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Biochemical Profile and Operational Phenotypes of Elevated Parathyroid Hormone in a Non-Renal Adult Hospital Laboratory Cohort

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Abstract **Original Research Article**

Background: Elevated parathyroid hormone (PTH) is a common laboratory finding, but its interpretation depends on calcium status, renal function, phosphate, and vitamin D. In hospital-based populations, chronic kidney disease and vitamin D deficiency are major confounders, particularly when PTH elevation occurs without hypercalcemia. This study aimed to describe the biochemical profile and operational phenotypes of elevated PTH in a non-renal adult hospital laboratory cohort. ***Methods:*** We performed a retrospective, single-center, laboratory-based study over January–December 2025. Adults with elevated PTH and same-date measurements of total calcium, albumin, phosphate, creatinine, and 25-hydroxyvitamin D [25(OH)D] were eligible. Patients with estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m² were excluded to minimize inclusion of chronic kidney disease-related secondary hyperparathyroidism. Albumin-corrected calcium was calculated, and operational phenotypes were defined according to corrected calcium and vitamin D status using a 30 ng/mL 25(OH)D cutoff. Associations between PTH and biochemical variables were assessed using Spearman rank correlation. ***Results:*** The final analytical cohort included 168 non-renal adults (78.6% female) with a median age of 55.0 years (IQR 43.0–62.0). Median PTH was 107.0 pg/mL (IQR 85.8–133.0), median corrected calcium was 88.6 mg/L (IQR 85.2–92.5), and median 25(OH)D was 14.35 ng/mL (IQR 10.0–24.0). Vitamin D deficiency [25(OH)D <30 ng/mL] was present in 83.3% of patients, including 23.2% with severe deficiency (<10 ng/mL). Most patients were normocalcemic (73.2%), while 22.6% were hypocalcemic and 4.2% were hypercalcemic. The predominant operational phenotype was an elevated-PTH profile likely related to vitamin D deficiency (79.2%). An operational normocalcemic hyperparathyroid phenotype without vitamin D deficiency accounted for 15.5% of cases, while a hypercalcemic phenotype compatible with probable primary hyperparathyroidism accounted for 4.2%. PTH showed weak but significant correlations with corrected calcium (ρ=0.163, p=0.035), phosphate (ρ=-0.208, p=0.007), and age (ρ=0.223, p=0.004). ***Conclusion:*** In this non-renal adult hospital laboratory cohort, low vitamin D status was the dominant biochemical context associated with elevated PTH, supporting secondary hyperparathyroidism as the most likely explanation in routine practice. A smaller subgroup with normocalcemic elevated PTH and no vitamin D deficiency remained present, but this pattern should be interpreted cautiously and viewed as an operational phenotype requiring further evaluation rather than a definitive diagnosis.

Keywords: parathyroid hormone; secondary hyperparathyroidism; vitamin D deficiency; normocalcemic hyperparathyroidism; corrected calcium; phosphate.

INTRODUCTION

Elevated parathyroid hormone (PTH) is a common laboratory finding with a broad differential diagnosis. It may reflect secondary hyperparathyroidism related to vitamin D deficiency or impaired calcium balance, but it may also point to primary hyperparathyroidism (PHPT) with autonomous PTH secretion. For that reason, an isolated increase in PTH should never be interpreted without considering calcium status (total calcium, albumin-corrected calcium, and ideally ionized calcium when available), renal function, phosphate, and vitamin D status [1,2].

Chronic kidney disease (CKD) is one of the main confounders in the evaluation of elevated PTH because CKD-related mineral and bone disorder (CKD-MBD) frequently leads to secondary hyperparathyroidism with disturbances in phosphate and

calcium metabolism [3]. Current recommendations therefore stress the importance of ruling out CKD and other secondary causes before considering PHPT, particularly in normocalcemic presentations [1,4]. In hospital-based populations, CKD-related secondary hyperparathyroidism often accounts for a substantial share of elevated-PTH cases; excluding patients with renal impairment can therefore provide a clearer view of non-renal biochemical patterns that might otherwise remain obscured.

