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Comprehensive biochemical profile of patients with calcium-based kidney stones in Kirkuk, Iraq: A case-control study of serum, urine and hormonal markers

Ibrahim Hadi Saleh *

College of Medicine, University of Kirkuk, Kirkuk, Iraq, 36001.

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Abstract

Background: Nephrolithiasis is a complex metabolic disorder and multifactorial urological disease. Awareness of its pathophysiology is important for patient management and prevention of recurrence. This carefully polarized case-control study aimed to rigorously assess within specific ranges the numerous serum biochemical markers, electrolytes, renal function markers, hormonal analyses including parathyroid hormone (PTH) and vitamin D (Vit D), as well as detailed urinary 24-hour excretion profiles in patients with calcium-based kidney stones against a strictly selected group of healthy controls without affecting disease.

Methods: This study was conducted on 150 subjects (120 patients and 30 controls) in Kirkuk Governorate from November 2024 and November 2025. Biochemical and hormonal indices were evaluated on fasting blood and 24-h urine samples, and characterization of stones was performed by FTIR.

Results: FTIR revealed 92.4% calcium oxalate stones. Patients had significantly lower serum calcium (Ca), sodium (Na) and potassium (K) but higher phosphate, urea and creatinine levels in comparison to controls ($p<0.05$). PTH was highly upregulated, whilst vitamin D showed no difference. Urine studies showed elevated urine calcium and oxalate; low urine citrate ($p<0.05$).

Conclusions: Patients exhibited systemic mineral derangements, renal dysfunction, secondary hyperparathyroidism and a lithogenic urinary profile with calcium oxalate stones and hypocitraturia. A thorough biochemical and urinary assessment of hyperparathyroidism is necessary for better risk stratification and personalized therapeutic strategies.

Keywords: Nephrolithiasis; Calcium Oxalate Stones; Metabolic Disturbances; Hyperparathyroidism; Urinary Biomarkers.

1. Introduction

The kidneys are essential organs that regulate homeostasis by controlling the balance of fluids, electrolytes and acid-base, as well as metabolism waste elimination. Perturbation of these roles are implicated in numerous renal diseases (Gao et al., 2023; Yamama et al., 2024). One of the most prevalent urological disorders around the world is nephrolithiasis (kidney stone disease) which affects 10-15% of the World's population and has a high rate of recurrence with up to 40% presenting a second stone within three years (Dunya et al., 2025; Rayner et al., 2020).

Most kidney stones (about 70-80%) are calcium-containing, with most of those being primarily composed of calcium oxalate (or less frequently, calcium phosphate). Stone formation results from supersaturation of the urine with lithogenic solutes (calcium, oxalate or phosphate) and a deficiency in inhibitory compounds (citrate and magnesium). (Evan and Lingeman; 2003). Randall's plaques, sub-epithelial calcium phosphate depositions at the tip of the renal

* Corresponding author: Ibrahim Hadi Saleh

papillae, are widely thought to be an important nidus for idiopathic calcium oxalate stone formation (Van den Berg et al., 2018; Callaghan and Bandyopadhyay, 2012).

