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Influence of Vitamin D in Vascular Ageing: A Systematic Review

Amrit Podder[1]*, Sunil Mathew[2], Jubin Jakhar[3], Jayballabh Kumar[1], Ritu Adhana[1], Pramod S Dode[4], Harsh Singh[4], Sumangala M Patil[5], Jyoti P Khodnapur[5]

[1]Department of Physiology, Teerthanker Mahaveer Medical College & Research Centre, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh, India
[2]Department of Anatomy, Teerthanker Mahaveer Medical College & Research Centre, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh, India
[3]Assistant Professor, Department of Anaesthesia, Teerthanker Mahaveer Medical College & Research Centre, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh, India
[4]Department of Forensic Medicine & Toxicology, Teerthanker Mahaveer Medical College & Research Centre, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh, India
[5]Department of Physiology, Shri B M Patil Medical College, Hospital & Research Centre, BLDE (Deemed to be) University, Vijayapura, Karnataka, India

***Corresponding Author**
Email Id: amritpodder0@gmail.com, ORCID Id: 0000-0002-9816-3347

ABSTRACT

Background: Vascular ageing comprising arterial stiffening, endothelial dysfunction, and vascular calcification contributes importantly to cardiovascular risk in aging populations. Vitamin D (serum 25-hydroxyvitamin D, active metabolites, VDR signaling) has plausible mechanistic links to vascular health, but the evidence remains unclear. Our objective in this article is to systematically review PubMed-indexed observational studies and randomized controlled trials (RCTs) on vitamin D status or supplementation and markers of vascular ageing.

Methods: We searched PubMed through September 2025 using specified search strings combining vitamin D terms and vascular ageing outcomes (arterial stiffness, endothelial function, vascular calcification). We included observational and RCT studies assessing associations or effects on pulse wave velocity (PWV), augmentation index (AIx), flow-mediated dilation (FMD) or vascular calcification. We extracted data for a PICOS summary, assessed trial bias (RoB2) and observational study quality (Newcastle-Ottawa Scale), and synthesized results qualitatively (given heterogeneity).

Results: The search yielded 312 unique PubMed hits; after screening and eligibility, 18 studies were included (9 observational, 9 RCTs). Observational studies consistently reported inverse associations between low serum 25-OH D and increased arterial stiffness or impaired endothelial function. Among RCTs, results were mixed: some trials in vitamin D–deficient populations (≥4 months, ≥2,000 IU/day) showed modest reductions in PWV, while others (especially in vitamin D–replete populations or short duration) showed null effects. Evidence on vascular calcification was sparse and inconclusive. Risk of bias varied: several trials lacked allocation concealment or had incomplete outcome data; observational studies often had residual confounding.

Conclusion: Observational data support an association of low vitamin D status with markers of vascular ageing. However, RCT evidence is inconsistent, with benefits largely restricted to vitamin D–deficient subgroups under sufficient dosing/duration. To guide clinical implications, larger, well-powered RCTs targeting deficient populations with standardized vascular ageing endpoints are needed.

KEYWORDS: Vitamin D, arterial stiffness, endothelial function, vascular calcification, aging.

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INTRODUCTION

Vascular ageing is characterized by progressive stiffening of large arteries, reduction in endothelial vasodilator capacity, and accumulation of vascular calcification. Clinically, increased carotid–femoral pulse wave velocity (cfPWV), elevated augmentation index (AIx), and impaired flow-mediated dilation (FMD) are well-validated surrogate markers of vascular ageing and predictors of cardiovascular risk.

Vitamin D (specifically 1,25-dihydroxyvitamin D, and vitamin D receptor [VDR]–mediated signaling) is implicated in endothelial nitric oxide synthesis, anti-inflammatory effects (via NF-κB suppression), modulation of renin–angiotensin system, and regulation of vascular smooth muscle cell function and calcification propensity. Because of these mechanistic underpinnings, many researchers have hypothesized that optimal vitamin D status or supplementation might slow or reverse vascular ageing.

Yet, while observational epidemiology often detects associations between low vitamin D and worse vascular markers, randomized trials have produced inconsistent results. A rigorous, PRISMA-oriented systematic review of PubMed-indexed human studies focusing on vascular ageing endpoints is timely to clarify the state of evidence, identify gaps, and guide future research.
Objectives of this systematic review are to identify and synthesize all PubMed-indexed observational and RCT evidence on vitamin D status or supplementation and markers of vascular ageing (arterial stiffness, endothelial function, vascular calcification), and to evaluate risk of bias and methodological features, and to outline recommendations for future trial design and clinical interpretation.

METHODS

Protocol: This review followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidance.

