Effect of vitamin D treatment in ANCA-associated vasculitis: results from an exploratory perspective, pragmatic, non-randomised study

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Abstract

Objective

Vitamin D deficiency has been linked with several autoimmune diseases. Data are limited in anti-neutrophil cytoplasmautoantibody (ANCA)-associated vasculitis (AAV), and it is unknown whether vitamin D could have a therapeutic role in AAV.

Methods

The prospective, pragmatic, non-randomised exploratory PRAVDA study with ITT and pre-protocol analyses aimed to enrol >100 patients with AAV at the Vasculitis Clinic (Toronto, Canada) from January to July 2021. 25-hydroxyvitamin D [25(OH)D] was measured at baseline by ELISA. Patients with low 25(OH)D (<75 nmol/L at baseline) were asked to increase vitamin D supplementation by 1000 IU/day (to a maximum 2000 IU/day). 25(OH)D was measured again at month 12. The primary endpoint was 12-month disease relapse. Secondary analyses included correlations between vitamin D status and disease-specific clinical features.

Results

The study included 101 patients, 41 (40.6%) of whom had low baseline vitamin D levels and were asked to increase vitamin D3 intake. Of these patients, 32 had vitamin D level reassessed at month 12; 62.5% (20/32) had achieved normal levels. Relapse rates at month 12 were similar between patients with low (n=3/41; 7.3%) and normal (n=6/60; 10%; p=0.64) baseline vitamin D levels. However, no relapses were observed in patients who corrected baseline vitamin D deficiency.

Conclusion

These findings can help designing larger studies on vitamin D supplementation in AAV patients, focusing mostly on those vitamin D deficient at baseline.

Key words

vasculitis, ANCA-associated vasculitis, vitamin D

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Introduction

Research conducted within the last decades has uncovered a potential immunomodulatory role of vitamin D in both the innate and adaptive immune responses. Indeed, vitamin D contributes to the regulation of the proliferation, differentiation, and function of several immune cells such as regulatory T cells (1). Notably, the inverse relationship between 25-hydroxyvitamin D [25(OH) D] levels and markers of inflammation in patients with various active rheumatic diseases has been reported, suggesting a potential pathophysiological and clinical role of vitamin D (2).

Vitamin D optimisation through supplementation has been shown to prevent disease progression of inflammatory bowel diseases or autoimmune encephalitis in mouse models (3). Trials investigating multiple sclerosis have commonly uncovered the protective effects of vitamin D3 in preventing the induction of multiple sclerosis (4). However, only a handful of human clinical studies on vitamin D supplementation have been conducted in other autoimmune diseases, such as lupus, rheumatoid arthritis or Hashimoto's thyroiditis, and these have shown minimal and mostly inconsistent trends (5-8).

There is limited data on vitamin D in anti-neutrophil cytoplasm autoantibody (ANCA) associated vasculitis (AAV) to date. AAV is a group of chronic inflammatory small-sized vessel vasculitides with remitting and relapsing course. It includes granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and eosinophilic granulomatosis with polyangiitis (EGPA) (9). A previous North American retrospective study from the Vasculitis Clinical Research Consortium (VCRC) showed insufficient/deficient vitamin D levels in one third of patients with AAV. In this study, a moderate association between low vitamin D levels and disease activity also supported that vitamin D deficiency could have a role in the initiation and/or progression of disease (10). Another study from Australia reported an increased incidence of GPA and EGPA with increasing latitudes and decreasing UV radiation levels, which results in lower vitamin D levels, also suggesting a possible association (11). Another study from Maastricht University identified a more frequent ANCA rise during the fall season, frequently followed by relapse (12). A more recent study from Ireland further determined an inverse relationship between AAV relapse risk and ultraviolet B exposure, similarly speculating a relationship with lower vitamin D status (13).

With this prospective, exploratory, pragmatic, non-randomised pilot study, we sought to explore the association between 25(OH)D and supplementation (the 'intervention'), and disease activity in AAV, and provide some experience and data to potentially design larger trials.

