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Journal of Parathyroid Disease

Journal of Parathyroid Disease 2014,2(1),7–8

Epidemiology and Prevention

Primary hyperparathyroidism and kidney; recent findings

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Disorders of the parathyroid glands most commonly present with abnormalities of serum calcium (1). Individuals with primary hyperparathyroidism as the the most common cause of hypercalcemia in outpatients, are frequently asymptomatic or may have bone disease, neuromuscular symptoms or nephrolithiasis (1-3). Patients with chronic renal failure may find secondary hyperparathyroidism with resultant chronic kidney disease-mineral and bone disorder (1,2). Assessment of patients with abnormal serum calcium levels includes a history and physical examination; repeat measurement of serum calcium level, and assessment of vitamin D, creatinine, magnesium and parathyroid hormone levels (1-4). It is accepted that, the treatment for symptomatic primary hyperparathyroidism is parathyroidectomy (2-4). Managing of asymptomatic primary hyperparathyroidism includes monitoring symptoms, serum creatinine and calcium levels and also bone mineral density. The name primary hyperparathyroidism denotes to the improper overproduction of the parathyroid hormone conducting to abnormal calcium homeostasis. High levels of parathyroid hormone lead to increased synthesis of 1,25(OH)2D (which increases intestinal calcium absorption), increased kidney resorption of calcium, increased resorption of the bone and phosphaturia (1-5). The classical clinical manifestation of primary hyperparathyroidism is the 'stone and bone' disease. Kidney manifestations of primary hyperparathyroidism include nephrocalcinosis, hypercalciuria, nephrolithiasis, chronic kidney disease, and kidney tubular dysfunction. Development of kidney failure in primary hyperparathyroidism was related to the extent and duration of hypercalcemia (1-7). The parathormone inhibits proximal tubular bicarbonate reabsorption, which tends to cause mild metabolic acidosis named proximal tubular acidosis. Nonetheless, this consequence is usually counterbalanced by the alkali released as a result of amplified bone resorption and tubular reabsorption of the bicarbonate, caused by hypercalcemia (1-4,7,8). On the other hand, the filtered load of calcium in the glomeruli increases proportionately with the quantity of serum hypercalcemia (3-8). Hypercalciuria is one of obvious factors in the complex pathophysiology of kidney stone formation. Most kidney stones in patients with primary hyperparathyroidism are comprised of calcium oxalate, although slightly alkaline urine may favor the precipitation of calcium phosphate stones. Stone producers are more likely to be hypercalciuric, but less than one-third of the hypercalciuric patients with primary hyperparathyroidism actually develop kidney stones (2-8). Currently, it is not possible to confidently predict which asymptomatic patients with primary hyperparathyroidism would develop a new onset of kidney stone disease, based on the biochemical measurements in the blood or urine including hypercalciuria (4-11). Male gender, young age, and high 1,25(OH)2D have been related to an increased risk of kidney stones. It is assumed that at a younger age, there is a relatively more viable kidney mass, and therefore, lower serum phosphorus. All these factors result in relatively higher 1,25(OH)2D levels, leading to increased intestinal calcium absorption which named as absorptive hypercalciuria. It was suggested that stage 3 of renal insufficiency is an indication for parathyroid adenomectomy (6-12).

Implication for health policy/practice/research/medical education

The name primary hyperparathyroidism denotes to the improper overproduction of the parathyroid hormone conducting to abnormal calcium homeostasis. High levels of parathyroid hormone lead to increased synthesis of $1,25(OH)_2D$ (which increases intestinal calcium absorption), increased kidney resorption of calcium, increased resorption of the bone and phosphaturia. The classical clinical manifestation of primary hyperparathyroidism is the 'stone and bone' disease. Kidney manifestations of primary hyperparathyroidism include nephrocalcinosis, hypercalciuria, nephrolithiasis, chronic kidney disease, and kidney tubular dysfunction.

Author's contribution

AB is the single author of the paper.

Received: 10 May 2013, Accepted: 27 January 2014, ePublished: 7 February 2014

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Conflict of interests

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

Funding/Support

None.

References

1. Pashtan I, Grogan RH, Kaplan SP, Devon K, Angelos P, Liu D, *et al*. Primary hyperparathyroidism in adolescents: the same but different. Pediatr Surg Int 2013; 29(3): 275-9.
2. Walker MD, Nickolas T, Kepley A, Lee JA, Zhang C, McMahon DJ, *et al*. Predictors of Renal Function in Primary Hyperparathyroidism. J Clin Endocrinol Metab 2014: jc20134192.
3. Baradaran A, Nasri H. Correlation of serum parathormone with hypertension in chronic renal failure patients treated with hemodialysis. Saudi J Kidney Dis Transpl 2005; 16(3): 288-92.
4. Grube M, Bech JN, Pedersen EB. Primary hyperparathyroidism as a cause of chronic renal failure. Ugeskr Laeger 2012; 174(8): 502-3.
5. Nasri H, Baradaran A. Long-lasting advanced primary hyperparathyroidism associated with end-stage renal failure in a diabetic patients. Acta Med Iran 2004; 42(6): 461-6.
6. Nasri H, Baradaran A. Secondary hyperparathyroidism in association with malnutrition-inflammation complex syndrome in chronic hemodialysis. Ann King Edward Med Coll 2005; 11(3): 301-6.
7. Nasri H, Baradaran A, Naderi AS. Close association between parathyroid hormone and left ventricular function and structure in end-stage renal failure patients under maintenance hemodialysis. Acta Med Austriaca 2004; 31(3): 67-72.
8. Baradaran A, Nasri H. Intensification of anaemia by secondary hyperparathyroidism in hemodialysis patients. Med J Islam Acad Sci 2001; 14:4, 161-6.
9. Baradaran A, Nasri H. Correlation of serum magnesium with serum parathormone levels in patients on regular hemodialysis. Saudi J Kidney Dis Transpl 2006; 17(3): 344-50.
10. Nasri H, Baradaran A, Doroudgar F, Ganji F. Relationship of conjunctival and corneal calcification with secondary hyperparathyroidism in hemodialysis patients. Iran J Med Sci 2003; 28(2): 86-9.
11. Salem MM. Hyperparathyroidism in the hemodialysis population: a survey of 612 patients. Am J Kidney Dis 1997; 29(6): 862-5.
12. Walker MD, Dempster DW, McMahon DJ, Udesky J, Shane E, Bilezikian JP, *et al*. Effect of renal function on skeletal health in primary hyperparathyroidism. J Clin Endocrinol Metab 2012; 97(5): 1501-7.

Please cite this paper as: Baradaran A. Primary hyperparathyroidism and kidney; recent findings. J Parathyr Dis 2014; 2(1): 7-8.