

# Recurrent Nephrolithiasis Secondary to Primary Hyperparathyroidism- A Diagnostic Dilemma

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## Citation

O Chan, H Aw, C Li Wai Suen, D Spernat, S Appu. *Recurrent Nephrolithiasis Secondary to Primary Hyperparathyroidism- A Diagnostic Dilemma*. The Internet Journal of Urology. 2012 Volume 9 Number 2.

## Abstract

Nephrolithiasis is common and can affect up to 10–15% of the population during their lifetime [13]. Following an episode of ureteric colic, the recurrence rate at 5 years ranges from 30 to 50%. [3] While recurrent nephrolithiasis may be idiopathic, it may also be secondary to metabolic or endocrinological causes such as hyperparathyroidism. We present the case of an elderly woman who suffered from recurrent nephrolithiasis for 40 years and was subsequently diagnosed with primary hyperparathyroidism secondary to a parathyroid adenoma

## INTRODUCTION

Nephrolithiasis is common and can affect up to 10–15% of the population during their lifetime [13]. Following an episode of ureteric colic, the recurrence rate at 5 years ranges from 30 to 50%. [3] While recurrent nephrolithiasis may be idiopathic, it may also be secondary to metabolic or endocrinological causes such as hyperparathyroidism.

We present the case of an elderly woman who suffered from recurrent nephrolithiasis for 40 years and was subsequently diagnosed with primary hyperparathyroidism secondary to a parathyroid adenoma.

## CASE SUMMARY

A 67-year-old female patient of Asian descent presented to our urology outpatient clinic with recurrent ureteric calculi. She reported a 40-year history of intermittent bilateral loin pain and haematuria secondary to renal calculi, and required multiple surgical interventions.

Her past medical history included chronic hepatitis B and cholecystectomy. She was not on any regular medication. Despite her long-history of recurrent calculi, no metabolic screen, including serum calcium and phosphate, had ever been performed.

Clinical examination revealed a well-looking patient with mild right loin tenderness. Her physical examination was otherwise unremarkable. Specifically there were no palpable masses or bony tenderness. Her vital signs were within normal range.

She had normal renal function, with an eGFR of >90. A non-contrast CT of the renal tracts showed a 4.5mm obstructing calculi in the left vesicoureteric junction (See Fig 1.).

## Figure 1

Fig 1. CT scan of renal tract showing calculi at left vesicoureteric junction



She had a recent serum calcium level, which was elevated at 2.67mmol/l (normal: 2.20-2.60). 24-hour urine collection showed significant elevation of urinary calcium excretion at 10.29mmol/day (normal: 2-7.5). More detailed biochemical analysis showed an elevated parathyroid hormone level (PTH) of 11.2 pmol/L (normal: 1.5-7.0).

On thyroid ultrasound, a nodule was found posterior to the right lobe measuring 4x5x7 mm with features suggestive of a parathyroid adenoma (See Fig 2).

**Figure 2**

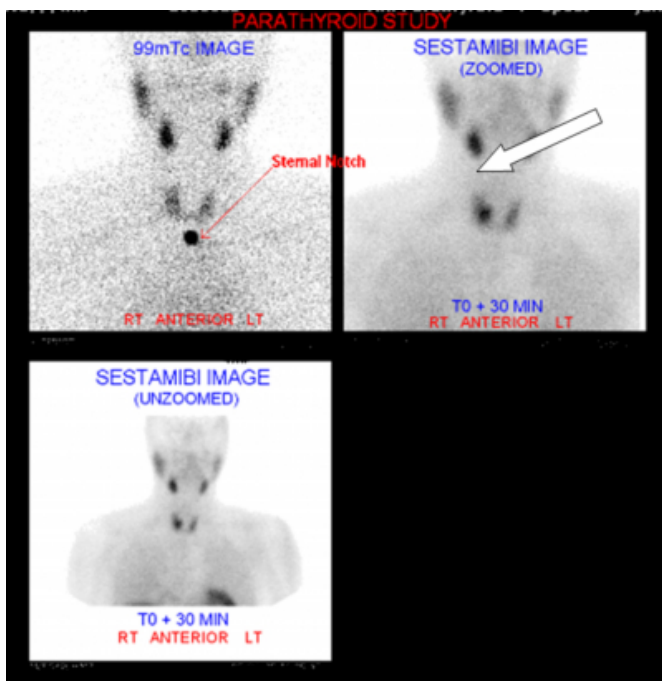
Fig 2. Ultrasound with findings suggestive of a parathyroid adenoma



A Sestamibi scan using Technetium 99-m was performed. Initial images demonstrated normal thyroid uptake with only slight asymmetry. Early Sestamibi images demonstrated slight prominence of the right lower lobe compared to the left, while delayed Sestamibi images demonstrate focal retention of tracer in the left inferior thyroid gland, consistent with a parathyroid adenoma.