Methods

Patients

The PRAVDA study (a PRagmatic Analysis of Vitamin D in ANCA-Associated Vasculitis) aimed to enrol 100 patients with AAV from the vasculitis clinic at Mount Sinai Hospital in Toronto, Ontario, Canada, from January to July 2021. The study was approved by the Research Ethics Board (MSH REB 19-0039-E) and all participants provided signed informed consent. The eligibility criteria encompassed a minimum age of 18 years at enrolment, and a diagnosis of GPA, MPA, or EGPA fulfilling the 1990 American College of Rheumatology criteria and/or the 2012 Chapel Hill definition. Participants were not eligible if they had a current or history of hypercalcemia, primary hyperparathyroidism, sarcoidosis, hypervitaminosis D, Williams syndrome, other autoimmune/chronic inflammatory/infectious conditions, malabsorptive disorders, cancer, type 1 diabetes mellitus, liver disease, or if pregnant.

Study design and endpoints

Serum 25(OH)D levels were measured using a high-quality antigen specific immunoassay (ELISA) at baseline and at follow-up 12 months later ± 6 months. Patients with low vitamin D levels at baseline, defined as 25(OH) D <75 nmol/L (30 ng/mL), were asked to take and/or increase oral vitamin D3 supplementation by 1000 IU/day to a maximum of 2000 IU/day (the study intervention'; dose increase beyond the advised +1000 IU or maximum 2000 IU/day was not controlled). Because vitamin D3 is routinely recommended for patients with AAV taking glucocorticoids and is available at low prices over the counter in Canada, it was not provided or reimbursed by the study. We recorded baseline and 12-month covariates, including laboratory data, clinical metrics, and relevant details on disease manifestation.

The primary study endpoint was relapse at month 12, defined by a Birmingham vasculitis activity score (BVAS) ≥1 and the need to increase glucocorticoid dose and/or initiate/re-institute other immunosuppressive therapy as per treating physician. The intention-to-treat group (ITT) included all enrolled patients; patients with low 25(OH)D at baseline (advised to increase vitamin D supplementation) were compared to patients who had normal 25(OH)D levels at baseline. A per-protocol subset analysis was also planned to account for compliance or absorption issues, restricted to patients with 25(OH)D levels normal at both baseline and month 12, and those with low levels at baseline but normalised at month 12 (effective study 'intervention').

Secondary endpoints and analyses included correlations between vitamin D status and disease activity, specific changes in vitamin D levels following the study intervention, impact of seasonal variations, sex/gender, association between vitamin D levels and selected specific clinical features (including renal disease, and lung fibrosis).

Statistics

The main recorded demographics, clinical characteristics, and biological parameters are reported as mean ± SD (or median and interquartile range (IQR)), or as number and proportion (%). Comparisons between groups used χ^2 or Fisher's exact test for categorical values, and Student t-test for continuous variables. Survival curves were constructed using the Kaplan-Meier method and compared using the proportional Cox-hazard model when feasible. Analyses were conducted using R and STATA statistical software. A p-value of <0.05 was considered statistically significant.

Table I. Clinical manifestations and main laboratory results at enrolment of the 101 patients with ANCA-associated vasculitis enrolled in the study.