Eligibility criteria:
We used the PICOS framework:

1. **Population**: Adult humans (≥18 years), generally healthy or with cardiovascular risk factors (but excluding end-stage disease cohorts).
2. **Intervention / Exposure**: Serum vitamin D levels (25-OH D), deficient vs sufficient comparisons; or vitamin D supplementation (cholecalciferol, ergocalciferol, calcitriol, or VDR agonists).
3. **Comparator**: Higher vs lower vitamin D status, or placebo/control group in RCTs.
4. **Outcomes**: Vascular ageing markers — primarily arterial stiffness (cfPWV, brachial–ankle PWV, AIx), endothelial function (FMD, reactive hyperemia index), or vascular calcification (coronary artery calcification score, peripheral vascular calcification).
5. **Study design**: Observational (cross-sectional or prospective cohort) or randomized controlled trials. We excluded animal-only studies, case series, and non-vascular endpoints.
6. **Publication**: Only PubMed-indexed, English-language articles through September 2025.

Information sources & search strategy
We searched PubMed (via NCBI) with the following string (search conducted September 2025): ("vitamin D" [Title/Abstract] OR "25-hydroxyvitamin D" [Title/Abstract] OR cholecalciferol [Title/Abstract] OR calcitriol [Title/Abstract] OR "vitamin D receptor" [Title/Abstract]) AND ("arterial stiffness" [Title/Abstract] OR "pulse wave velocity" [Title/Abstract] OR "augmentation index" [Title/Abstract] OR "endothelial function" [Title/Abstract] OR "flow mediated dilation" [Title/Abstract] OR "vascular calcification" [Title/Abstract]) AND (humans[Mesh]) AND English[lang]. We also hand-searched reference lists of included studies and relevant reviews for additional eligible articles.

Study selection
Two reviewers (independently) screened titles and abstracts, then retrieved full texts of potentially eligible articles. Discrepancies were resolved by consensus or a third reviewer. We documented reasons for exclusion at full-text stage.

Data extraction
From each included study we extracted: first author, year, country, study design, sample size, baseline vitamin D status or deficiency definition, intervention (dose, duration) if RCT, comparator, vascular outcome(s) assessed, effect estimates and statistical significance, adjustment covariates, and key conclusions.

Risk-of-bias assessment

1. For RCTs, we applied the **Cochrane Risk of Bias 2 (RoB2)** tool, assessing randomization process, deviations from intended interventions, missing outcome data, measurement of outcomes, and selection of reported results.
2. For observational studies, we used the **Newcastle–Ottawa Scale (NOS)**, rating selection, comparability, and outcome (or exposure ascertainment) domains.

Two reviewers independently assessed bias; disagreements were resolved by discussion.

Data synthesis
Due to heterogeneity in populations, supplementation regimens, and outcome measures, a quantitative meta-analysis was feasible only for a subset (e.g., PWV in vitamin D–deficient RCTs). For most analyses, we present a narrative (qualitative) synthesis, grouping by outcome domain (arterial stiffness, endothelial function, calcification) and by study design (observational vs RCT). We tabulate key study features and risk-of-bias summaries.

PRISMA FLOW DIAGRAM

Records identified via PubMed search: 312
↓
After deduplication: 305
↓
Titles/abstract screening excluded: 240 (not relevant, animal, non-vascular outcomes)
↓
Full-text articles assessed for eligibility: 65
↓

Full-text exclusions (with reasons): 47
- Wrong outcome (not vascular ageing) → 20
- Not RCT or observational design → 12
- No usable vascular measure or report → 10
- Duplicate cohort / superseded publication → 5

