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Universal Hypovitaminosis D and Its Clinicobiological Correlates in Hormone Receptor-Positive Breast Cancer: A Single-Center Moroccan Study

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Abstract

Background and aim: Vitamin D deficiency is a widespread public health issue, yet data regarding its prevalence and biological correlates in North African women with hormone receptor-positive (HR+) breast cancer remain limited. This study aimed to assess serum 25-hydroxyvitamin D (25{OH}D) levels and evaluate their associations with clinicopathological features in women diagnosed with HR+ breast cancer in Marrakech, Morocco.

Methods: A cross-sectional study was conducted on 67 consecutive HR+ breast cancer patients treated at the Oncology Center of the Mohammed VI University Hospital in Marrakech between 2024 and 2025. Serum 25(OH)D concentrations were measured, and parametric (Pearson's r) and non-parametric (Spearman's ρ) correlation analyses were performed against clinicobiological variables.

Results: Hypovitaminosis D (<30 ng/mL) was present in 100% of the cohort (mean serum level: 12.26±9.4 ng/mL). Severe deficiency (<10 ng/mL) was detected in 47% (n=31), insufficiency (10-20 ng/mL) in 47% (n=32), and borderline insufficiency (20-30 ng/mL) in 6% (n=4). No patient had optimal levels (≥30 ng/mL). Serum 25(OH)D concentrations correlated positively with progesterone receptor (PR) expression (r = +0.616, p=0.005) and the Ki-67 index (ρ = +0.699, p=0.001), while demonstrating a significant inverse correlation with metastatic stage (M1 versus M0; ρ = -0.472, p=0.041).

Conclusion: Hypovitaminosis D is universal and severe among HR+ breast cancer patients in Marrakech, Morocco. Lower vitamin D status is associated with metastatic disease. These findings underscore an urgent need for systematic vitamin D screening and prospective interventional trials in this population.

Categories: Obstetrics/Gynecology, Radiation Oncology, Epidemiology/Public Health
Keywords: 25-hydroxyvitamin d, breast cancer, hormone receptor-positive, ki-67, progesterone receptor, vitamin d

Introduction

Hormone receptor-positive (HR+) breast cancer is the most common molecular subtype, accounting for roughly 70-75% of all breast cancers. Beyond its classical role in calcium-phosphate metabolism, vitamin D exerts pleiotropic effects through the vitamin D receptor (VDR), which is widely expressed in breast tissue and regulates proliferation, apoptosis, angiogenesis, and differentiation [1,2]. Low vitamin D status is frequent in breast cancer populations, but the reported prevalence varies widely. Pooled meta-analytic data indicate a mean baseline 25-hydroxyvitamin D of 26.88 ng/mL, with 45.28% of patients below 20 ng/mL and 67.44% below 30 ng/mL [1]. A prospective German cohort of newly diagnosed, non-metastatic patients reported a median of 24 ng/mL and 64.8% below 30 ng/mL [3]. Additional single-center cohorts, including studies in postmenopausal HR+ disease, have confirmed that deficiency is common [4,5]. Associations with tumor biology are mixed. Some studies link lower vitamin D to higher Ki-67, positive nodal status, estrogen receptor (ER) or progesterone receptor (PR) negativity, metastasis, or worse recurrence outcomes [6-9]. Others have found no significant association with Ki-67 or with clinicopathological factors [3,10].

Despite abundant sunlight, hypovitaminosis D remains frequent in Morocco, a pattern commonly attributed to veiling, higher skin pigmentation, sedentary lifestyle, and insufficient dietary intake. Limited data address Moroccan women with HR+ disease. This study evaluated the prevalence of vitamin D deficiency in Moroccan women with HR+ breast cancer and explored its correlations with clinicobiological, anatomopathological, and tumor variables. The cohort was restricted to HR+ tumors for the following two reasons: in our department, serum 25(OH)D is measured systematically as part of the pre-therapeutic work-up preceding endocrine therapy, ensuring a complete and homogeneous dataset for this subgroup; and HR+

tumors are the most common subtype, in which the VDR interacts with hormone-receptor signaling, making this population particularly suited to studying the vitamin D-hormone receptor axis [2].