	Total n=101	Patients with normal 25(OH)D >75nmol/L n=60	Patients with low 25(OH)D <75nmol/L n=41	*p*-value
Female, n (%)	59 (58.4)	28 (46.7)	21 (51.2)	0.33
Age, mean ± SD				
Age at diagnosis, years	47.6 ± 20	50.0 ± 21	43.7 ± 19	0.117
Age at enrolment, n (%)	54.7 ± 20	58.1 ± 10	49.6 ± 17	**0.016**
Ethnicity, n (%)				0.173
White	66 (65.3)	43 (71.7)	23 (56.1)	
Asian	3 (3.0)	2 (3.3)	1 (2.4)	
Black	3 (3.0)	2 (3.3)	1 (2.4)	
East Indian	13 (12.9)	6 (10.0)	7 (17.1)	
Middle Eastern	11 (10.9)	7 (11.7)	4 (9.8)	
Pacific	2 (2.0)	1 (1.7)	1 (2.4)	
Smoking history, n (%)				**0.042**
Ex-smoker	18 (17.8)	13 (21.7)	5 (12.2)	
Current smoker	2 (2.0)	0 (0.0)	2 (4.9)	
Forms of vasculitis, n (%)				0.89
EGPA	24 (23.8)	15 (25)	9 (21.9)	
GPA	50 (49.5)	31 (51.7)	19 (46.3)	
MPA	28 (27.7)	17 (28.3)	11 (26.8)	
Positive ANCA, n (%)	87 (86.1)	56 (91.3)	31 (75.6)	0.19
Treatments at enrolment, n (%)				0.38
Prednisone	52 (51.5)	29 (48.3)	23 (56.1)	
Rituximab	45 (44.5)	29 (48.3)	16 (39)	
Dose of prednisone, mean ± SD				
Dose of Prednisone at enrolment	11.5 ± 14.1	9.7 ± 12.6	13.9 ± 15.6	0.153
Dose of Prednisone at month 12	6.3 ± 8.11	5.04 ± 1.66	7.05 ± 10.2	0.191
Manifestations, n (%)				
Lung (includes asthma)	79 (78.2)	50 (83.3)	29 (70.7)	0.131
Renal	63 (62.4)	38 (63.3)	25 (61)	0.818
Gastrointestinal	17 (16.8)	13 (21.7)	4 (9.76)	0.116
Laboratory, mean ± SD				
Haemoglobin, g/dL ± SD	133 ± 17	132 ± 18	136 ± 17	0.27
Mean White blood cells, 10^9/L	7.9 ± 3	7.6 ± 3	8.5 ± 3	0.14
Mean Platelets, 10^9/L	262 ± 84	261 ± 94	268 ± 65	0.68
Mean C-reactive protein, mg/L	4.7 ± 97	5.1 ± 11	4.0 ± 8	0.59
Mean Creatinine µmol/L	121 ± 131	130 ± 159	105 ± 67	0.35
25-Hydroxyvitamin D3 (25(OH)D), mean ± SD				
All, nmol/L	89.8 ± 44	117 ± 37	51.7 ± 15	0.1
(April-September), nmol/L	95.0 ± 40	115 ± 32	53.7 ± 13	0
(October-March), nmol/L	85.6 ± 47	118 ± 42	51.8 ± 18	0
Patients recruited per season, n (%)				
April-June	36 (35.6)	10 (16.7)	26 (63.4)	**<0.001**
July-September	4 (4.0)	2 (3.33)	2 (4.88)	0.696
October-December	18 (17.8)	7 (11.7)	11 (26.8)	**0.051**
January-March	42 (41.6)	22 (36.7)	20 (48.8)	0.226
Disease status at enrolment, n (%)				0.21
Remission at enrolment	89 (88.1)	57 (95.0)	32 (78)	**<0.01**
Active disease at enrolment	12 (11.9)	5 (8.33)	7 (17.1)	0.18
Prior VitD supplementation at recruitment				
Patients already supplementing vitaminD, n (%)	82 (81.2)	56 (93.3)	26 (63.4)	**<0.001**
VitD dose of those supplementing, nmol ± SD	1367.1 ± 745	1535.3 ± 793	1004.6 ± 451	**<0.001**

EGPA: eosinophilic granulomatosis with polyangiitis; GPA: granulomatosis with polyangiitis; MPA: microscopic polyangiitis; SD: standard deviation.

Results

Baseline characteristics

Enrolment took place between January and December 2021; the enrolment period was longer than planned due to the COVID-19 pandemic. A total of 103 patients were consented, but one patient was excluded due to a history of

