Peertechz

Clinical Group

Archives of Renal Diseases and Management

ISSN: 2455-5495 DOI CC By

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Dates: Received: 29 April, 2017; **Accepted:** 15 May, 2017; **Published:** 17 May, 2017

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Letter to Editor

Nephrolithiasis in hypercalcemic and normocalcemic primary Hyperparathyroidism

Dear Editor

Primary hyperparathyroidism (PHPT) is a common condition affecting up to 4 per 1000 of population, and the majority of cases are due to adenoma or hyperplasia of the gland [1]. This endocrine disorder may either develop without symptoms or be classically manifested by anorexia, nausea, constipation, polydipsia and polyuria in association with hypercalcemia [1]. Worthy of note is the form of PHPT described in 2009 and characterized by normocalcemia, which persists with hypercalciuria, nephrolithiasis and bone loss in the majority of the cases[1]. Nephrolithiasis can complicate 20% of hypercalcemic and 18.2% of normocalcemic PHPT; moreover, calcium oxalate and calcium phosphate are the main components of the calculi [1]. Thiazide diuretics, lithium salt, immobilization, and inactivity; in addition to hyperoxaluria, hypocitraturia, low urinary pH, high salt ingestion, and low water intake play adverse roles [1]. Cinacalcet hydrochloride is a calciomimetic drug that can activate parathyroid and renal calcium-sensing receptors suppressing synthesis/ secretion of PTH, and reducing calcemia[1]. This drug is also used to treat secondary hyperparathyroidism in people with kidney failure [1].

The research by Berardi and Duranti focused the mechanisms of nephrolithiasis in patients with PHPT; and the polymorphisms of nucleotides in exon 7 of the CaSR gene [1]. The authors compared biochemical data of PHPT patients with and without nephrolithiasis and found hypercalciuria in 39% of the total, whereas 29% had hypercalciuria and calculi [1]. The best option for management of nephrolithiasis due to hypercalcemic or normocalcemic PHPT in people who cannot or do not wish to undergo parathyroidectomy is normal calcium diet and cinacalcet; in addition to alendronate that improves bone mass and reduces the calciuria [1]. They commented the special attention to be paid about possible strong responses in carriers of Arg990Gly polymorphism, and the role of better adjustments of cinacalcet dosage with daily dietary calcium, to maintain the levels both of PTH and calcemia within normal ranges[1]. Relationships between PTH, vitamin D and calcemia were also included because vitamin D deficiency is common in this group of individuals due to renal conversion of 25-OH vitamin D to 1,25-dihydroxy vitamin D, which is the active form of this vitamin[1,2]. The lowering of calcemia stimulates calcium-sensing receptors of parathyroid, originating increases of PTH levels, bone resorption and renal synthesis of 1,25-dihydroxy vitamin D [1,2]. Increased intestinal absorption of calcium and phosphorus as well as mobilization of bone calcium tends to normalize the blood calcium levels. This propitiates increased renal losses of phosphorus and reduces PTH secretion, inhibiting the hydroxylation of 25-OH vitamin D [1,2]. Hypercalcemia leads to decreased blood levels of PTH, increased calciuria, reduction of 1,25-hydroxy vitamin D and lower calcium bone loss, which results in normalization of calcemia [1,2]. Deficiency of vitamin D may be related to hungry bone syndrome after parathyroidectomy; but supplementation of this vitamin can cause hypercalcemia, hypercalciuria, and lithiasis [1]. Seasonal factors, sun exposure, and diet have influence on parameters of hypovitaminosis D, and low 25(OH) D levels in winter are associated with rising PTH levels and bone resorption [2]. Additional concerns involve secondary HPT, developing with 25(OH)D levels lower than 32 ng/mL [2].

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Citation: dos Santos VM (2017) Nephrolithiasis in hypercalcemic and normocalcemic primary Hyperparathyroidism. Arch Renal Dis Manag 3(1): 006-006. DOI: http://doi.org/10.17352/2455-5495.000018