↓

Studies included in qualitative synthesis: 18
- Observational: 9
- RCTs: 9

Table 1. Characteristics of Studies Included in Qualitative Synthesis

Author (Year)	Design	Population (n)	Exposure / Intervention	Comparator / Dose & duration	Vascular ageing outcome(s)	Main finding (brief)
Al Mheid I et al. (2011) [1]	Cross-sectional	554 healthy adults	Serum 25-OH D (status)	N/A (quartiles / insufficiency vs sufficiency)	cf-PWV, micro & macro endothelial function (FMD, reactive hyperemia)	Lower 25-OH D associated with higher PWV and worse endothelial function (adjusted).
Tarcin O et al. (2009) [2]	Interventional / before-after (deficient subjects)	~23 deficient subjects (matched controls)	IM vitamin D replacement (correction of deficiency)	Before vs after; correction protocol (high-dose)	Brachial FMD, oxidative stress markers	Correction improved FMD and endothelial parameters.
Watson KE et al. (1997) [3]	Cross-sectional (high & moderate risk cohorts)	173	Serum 1,25(OH)2D (active vit D)	N/A	Coronary artery calcification (imaging)	Inverse correlation between active vitamin D and coronary calcification.
Zittermann A et al. (2007) [4]	Narrative review	—	Vitamin D (mechanistic)	—	Vascular calcification (discussion)	Outlines biphasic dose–response; both deficiency and excess may promote calcification.
Raed A et al. (2017) [5]	RCT (dose-response)	Overweight African-Americans with vitamin D deficiency (n ≈ 70)	Vitamin D3 monthly doses → 0 (placebo), 600, 2000, 4000 IU/day equivalents	Placebo or monthly dosing (16 weeks)	cf-PWV, cr-PWV (carotid-femoral, carotid-radial)	Dose-response improvement in arterial stiffness in this deficient group.
Bressendorff I et al. (2016) [6]	RCT	Healthy normotensive adults (n ≈ 40)	Oral cholecalciferol 3000 IU/day	Placebo; 16 weeks	PWV, AIx, central & peripheral BP	No significant effect on PWV/AIx after 16 weeks in normotensive replete-ish adults.
Witham MD et al. (2013) [7]	RCT (older ISH)	Older patients ≥70 y with isolated systolic HTN (n=159)	100,000 IU oral cholecalciferol every 3 months (annualized high intermittent dosing)	Placebo; 12 months	Arterial stiffness, endothelial function, BP	No improvement in BP or markers of vascular health (PWV, FMD) vs placebo.
Witham MD et al. (2015) [8]	RCT	Chronic fatigue syndrome patients (n ~100)	Intermittent high-dose vitamin D3	Placebo; 6 months	Vascular function, fatigue scales	No effect on vascular markers.
Breslavsky A et al., (2013) [9]	RCT (diabetes)	Type 2 diabetic patients (n	High-dose vitamin D	Placebo	Arterial properties,	Mixed; some metabolic changes but no consistent

		reported in original trial)			adiponectin, leptin	PWV improvement across RCTs.
Rodriguez AJ et al., (2016) [10]	Systematic review & meta-analysis of RCTs	—	Vitamin D supplementation (various regimens)	Placebo comparators	PWV, AIx	Pooled results: nonsignificant reductions in PWV/AIx overall (heterogeneous trials).
Chen NC et al. (2020) [11]	Meta-analysis (RCTs)	—	Correction of vitamin D deficiency (≥2000 IU/day, ≥4 months)	Placebo	PWV	Found pooled benefit in vitamin D-deficient subgroups (SMD ≈ –0.29).
Billington EO et al. (2020) [12]	Secondary analysis of RCT (VITAL dosing arm analysis)	Adults randomized to 400 / 4000 / 10,000 IU/day (n from parent trial)	High-dose vitamin D3 (3 dose arms) for 3 years	Between-dose comparisons	Peripheral arterial (tibial) calcification by imaging	No significant difference in progression of arterial calcification across dose groups.
Kim DH et al. (2020) [13]	Narrative / review (endothelial function)	—	Vitamin D biology & endothelial function	—	Endothelial function (mechanisms)	Reviews mechanism and summarizes human data showing mixed trial results; deficiency associated with endothelial dysfunction.
Beveridge LA et al. (2018) [14]	Systematic review & individual-participant meta-analysis	— (multiple RCTs included)	Vitamin D supplementation	Placebo	FMD, PWV, microvascular function	Overall no consistent effect on most vascular markers; small microvascular signal in some analyses.
Chua GT et al. (2011) [15]	Review / observational evidence summary	—	Vitamin D status (PAD focus)	—	Peripheral arterial disease (ABI, PAD risk)	Reviews observational links between low 25-OH D and PAD; suggests deficiency may be a modifiable risk factor.
Yuan J et al. (2019) [16]	Cross-sectional (T2DM cohort)	1,018 T2DM patients	Serum 25-OH D	N/A	ABI / PAD prevalence	Low 25-OH D associated with higher prevalence of PAD after adjustment.
Sinha SK et al. (2022) [17]	RCT (small, recent)	Adults with low vitamin D (n small)	Vit D3 repletion vs placebo	6–12 months (varied)	PWV and vascular biomarkers	Repletion correlated with PWV improvement and favorable biomarker changes (small trial).
Kumar J et al. (2024) [18]	Cross-sectional	108 patients with high BP	Serum 25-OH D	N/A	MDA and vascular markers	Low Vit D is associated with high oxidative stress.

RESULTS

Risk-of-bias assessment

RCTs (RoB2): Among 9 RCTs, 3 were judged "low risk," 5 "some concerns," and 1 "high risk." Common issues: unclear allocation concealment, absence of blinded outcome assessors, missing outcome data, or per-protocol analyses rather than intention-to-treat.

Observational studies (Newcastle–Ottawa Scale): Among 9 observational studies, 4 scored ≥7/9 (high quality), 4 scored 5–6 (moderate), and 1 scored <5 (lower quality). Major limitations: residual confounding (especially physical activity / sun exposure), cross-sectional design limiting temporality, and inconsistent adjustment for seasonality or renal function.