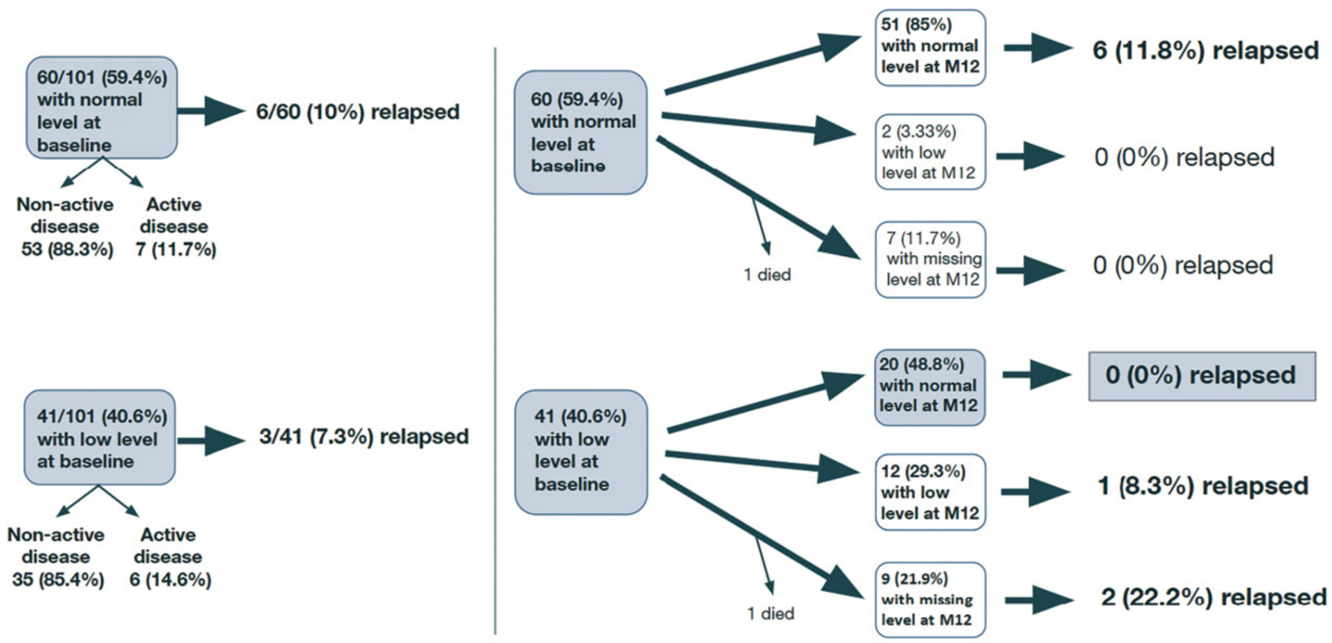

Fig. 1. Study flowchart based on vitamin D status and primary outcome (relapse).

hyperparathyroidism, and a second patient was excluded due to non-compliance (missed baseline vitamin D measurement and follow-up appointments). The mean age of the 101 (ITT) included patients was 47.6±20 years at AAV diagnosis, and 54.7±20 years at enrolment. Fifty-nine patients (58.4%) were female; 49 had GPA, 28 had MPA, and 24 EGPA. A history of positive ANCA was present in 87/101 (86.1%) patients; disease manifestations included lung (n=79), renal (n=63), peripheral nervous system (n=25), and gastrointestinal (n=17) involvement. At enrolment, 89 (88.1%) patients were in remission; 52 were on prednisone, and 45 on rituximab (Table I).

Forty-one (40.6%) patients had low vitamin D at baseline and were asked to take vitamin D supplementation according to the study protocol. Patients with low vitamin D levels were younger (49.6±17 years *vs*. 58.1±10 years in patients with sufficient levels; p=0.016) (Table I) and showed active disease at a numerically higher frequency [7/41 (17.1%) with active disease *vs*. 5/60 (8.33%) in patients with sufficient vitamin D levels; p=0.18] (Table I). Fewer patients with low vitamin D were already supplementing at baseline [26/41 (63.4%) patients *vs*. 56/60 (93.3%) patients with sufficient levels; p<0.001]. Among patients already supplementing with vitamin D3, those with normal vitamin D were on a greater average dose at 1535.3±793 IU/day than those with low vitamin D, supplementing at 1004.6±451 IU/day (p<0.001).

Primary endpoints

Nine (8.9%; 95%CI 4.6–16.2%) patients in total relapsed throughout the study period (Fig. 1, 2). There was no difference in relapse rates between groups with normal or low baseline vitamin D status in the ITT analysis (p=0.66). Five (55.6%) of these nine relapsers had GPA; two (22.2%) had active disease at baseline; and two (22.2%) were not on vitamin D3 supplementation before enrolment. One (11.1%) of these 9 relapses was severe (systemic manifestations, pulmonary embolism, alveolar haemorrhage); the remaining relapses included ENT and/or subglottic stenosis flares, purpura, and/or asthma.

