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Original article

The Relationship Between Renal Stones and Parathyroid Hormone

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A B S T R A C T

This study aimed to evaluate the relationship between biologic markers and parathyroid hormone (PTH) in stone patients. Two hundred patients (one hundred renal stones and one hundred healthy controls) formed this hospital-based case-control study. Tests for serum levels of PTH, calcium, phosphorus, and creatinine were performed and analyzed statistically. Phosphorus concentrations were significantly reduced while PTH and calcium concentrations were significantly increased in renal stone patients versus controls ($p < 0.001$). Creatinine levels showed no obvious differences. PTH had a moderately positive correlation with creatinine ($r = 0.35$), a negative correlation with phosphorus ($r = -0.48$), and a strong, positive correlation with calcium ($r = 0.62$). PTH excess stimulates renal stone formation by deranging calcium and phosphate metabolism. Conclusion: Regular PTH measurements could improve the diagnosis and management of nephrolithiasis.

Introduction

Nephrolithiasis (also referred to as renal stone disease) is a worldwide growing urological disorder with significant morbidity and recurrence potential. This indicates the importance of calcium metabolism in stone formation, since renal stones are mainly composed of calcium salts (mainly oxalate and phosphate). The mechanism of nephrolithiasis is poorly understood; however, it can be based on a variety of metabolic disorders such as hypocitraturia, hypercalciuria, and hyperoxaluria. Calcium regulatory problems are one of the most important among these because they directly affect urine supersaturation and thus crystal formation.

Recent epidemiological data indicating that the global incidence of kidney stones is rising in relation to dietary, environmental or metabolic factors (1) underscores the need for an improved understanding of how underlying hormonal effects, such as parathyroid hormone (PTH), influence stone pathogenesis. Parathyroid hormone is responsible for maintaining calcium and phosphate homeostasis. Released into circulation from parathyroid glands in response to low serum calcium levels, it acts on the kidneys, bones and intestines to normalize the calcium balance. PTH alters urine composition by increasing phosphate excretion and calcium reabsorption in the kidney. Conversely, high PTH concentrations in patients with chronic high PTH, such as those seen in cases of hyperparathyroidism, can lead to fracture and reduction in filtered charge since the amount of circulating calcium load will be excessive; these factors also predispose to hypercalciuria. This imbalance is the chief cause of renal calculi. The action of PTH on the tubular transport systems of the kidney is a typical example of the direct involvement of this hormone in lithogenic activity (2).

One of the most important endocrine conditions that are associated with nephrolithiasis is primary hyperparathyroidism (PHPT). It is associated with an increased blood calcium level that is caused by pathological overproduction of PTH, typically due to an adenoma or hyperplasia of the parathyroid gland. One of the more frequent complications of PHPT (seen in many patients) is renal stones. There are 3–5 per cent of patients who present with kidney stones who have pre-existing PHPT, and imaging studies show even higher rates of stones in hyperparathyroid patients without symptoms. PHPT is also known to be a potentially reversible cause of nephrolithiasis, and early detection is crucial to avoid renal impairment and recurrence of urolithiasis (3).

Both kidney stones and PTH affect one another and their impact may vary depending on the other chemicals in the body and the environment. Although not all people with PHPT develop stones, a high amount of calcium in the urine shows that there is more than one process going on, which is why its diagnosis is linked to excess PTH. Genetics, level of vitamin D, urine acidity, and citrate levels also play a role in determining the risk of kidney stones. The studies have indicated that high calcium increases the risk of developing kidney stones, but other urinary issues and personal factors are the final determining factors in whether or not kidney stones will develop. PTH and vitamin D work closely together, and

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scientists are still studying how the metabolism of vitamin D is related to the development of kidney stones (4).
An early sign of impaired functioning of the parathyroid may be the presence of kidney stones. Even with this connection, not many nephrolithiasis patients receive enough screening for PHPT, and therefore, the diagnosis and treatment are delayed. Treatment, such as parathyroidectomy and early diagnosis of the condition, helps to decrease urine calcium levels and helps to decrease the risk of recurrence of kidney stones. Therefore, it is necessary to know the influence of PTH on the formation of kidney stones so that doctors can be better at caring for their patients and making more accurate diagnoses. To spot reversible hormone causes like hyperparathyroidism, today's research says stone formers should get regular metabolic testing (5).

Methods

Study Design and Population

This hospital-based case-control study was conducted in individuals with kidney stones, and linked those patients to laboratory markers of parathyroid hormone (PTH). There were 200 people, 100 of whom had kidney stones and 100 healthy controls.

