



Renal calcifications and primary hyperparathyroidism in a tertiary care hospital center of North Africa: a single-center experience

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Abstract

Background and aims. Primary hyperparathyroidism (PHPT) is a common endocrine disorder; one of its common complications is renal calcifications. Our study aimed to determine the prevalence of renal calcifications (nephrolithiasis and nephrocalcinosis) in patients with primary hyperparathyroidism, to elucidate potential demographic, biochemical, radiological, and histological differences between patients with and without renal calcifications, and to assess potential risk factors associated with its occurrence.

Methods. This was a retrospective, single-center, descriptive, and analytical study involving 62 patients with primary hyperparathyroidism (PHPT). Participants were assessed through serum and urinary parameters, kidney-urinary tract ultrasonography, and abdominal computed tomography (CT) scans to evaluate the presence or absence of renal calcifications.

Results. We found 30 patients (48.4%) with renal calcifications, all of them nephrolithiasis, and only 2 cases (6.7%) had nephrocalcinosis associated with nephrolithiasis; and they were all detected by abdominal CT scan. Compared to patients without renal calcifications, those who had renal calcifications had a significantly higher prevalence of diabetes, BMI, waist circumference, PTH 1-84, alkaline phosphatase, and lower 25-hydroxy vitamin D (25-(OH)-D). No significant difference was found between the two groups in other studied parameters. Higher BMI and lower 25-(OH)-D were potential risk factors for renal calcifications in multivariate regression analysis.

Conclusion. The combination of PHPT and renal calcifications is still common and often asymptomatic.

Keywords: primary hyperparathyroidism, renal calcifications, nephrolithiasis, nephrocalcinosis.

Introduction

Primary hyperparathyroidism (PHPT) is the third most common endocrine disorder, characterized by autonomous production of parathyroid hormone (PTH). Classically defined as hypercalcemia in the presence of elevated or inappropriately normal concentrations of PTH [1]. However, a newer phenotype of the disease has been described, primarily in patients presenting nephrolithiasis or osteoporosis; normocalcemic PHPT

is diagnosed in patients with elevated parathyroid hormone concentrations with persistently normal serum total and ionized calcium levels [2]. With the introduction of automated chemistry panels that routinely measure serum calcium concentration, the prevalence of PHPT has increased and is reported to vary between 0.2% and 1.3% of the population across the world from the United States, Europe, Bahrain, and Korea [3-7]. Today PHPT is mostly an asymptomatic disease randomly diagnosed by biochemical screening in

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western countries [8], meanwhile, PHPT patients still demonstrate target organ involvement in other parts of the world where screening is not performed systematically [9]. One of the most common complications of PHPT is renal calcifications, which include nephrolithiasis with stone formation in the calyx, pelvis, and ureters, and nephrocalcinosis with diffuse deposition of calcium-phosphate complexes in the renal parenchyma [10].

Few studies have focused on the impact of primary hyperparathyroidism in terms of nephrocalcinosis and renal lithiasis, and the factors exposing the patient to these two entities. The aim of this study is to determine the prevalence of renal calcifications (nephrolithiasis and nephrocalcinosis), as well as to elucidate demographic, biochemical, radiological, and histological differences between patients with renal calcifications and without, and to assess potential risk factors associated with its occurrence.

Methods

Study design

This was a retrospective, single-center, descriptive, and analytical study, involving patients with PHPT admitted to the Endocrinology-Diabetology and Nutrition department, from April 2018 to April 2024.

Study population

We included 62 adult patients diagnosed with PHPT aged between 18 and 82 years, followed-up at our University Hospital Center, in whom the diagnosis of PHPT was made by both hypercalcemia and an elevated or inappropriately normal PTH level, or normocalcemia with elevated PTH level confirmed on more than one occasion, and had complete medical records. We excluded all the patients diagnosed with chronic kidney disease before the PHPT diagnosis, and other secondary causes of parathyroid stimulation, familial hypocalciuric hypercalcemia, and patients in whom imagery of kidneys and urinary tract were not performed.