Fourteen (13.9%) patients did not proceed with their month 12 vitamin D level measurement, and two patients had died during the study period (one from sepsis, and one from cancer). Of the 85 patients with a follow-up month-12 25(OH)D available, repeat measurement occurred at a median of 13 months. Fifty-one of these patients maintained normal levels compared to enrolment (normal to normal 25(OH)D, n=51), of which 6 (11.8%; 95%CI 5.5–23.1%) patients relapsed; and 20 patients with low vitamin D at enrolment improved to normal levels at month 12 (low to normal 25(OH)D n=20), of which none (0%; 95%CI 0–16.6%) relapsed (Fig. 1, 3) (per protocol subgroup analysis, n=71; Cox models were fitted but not emphasised due to low event count). Patients missing month 12 measurements included 9 patients with low vitamin D at enrolment, of which 2 (22.2%; 95%CI 6.4–55.6%) relapsed; and 7 patients who had normal vitamin D levels at enrolment, of which none (0%; 95%CI 0–34.6%) relapsed during the study period (Fig. 1). The patient who presented a severe relapse had normal vitamin D at enrolment but was among those missing a month 12 vitamin D level measurement.

Secondary endpoint and analyses

Twenty (23.5%) of the 85 patients with a follow-up vitamin D measurement, presented an increase in serum levels by ≥30 nmol/L (Supplementary Fig. S1). Among the latter patients, fewer relapses occurred than in those who did not present such an increase [1/20 (5%; 95%CI 0.9–23.2%) relapses, *vs*. 6/65 (9.2%: 95%CI 4.3–18.7%) relapses, respectively; I=0.552]. More specifically, none of the 16 patients with low baseline vitamin D levels who increased

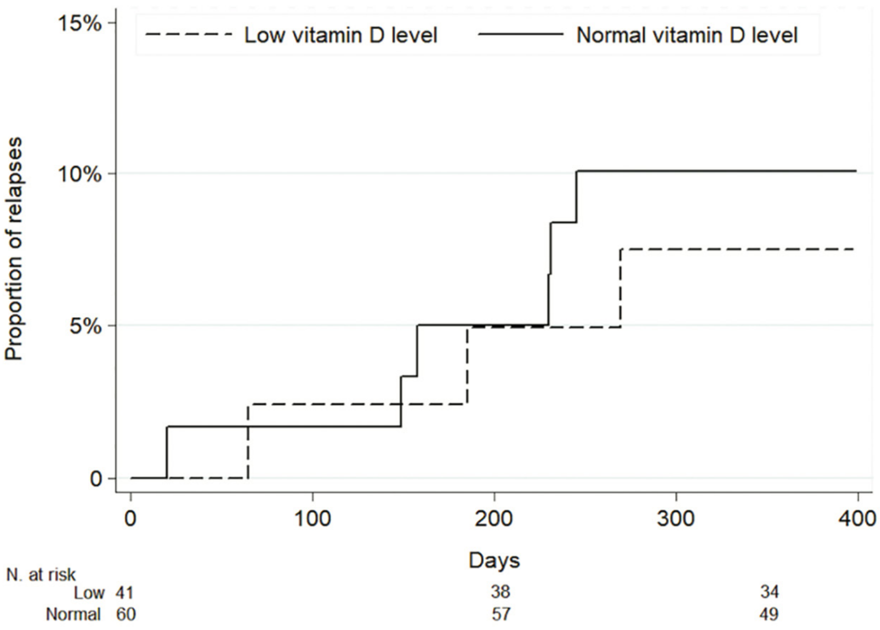

Fig. 2. Cumulative proportion of relapsing patients, according to vitamin D status at enrolment (low at baseline, n=41; normal at baseline, n=60). ITT analysis.
Low vitamin D status defined as <75 nmol/L at baseline.