Inclusion and Exclusion Criteria

The scientists analysed those with confirmed kidney stones who were 18 or older. Chronic kidney disease, hormone disorders such as thyroid problems and primary hyperparathyroidism, and medicines that affect calcium levels (such as calcium or vitamin D) were exclusion criteria, as were pregnant women. The healthy controls were age- and sex-matched, and did not have a history of metabolic disease or kidney stones.

Data Collection

Basic information, such as sex and age, was recorded for each of the participants. Patients' history and important health information were gathered through interviews with doctors and medical records.

Biochemical Analysis

Aseptic venous blood samples (5 mL) were taken from each participant. Serum was extracted for biochemical examination after the samples were centrifuged.
The parameters that were measured were as follows:
• The enzyme-linked immunosorbent assay (ELISA) was used to measure the levels of parathyroid hormone (PTH).
• Standard colorimetric techniques were used to measure serum calcium and serum phosphorus.
• An automated analyzer based on the Jaffe method was used to measure serum creatinine. Every measurement was carried out in compliance with accepted lab procedures.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS) version was used to examine the data. The mean ± standard deviation (SD) was used to express continuous variables. The means of renal stone patients and controls were compared using the independent samples t-test. The association between PTH and biochemical parameters (creatinine, phosphorus, and calcium) was assessed using the Pearson correlation coefficient (r). Statistical significance was defined as a p-value of less than 0.05.

Results

The study comprised 200 patients with a mean age of 44.6 ± 12.3 years, 120 of whom were male (60%) and 80 of whom were female (40%). One hundred patients with renal stones and one hundred healthy controls made up the two groups of patients .
According to table (1), patients with renal stones had a substantially higher mean serum parathyroid hormone (PTH) level (79.5 ± 17.6 pg/mL) than the control group (46.2 ± 11.1 pg/mL) ($p < 0.001$); also, patients with renal stones had a substantially higher mean serum calcium level (11.8 ± 0.8 mg/dL) than the control group (8.5 ± 0.7 mg/dL) ($p < 0.001$). The mean serum phosphorus level of the patients (2.9 ± 0.6 mg/dL) was significantly lower than that of the control group (3.9 ± 0.8 mg/dL) ($p < 0.001$), while the mean serum creatinine level of the patients (1.2 ± 0.3 mg/dL) was slightly higher than the control group (1.0 ± 0.3 mg/dL), but not statistically significant ($p > 0.05$).

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Table 1. Comparison of Biochemical Parameters Between Renal Stone Patients and Controls

Parameters	Renal Stone Patients (n=100)	Control Group (n=100)	P-value	Significance
Age (years)	47.2 ± 11.8	44.0 ± 12.7	0.52	Not significant
Parathyroid Hormone (pg/mL)	79.5 ± 17.6	46.2 ± 11.1	<0.001	Significant
Serum Calcium (mg/dL)	11.8 ± 0.8	8.5 ± 0.7	<0.001	Significant
Serum Phosphorus (mg/dL)	2.9 ± 0.6	3.9 ± 0.8	<0.001	Significant
Serum Creatinine (mg/dL)	1.2 ± 0.3	1.0 ± 0.3	0.08	Not significant

Correlation coefficient relationship between PTH and Serum Calcium

Figure 1 shows significantly strong positive correlation between PTH and Calcium (r= +0.62) in kidney stone.

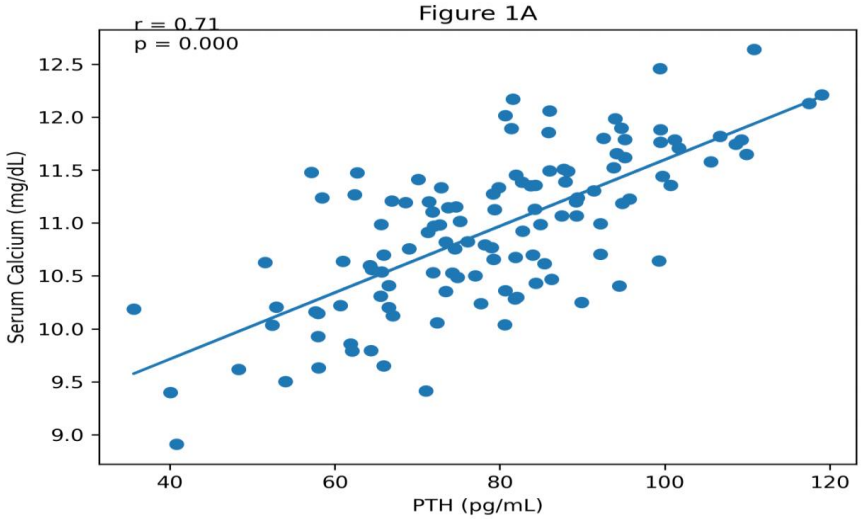

Figure (1) Correlation coefficient relationship between PTH and Calcium

Correlation coefficient relationship between PTH and Phosphorus

Figure 2 shows a significant negative correlation between PTH and Phosphorus (r = −0.48) in kidney stone.