Materials And Methods

Study design and setting

This was a single-center, cross-sectional, descriptive, and analytical study conducted in the Department of Radiation Oncology at Mohammed VI University Hospital, Marrakech, Morocco, between 2024 and 2025. All eligible patients diagnosed during this period were evaluated consecutively, without selection according to the season of blood sampling.

Participants

All women with a histologically confirmed diagnosis of hormone receptor-positive breast cancer who had a serum 25-hydroxyvitamin D measurement obtained in the absence of any ongoing vitamin D supplementation were included, yielding 67 consecutive patients. A serum 25(OH)D measurement was part of the routine pre-therapeutic work-up for all eligible patients during the study period. Patients receiving vitamin D supplementation at the time of sampling were excluded.

Variables and definitions

Collected variables were age, body mass index, menopausal status, full tumor, node, metastasis (TNM) stage, Scarff-Bloom-Richardson grade, estrogen receptor expression (%), progesterone receptor expression (%), human epidermal growth factor receptor 2 (HER2) status, Ki-67 index (%), serum 25-hydroxyvitamin D (ng/mL), and treatment received [11]. Estrogen receptor (ER) and PR were considered positive at 1% or more of stained tumor nuclei, and HER2 was scored from 0 to 3+ with in situ hybridization for equivocal (2+) cases, per American Society of Clinical Oncology (ASCO)/College of American Pathologists (CAP) guidelines [12,13].

Vitamin D assessment

Serum 25-hydroxyvitamin D status was classified into the following three categories: severe deficiency (below 10 ng/mL), insufficiency (10-20 ng/mL), and borderline insufficiency (20-30 ng/mL); levels of 30 ng/mL or above were considered normal, in line with international endocrine recommendations [14]. Blood samples were collected as part of the standard pretherapeutic workup before the initiation of oncological treatment, and patients were not selected based on the season of sample collection. Serum 25-hydroxyvitamin D was measured by an automated chemiluminescence immunoassay, the technique most commonly used in comparable breast cancer cohorts. This is an observational study; serum 25(OH)D was measured once at baseline, and no vitamin D was administered as part of the study; consequently, no dose, commercial brand, or route of administration applies.

Ethics

This study used fully anonymized data. Approval from the head of the Department of Radiation Oncology was obtained before data collection. Under local regulations, formal ethics committee approval was not required because the data were completely anonymized; therefore, the requirement for individual informed consent was waived. The study was conducted in accordance with the Declaration of Helsinki.

Statistical analysis

Analyses were performed with IBM SPSS Statistics (Armonk, NY: IBM Corp.). Descriptive statistics included means, standard deviations, and ranges. Bivariate associations were assessed with Pearson's correlation and non-parametric Spearman's rank correlation. Correlations were computed on a complete-case (pairwise) basis for each variable, so the number of patients with available data varied across variables. No adjustment for multiple comparisons and no multivariable modeling were applied, consistent with the exploratory aim of the analysis. A two-sided $p<0.05$ was considered statistically significant.

Results

Baseline characteristics

The baseline clinical and tumor characteristics of the 67 patients are summarized in Table *1*. Mean age was 51.7±8.7 years (range: 34-70 years), mean BMI was 26.9±5.2 kg/m^2, and 42 patients (62.7%) were postmenopausal. T2 and N1 were the most frequent T and N stages, and 28 patients (41.8%) had metastatic (M1) disease at diagnosis.

Characteristics	Values
Patients	67
Mean age, years	51.7±8.7 (range: 34-70)
Mean body mass index, kg/m²	26.9±5.2
Postmenopausal, n (%)	42 (62.7)
Predominant T stage	T2
Predominant N stage	N1
Metastatic (M1) at diagnosis, n (%)	28 (41.8)
Mean ER expression	55.8±30.9%
Mean PR expression	31.3±30.3%
Most frequent HER2 category	3+
Mean Ki-67 index	24.3±14.4%

TABLE 1: Baseline clinical and tumor characteristics of the cohort (n=67).