Vitamin D deficiency is also highly prevalent in many settings and is a well-recognized cause of secondary PTH elevation in adults [5,6]. In routine hospital practice, many patients with elevated PTH do not present with overt hypercalcemia, which makes interpretation more challenging and increases the risk of overcalling primary parathyroid disease if renal dysfunction and vitamin D status are not assessed systematically.

Against this background, we sought to describe the biochemical profile of elevated PTH in a non-renal adult cohort from a tertiary hospital laboratory and to propose a practical laboratory-based phenotypic stratification using corrected calcium and vitamin D status after exclusion of CKD-related cases.

MATERIALS AND METHODS

Study design and setting

We conducted a retrospective, observational, single-center laboratory-based study in the Biochemistry Laboratory of Arrazi Hospital, CHU Mohammed VI (Marrakech, Morocco), using routine testing data generated during the study period. All biochemical analyses were performed on the Abbott Alinity ci platform according to the manufacturer's procedures and internal laboratory quality practices.

Study period and eligibility criteria

Patients with elevated intact PTH (iPTH) were identified from laboratory records collected between January and December 2025. For this analysis, we focused on non-renal adults with complete biochemical data allowing standardized calcium correction and vitamin D stratification. Restricting the study to adults was intended to create a more homogeneous population and to avoid age-related physiological differences in PTH and mineral metabolism, while exclusion of renal impairment reduced the predominance of CKD-related secondary hyperparathyroidism and allowed a clearer description of non-renal elevated-PTH profiles.

Inclusion criteria:

1. Elevated intact PTH (iPTH) >65 pg/mL.
2. Adult age ≥18 years.
3. Available measurements on the same sampling date for iPTH (pg/mL), total calcium (mg/L), albumin (g/L), phosphate (mg/L), creatinine (mg/L), and 25-hydroxyvitamin D [25(OH)D] (ng/mL).
4. Non-renal status defined as eGFR ≥60 mL/min/1.73 m², calculated from serum creatinine.

For patients with multiple eligible laboratory records, a single record per patient was retained (one measurement per patient), selecting the earliest eligible sampling date within the study period.

From the initial dataset of patients with elevated iPTH during January–December 2025, we selected adults with same-date availability of total calcium, albumin, phosphate, creatinine, and 25-hydroxyvitamin D. We then excluded patients with eGFR <60 mL/min/1.73 m² to minimize inclusion of CKD-related secondary hyperparathyroidism. After applying these criteria and retaining only one eligible record per patient, the final analytical cohort consisted of 168 non-renal adult patients.

Exclusion criteria:

- eGFR <60 mL/min/1.73 m² (to exclude CKD-associated secondary hyperparathyroidism) [3,4].
- Age <18 years.
- Missing 25(OH)D result.

Laboratory methods and derived variables

Albumin-corrected calcium (Ca_corr) was computed to standardize calcium interpretation using the following formula (units as measured in our laboratory): Ca_corr (mg/L) = total calcium (mg/L) + 0.8 × (40 − albumin [g/L]).

Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI 2021 (race-free) creatinine equation. Creatinine values measured in mg/L were converted to mg/dL prior to eGFR computation. The threshold of eGFR ≥60 mL/min/1.73 m² was used operationally to minimize inclusion of CKD-related secondary hyperparathyroidism in this laboratory-based dataset, consistent with commonly used thresholds for excluding CKD in evaluations of elevated PTH and normocalcemic phenotypes [1,4].

Biochemical categories (threshold definitions)

Corrected calcium (mg/L): hypocalcemia <85; normocalcemia 85–105; hypercalcemia >105. Phosphate (mg/L): hypophosphatemia <25; normal 25–45; hyperphosphatemia >45. 25(OH)D (ng/mL): deficiency <30; severe deficiency <10. A 30 ng/mL cutoff was used to improve exclusion of secondary hyperparathyroidism related to vitamin D insufficiency and to reduce misclassification of normocalcemic hyperparathyroid phenotypes [4-6].