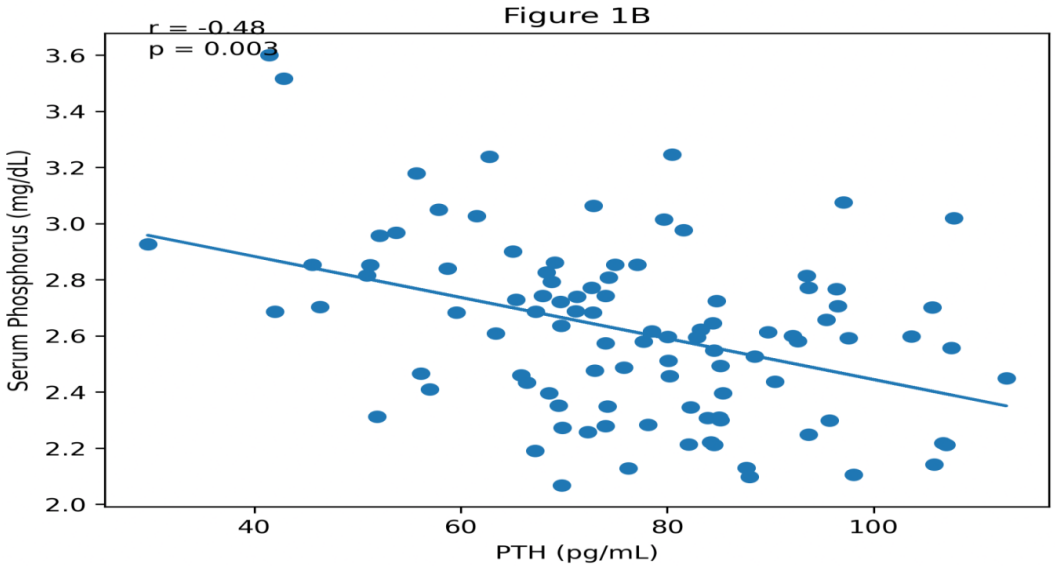

Figure 2. Correlation coefficient relationship between PTH and Serum Phosphorus

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Correlation coefficient relationship between PTH and Serum Creatinine

Figure 3 shows a significant positive correlation between PTH and Creatinine (r = +0.35) in kidney stone.

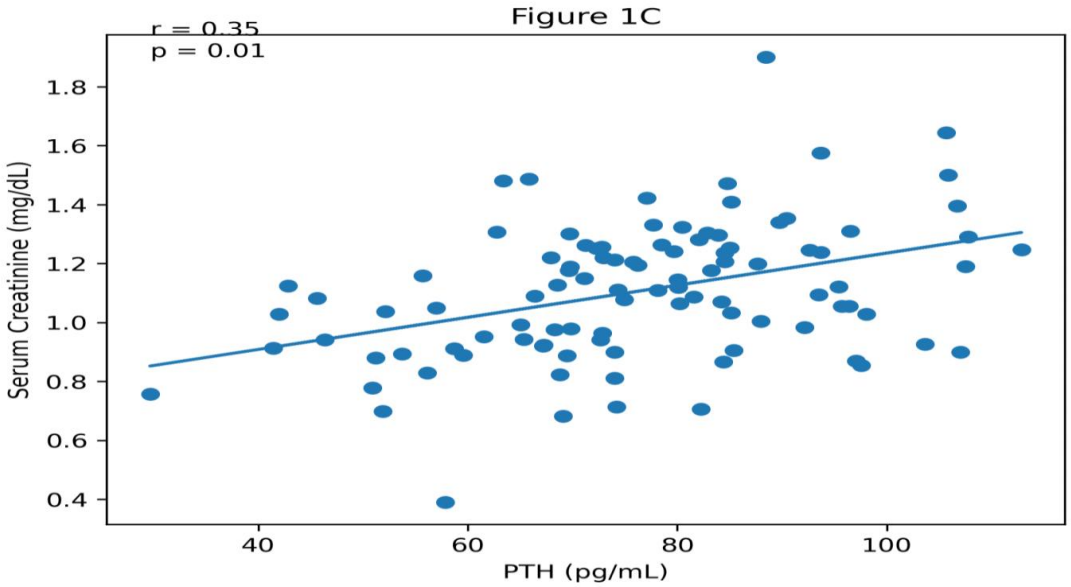

Figure 3. Correlation coefficient relationship between PTH and Serum Creatinine

Discussion

In comparison to healthy controls, the current investigation showed that patients with renal stones had significantly higher serum levels of calcium and parathyroid hormone (PTH) and lower serum levels of phosphorus. Significant relationships between PTH and important biochemical markers were also found, indicating that PTH plays a critical role in the pathogenesis of nephrolithiasis. The elevated PTH levels seen in patients with renal stones are in line with earlier research showing that hyperparathyroidism plays a significant role in the development of calcium stones. Hypercalcemia and hypercalciuria are two known risk factors for nephrolithiasis that are caused by increased PTH.

