Abstract citation ID: qdag063.083

(090) VITAMIN D SUPPLEMENTATION AND RISK OF POSTMENOPAUSAL ATROPHIC VAGINITIS: A GLOBAL REAL-WORLD COHORT STUDY OF VITAMIN D-DEFICIENT POSTMENOPAUSAL WOMEN

E.N. Chaudhri[1], M.A.M. Hammad[2], R. Pope[1], J.M. Yih[2]
[1]University Hospitals Cleveland Medical Center/Case Western Reserve University
[2]University of California, Irvine

Introduction: Female sexual dysfunction (FSD), encompassing disorders of desire, arousal, orgasm, and sexual pain, affects an estimated 40-50% of women globally. In postmenopausal women, declining sex steroid levels, vaginal epithelial atrophy, and altered vascular function heighten vulnerability to FSD and are closely linked to genitourinary syndrome of menopause (GSM) and recurrent urinary symptoms. Vitamin D contributes to estrogen and testosterone synthesis, vaginal cell maturation, and endothelial function. Vitamin D deficiency (VDD) has been linked to impaired sexual function, yet real-world evaluations of whether vitamin D supplementation influences the risk of postmenopausal atrophic vaginitis (PMAV), a common manifestation of GSM, in vitamin D-deficient postmenopausal women remain limited.

Objective: To determine the association between vitamin D supplementation and PMAV outcomes among postmenopausal women with VDD, utilizing a real-time, global, real-world database.

Methods: We queried the TriNetX Research Network, a global federated real-world database encompassing over 100 healthcare systems, to assess PMAV outcomes in postmenopausal women with VDD. A retrospective cohort analysis (2014-2025) including females aged >50 years with VDD was performed using ICD-10 codes. Two study arms were defined: vitamin D non-supplemented VDD (nsVDD) and supplemented VDD (sVDD). Patients on any medications or with any conditions that may affect FSD outcomes or vitamin D metabolism were excluded from our analysis (Fig. 1). Cohorts were propensity-score matched on age, race, and ethnicity. Both cohorts were followed up for PMAV. Outcomes were compared using risk ratios (RR) with 95% CI and Kaplan–Meier survival analyses.

Results: Our search yielded 58 614 patients in each cohort with similar baseline characteristics (Fig. 2). The average follow-up periods were 3.7 and 3.9 years for the nsVDD and sVDD cohorts, respectively. PMAV was significantly higher in the nsVDD cohort compared with the sVDD cohort, although modestly (0.941% vs 0.803%; RR 1.173; 95% CI 1.037, 1.326; p = 0.010). By the end of follow-up, our Kaplan–Meier analysis showed that PMAV-free survival probability was 93.275% in the nsVDD cohort versus 95.835% in the sVDD cohort, corresponding to a significantly elevated hazard (23% higher) of PMAV among unsupplemented women (HR 1.231; 95% CI 1.088, 1.392; p = 0.009) (Fig. 3).

Conclusions: Vitamin D supplementation was associated with a reduced risk of PMAV in postmenopausal women with VDD, suggesting a potential protective role against GSM-related sexual dysfunction. As a low-risk and widely available intervention, vitamin D optimization may offer a valuable adjunct in PMAV prevention and care. Further prospective studies utilizing validated sexual function questionnaires and endocrine biomarkers are warranted to clarify its therapeutic role.

Disclosure: No.

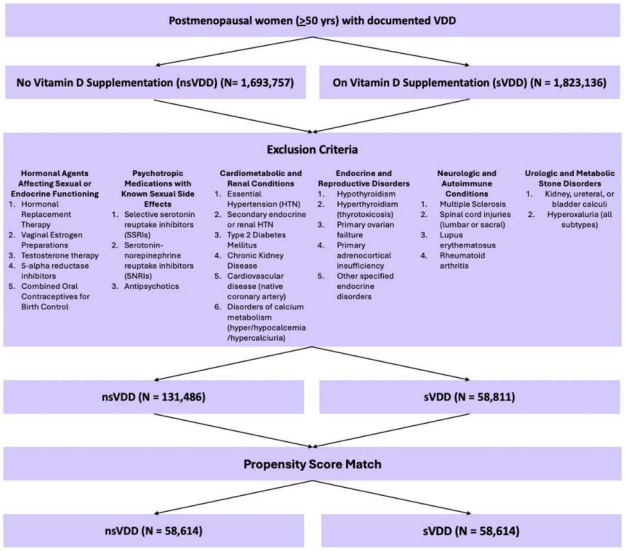

Figure 1: Identification Flowchart of Vitamin D Non-Supplemented (nsVDD) and Supplemented (sVDD) Postmenopausal Women with Vitamin D Deficiency (VDD).

Baseline Characteristics	nsVDD	sVDD	p-value
Patients (n)	58,614	58,614	1
Age at index [Mean ± SD]	53.8 ± 9.64	53.8 ± 9.66	0.536
Mean follow-up (years)	3.7	3.9	1
White (%)	59.0	59.0	0.957
Black or African American (%)	17.3	17.2	0.835
Non-Hispanic or Latino (%)	70.0	70.0	0.863
Hispanic or Latino (%)	9.2	9.1	0.746

Figure 2: Baseline characteristics of propensity-score-matched nsVDD and sVDD cohorts

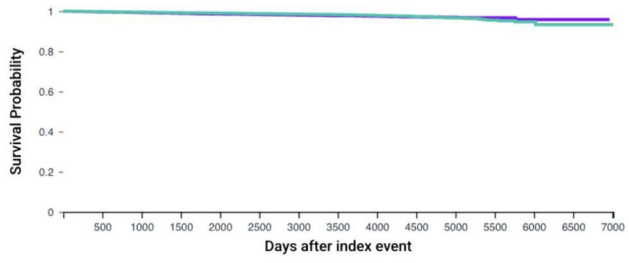

Figure 3: Kaplan—Meier Survival Curve comparing postmenopausal atrophic vaginitis (PMAV)-free survival between nsVDD and sVDD cohorts. The nsVDD cohort demonstrated lower PMAV-free survival over time as compared to the sVDD group (HR 1.231; 95% CI 1.088-1.392; p=0.009).