Operational phenotype classification

To describe patterns of elevated PTH in this non-renal cohort, we used a pragmatic laboratory-based

classification that combined corrected calcium status and vitamin D status: (1) elevated-PTH profile likely related to vitamin D deficiency: 25(OH)D <30 ng/mL with normocalcemia or hypocalcemia; (2) operational normocalcemic hyperparathyroid phenotype without vitamin D deficiency: corrected calcium 85–105 mg/L with 25(OH)D ≥30 ng/mL; (3) hypercalcemic phenotype compatible with probable primary hyperparathyroidism: corrected calcium >105 mg/L; and (4) hypocalcemic elevated-PTH phenotype without vitamin D deficiency: corrected calcium <85 mg/L with 25(OH)D ≥30 ng/mL. This classification was intended for descriptive stratification rather than definitive etiologic diagnosis [1,2]. In particular, the hypercalcemic phenotype was considered only biochemically compatible with probable primary hyperparathyroidism and not diagnostic in itself, as confirmation would require repeat biochemical testing and clinical evaluation.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 29.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as median and interquartile range (IQR) because the biochemical variables were not assumed to follow a normal distribution. Categorical variables were expressed as counts and percentages. Associations between PTH and continuous variables were assessed using Spearman rank correlation coefficients. All tests were two-sided, and p-values <0.05 were considered statistically significant. Only patients with complete data for the variables required for the present analysis were included in the final analytical cohort.

RESULTS

Cohort selection and demographic characteristics

After excluding patients with reduced eGFR consistent with chronic kidney disease (eGFR <60 mL/min/1.73 m²) and restricting the analysis to adults (≥18 years) with available 25-hydroxyvitamin D [25(OH)D], the final analytical cohort included 168 patients with elevated PTH. The cohort comprised 132 females (78.6%) and 36 males (21.4%), with a median age of 55.0 years (IQR 43.0–62.0) and a range of 18–86 years. Median eGFR was 105.0 mL/min/1.73 m² (IQR 92.4–112.8). Baseline characteristics and categorical distributions are summarized in Table 1.

Table 1: Baseline characteristics and biochemical categories (n=168)

Variable	Value
Patients, n	168
Age (years), median (IQR)	55.0 (43.0–62.0)
Female, n (%)	132 (78.6%)
PTH (pg/mL), median (IQR)	107.0 (85.8–133.0)
Corrected calcium (mg/L), median (IQR)	88.6 (85.2–92.5)
Phosphate (mg/L), median (IQR)	35.0 (31.0–39.0)
25(OH)D (ng/mL), median (IQR)	14.35 (10.00–24.00)
Creatinine (mg/L), median (IQR)	6.0 (5.0–8.0)
eGFR (mL/min/1.73 m²), median (IQR)	105.0 (92.4–112.8)
Corrected calcium categories	
Hypercalcemia (>105 mg/L), n (%)	7 (4.2%)
Normocalcemia (85–105 mg/L), n (%)	123 (73.2%)
Hypocalcemia (<85 mg/L), n (%)	38 (22.6%)
Phosphate categories	
Hyperphosphatemia (>45 mg/L), n (%)	11 (6.5%)
Normal phosphate (25–45 mg/L), n (%)	141 (83.9%)
Hypophosphatemia (<25 mg/L), n (%)	16 (9.5%)
25(OH)D categories	
Deficiency (<30 ng/mL), n (%)	140 (83.3%)
Severe deficiency (<10 ng/mL), n (%)	39 (23.2%)
PTH categories	
65–149 pg/mL, n (%)	134 (79.8%)
150–299 pg/mL, n (%)	26 (15.5%)
300–599 pg/mL, n (%)	5 (3.0%)
≥600 pg/mL, n (%)	3 (1.8%)

Abbreviations: eGFR, estimated glomerular filtration rate; IQR, interquartile range; PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D.

Overall biochemical profile

In the overall cohort, median PTH was 107.0 pg/mL (IQR 85.8–133.0). Median total calcium was 89.0 mg/L (IQR 85.0–93.4) and median albumin was 43.0 g/L (IQR 39.0–46.0), yielding a median albumin-corrected calcium (Ca_corr) of 88.6 mg/L (IQR 85.2–92.5).