New research has indicated that increases in PTH, even at the upper end of normal range, may increase the risk of kidney stones [6-10]. In the case of stone formers, the calcium levels were significantly higher, which we found. The same was observed by Ferraro [7] with a special emphasis on the fact that renal stones are primarily formed as a result of disordered calcium metabolism, mainly by the formation of calcium oxalate and calcium phosphate crystals that tend to clump together. They stated that in 2021, they related kidney stones to calcium balance issues. On the other hand, people with kidney stones had much lower serum phosphorus levels. The opposite link may be due to PTH reducing the amount of phosphate excreted by the kidneys. A negative relationship between PTH and phosphorus was found, with opposite movement between these two variables (r = −0.48), supporting this. The same pattern has been observed in new studies conducted on clinics, where it is proven how PTH and phosphate are correlated in kidney stone disease [8]. The study found a strong positive link (r = +0.62) between PTH and calcium, supporting PTH's normal role in regulating calcium levels. If stones do form, then the higher calcium levels appear to be directly related to the higher PTH levels in patients.

A moderately positive correlation (+0.35) was found between serum creatinine and PTH. Although blood creatinine remained similar across all groups, it could indicate that the stone formers had early kidney disease or various reactions to PTH treatment. A new study of the kidneys has revealed that repeated stone formation can occur silently over time, causing the kidneys to gradually lose function, which could be the cause of the tiny decreases in kidney function [9]. In conclusion, this study demonstrates the significance of PTH in the renal stone-forming processes in the body chemistry. The combination of hyperparathyroidism, high calcium and low phosphate can lead to stones. These findings highlight the importance of regularly monitoring PTH and blood markers in patients with kidney stones, especially in cases of recurrent stones and/or other unexplained features.

Conclusion

Some people with kidney stones have high calcium and/or high phosphorus levels, and these levels correlate closely with their PTH level. PTH might be a useful indicator to assess and control risk and the

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strong correlations identified indicate that it has an important role in the pathogenesis of the formation of kidney stones.

Conflict of interest. Nil

References

1. Ferraro PM, Curhan GC, Gambaro G. Epidemiology of kidney stones: Global trends and risk factors. Urology Rep. 2026;18(2):100-12.
2. Minisola S, Pepe J, Scillitani A, Cipriani C. The diagnosis and management of hyperparathyroidism. Lancet Diabetes Endocrinol. 2023;11(3):215-28.
3. Yeh MW, Ituarte PHG, Zhou HC, Nishimoto S, Liu ILA, Harari A, Haigh PI. Incidence and prevalence of primary hyperparathyroidism in patients with nephrolithiasis. J Clin Endocrinol Metab. 2022;107(5):e1901-10.
4. Ticinesi A, Nouvenne A, Borghi L. The complex relationship between vitamin D, parathyroid hormone, and kidney stone disease. Metabolites. 2024;14(1):39.
5. Rule AD, Lieske JC, Pais VM. Evaluation and management of kidney stones in adults. Mayo Clin Proc. 2024;99(4):789-803.
6. Bilezikian JP, Bandeira L, Khan A, Cusano NE. Hyperparathyroidism. Lancet. 2022;399(10331):168-78.
7. Ferraro PM, Bargagli M, Trinchieri A, Gambaro G. Risk of kidney stones: Influence of dietary factors, dietary patterns, and vegetarian–vegan diets. Nutrients. 2021;13(3):779.
8. Khan SR, Pearle MS, Robertson WG, et al. Kidney stones. Nat Rev Dis Primers. 2020;6(1):1-23.
9. Rule AD, Lieske JC, Li X, et al. The ROKS nomogram for predicting a second symptomatic stone episode. J Am Soc Nephrol. 2019;30(6):1111-9.
10. Walker MD, Silverberg SJ. Primary hyperparathyroidism. Nat Rev Endocrinol. 2018;14(2):115-25.
11. Yavropoulou MP, Yovos JG. The role of the calcium-sensing receptor in the pathogenesis of hypercalcemia. Endocrine. 2021;71(2):243-51.
12. Kadhim HM, Al-Ghanimi HH, Al-Dedah RM. Haematological parameters and biochemical indices in patients with chronic kidney disease before haemodialysis Al-Furat Al-Awsat Governorates/Iraq. AIP Conf Proc. 2020 Dec;2290(1):020004.

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