**Figure 3**

Fig 3. Technetium (Tc) Sestamibi scan



The patient underwent a right inferior parathyroidectomy. Histological confirmation of a 160mg parathyroid adenoma was obtained. Following this procedure the patient's PTH

levels normalised as did her serum and urine calcium levels. The patient remained symptom-free and did not have a recurrence of renal calculi for the subsequent 12 months. Subsequently the patient was discharged from the Urology service.

### DISCUSSION

Nephrolithiasis is common and affects 0.131% of the population at any time [13]. However, urolithiasis can affect up to 10–15% of the population during their lifetime [13]. Following an episode of ureteric colic there is a high risk of recurrence (30 to 50% at 5 years) [3, 4]. In patients with recurrent ureteric colic a reversible metabolic abnormality can be identified in over 90% [13].

First-time stone formers do not regularly have a full urine and electrolyte evaluation due to the low incidence of a reversible metabolic cause [13]. However, initial biochemical evaluations including serum electrolytes, calcium, uric acid, fresh urine pH, and urine culture are essential first-line tests. Any calculi that are passed should be sent for analysis.

Patients with elevated or high-normal serum calcium should have hyperparathyroidism excluded as approximately 5% of patients with nephrolithiasis are found to have primary hyperparathyroidism [5, 8]. This is best achieved with simultaneous measurement of serum PTH and calcium levels. [7] Following biochemical diagnosis of hyperparathyroidism imaging of the parathyroid glands is best achieved with ultrasound and Technetium Sestamibi scan. This will determine the location of an overactive parathyroid adenoma and aid surgical planning.

PTH regulates calcium homeostasis through bone resorption, renal reabsorption and absorption of calcium from the gastrointestinal tract. Primary hyperparathyroidism results from increased secretion of PTH directly from the parathyroid glands. A European study published in 2002, quoted the incidence of primary hyperparathyroidism in the general population to be 3 in 1000 [9] with a female preponderance [10].

Most cases of primary hyperparathyroidism occur from a solitary parathyroid adenoma (88.9% of cases) [11]. Primary hyperparathyroidism is often asymptomatic and commonly detected as an incidental finding of hypercalcaemia on routine blood tests. Primary hyperparathyroidism, if symptomatic, may present with non-specific manifestations of hypercalcaemia, including symptoms of fatigue, weakness, constipation, or mood

disturbances. End organ complications of primary hyperparathyroidism include cortical bone disease and nephrolithiasis with or without nephrocalcinosis [12].

## CONCLUSION

Metabolic causes of nephrolithiasis should be considered and screened for in all patients who are recurrent stone formers.

## References

1. Johnson CM, Wilson DM, O'Fallon WM, Malek RS, Kurland LT: Renal stone epidemiology: a 25-year study in Rochester, Minnesota. *Kidney Int.* 1979;16(5): 624-31.
2. Hiatt RA, Dales LG, Friedman GD, Hunkeler EM: Frequency of urolithiasis in a prepaid medical care program. *Am J Epidemiol.* 1982; 115(2): 255-65.
3. Hall PM: Nephrolithiasis: treatment, causes, and prevention. *Cleve Clin J Med.* 2009; 76(10): 583-91.
4. Uribarri J, Oh MS, Carroll HJ: The first kidney stone. *Annals of Internal Medicine;* 1989; 111(12): 1006-1009.
5. Morton AR, Iliescu EA, Wilson JW: Nephrology: 1. Investigation and treatment of recurrent kidney stones. *CMAJ.* 2002; 166(2): 213-8.
6. Moe OW: Kidney stones: pathophysiology and medical management. *Lancet.* 2006; 367(9507): 333-44.
7. Sorensen MD, Duh Q-Y, Grogan RH, Tran TC, Stoller ML: Urinary Parameters as Predictors of Primary Hyperparathyroidism in Patients With Nephrolithiasis. *Journal of Urology.* 2012; 187(2): 516-21.
8. Parks J, Coe F, Favus M: Hyperparathyroidism in nephrolithiasis. *Arch Intern Med.* 1980; 140(11): 1479-81.
9. Adami S, Marcocci C, Gatti D: Epidemiology of primary hyperparathyroidism in Europe. *J Bone Miner Res.* 2002; 17 Suppl 2: N18-23.
10. Miller BS, Dimick J, Wainess R, Burney RE: Age- and sex-related incidence of surgically treated primary hyperparathyroidism. *World J Surg.* 2008; 32(5): 795-9.
11. Ruda JMB, Hollenbeak CSP, Stack BCJMF: A systematic review of the diagnosis and treatment of primary hyperparathyroidism from 1995 to 2003. *Otolaryngology Head & Neck Surgery.* 2005; 132(3): 359-72.
12. Silverberg SJ, Shane E, de la Cruz L, Dempster DW, Feldman F, Seldin D, et al.: Skeletal disease in primary hyperparathyroidism. *J Bone Miner Res.* 1989; 4(3): 283-91.
13. Spornat, D. And Kourambas, J.(2011), Urolithiasis – medical therapies, *BJU International*, 108: 9-13. Doi: 10.1111/i.1464-410x.2011.10688.x

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