Median phosphate was 35.0 mg/L (IQR 31.0–39.0). Median 25(OH)D was 14.35 ng/mL (IQR 10.0–24.0), and median creatinine was 6.0 mg/L (IQR 5.0–8.0). The distribution of PTH values is shown in Figure 1.

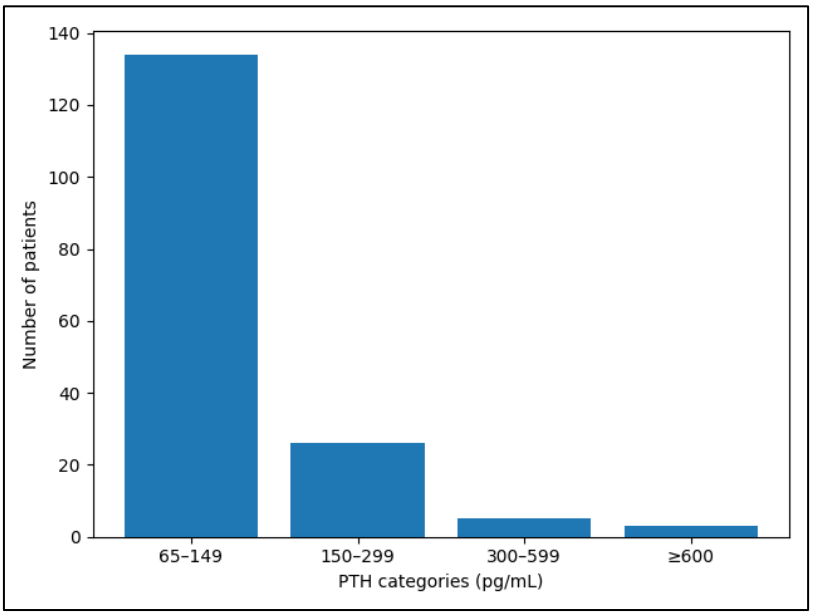

Figure 1: Distribution of PTH values in the non-renal adult cohort (n=168)

Corrected calcium categories

Based on Ca_corr, most patients were normocalcemic: 123/168 (73.2%) had Ca_corr between 85–105 mg/L. Hypocalcemia (Ca_corr <85 mg/L) was observed in 38/168 (22.6%), whereas hypercalcemia (Ca_corr >105 mg/L) was uncommon (7/168; 4.2%). These categories are detailed in Table 1. The relationship between PTH and Ca_corr is presented in Figure 2.

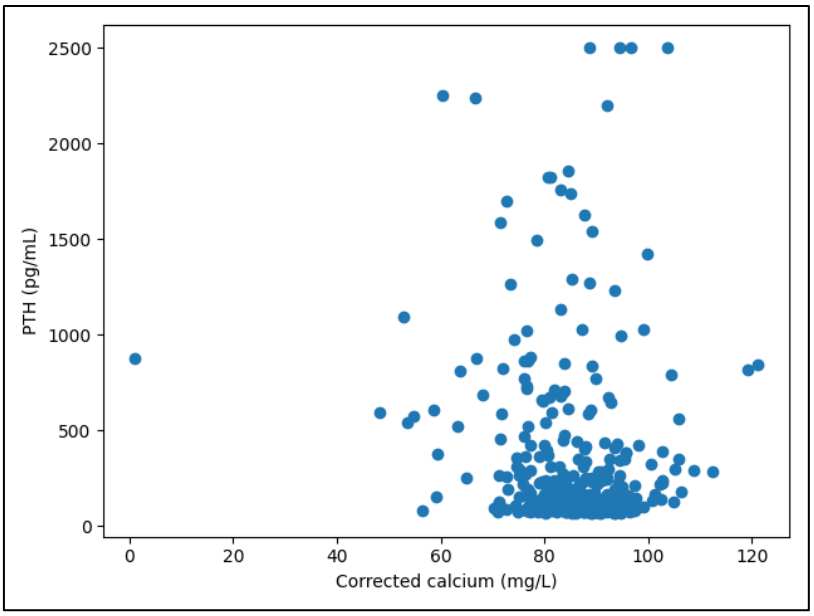

Figure 2: Scatter plot of PTH versus albumin-corrected calcium (n=168)

Phosphate abnormalities

Phosphate values were within the 25–45 mg/L range in 141/168 patients (83.9%). Hyperphosphatemia (>45 mg/L) was observed in 11/168 (6.5%), and hypophosphatemia (<25 mg/L) in 16/168 (9.5%) (Table 1). The association between PTH and phosphate is illustrated in Figure 3.