HER2: human epidermal growth factor receptor 2; PR: progesterone receptor; ER: estrogen receptor

Vitamin D status

Mean serum 25-hydroxyvitamin D was 12.26±9.4 ng/mL. No patient had a level within the reference range (above 30 ng/mL); 31 patients (46.3%) had severe deficiency (below 10 ng/mL), 32 (47.8%) had insufficiency (10-20 ng/mL), and four (6.0%) were in the borderline range (20-30 ng/mL) (Table *2*, Figure *1*). This burden is more severe than the pooled meta-analytic estimate (67.44% below 30 ng/mL) and the German prospective cohort (64.8% below 30 ng/mL), in which a fraction of patients still had normal levels [1,3].

25-hydroxyvitamin D category	Patients
Severe deficiency (<10 ng/mL), n (%)	31 (46.3)
Insufficiency (10-20 ng/mL), n (%)	32 (47.8)
Borderline insufficiency (20-30 ng/mL), n (%)	4 (6.0)
Normal (≥30 ng/mL), n (%)	0 (0)
Mean level, (ng/mL)	12.26±9.4

TABLE 2: Distribution of vitamin D status in the cohort.

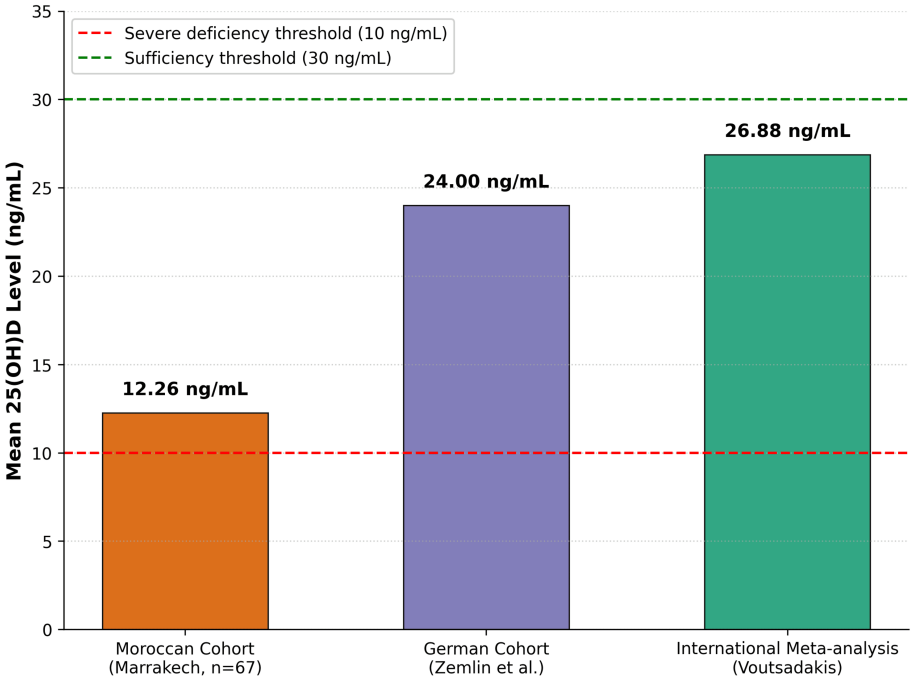


FIGURE 1: Mean serum 25-hydroxyvitamin D (25{OH}D) in the present Moroccan HR+ breast cancer cohort (n=67) compared with international reference values from a pooled meta-analysis of baseline 25(OH)D at breast cancer diagnosis and a German cohort of newly diagnosed breast cancer patients.

Dashed lines indicate the severe-deficiency threshold (10 ng/mL) and the sufficiency threshold (30 ng/mL) [1,3].