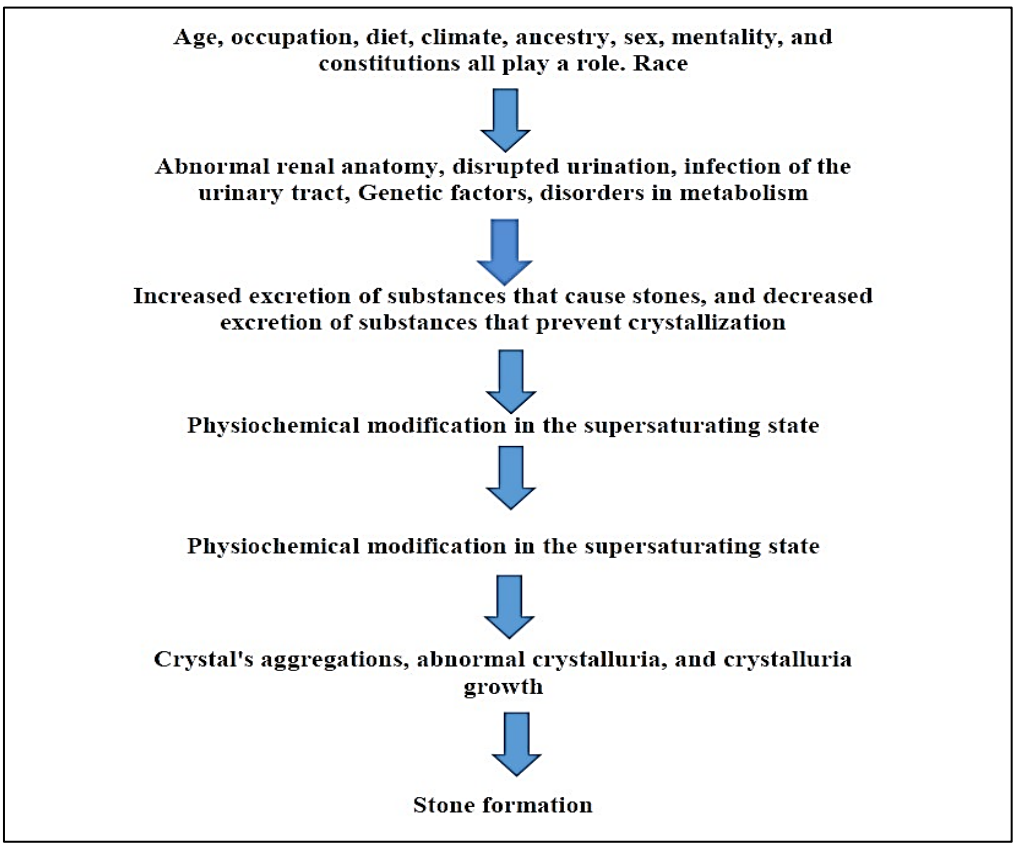

Figure 1 Illustration of the three types of urinary stone formations

Clinical presentations vary from asymptomatic passage of stone to severe renal colic, hematuria, recurrent urinary tract infections and - in overt or obstructive cases - progressive renal failure (Peacock, 2010; Thongprayoon et al., 2020). Recognized risk factors include hypercalciuria, hyperoxaluria, hypocitraturia, hyperuricosuria, primary hyperparathyroidism, type 2 diabetes, obesity, inflammatory bowel disease, and certain dietary patterns (Tasian et al., 2018; Alelign and Petros, 2018).

Figure 2 Pathophysiology of uric acid stone formation

Figure 3 Interaction between uric acid and calcium stone formation pathways

Figure 4 Role of magnesium and calcium oxalate stone formation.

Figure 5 Magnesium and calcium phosphate precipitation in the presence of magnesium

Parathyroid hormone (PTH) plays a central role in calcium homeostasis. Elevated PTH, as seen in primary or secondary hyperparathyroidism, promotes renal tubular calcium reabsorption while increasing calcitriol (active Vitamin D) synthesis, which in turn enhances intestinal calcium absorption. However, in the setting of renal calcium leak or impaired renal function, these compensatory mechanisms may paradoxically contribute to hypercalciuria and stone formation (Peacock, 2010; Sakhaee et al., 2012). Despite the well-established pathophysiology, population-specific data from Iraq - and from Kirkuk specifically - remain limited.

This study therefore aimed to characterize the comprehensive biochemical, hormonal, and urinary profile of patients with calcium-based kidney stones in Kirkuk, Iraq, in order to better understand the local metabolic milieu contributing to stone formation and to provide a foundation for evidence-based clinical management in this region.

2. Materials and methods

2.1. Study Design and Ethics

A case-control study design was employed. The study was approved by the Institutional Review Committee of the College of Medicine/University of Kirkuk, Iraq - Ethics Approval Reference Number: issue number 946, in 12/3/2025.

2.2. Study Population and Sampling

The 150 subjects were recruited between November 2024 and November 2025. Patients The study group included 120 patients who had been diagnosed with renal calculi and were randomly selected from private laboratories and hospitals of Kirkuk governorate. The control group included 30 healthy subjects and was age- and sex-matched to patients, with no history of kidney stones or any related metabolic abnormalities. Clinical history, including medication (such as diuretic) use and comorbidities were obtained for all patients.