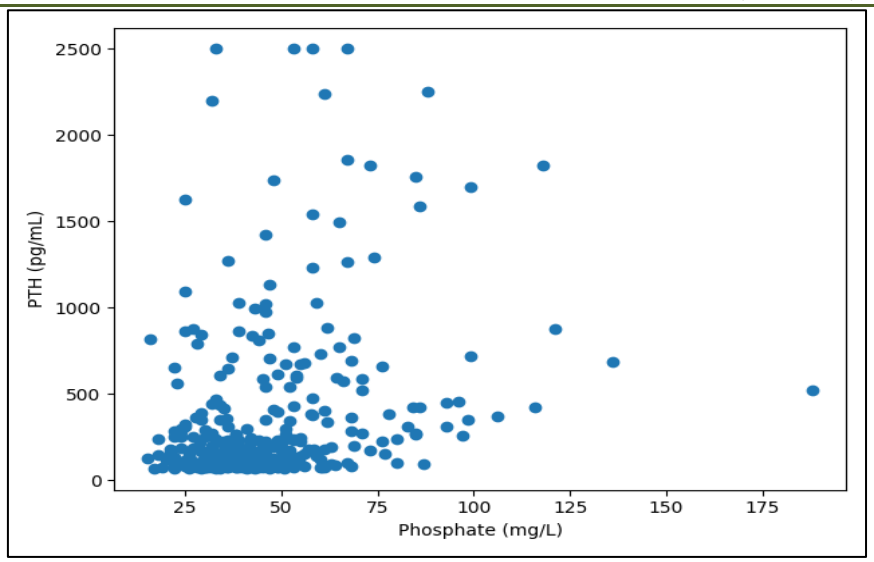

Figure 3: Scatter plot of PTH versus phosphate (n=168)

Vitamin D status

Vitamin D deficiency was very frequent in this non-renal adult cohort. 25(OH)D <30 ng/mL was found in 140/168 patients (83.3%), and severe deficiency (<10 ng/mL) in 39/168 patients (23.2%) (Table 1). These findings were consistently observed across the cohort and informed the operational phenotype classification described below.

Operational biochemical phenotypes

Using a laboratory-based classification that combined corrected calcium and vitamin D status, the most common pattern was an elevated-PTH profile likely related to vitamin D deficiency, observed in 133/168 patients (79.2%). An operational normocalcemic hyperparathyroid phenotype without vitamin D deficiency was identified in 26/168 patients (15.5%). A smaller subgroup showed a hypercalcemic phenotype compatible with probable primary hyperparathyroidism (7/168; 4.2%), while hypocalcemic elevated-PTH cases without vitamin D deficiency accounted for 2/168 patients (1.2%). These categories were used for descriptive biochemical stratification only and should not be interpreted as definitive etiologic diagnoses. In particular, the normocalcemic and hypercalcemic subgroups require cautious interpretation in the absence of repeat biochemical confirmation and complementary clinical evaluation. Phenotype-specific summary statistics are provided in Table 2, and the distribution of phenotypes is shown in Figure 4.