We collected data on the medical history of diabetes and arterial hypertension, duration of PHPT, anthropometric characteristics, and laboratory tests that included plasma levels of albumin-adjusted calcium, phosphate, alkaline phosphatase (ALP), magnesium, creatinine, 24-hour urinary calcium, and phosphate using Architect ci8200. PTH 1- 84, and serum 25-(OH)-D were measured by Immunoanalyse. The estimated glomerular filtration rate (eGFR) was calculated using the chronic kidney disease epidemiology collaboration (CKD-EPI) formula [11]. The reference ranges of adult female and male participants are reported in table I. The preoperative localization of the abnormal parathyroid gland was done with neck ultrasonography (US), and for some cases with cervical CT scan and ^{99m}Tc-sestamibi scintigraphy.

The imaging modalities used for the evaluation of renal calcifications were kidney and urinary tract US

using a 2–5-MHz-wide band convex transducer and abdominal CT scan. The presence of kidney stones or nephrocalcinosis, the size of stones (diameter < 5 mm, between 5 and 10 mm, > 10 mm), and unilateral or bilateral renal involvement was noted. We considered patients with a history of calculi emission or ongoing symptoms associated with the presence of kidney stones or nephrocalcinosis as symptomatic, whereas those with positive imaging findings alone as asymptomatic.

Following a baseline evaluation, patients were categorized into two groups: group A, which included those with renal calcifications, and group B, which comprised those without renal calcifications.

Statistical analysis

We used the Statistical Package for the Social Sciences, version 21 (IBM, Armonk, NY) for all analyses. The Kolmogorov-Smirnov and Shapiro-Wilk tests were used to test the normality of the data. Continuous variables are presented as means and standard deviations (SDs) or medians and interquartile range (IQR) as appropriate, while dichotomous variables are presented as absolute numbers and percentages. Levene's test was used to test for variance homogeneity. For comparison between the two groups, Student's t-test or the Mann-Whitney U test was used for continuous variables, and for categorical variables, the chi-square test or Fisher's exact test was used. We performed a backward multivariate analysis using binary logistic regression models on predictor variables with a p -value < 0.10 (a pre-determined cut-off from univariate analysis). We then sequentially removed variables starting with those having the highest p -values, retaining only those with a significant p -value in the final model. Statistical significance was defined by a p -value < 0.05.

Ethics approval

This study is registered in the Research registry under the number: researchregistry7745, and was approved by the Ethics Board Committee of Biomedical Research at our local faculty (CERBO) under the reference number: 22/2020. Considering the retrospective type of study, we worked on medical records, and all involved patients provided oral informed consent to the use of their medical data.

Results

The mean age of studied patients was 54.7±13.2 years, represented mainly by females (80.6%). Postoperative pathological examination revealed parathyroid adenoma in 91.1% of patients, and hyperplasia and parathyroid carcinoma in 6.7% and 2.2% of patients respectively. The baseline characteristics of this PHPT cohort are described in table I.

Among the 62 patients, 30 (48.4%) had renal calcifications, all of which were nephrolithiasis (100%), and only 2 cases (6.7%) had nephrocalcinosis associated with nephrolithiasis. The majority had occult kidney stones (73.3%) and the two cases of nephrocalcinosis were

asymptomatic.

Abdominal CT scan detected all the renal stones (100%) and the two cases of nephrocalcinosis, meanwhile, kidney US identified only 21 cases (70%). Renal calculi size ranged from 5 to 10 mm in more than half of patients. Most patients had unilateral stones mainly in the left kidney on both imaging techniques: 12 patients (57.2%) had unilateral stones and 9 (42.8%) bilateral stones in the US, while CT identified 17 (56.7%) cases with unilateral and 13 (43.3%) bilateral stones.

Compared to patients without renal calcifications, those with renal calcifications were generally younger, although this difference was not statistically significant. There were no differences in gender, duration of PHPT, or the presence of arterial hypertension. However, they had significantly higher BMI ($p=0.028$), waist circumference ($p=0.028$), and prevalence of diabetes ($p=0.04$). In terms of biochemical findings, they tended to have higher levels

of serum calcium, and 24-hour urinary calcium, although not statistically significant, and significantly higher levels of PTH 1- 84, ALP and lower 25-(OH)-D. Meanwhile, there was no significant difference between the two groups regarding the level of eGFR, serum phosphate, magnesium, and 24-hour urinary phosphate (Table I).

All the patients had preoperative parathyroid imaging to determine the localization of the abnormal parathyroid gland. We excluded those with double localization (one patient in group A and two patients in group B). We observed that most of the abnormal parathyroid gland was located in the inferior glands (82.2%) in all our PHPT patients. However, in group A, the affected parathyroid gland was located particularly on the left side in 19 cases (65.5%). Meanwhile, patients of group B had their abnormal parathyroid gland primarily located on the right side in 17 cases (56.7%), without statistically significant difference ($p=0.087$) (Table II).