2.3. Inclusion and Exclusion Criteria

For Patients: Adults (25-70 years old); confirmed diagnosis of kidney stones diagnosed using ultrasonography or X-ray KUB, with the backing of a CT scan; primarily calcium stone composition as determined by stone analysis or biochemical profile; consent to participate in the study.

Patients Aged >18 Years (Men and Women) Patients with primary endocrine diseases other than those associated with calcium metabolism; patients on dialysis; active malignancy; pregnancy, incomplete laboratory data; refusal to give informed consent.

Control Inclusion Criteria: Healthy adults aged 25-70 years; no previous kidney stone, urinary tract disease, chronic kidney disease or endocrine drug history that affects mineral metabolism; none of the contraindicating medications at interview check list and performed informed consent.

2.4. Sample Collection

Venous blood (5 ml) was drawn from fasting healthy individuals using a sterile disposable syringe after overnight fast 8-12 h. Clotted samples were then centrifuged at 3,000 × g for 15 min and serum was separated and transferred into labelled polypropylene tubes and stored at -20°C pending analysis.

All subjects were instructed on the correct way to collect a full 24-hour urine specimen using standardized collection vessels with preservative/antioxidant agent (e.g., acidification to pH < 3 for prevention of calcium precipitation). Cumulative 24-hour urine collection was measured and aliquots kept at 4°C for subsequent determination.

Stone fragments were collected for compositional analysis when available from patients undergoing stone removal procedures (e.g., extracorporeal shock wave lithotripsy, ureteroscopy, or percutaneous nephrolithotomy).

2.5. Biochemical and Hormonal Analyses

Serum calcium, phosphate, magnesium, sodium, potassium, urea, creatinine was estimated in an autoanalyzer by spectrophotometry (standard colorimetric assays had been run on an automated clinical chemistry machine). Serum parathyroid hormone (PTH) and 25-hydroxyvitamin D were quantified using enzyme-linked immunosorbent assay (ELISA) kits as per manufacturers' protocol.

Urinary calcium, oxalate, phosphate, citrate and uric acid were determined based on 24-hour urine collections using commercially available enzymatic and colorimetric assay kits. The 24-hour urine volume was then multiplied by the concentration of analyte to obtain the urinary excretion rate.

2.6. Stone Composition Analysis

All the stone fragments were subjected to Fourier Transform Infrared Spectroscopy (FTIR) for chemical analysis. Stones were further characterized as being calcium oxalate, calcium phosphate, mixed calcium based, uric acid, struvite or other by their predominant crystal content.

2.7. Statistical Analysis

All values are shown as means ± SD. Distribution normality was tested with the Shapiro-Wilk test before performing group comparisons. Statistical comparisons between patient and control groups were calculated with the unpaired Student's t-test or Mann-Whitney U test for normally versus non-normally distributed data. Correlations among biochemical variables within each group were examined by calculating Pearson's (or Spearman's when appropriate) correlation coefficients. Statistical analysis All the statistical analyses were carried out by GraphPad Prism 8 (GraphPad Software, San Diego, CA). A p-value < 0.05 was considered significant.

3. Results

3.1. Demographic Characteristics

The age distribution of study participants is presented in Table 1. The patient group (n = 120) and the control group (n = 30) were comparable across age categories. The highest prevalence of kidney stones was observed in the 40-54-year age group, which accounted for 45.8% of patients.

Table 1 Age Distribution of Kidney Stone Patients and the Control Group

Age Group (years)	Patients (n)	Patients (%)	Control (n)	Control (%)	Total
25-39	36	30.0	7	23.3	43
40-54	55	45.8	10	33.3	65
55-70	29	24.2	13	43.4	42
Total	120	100	30	100	150

Values are expressed as number (n) and percentage (%).

3.2. Stone Composition Analysis

Stone analysis was successfully performed in 120 of the available stone samples from the patient arm. The most common was predominantly calcium oxalate in 85 stones (92.4%). The remaining 7 stones (7.6%) were calcium phosphate or calcium-containing mixed stones. No pure uric acid and/or struvite stones were found in this group.