HR+: hormone receptor-positive

Correlations

Vitamin D was analyzed as a continuous serum concentration (ng/mL), so a positive coefficient indicates that higher serum vitamin D accompanied higher values of the parameter and a negative coefficient the opposite. Serum vitamin D correlated positively with progesterone receptor expression (Spearman's ρ = +0.559, p=0.013; Pearson's r = +0.616, p=0.005) and with the Ki-67 index (Spearman's ρ = +0.699, p=0.001; Pearson's r = +0.606, p=0.006), and negatively with metastatic (M) stage, coded 0 for M0 and 1 for M1 (Spearman's ρ = -0.472, p=0.041; Pearson's r = -0.472, p=0.041). In other words, lower serum vitamin D was associated with a higher likelihood of metastatic disease, whereas higher serum vitamin D was associated with higher PR expression and higher Ki-67. A trend was observed for nodal (N) involvement (Spearman's ρ = -0.447, p=0.055) without reaching significance. No significant correlation was found with age, BMI, ER expression, HER2 status, T stage, menopausal status, or treatment type (Table *3*).

Variables	Spearman's ρ (p-Value)	Pearson's r (p-Value)	Interpretation
Progesterone receptor (PR)	+0.559 (0.013)	+0.616 (0.005)	Significant
Ki-67 index	+0.699 (0.001)	+0.606 (0.006)	Significant
Metastatic (M) stage	-0.472 (0.041)	-0.472 (0.041)	Significant
Nodal (N) stage	-0.447 (0.055)	-	Trend
Age	-0.299 (0.213)	-	Not significant
Body mass index	-0.309 (0.198)	-	Not significant
Estrogen receptor (ER)	+0.213 (0.381)	-	Not significant
HER2 status	-0.109 (0.657)	-	Not significant
T stage	-0.031 (0.900)	-	Not significant
Menopausal status	-0.288 (0.233)	-	Not significant
Treatment type	+0.257 (0.288)	-	Not significant

TABLE 3: Correlations between the continuous serum 25-hydroxyvitamin D concentration and clinicobiological parameters (metastatic stage coded M0=0, M1=1).

HER2: human epidermal growth factor receptor 2

Discussion

The principal finding is the universal hypovitaminosis D observed in this Moroccan HR+ cohort. This burden exceeds pooled international estimates and several single-center reports, although deficiency is consistently frequent across studies [1,3,5]. To situate these findings, hypovitaminosis D is already highly prevalent in the general Moroccan female population. Among healthy Moroccans aged 50 years and older, insufficiency reached 77.4% in women [15], and in a cohort of 178 postmenopausal Moroccan women, the mean 25(OH)D was 15.8 ng/mL, with 85.3% below 30 ng/mL and 51.6% below 10 ng/mL [16]. Against this already unfavorable background, our HR+ cohort showed an even more severe profile (100% below 30 ng/mL; mean: 12.26 ng/mL; 46.3% below 10 ng/mL). This pattern is consistent with case-control studies reporting significantly lower 25(OH)D in breast cancer patients than in matched controls, for example, 9.3 versus 14.9 ng/mL, with deficiency in 95.6% of cases versus 77% of controls [17], and with data linking 25(OH)D below 20 ng/mL to an increased risk of breast cancer [18]. Because our study lacked an internal control group, these comparisons rely on published reference data rather than a concurrent control arm.

The inverse association with metastatic stage indicates that lower serum vitamin D is associated with a higher likelihood of metastatic (M1) disease. This is compatible with observational data linking lower vitamin D to metastasis, locally advanced disease, or worse late recurrence-free survival in HR+ breast cancer [6-8]. The evidence nonetheless remains mixed, and some cohorts did not support vitamin D deficiency as a major prognostic factor [3]. Consistent with these findings, a dose-response meta-analysis reported that higher circulating 25-hydroxyvitamin D is associated with improved overall survival in breast cancer [19], and reduced tumor vitamin D receptor (VDR) expression has been linked to bone metastasis and poorer clinical outcome [20].