Table I. Baseline characteristics and comparison of PHPT patients with and without renal calcifications.

Variables	Baseline population (n=62)	Renal calcifications (+) (30 cases)	Renal calcifications (-) (32 cases)	p value
Age, years	54.7±13.2 (18 – 82)	51.4±10.7	57.7±14.7	0.063
Sex				
Male, n (%)	12 (19.4%)	6 (20%)	6 (18.8%)	0.901
Female, n (%)	50 (80.6%)	24 (80%)	26 (81.3%)	
Menopause, n (%)	39 (78%)	18 (75%)	21 (80.8%)	0.62
BMI, kg/m ² (18.5 – 24.99)	26.8±5.4	28.5±4.8	25.5±5.5	0.028
Waist circumference				
M ≤ 94 cm	93.4±13.3	97.7±11.7	89.8±13.7	0.028
F ≤ 80 cm				
Duration of PHPT, months	5.5 (2 – 24)	8 (2 – 24)	5 (2.5 – 24)	0.858
Diabetes, n (%)	12 (19.3%)	9 (30%)	3 (9.4%)	0.040
Arterial hypertension, n (%)	21 (33.9%)	13 (43.3%)	8 (25%)	0.127
Albumin-adjusted serum calcium, mg/l (84 – 102) ^a	121 (113.5 – 135)	126 (116.5 – 148.5)	120 (111 – 130.7)	0.146
Serum phosphate, mg/l (23 – 47) ^a	22.5±5.7	21.4±6.7	23.6±4.5	0.155
Serum magnesemia, mg/l (16 – 26) ^a	19.7±3.8	20±4.7	19.5±3.1	0.746
Serum 25- (OH)-D, ng/ml (≥30) ^a	14 (8 – 24.05)	9.6 (7.6 – 19.2)	19.5 (12.9 – 27.1)	0.018
PTH 1- 84, pg/ml (7 – 37) ^a	224 (138 – 541.1)	253.8 (184 – 694.3)	172.5 (125.2 – 372.5)	0.032
Creatinine, mg/l (M: 7.2 – 12.5 F: 5.7 – 11.1)	7.2 (6 – 10.04)	7.4 (5.9 – 10.01)	7.1 (6.2 – 11)	0.29
eGFR, ml/min/1.73m ² (≥90)	90.3±29.8	93.1±28.8	87.6±30.8	0.469
ALP, ui/l (40 – 150) ^a	156 (98 – 230)	190 (129 – 290)	117 (71.2 – 193.5)	0.005
24-hour urinary calcium, mg/24h (100 – 300) ^a	416±201	463.4±182.1	374.2±214.8	0.098
24-hour urinary phosphate, mg/24h (0.40 – 1.30) ^a	0.45 (0.26 – 0.63)	0.41 (0.26 – 0.55)	0.55 (0.25 – 0.67)	0.489

^a: Reference range at our laboratory; n: number; %: percentage; M: males; F: females

Table II. Localization of abnormal parathyroid gland of PHPT patients with and without renal calcifications.

Variables	Renal calcifications (+)	Renal calcifications (-)	p value
Right side, n (%)	10 (34.5%)	17 (56.7%)	0.087
Left side, n (%)	19 (65.5%)	13 (43.3%)	

Table III. Cervical ultrasound characteristics of abnormal parathyroid gland of PHPT patients with and without renal calcifications.

Variables	Renal calcifications (+)	Renal calcifications (-)	p value	
Echogenicity	Hypochoic, n (%)	20 (66.7%)	22 (78.6%)	
	Isoechoic, n (%)	3 (10%)	3 (10.7%)	
	Hyperechoic, n (%)	3 (10%)	None	0.503
	Anechoic, n (%)	1 (3.3%)	None	
	Heterogeneous, n (%)	3 (10%)	3 (10.7%)	
Size, mm	18.4±8.5	18.5±8.3	0.957	

Table IV. Histopathological evaluation of the excised abnormal parathyroid gland of PHPT patients with and without renal calcifications.