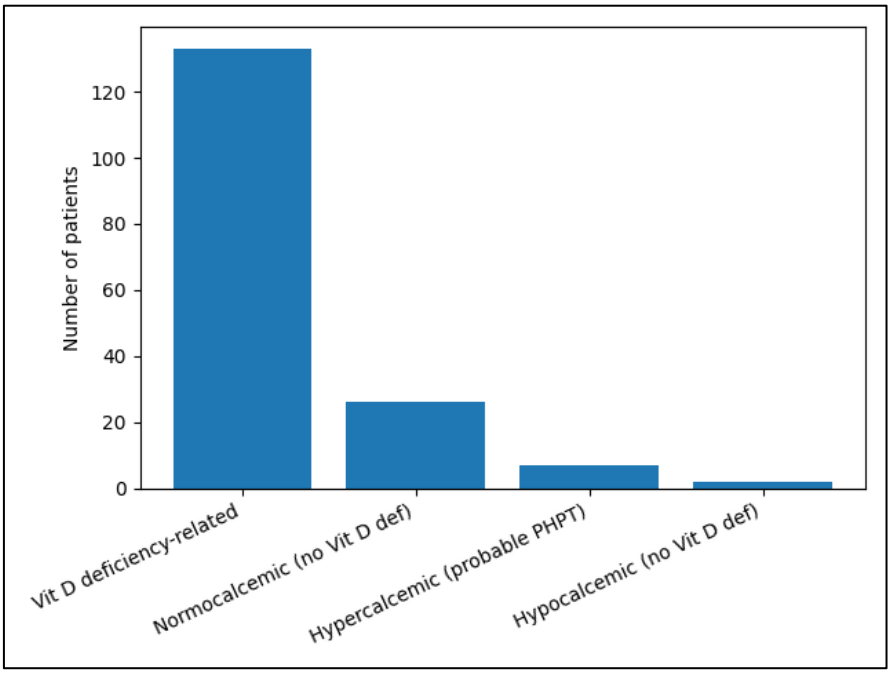

Figure 4: Distribution of phenotypes of elevated PTH in the non-renal adult cohort (n=168)

Table 2: phenotypes of elevated PTH and corresponding biochemical characteristics

Phenotype	n (%)	Age, median (IQR)	Female, n (%)	PTH, median (IQR) pg/mL	Corrected Ca, median (IQR) mg/L	Phosphate, median (IQR) mg/L	25(OH)D, median (IQR) ng/mL
Hypocalcemic elevated-PTH phenotype without vitamin D deficiency	2 (1.2%)	50.0 (39.5–60.5)	2 (100.0%)	109.5 (100.2–118.8)	81.7 (81.2–82.1)	28.0 (25.0–31.0)	34.00 (33.00–35.00)
Operational normocalcemic hyperparathyroid phenotype without vitamin D deficiency	26 (15.5%)	58.5 (52.2–62.0)	21 (80.8%)	102.5 (83.5–113.5)	90.1 (87.8–93.0)	36.5 (34.0–41.8)	34.00 (32.00–42.25)
Hypercalcemic phenotype compatible with probable primary hyperparathyroidism	7 (4.2%)	57.0 (50.0–60.0)	6 (85.7%)	300.0 (286.5–457.0)	106.3 (105.8–110.6)	24.0 (23.5–26.5)	12.00 (8.00–13.50)
Elevated-PTH profile likely related to vitamin D deficiency	133 (79.2%)	53.0 (41.0–62.0)	103 (77.4%)	107.0 (85.0–127.0)	87.6 (84.4–91.8)	35.0 (31.0–39.0)	13.00 (9.00–19.00)

Data are presented as median (IQR) unless otherwise specified.

Correlation analyses

Spearman correlation analyses (n=168 for each pair) showed a weak positive association between PTH and corrected calcium ($\rho = 0.163$, $p = 0.035$) and a weak inverse association between PTH and phosphate ($\rho = -0.208$, $p = 0.007$). PTH was positively correlated with age ($\rho = 0.223$, $p = 0.004$). The correlations between PTH and 25(OH)D ($\rho = -0.141$, $p = 0.068$) and between PTH and creatinine ($\rho = 0.125$, $p = 0.105$) did not reach statistical significance. All correlation results are summarized in Table 3.