3.3. Serum Biochemical, Renal Function, and Hormonal Profile

The serum concentrations of important biochemical parameters, renal functional indices and hormones in the two groups are shown in Table 2. Serum calcium was also significantly lower in patients (8.828 ± 0.348 mg/dl) as compared to controls (9.393 ± 0.471 mg/dl; p = 0.037). Patients had significantly higher serum phosphate (5.381 ± 0.922 mg/dl) compared with controls (3.897 ± 0.426 mg/dl; p < 0.0001). The serum magnesium was lower among the patients (1.566 ± 0.322 mg/dl) when compared to control group (2.140 ± 0.256 mg/dl), but this difference did not reach statisticl significant level (p = 0,169).

Serum sodium (134.5 ± 6.48 mM/l vs. 142.1 ± 3.86 mM/l; p = 0.007) and serum potassium (3.321 ± 0.378 mM/l vs. majority, that is, median [+30 -10]; p =0.011) were the two electrolytes that were significantly lower in cases compared to controls). The hyponatremia and hypokalemia present in these patients may have resulted from stone prophylaxis use or presence of comorbid conditions, as 45% were using diuretics.

Renal function indices were markedly damaged in the patients. Serum urea was significantly higher (48.5 ± 15.2 mg/dl vs 25.1 ± 5.8 mg/dl; p <0,0001), and serum creatinine was also increased (1.6 ± 0.7 mg/dl vs 0.9 ± 0.2 mg /dl; p <0,0001). These results are consistent with extensive reduction of glomerular filtration in a large proportion of the population studied.

Hormonal measurements the group of patients showed significantly higher levels of parathyroid hormone (PTH: 85.3 ± 25.1 pg/ml) than the controls values (42.6 ± 11.5 pg/ml, p <0.0001), indicating a secondary hyperparathyroidism condition (Table 2). Importantly, serum 25-hydroxyvitamin D concentrations were similar in both groups (22.4 ± 8.1 vs. 24.9 ± 7.5 ng/ml; p = 0.215), indicating that Vitamin D insufficiency alone does not account for the detected elevation of PTH levels.

Table 2 Serum Biochemical, Renal Function, and Hormonal Profile in Kidney Stone Patients and the Control Group

Parameter	Patients (n=120) Mean ± SD	Controls (n=30) Mean ± SD	t-value	P-value
Calcium (mg/dl)	8.828 ± 0.348	9.393 ± 0.471	2.107	0.0370*
Phosphate (mg/dl)	5.381 ± 0.922	3.897 ± 0.425	9.803	<0.0001***
Magnesium (mg/dl)	1.566 ± 0.322	2.140 ± 0.256	1.382	0.1689 (NS)
Sodium (mM/l)	134.5 ± 6.482	142.1 ± 3.859	7.620	0.0073**
Potassium (mM/l)	3.321 ± 0.378	4.1 ± 2.05	2.563	0.0109*
Urea (mg/dl)	48.5 ± 15.2	25.1 ± 5.8	10.88	<0.0001***
Creatinine (mg/dl)	1.6 ± 0.7	0.9 ± 0.2	7.143	<0.0001***
PTH (pg/ml)	85.3 ± 25.1	42.6 ± 11.5	12.48	<0.0001***
Vitamin D (ng/ml)	22.4 ± 8.1	24.9 ± 7.5	1.245	0.2150 (NS)

* p ≤ 0.05; ** p ≤ 0.01; *** p ≤ 0.001; NS = not significant.

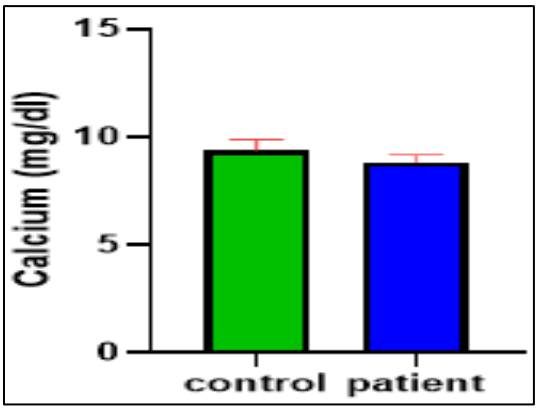

Figure 6 Comparison of serum calcium levels between kidney stone patients and controls

