откривме дека орално администрираниот калцитриол е исто така ефикасен во терапијата на уремична анемија. Потребни се подетални студии so поголем број пациенти за да се donese конечен заклучок.

**Клучни зборови:** уремична анемија, PTH, калцитриол.