Table 3: Spearman correlations with PTH (n=168)

Association	Spearman rho	p-value	n
PTH vs corrected calcium	0.163	0.035	168
PTH vs phosphate	-0.208	0.007	168
PTH vs 25(OH)D	-0.141	0.068	168
PTH vs age	0.223	0.004	168
PTH vs creatinine	0.125	0.105	168

DISCUSSION

Principal findings in context

In this non-renal adult hospital cohort with elevated PTH and preserved renal function, the overall biochemical picture was dominated by low vitamin D status and by profiles consistent with secondary hyperparathyroidism. Most patients were normocalcemic, while clearly hypercalcemic presentations were uncommon. This pattern fits with current diagnostic frameworks, which recommend interpreting elevated PTH in light of calcium, renal function, phosphate, and vitamin D before considering primary hyperparathyroidism, particularly in normocalcemic settings [1,2].

Vitamin D deficiency burden: comparison with Morocco and the region

Low vitamin D status was strikingly common in our cohort: 83.3% of patients had 25(OH)D concentrations below 30 ng/mL, and 23.2% had severe deficiency below 10 ng/mL. Although direct comparisons across studies must be made with caution because of differences in thresholds, assays, seasonality, and case mix, this finding is in keeping with the high burden of hypovitaminosis D reported in Morocco and across the broader Arab region [7,8]. In practical terms, such a background prevalence creates a strong biochemical setting for secondary PTH elevation even in patients without reduced eGFR.

Secondary hyperparathyroidism outside CKD

The predominance of the vitamin D-related phenotype suggests that secondary hyperparathyroidism remains the most likely explanation for elevated PTH even after patients with reduced eGFR are excluded. This matters clinically because elevated PTH in non-renal patients may be interpreted too quickly as evidence of intrinsic parathyroid disease, whereas in many cases it is more plausibly an adaptive response to impaired calcium balance in the setting of inadequate vitamin D status [4,5,9]. Our findings therefore support systematic assessment—and, when appropriate, correction—of vitamin D status as a key step in the evaluation of elevated PTH in routine hospital practice.

Normocalcemic hyperparathyroidism without vitamin D deficiency

A clinically meaningful minority of patients showed a normocalcemic phenotype without vitamin D deficiency. However, this subgroup should not be equated with confirmed normocalcemic primary hyperparathyroidism. In routine laboratory datasets, elevated PTH with normal corrected calcium at a single time point is not sufficient for diagnosis, especially because reported prevalence varies widely depending on whether repeated measurements and rigorous exclusion of other secondary causes have been performed [4,10,11]. Accordingly, this subgroup is better viewed as an operational biochemical phenotype that identifies patients who warrant further evaluation rather than as a definitive disease category.

Some cases in this subgroup may represent early or mild primary hyperparathyroidism, whereas others may reflect residual secondary mechanisms or transient biological variation that cannot be resolved from a single retrospective measurement [1,2,4].

Hypercalcemic phenotype and probable primary hyperparathyroidism

Only a small fraction of patients showed a hypercalcemic phenotype compatible with probable primary hyperparathyroidism. This low proportion is not surprising in a general hospital laboratory cohort and is likely lower than what would be observed in endocrine referral or surgical series. Even so, this category should still be interpreted with caution, because retrospective biochemical classification at a single time point cannot replace a full diagnostic work-up. Definitive diagnosis would require repeat biochemical confirmation together with appropriate complementary evaluation, including clinical assessment, urinary calcium measurement, and assessment for skeletal or renal complications when indicated [1,2].

Correlation patterns after excluding CKD

The correlations observed in this study were statistically significant but modest, suggesting that elevated PTH in non-renal adults arises within a heterogeneous biochemical setting rather than from a single dominant mechanism. The weak positive correlation between PTH and corrected calcium and the weak inverse correlation with phosphate are biologically plausible, but their limited strength highlights substantial inter-individual variability. The absence of a significant association between PTH and creatinine is consistent with the deliberate exclusion of reduced eGFR, while the positive association with age agrees with the broader literature on age-related changes in mineral metabolism [1,2,9].