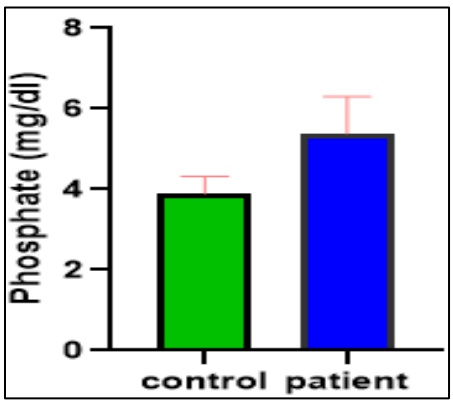

Figure 7 Comparison of serum phosphate levels between kidney stone patients and controls

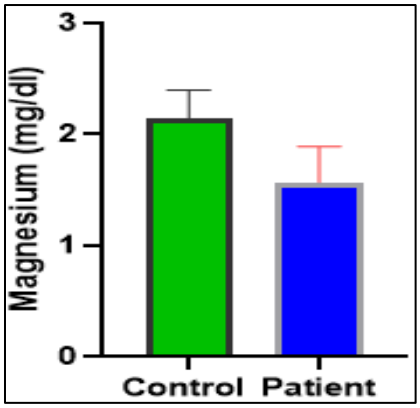

Figure 8 Comparison of serum magnesium levels between kidney stone patients and controls

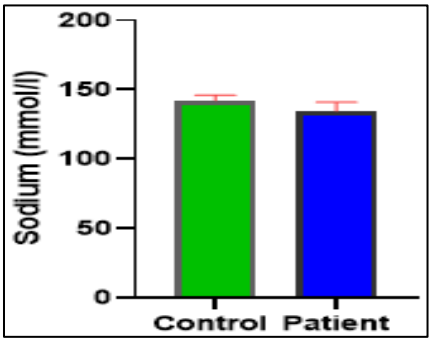

Figure 9 Comparison of serum sodium levels between kidney stone patients and controls

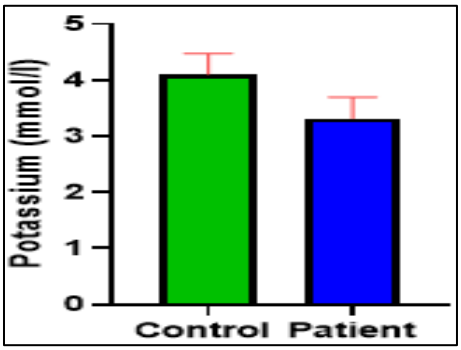

Figure 10 Comparison of serum potassium levels between kidney stone patients and controls

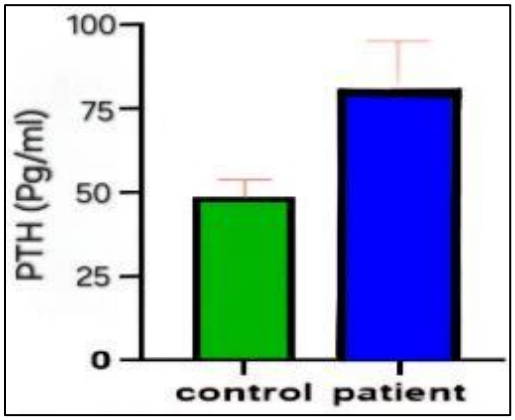

Figure 11 Comparison of serum PTH levels between kidney stone patients and controls

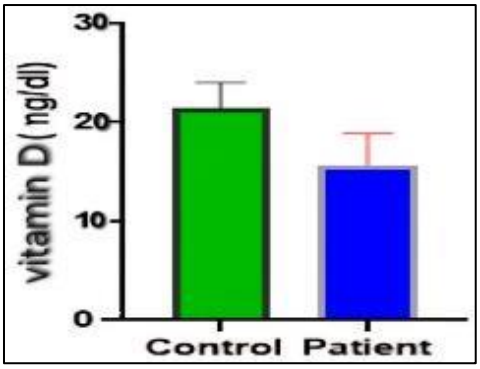

Figure 12 Comparison of serum Vitamin D levels between kidney stone patients and controls

3.4. Twenty-Four-Hour Urinalysis Profile

Analysis of 24-hour urine samples Patients' 24-hour urine was pro-lithogenic in nature, as demonstrated by the results (Table 3). The mean urinary calcium (hypercalciuria) and oxalate (hyperoxaluria) excretion were also significantly greater in the patients compared to Controls (310.5 ± 85.2 mg/24h versus 175.4 ± 45.1 mg/24h; P <0.0001), (55.8 ±18.3 md/24h; P<0.0001).