Variables	Renal calcifications (+)	Renal calcifications (-)	p value	
Histology	Adenoma, n (%)	21 (87.5%)	20 (95.2%)	
	Hyperplasia, n (%)	2 (8.3%)	1 (4.8%)	1.00
	Carcinoma, n (%)	1 (4.2%)	None	
Weight, g	3 (2 – 5.9)	4 (3.3 – 4.8)	0.324	

Table V. Binary logistic regression analysis of PHPT patients with and without renal calcifications.

Variables	OR	95% CI	p value
BMI, kg/m ²	1.214	(1.051 – 1.401)	0.008
PTH 1- 84, pg/ml	1.002	(1.000 – 1.004)	0.133
Serum 25- (OH)-D, ng/ml	0.927	(0.860 – 1.000)	0.049

The ultrasound presentation of the majority of abnormal parathyroid glands was hypoechoic for both groups A and B (66.7% and 78.6% respectively) (Table III). No significant difference between the two groups was found in terms of ultrasound echogenicity ($p=0.503$), and size of the diseased gland on parathyroid imaging (18.4 ± 8.5 vs 18.5 ± 8.3 , $p=0.957$) (Table III).

Six patients were excluded from group A due to missing data or refusal to undergo surgery, while 11 patients were excluded from group B. Histopathological evaluation in both groups A and B has confirmed the presence of parathyroid adenoma as the principal etiology (87.5% and 95.2% respectively). However, hyperplasia was found in 8.3% and 4.8% of cases respectively. Only one case (4.2%) had parathyroid carcinoma and was complicated by both nephrocalcinosis and nephrolithiasis. The median weight of the removed parathyroid gland of patients with renal calcifications was non-significantly lower than the other

group ($p=0.324$) (Table IV).

In multivariate regression analysis, only BMI and 25-(OH)-D were found to be risk factors of renal calcifications (Table V).

Discussion

In this study, we found a prevalence of renal calcifications to be 48.4%, all of them were cases of nephrolithiasis (100%), and among them, 2 (6.7%) had both nephrolithiasis and nephrocalcinosis. The majority were asymptomatic and all cases of renal calcifications were identified through abdominal CT scans. When compared to patients without renal calcifications, patients with renal calcifications had significantly higher BMI, WC, prevalence of diabetes, levels of PTH 1- 84, ALP and lower levels of 25-(OH)-D. However, only a higher BMI and a lower 25-(OH)-D were potential risk factors for renal calcifications in multivariate regression analysis.

The prevalence of renal calcifications in studies is variable, with a rate ranging from less than 10% to over 50% [10,12–18]. The high prevalence in our cohort is explained by our systematic screening strategy for renal calcifications even in asymptomatic patients, which strengthens the recommendation of the latest guidelines [19].

In our study, all the patients underwent a routine screening of renal complications with kidney US, and abdominal CT scan. The latter test detected all the cases of renal stones and nephrocalcinosis. Meanwhile, kidney US identified only 70% of stones and none of the 2 cases of nephrocalcinosis. Our results are in line with a meta-analysis evaluating the diagnostic performance of low-dose CT scan as a diagnostic tool for nephrolithiasis and reported a pooled sensitivity of 97% and a pooled specificity of 95% [20]. Another study by Selberherr A et al. found a significantly higher number of detected stones with CT scans than with US (41 vs 4, $p=0.00124$) [21]. Our result confirms the higher performance of CT scans in detecting renal calcifications and should be used for the routine evaluation of patients. Most of our patients had unilateral stones, consistent with the findings of Suh et al. [12] and Ejlsmark-Svensson et al. [10]. The size of the stones ranged from 5 to 10 mm, whereas Suh et al. reported that the majority of renal stones were smaller than 5 mm [12].

We found that patients with renal calcifications were younger compared to those without renal calcifications, though not statistically significant, and no difference in gender was found between these two groups. In most studies, male gender and younger age were risk factors [16,17,22], suggesting that younger individuals may be at higher risk of nephrolithiasis because of higher renal mass, greater renal production of 1,25-dihydroxyvitamin D than elderly patients, causing a higher intestinal calcium absorption, and consequently, greater calciuria [23]. Former studies have shown a positive association between higher BMI and risk of renal stones in the general population [24], and increased BMI in patients with PHPT [25]. Tran H et al. reported that obese patients with PHPT are more likely to present with hypercalciuria and nephrolithiasis when compared with non-obese PHPT patients independently of serum calcium and PTH concentration, which follows our study [26].