Strengths and limitations

This study has several strengths, including its focus on a non-renal adult cohort, the simultaneous availability of key biochemical variables, the use of albumin-corrected calcium, and the analysis of real-world hospital laboratory data. Nevertheless, several limitations should be acknowledged. The retrospective design did not allow access to clinical history, treatment exposure, calcium intake, symptoms, urinary calcium excretion, magnesium, or longitudinal follow-up. The phenotypes used here were operational and descriptive rather than diagnostic, and classification was based on a single time point. In addition, ionized calcium was not available, so calcium status was assessed using albumin-corrected total calcium rather than direct measurement of biologically active calcium. Restricting the analysis to patients with complete biochemical data may also have introduced selection bias toward individuals undergoing more extensive metabolic evaluation [4,10].

CONCLUSION

In this non-renal adult hospital laboratory cohort with elevated PTH, low vitamin D status was the predominant biochemical context associated with PTH elevation, supporting secondary hyperparathyroidism as the most common explanation in routine practice. A smaller normocalcemic subgroup without vitamin D deficiency remained present, but this pattern is best regarded as an operational phenotype requiring structured follow-up rather than as established primary disease. Overall, these findings support systematic vitamin D assessment and a stepwise laboratory-clinical approach to the evaluation of elevated PTH in adults without overt renal impairment.

Additional Information

Ethics approval and consent: This retrospective laboratory-based study used de-identified data obtained from routine care and was conducted with the agreement of the head of the Biochemistry Laboratory of Arrazi Hospital, CHU Mohammed VI, Marrakech, Morocco. Because the study involved retrospective analysis of fully de-identified laboratory data, with no direct patient contact or intervention, informed consent was waived in accordance with local institutional policy.

Conflicts of interest: The authors declare no competing interests.

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Data availability: The dataset is available from the corresponding author upon reasonable request, subject to institutional approval.

REFERENCES

1. Bilezikian JP, Khan AA, Silverberg SJ, *et al.,* Evaluation and Management of Primary Hyperparathyroidism: Summary Statement and Guidelines from the Fifth International Workshop. J Bone Miner Res. 2022;37(11):2293-2314. doi:10.1002/jbmr.4677.

2. Bollerslev J, Rejnmark L, Zahn A, *et al.,* European expert consensus on practical management of specific aspects of parathyroid disorders in adults and in pregnancy: recommendations of the ESE Educational Program of Parathyroid Disorders (PARAT 2021). Eur J Endocrinol. 2022;186(2): R33-R63. doi:10.1530/EJE-21-1044.
3. Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group. KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). Kidney Int Suppl (2011). 2017;7(1):1-59. doi: 10.1016/j.kisu.2017.04.001.
4. Cusano NE, Cetani F. Normocalcemic primary hyperparathyroidism. Arch Endocrinol Metab. 2022;66(5):666-677. doi:10.20945/2359-3997000000556.
5. Holick MF, Binkley NC, Bischoff-Ferrari HA, *et al.,* Evaluation, Treatment, and Prevention of Vitamin D Deficiency: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2011;96(7):1911-1930. doi:10.1210/jc.2011-0385.
6. Demay MB, *et al.,* Vitamin D for the Prevention of Disease: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2024;109(8):1907-1947. doi:10.1210/clinem/dgae290.
7. Zouine N, Lhilali I, Messaoudi A, El Jaafari S, Filali-Zegzouti Y. Gaps in Vitamin D Intake and Status in Moroccan Women. Epidemiologia. 2025;6(4):66. doi:10.3390/epidemiologia6040066.
8. Hassan AB, *et al.,* Prevalence of vitamin D deficiency in the Arab countries: a systematic review and meta-analysis. Discover Public Health. 2025. doi:10.1186/s12982-025-00993-w.
9. Lips P. Vitamin D deficiency and secondary hyperparathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. Endocr Rev. 2001;22(4):477-501. doi:10.1210/edrv.22.4.0437.
10. Zavatta G, Clarke BL. Normocalcemic Hyperparathyroidism: A Heterogeneous Disorder Often Misdiagnosed? JBMR Plus. 2020;4(8): e10391. doi:10.1002/jbm4.10391.
11. Schini M, Jacques RM, Oakes E, Peel NFA, Walsh JS, Eastell R. Normocalcemic Hyperparathyroidism: Study of its Prevalence and Natural History. J Clin Endocrinol Metab. 2020;105(4): e1171-e1186. doi:10.1210/clinem/dgaa084.