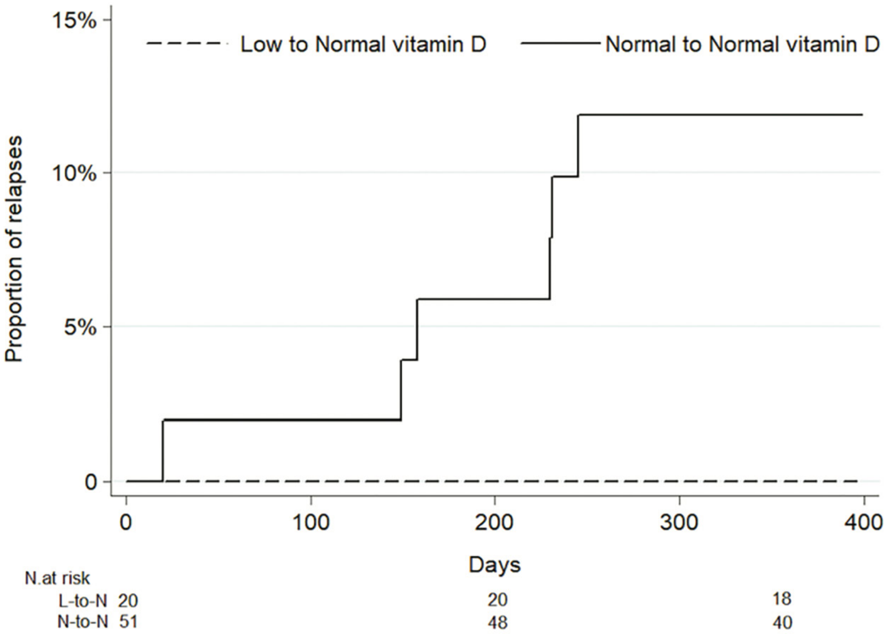

Fig. 3. Cumulative proportion of relapsing patients, according to vitamin D status from enrolment to month 12 in patients considered compliant and with good vitamin D absorption (low to normal, n=20; normal to normal, n=51; per protocol subgroup, n=71). Per protocol analysis.
Low vitamin D status defined as <75 nmol/L at baseline.

their levels by ≥30 nmol/L had a relapse. The mean vitamin D levels at baseline and month 12 during months with higher sun exposure (April to September; 95±40 nmol/L baseline, 112.3±40 nmol/L at month 12) were similar to that during months with lower sun exposure (October to March; 85.6±47nmol/L baseline, 99.8±35 nmol/L at month 12; *p*=0.13 and 0.166, respectively (Table I, and Suppl. Table S1). Only one patient visited a destination with relatively high intensity sun exposure for over 30 days during the study period.

No correlation was observed between vitamin D status at baseline and/or month-12 and selected organ-specific manifestations ever present since diagnosis (renal involvement, gastrointestinal manifestations, peripheral neuropathies, or lung fibrosis), laboratory results (haemoglobin levels, white blood cell or platelet counts, C-reactive protein, or serum creatinine at baseline and/or month 12 levels), and the use of prednisone or rituximab (Table I). Patients who relapsed during the study period were taking a mean prednisone dosage at the time of enrolment of 5.7±10 mg, compared to 6.9±15 mg in patients who did not relapse (*p*=0.331) (Suppl. Table S2). However, only 4/9 (44.4%) patients who relapsed were on other immunosuppressants during the study period *versus* 68/92 (73.9%) patients who did not relapse (*p*=0.031) (Suppl. Table S2). Other baseline predictors of relapse in univariate analysis included history of subglottic stenosis (*p*=0.015); lung manifestations (*p*=0.048); pleural effusion (*p*=0.046); ground glass opacity (*p*=0.007), and mononeuritis multiplex (*p*= 0.014) (Suppl. Table S3).

Discussion

Our study aimed to provide more specific data on the possible association between vitamin D status and relapse risk in patients with AAV, and to assist in determining whether further investigation in a larger study is worthy. Overall, about 40% of the study patients with AAV had baseline vitamin D levels <75 nmol/L, like in the previous VCRC study in North America, but baseline vitamin D status alone was not associated with relapse risk at month 12 (a total of 8.9% of the patients had a relapse over the study period), unlike the VCRC study. Increasing vitamin D supplementation dose by 1000 IU/d in patients with low baseline vitamin D did not decrease the risk of relapse in the ITT analysis (Fig. 2). However, a potential signal emerged in the specific subgroup of patients whose vitamin D status improved from low at baseline to normal (or increased by ≥30 nmol/L) at month 12, as none of them relapsed. This remains a hypothesis-generating finding, given the small sample size (n=20 for low to normal 25(OH)D) (Fig. 3).

