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SECONDARY AND TERTIARY HYPERPARATHYROIDSIS IN CHRONIC KIDNEY DISEASE

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Abstract: Treatment goals for secondary hyperparathyroidism in patients with chronic kidney disease are aimed at preventing disease progression and suppressing parathyroid activity through modulation of vitamin D receptors and calcium-sensing receptors. However, treatment options are limited in severe hyperparathyroidism; monoclonal changes and nodular transformation of glands with loss of receptors for vitamin D and calcium form resistance to this therapy with the development of drug-uncontrolled hyperparathyroidism.

Keywords: Clinical case; secondary hyperparathyroidism; tertiary hyperparathyroidism; cinacalcet; parathyroidectomy; hemodialysis; kidney transplant.

INTRODUCTION

Chronic kidney disease (CKD) today is considered as a general medical problem, and not just a nephrological problem. Renal replacement therapy (dialysis) and kidney transplantation are considered complementary treatments for end-stage CKD. In CKD, a decrease in the number of active nephrons causes a cascade of complications, including vitamin D deficiency, hyperphosphatemia, changes in the function of the calcium-sensing receptor (CSR) of the parathyroid glands (PSG), decreased absorption of calcium from the gastrointestinal tract, increased synthesis of parathyroid hormone (PTH) and hypertrophy with hyperplasia of PTG cells.

MATERIALS AND METHODS

In most patients, with timely initiation of therapy, secondary hyperparathyroidism (SHPT) can be controlled by dietary restrictions on phosphorus content and pharmacotherapy (phosphate-lowering drugs, oral or parenteral vitamin D preparations, calcimimetics) [1]. However, despite the progress achieved in conservative treatment and the presence of clearly developed criteria for its prescription and monitoring, there remains a cohort of patients resistant to this therapy. In these patients, even after successful kidney transplantation, normalization of mineral-bone disorders does not occur and persistence of hyperparathyroidism is observed with the development of tertiary hyperparathyroidism with hypercalcemia and hypophosphatemia.

In 2023, patient M., born in 1966, applied. with stage 5 CKD, on renal replacement therapy (programmed hemodialysis).

At the time of treatment, the patient complained of muscle weakness, pain in bones and joints, “stiffness” of the whole body when moving, and itchy skin.

From the anamnesis it is known that in 1976 he was diagnosed with Periodic Disease, in 1988 - renal amyloidosis (according to biopsy). In 2006, stage 5 CKD was diagnosed and renal replacement therapy with program hemodialysis was started.

The above complaints began to be a concern in 2018; upon examination, mineral and bone disorders in CKD were diagnosed: secondary hyperparathyroidism with a PTH level of 634 pg/ml. Since that time, he has been receiving active metabolites of vitamin D (Alfacalcidol 4 mcg per week) irregularly.

RESULTS AND DISCUSSION

Based on the examination, therapy was recommended aimed at [2]:

1. correction of secondary hyperparathyroidism – cinacalcet 30 mg per day;

2. reduction of hyperphosphatemia - a strict hypophosphate diet (up to 800 mg per day) and lengthening the program hemodialysis procedure;

3. maintaining the level of calcium in the target range in accordance with the American Clinical Practice Guidelines for Bone Metabolism and Skeletal Pathology in Chronic Kidney Disease (KDOQI, 2.13–2.37 mmol/l) – reducing the calcium concentration in dialysis solution up to 1.25 mmol/l.

The levels of PTH, serum calcium with albumin and phosphorus were monitored monthly.

During the observation period:

1. the dose of cinacalcet was titrated to 60 mg per day;

2. after normalization of phosphorus levels, Colecalciferol 50,000 IU per week was added to therapy to correct vitamin D deficiency;

3. only after correction of vitamin D deficiency (level 36 ng/ml) and subject to normocalcemia, normophosphatemia, and PTH level above the target range according to KDOQI 2003 (more than 300 pg/ml), alfacalcidol was added to therapy in order to correct SHPT 1 mcg 3 times a week.

CONCLUSION

The above clinical case clearly demonstrates the complexity of managing patients with mineral-bone disorders in chronic kidney disease.

At the dialysis stage of CKD, drug therapy for SHPT with vitamin D preparations and a calcimimetic showed significant positive dynamics with normalization of phosphorus-calcium metabolism parameters; improvement of the course of hyperparathyroid renal osteodystrophy with restoration of BMD and a decrease in markers of bone metabolism. Against the background of an integrated approach, the reverse development of hyperplasia and/or hypertrophy of cells of three parathyroid glands was also revealed; the fourth gland, on the contrary, increased in volume, which indirectly indicated nodular transformation of parathyroid cells and monoclonal growth. After successfully treated SHPT at the dialysis stage and adequate functioning of the renal graft after transplantation, cases of hypercalcemia developing against the background of persistent SHPT are relatively rare. Our clinical case is an example of the transition of SHPT, successfully treated before kidney transplantation, into TGPT after kidney allotransplantation, which indicates the importance of assessing not only laboratory parameters, but also the volume of the PTG before kidney transplantation. Despite the stabilization of calcemia and a tendency to decrease PTH levels during the use of a calcimimetic, a decision was made to perform

parathyroidectomy due to suspected nodular hyperplasia of PTG cells, which was confirmed histologically.

REFERENCES

1. Abe M, Okada K, Soma M. Mineral Metabolic Abnormalities and Mortality in Dialysis Patients. Nutrients. 2013;5(3):1002-1023. doi: 10.3390/nu5031002.

2. Goodman WG, Quarles LD. Development and progression of secondary hyperparathyroidism in chronic kidney disease:

lessons from molecular genetics. Kidney Int. 2018;74(3):276-288. doi: 10.1038/sj.ki.5002287.

3. Chertow GM, Dillon M, Burke SK, et al. A randomized trial of sevelam-er hydrochloride (RenaGel) with and without supplemental calcium. Strategies for the control of hyperphosphatemia and hyperparathyroidism in hemodialysis patients. Clin Nephrol. 2019;51(1):18-26.

4. Chertow GM, Lu ZJ, Xu X, et al. Self-reported symptoms in patients on hemodialysis with moderate to severe secondary hyperparathyroidism receiving combined therapy with cinacalcet and low-dose vitamin D sterols. Hemodialysis International. 2012;16(2):188-197. doi: 10.1111/j.1542-4758.2011.00642.x.