Hypocitraturia was pronounced in patients (250.6 ± 110.5 mg/24h) compared with controls (610.2 ± 155.8 mg/24h; p < 0.0001).

Urinary uric acid excretion was also significantly greater in patients (780.1 ± 130.4 mg/24h vs. 650.5 ± 115.3 mg/24h; P = 0.0012).

Table 3 Twenty-Four-Hour Urinalysis Profile in Kidney Stone Patients and the Control Group

Parameter (mg/24h)	Patients (n=120) Mean ± SD	Controls (n=30) Mean ± SD	t-value	P-value
Urinary Calcium	310.5 ± 85.2	175.4 ± 45.1	11.94	<0.0001***
Urinary Oxalate	55.8 ± 18.3	28.6 ± 9.7	10.68	<0.0001***
Urinary Citrate	250.6 ± 110.5	610.2 ± 155.8	14.26	<0.0001***
Urinary Uric Acid	780.1 ± 130.4	650.5 ± 115.3	3.282	0.0012**

** p ≤ 0.01; *** p ≤ 0.001. All urinary parameters are expressed as total excretion per 24 hours (mg/24h).

3.5. Correlation Analysis of Serum Biochemical Variables

Tables 4 and 5 present Pearson correlation coefficients among serum biochemical variables in the patient group and the control group, respectively. In the patient group, modest positive correlations were observed between serum calcium and both phosphate (r = 0.120) and magnesium (r = 0.142), along with a positive correlation between magnesium and potassium (r = 0.202). In the control group, a strong positive correlation was found between calcium and potassium (r = 0.527), and moderate positive correlations were observed between calcium and sodium (r = 0.289) and calcium and magnesium (r = 0.266). A notable negative correlation was observed between phosphate and sodium in the control group (r = -0.411).

Table 4 Pearson Correlation Coefficients Among Serum Biochemical Variables - Patient Group (n = 120).

Variable	Ca^{2+}	PO_4	Mg^{2+}	Na^+
PO_4	0.120	-	-	-
Mg^{2+}	0.142	-0.078	-	-
Na^+	-0.157	0.108	0.004	-
K^+	0.104	-0.096	0.202	0.062

r = Pearson correlation coefficient. Values represent the strength and direction of linear relationships between variables.

Table 5 Pearson Correlation Coefficients Among Serum Biochemical Variables - Control Group (n = 30)

Variable	Ca^{2+}	PO_4	Mg^{2+}	Na^+
PO_4	-0.046	-	-	-
Mg^{2+}	0.266	0.026	-	-
Na^+	0.289	-0.411	-0.037	-
K^+	0.527	0.041	0.010	0.169

r = Pearson correlation coefficient. Values represent the strength and direction of linear relationships between variables.

4. Discussion

The present study is the first one documenting the biochemical, hormonal and urinary profile of CKS patients in Kirkuk city, Iraq; showing that there are marked changes in various metabolic axes compared to healthy controls. Together, the results indicate a rich and untoward clinical phenotype underscored by a complex, multivariable mechanism that includes disturbance in systemic minerals, compromised kidney function, secondary hyperparathyroidism, and an extremely pro-lithogenic urinary milieu.

4.1. Stone Composition and Clinical Relevance

The strikingly higher prevalence of calcium oxalate stones in this study (92.4%) is in line with worldwide epidemiology reporting that calcium oxalate is the most common stone type (worldwide) [6; 8]. This compositional homogeneity also enhances the legitimacy of our biochemical results, as it is likely that the metabolic defects, we detected are directly related to calcium oxalate nephrolithiasis itself.