The progesterone receptor association was positive, meaning that higher serum vitamin D accompanied higher PR expression. This direction is consistent with reports that higher vitamin D status tracks with more hormone-receptor-positive biology [2,7], and with immunohistochemical data showing that higher tumor VDR expression is associated with ER and PR positivity and with lower Ki-67 [21]. A study by Ünlü et al. assessed tumor VDR expression by immunohistochemistry rather than serum 25-hydroxyvitamin D, so it supports the receptor-VDR relationship rather than a direct serum-level claim [21]. A large cohort also found no association between serum vitamin D status and receptor status at diagnosis [22]. Because the present analysis was restricted to HR+ tumors and the sample was small, the PR correlation remains exploratory.

By contrast, the direction of the Ki-67 association was unexpected; higher serum vitamin D accompanied higher Ki-67, so lower vitamin D accompanied lower proliferative activity. This runs opposite to the inverse relationship reported in Brazilian and Iranian cross-sectional studies, where lower vitamin D was associated with higher proliferation [6,7], and it is not explained by a recent Polish study that found no association between circulating 25-hydroxyvitamin D and Ki-67 (Spearman's ρ=0.035, p=0.730) [10]. Given the small

sample, possible reverse causation in advanced disease, and uncontrolled confounding, this paradoxical finding should be regarded as hypothesis-generating and interpreted with particular caution.

A biologically plausible pathway links low 25(OH)D status to metastatic dissemination. VDR signaling in breast tissue has antiproliferative and pro-apoptotic effects, and translational work has suggested links to proliferation, apoptosis, estrogen signaling, and recurrence biology, particularly in ER-positive disease [8,9]. Circulating 25(OH)D is the substrate for local calcitriol synthesis, the ligand for the tumor VDR, so low serum levels limit VDR activity [23]. Loss of VDR signaling induces epithelial-to-mesenchymal transition, with loss of E-cadherin and activation of beta-catenin, thereby enhancing migration, invasion, and skeletal colonization [24]. Vitamin D signaling also represses the tumor-progression gene Id1, a pathway abrogated under deficiency, with an inverse correlation between serum 25(OH)D and tumor ID1 in patients, and it down-regulates pro-invasive and pro-metastatic genes such as Plau, Hbegf, Postn, and Has2, while modulating long non-coding RNAs that inhibit this transition [23,25,26]. Clinically, VDR expression is lower in bone metastases than in matched primary tumors and predicts bone metastasis and poorer survival [20]. The inverse association between 25(OH)D and M-stage observed here is therefore consistent with this framework, while remaining hypothesis-generating given the cross-sectional design. A meta-analysis of randomized controlled trials found that vitamin D supplementation reduced total cancer mortality but not total cancer incidence [27].

Limitations

The cross-sectional design prevents causal inference and leaves open reverse causation, whereby advanced disease lowers serum vitamin D rather than the reverse [3,10]. The single-center sample of 67 patients limits precision and generalizability, and the absence of an internal control group means that deficiency cannot be shown to be specific to breast cancer; comparison with cancer-free Moroccan women is a necessary next step. The number of parallel correlations raises the risk of type I error, and because no multivariable adjustment or correction for multiple testing was applied, the correlations should be regarded as exploratory. Correlations were computed on a complete-case basis, so the effective sample size varied across variables. The specific immunoassay kit and the season of sampling were not recorded, which limits reproducibility, and a single vitamin D measurement may not reflect long-term status [9]. Unmeasured confounders, including diet, skin pigmentation, veiling, and sun exposure, can all affect serum vitamin D [3,10]. Finally, the unexpected direction of the Ki-67 association, opposite to most prior reports, underscores that these correlations are hypothesis-generating rather than confirmatory.

Conclusions

In this cohort of women with hormone receptor-positive breast cancer, vitamin D deficiency was extremely prevalent and frequently severe, with no patient reaching the reference range. The observed correlations with progesterone receptor expression, Ki-67, and metastatic status are exploratory and hypothesis-generating. Resting primarily on the striking prevalence data, these findings support systematic screening for vitamin D deficiency in this population and justify larger prospective, multicenter, and interventional studies. These observational data are not treatment-directing.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have

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