A survey of the literature over the past years has revealed a considerable disparity in the biochemical presentation of PHPT between stone-formers and non-formers. In our study, patients with renal calcifications had significantly higher levels of PTH 1-84, which is in line with some studies [10,13,27–29]. However, other studies showed no difference between the two groups [16,17]. We were unable to find any statistically significant difference concerning serum calcium levels between the two groups, a result that is supported by other studies [16,17,22,30–33]. A lower value of phosphatemia, even though not

significant, was noted in patients with renal calcifications, which corroborates the results of Düger et al. who found PHPT patients with hypophosphatemia to have higher rates of renal stones [34].

A lower vitamin D is associated with greater levels of PTH in patients with PHPT and consequently a more severe presentation [35]. Following Elkoushy et al. [16], we have found PHPT stone formers to have significantly lower stores of vitamin D when compared with non-stone formers. However, in other reports, no difference was noted in terms of levels of 25-(OH)-D [10,13,14,17,28,31,36,37].

An increased level of ALP was significantly associated with renal calcifications in our report matching the result of F. Valle Díaz de la Guardia et al. [29] and Perez et al. [36], reflecting a greater rate of bone turnover in this group.

In terms of renal function, our findings although not statistically significant support the conclusion of earlier reports that found a higher eGFR in patients with renal calcifications [10,14,17,28,31,32]. Nevertheless, higher eGFR is associated with a higher renal excretion and consequently hypercalciuria [38], which explains the higher average value in stone formers than in non-stone formers in our study. The results in the literature have been discrepant regarding hypercalciuria; some studies have reported hypercalciuria as a risk factor for renal calcifications [10,27,29,31,33,36,39,40], but not others [16–18,30,32], as nephrolithiasis may occur even in the absence of hypercalciuria. Khan et al. [41] recommended the assessment of other urinary factors such as uric acid, magnesium, oxalate, citrate, and cystine. Hypomagnesuria, hypocitraturia, hyperoxaluria, hyperuricosuria, and cystinuria are associated with an increased risk of nephrolithiasis, which suggests further evaluation of other urinary parameters in PHPT patients as they may contribute to kidney stones [27,31,42]. Moreover, genetic factors may contribute to the development of renal calculi in PHPT, Scillitani et al. reported that patients harboring the AGQ haplotype of the CaSR are at a greater risk of developing renal calculi, whereas those with the SRQ haplotype having a lower risk, supporting the multifactorial etiology behind renal calcifications [43].

Parathyroid imaging has located the abnormal parathyroid gland mainly in the inferior glands (82.2%). This finding supports previous studies, which have also observed a tendency for abnormal glands to be located in the inferior glands [44,45]. We found that patients with renal calcifications had their affected parathyroid gland mainly in the left inferior parathyroid gland, in contrast to those without renal calcifications who had their abnormal parathyroid gland located mainly in the right inferior gland. Our findings support the conclusion of Csupor et al., who linked the location of the adenoma in the left inferior gland with urolithiasis [46]. Regarding the size and ultrasound echogenicity of the diseased gland, no difference was found

between the two groups, and to our knowledge echogenicity has not been compared before in studies.

In accordance with literature data [47], the principal etiology of PHPT was a parathyroid adenoma in both groups. The groups did not differ in the weight of the removed gland, similarly to the results of Mollerup et al. [22] and Agrawal et al. [48].

Our study has several strengths. To our knowledge, this is the first study comparing PHPT patients with and without renal calcifications in North Africa. All patients had their laboratory measurements, and renal imaging performed at the same hospital laboratory and radiology units, and all the PHPT patients had a routine screening of renal calcifications as recommended [19], which minimizes selection bias. Extensive demographic, clinical, biological, radiological, and histological data were included in this study.

The limitations of our study were the fact that it was a monocentric study with a small size of PHPT patients included and the limiting urinary profile of our PHPT patients who did not include all the parameters involved in the causative mechanism of stone formation.

Conclusions

To conclude, we found a high prevalence of 48.4% of renal calcifications in patients with primary hyperparathyroidism, mainly asymptomatic, and all of them detected by CT scan, suggesting its higher performance and its use routinely as the first-line imaging device even in asymptomatic patients. The principal etiology of PHPT was parathyroid adenoma in both groups, located mainly in the left inferior parathyroid gland in those with renal calcifications. Risk factors associated with the occurrence of renal calcifications were higher BMI, and lower 25-(OH)-D.

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