4.2. Evidence of Impaired Renal Function

High serum levels of urea 48.5 (25.1; $p < 0.0001$) and creatinine 1.6 (0.9; $p < 0.0001$), as well as hyperphosphatemia, clearly indicates the renal failure status of the patient population studied here. This indicates that nephrolithiasis in these patients may not represent a benign state, but somehow reflects a reduction in glomerular filtration ability. Potential mechanisms involved are an obstruction (repeatedly) of the urinary tract by stones, chronic inflammation, infections and direct injury to tubules due to crystal-forming substances, these findings are consistent with data from contemporary end-stage kidney disease populations (Van den Berg et al., 2018)

The renal are the main site of phosphate elimination. Hyperphosphatemia (5.381 versus 3.897 mg/dl; $p < 0.0001$) is a sensitive indicator of decreased renal filtration, and clearance of phosphate can be exceeded by gains in dietary intake leading to precipitation with calcium within the tubules to favor development of both calcium phosphate stones and Randall's plaque (Evan & Lingeman, 2003; Coe et al., 2016).

4.3. The Calcium-PTH Axis and the Urinary Calcium Paradox

A clinically significant, core result of this study is the paradoxical coexistence of low-serum calcium (8.828 versus 9.393 mg/dl; $p = 0.037$) with extremely high urinary-excretion-calcium values (hypercalciuria: 310.5 versus 175.4 mg/24 h; $p < 0.0001$). Although the serum calcium levels are at or near the lower end of normal (8.5-10.5 mg/dl), This seeming contradiction strongly implies a renal loss of calcium, ie primary renal Ca wasting with inadequate tubular reabsorption of filtered Ca. In addition, calcium is excreted in the urine at high rates (leading to supersaturation and stone generation) while its plasma level is diminished (Bushinsky & Monk, 1998)

In response to this low circulating calcium state, the body strives to normalize serum P by releasing more parathyroid hormone (PTH), indeed our patients show a significantly higher levels of PTH 85.3 versus 42.6 pg/ml ($p < 0.00001$). This represents secondary hyperparathyroidism. In the absence of primary hyperparathyroidism (which typically results in hypercalcemia) where PTH usually increases serum calcium by increasing bone resorption, renal calcium reabsorption and calcitriol synthesis (and thus intestinal calcium absorption), calculi can form if these compensatory responses are ineffective or inadequate due to intrinsic renal tubular dysfunction or recurrent stone disease. Conversely, the PTH-stimulated bone resorption adds to the total body calcium load filtered by the kidneys which may exacerbate hypercalciuria and continue a cycle of forming stones (Peacock, 2010; Prince et al., 2022; Van der Wijst et al., 2019).

Notably, serum Vitamin D levels were not significantly different in patients versus controls (22.4 vs 24.9 ng/ml; p = 0.215). This observation implies that degree of Vitamin D deficiency by itself is unlikely to account for the high PTH in this population, and other contributors including renal calcium wasting, PTH stimulation from hyperphosphatemia, or intrinsic parathyroid gland end-organ resistance to hypovitaminosis D effects may be more significant influences upon secondary hyperparathyroidism in this group.

4.4. The Pro-Lithogenic Urinary Milieu

The 24-h urine panel demonstrates the specific chemical risk factors for stone formation in this cohort. The association of hypercalciuria (310.5 mg/24h) and hyperoxaluria (55.8 mg/24h) leads to high values of urinary supersaturation for calcium oxalate, the major crystalline phase found in the patients' stones. Physiological urinary calcium excretion is usually < 200 mg/24h in females and < 250 mg/24h in males, and physiological oxalate excretion is <40 mg/24h; thus, patients substantially exceed both (Prince et al., 2022; Xie et al., 2020).

This nephrolithiasis risk is profoundly enhanced by the significant hypocitraturia found in patients (250.6 vs 610.2 mg/24 h; p < 0.0001). Citrate as a powerful endogenous inhibitor of calcium stone formation. By two mechanisms and by inhibiting nucleation and aggregation of calcium oxalate crystals, thereby reducing crystal growth and adherence to the urothelium (Sakhaee et al., 2002). The stark lack of this guardian molecule in our patients puts them at substantial risk for crystal formation, growth and aggregation.