Many patients in our study with normal vitamin D levels at baseline and month 12 relapsed, and conversely, some deficient patients did not. However, our findings tentatively support the idea that normalising vitamin D levels in deficient patients may reduce relapse risk in comparison to simply maintaining sufficient vitamin D levels. This finding and hypothesis align with recent data in multiple sclerosis (MS), such as the D-Lay MS trial, where targeted supplementation in deficient patients showed signals of benefit (14-17). Many studies in other autoimmune diseases, including systemic lupus erythematosus, rheumatoid arthritis, and Behçet's disease, have shown mixed results on vitamin D supplementation, but were most often limited by methodological variability, lack of baseline stratification for vitamin D status, and thereby included heterogeneous and pooled populations with both normal and low baseline levels (14, 15, 17-20).

Through its limitations, partly inherent to its original prospective, pragmatic, non-randomised and exploratory design, this study conducted under real-world conditions helped determine many important aspects to better handle in any subsequent trial. Larger sample is obviously needed, but with a narrower population of only patients with low baseline vitamin D. Sensitivity and imputation analyses to account for missing month-12 vitamin D levels lacked statistical power (data not shown) and would still not account for all potential confounders, particularly co-interventions during the study period such as glucocorticoids (GC, and dose) and other immunosuppressive agents. The recording and analysis of these confounders need to be carefully pre-planned in future trials. Observance should be better accounted and recorded, including with serial vitamin D measurements rather than at study end only. A placebo-controlled study was not feasible because the risk of GC-induced osteoporosis mandates the use of vitamin D and calcium supplementation (although many patients still do not take their vitamin D and/or are deficient). However, with the increasing use of avacopan and other GC-sparing therapies in AAV, allowing for GC-free regimens at least in GPA and MPA, there is a potential window to now ethically conduct a placebo-controlled trial of vitamin D supplementation.

Further, it should be noted that vitamin D thresholds used in this study were based on guidelines accounting for the high latitude that contributes to lower sun exposure in regions like Canada, as well as the specific population health needs (21, 22). With the average vitamin D level in Canada being 70 nmol/L, this cohort presented relatively high levels of vitamin D (23). In our study, lower vitamin D levels were associated with a younger age at enrolment, like in another study from Turkey, but not as clearly with disease activity, contrasting with the previous VCRC study, and thereby adding to the acknowledged reservations about the role and impact of vitamin D in auto-immune diseases (24). Lifestyle factors prevalent in younger individuals such as increased indoor activity, higher fat percentages due to dietary differences, sun exposure, and supplementation behaviour may confound the observed association of younger age with low vitamin D status, along with genetic differences. While seasonal variation is typically a concern within a 12-month enrolment period, our study presented no consistent seasonal difference between the mean baseline vitamin D levels. This may be attributed to the COVID-19 pandemic with mobility and travel restrictions, and to fact that 81.2% of our patients were on vitamin D supplementation at baseline, for prevention of GC-induced osteoporosis. We also did not observe a clear association between vitamin D status and specific clinical manifestations such as subglottic stenosis, renal, or pulmonary involvement, which we looked at because of the possible anti-fibrotic or anti-inflammatory effects of vitamin D.

In conclusion, while the broad effects of vitamin D on AAV disease activity appears limited, there may still be a therapeutic signal in correcting deficiency among selected patients. The evolving evidence on novel biomarkers and unmet needs in AAV supports further exploration of vitamin D status as a potential factor for more nuanced prognostic assessment and improved patient outcomes (25, 26). Given the simplicity, safety and low cost of vitamin D supplementation, and the potential to unmask its effects in the context of reduced steroid use (thereby, a less systematic vitamin D-calcium supplementation), further investigation is justified, provided it is carefully targeted and methodologically robust. This study helps identify key considerations for such future research and supports a more nuanced, individualised approach to vitamin D in AAV care.

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