Disorders of the parathyroid glands most commonly present with abnormalities of serum calcium (1). Individuals with primary hyperparathyroidism as 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 hypercalcemia (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).

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References

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