4.5. Hyponatremia, Hypokalemia, and Diuretic Use

According to the clinical notes, 45% of this group of patients were using diuretics, typically either for hypertension or stone prophylaxis. Diuretics - thiazide and loop diuretics in particular are known to enhance renal sodium and potassium excretion, thus causing alkalosis, hyponatremia and hypokalemia (Herrington et al., 2018). Furthermore, most stone formers are advised to limit dietary sodium as an approach to lower urinary calcium excretion since sodium is co-transported with calcium in the proximal tubule.

Whilst mean serum sodium (134.5 mM/l) and potassium levels (3.321 mM/l), in the patients, fall within the mild hyponatremia and borderline hypokalemia ranges respectively (normal range: Na 135-145mM/l; K 3.5-5.0mM/l), we believe these are probably chronic, asymptomatic findings in most of our patient population. However, on the contrary, loss of body potassium can result in an increased citrate excretion due to net alkali gain through bone buffering with resultant hypocitraturia (Swaminathan, 2003) which may suggest that stone patients on diuretics be followed up regularly for electrolytes and given potassium supplement if necessary.

4.6. Magnesium and Stone Inhibition

Serum magnesium was lower in patients than controls (1.566 vs 2.140 mg/dl), but not significantly different (p = 0.169). Magnesium is a widely known inhibitor of calcium oxalate crystallization in the urine and it prevents stone formation by forming soluble complexes with, which will decrease free form of oxalate facilitating binding conceivably to an apparently common site and interacting even forming weak complexes, rendering oxalate less available for calcium excretion (Johansson & Danielson, 1980 , Malcik et al., 2005). Thus, the clinically meaningful tendency toward decreased serum magnesium in patients is likely indicative of diminished availability of this protective ion if not statistically significant. A large prospective study with sufficient power would be useful to conclusively demonstrate this association.

4.7. Clinical Implications and Therapeutic Strategies

Collectively, the biochemical profile in this study implies that kidney stone patients in Kirkuk carry several co-existing and interacting metabolic risk factors that together create an extremely favorable ground for stone development. Physicians in charge of these patients should follow an integrated diagnostic approach, which is based on a serum-biochemical and 24-h urine-metabolomic profile (with particular focus on PTH, renal function markers [urea-creatinine], urinary calcium-oxalate-citrate excretion).

Specific interventions should be tailored according to these biochemistry profiles. Potassium citrate therapy, typically 30-60 mEq/day in divided doses, is a very effective and evidence-based treatment of hypercalciuric and hypocitraturic stone formers. Dietary restrictions in oxalate intake as well as sufficient calcium intake (to bind gut oxalate) are recommended for patients with hyperoxaluria. In cases of renal dysfunction, intensive observation and nephrology consultation are required. In patients with secondary hyperparathyroidism, correcting the calcium leak and getting adequate Vitamin D repletion (if deficient) could potentially normalize PTH levels. Higher fluid intake to allow at least 2-2.5 total liters of urine per day, is a common recommendation for all stone former patients (Türk et al., 2016).

Prospective studies should include long term follow-up to determine recurrent stone rates in relation to baseline biochemical data, intervention studies with selective metabolic treatments in the patient population, genetic studies of inherited risk and dietary inquiry into locally-consumed foods that may predispose to the observed metabolic disorders.

5. Conclusion

This case-control study demonstrates that patients with calcium-based nephrolithiasis in Kirkuk, Iraq have a unique metabolic abnormality characterized by a very lithogenic environment. Major results include deterioration of renal function (increased urea, creatinine and phosphate), a secondary hyperparathyroidism associated to hypocalcemia and hypercalciuria, and lithogenic urinary profile with hyperoxaluria and hypocitraturia. Calcium oxalate stones predominated (92.4%). While most clinicians focus on risk stratification and predisposing clinical factors, comprehensive assessment of both serum and 24-h urine allows for effective management, and the correction of modifiable abnormalities such as hypocitraturia offers significant preventative potential. The results highlight the importance of personalized metabolic evaluation for decreasing kidney stone recurrence and associated renal injury.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The author declares no conflicts of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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