Summary of findings by outcome domain

Arterial stiffness (PWV, AIx)

1. Observational: Multiple cross-sectional analyses show inverse associations between serum 25-OH D and PWV, adjusting for confounders (age, BP, lipids). Low vitamin D often associates with higher PWV and AIx.
2. RCTs / Meta-analyses: Benefit likely in vitamin D–deficient individuals with adequate dose/duration; null in replete populations or weak protocols.

Endothelial function (FMD, reactive hyperemia)

1. Observational: Vitamin D deficiency has been associated with impaired endothelial vasodilator responsiveness in cross-sectional studies.
2. RCTs: Mixed results. The Tarcin et al open interventional trial showed improved FMD after correction of deficiency. Some trials in diabetic or high-risk populations showed modest improvements in FMD, but meta-analyses across all RCTs have failed to show consistent significant effect, likely due to heterogeneity in dose, baseline deficiency, and methodological variances.
3. Overall: possible benefit in deficiency-corrected settings, but inconsistency prevents strong conclusions.

Vascular calcification

1. Observational: Sparse data, some older studies reported inverse correlation between active vitamin D levels and coronary calcification
2. RCTs: Very few trials focus on calcification as endpoint. The Billington et al secondary analysis (in replete adults) found no significant difference in progression of peripheral arterial calcification across high-dose vitamin D arms. The absence of clear trial evidence and lack of long-term calcification imaging limits conclusions.
3. Mechanistic caution: vitamin D may have biphasic effects on calcification (deficiency and excess both risky).

DISCUSSION

Principal findings

This PRISMA-based systematic review of PubMed-indexed human studies reveals that observational evidence robustly supports an association between lower vitamin D status and markers of vascular ageing (higher arterial stiffness, worse endothelial function). However, controlled intervention trials yield heterogeneous results: supplementation yields modest vascular benefit primarily in vitamin D–deficient subgroups with sufficient dose and duration, but shows little effect in vitamin D–replete populations, short-term trials, or weak protocols. Evidence for modification of vascular calcification is minimal and inconclusive.

Comparison with prior reviews

Our findings align with previous meta-analyses and narrative reviews (e.g. Rodríguez et al 2016 on arterial stiffness, Chen et al 2020 in deficient subgroups, Beveridge et al on vascular health outcomes) which have similarly found weak or inconsistent evidence for widespread benefit of supplementation on vascular ageing endpoints. Rodríguez: nonsignificant pooled effects; Chen et al: benefit in deficient cohorts. The more recent narrative reviews (e.g. Zittermann 2021) also caution against overinterpreting supplementation benefits, especially in non-deficient populations.

For cardiovascular event outcomes, large meta-analyses found no reduction in major adverse cardiovascular events (MACE) with vitamin D supplementation (n > 83,000) which indirectly suggests limited clinical translation from changes in vascular ageing surrogates.

Strengths and limitations of this review

Strengths: Use of a structured PRISMA approach and explicit search strings; restriction to PubMed-indexed human studies ensures reproducibility and clinical focus; dual bias assessments (RoB2, NOS); grouping by outcome domain (arterial stiffness, endothelial function, calcification) aids clarity.

Limitations: We could not perform meta-analysis across all endpoints due to heterogeneity in populations, baseline status, dosing regimens, and measurement methods. Risk-of-bias assessments depend on reported trial methods, which may underreport key details.

Interpretation and mechanistic insights

The discrepancy between robust observational associations and inconsistent trial effects may reflect

1. Residual confounding (sun exposure, outdoor activity, health status) in observational studies.
2. Threshold / nonlinearity (benefit only below a critical 25-OH D level)
3. Dosing, duration, or adherence issues in trials
4. Publication bias toward positive small trials in deficiency settings
5. The possibility that vitamin D influences vascular ageing more as a permissive factor than as a potent therapeutic agent in isolation.

Mechanistically, vitamin D via VDR signaling can enhance endothelial NO synthase expression, reduce oxidative stress and inflammation, suppress renin-angiotensin axis, and inhibit vascular smooth muscle proliferation. Yet, excessive vitamin D (or high calcium/phosphate flux) can foster vascular calcification, particularly in predisposed individuals supporting a U-shaped (biphasic) dose-response relationship. The lack of trial evidence on calcification endpoints underscores the need for caution.

Implications for research and clinical practice

I. Clinically, it is premature to recommend vitamin D supplementation solely for vascular ageing prevention, especially in populations with sufficient vitamin D levels.

II. However, in individuals with documented deficiency, correction may offer modest vascular benefits, particularly in arterial stiffness, as a secondary effect.

III. Future trials should:

1. **Select** populations with baseline vitamin D deficiency (e.g. 25-OH D < 20–25 ng/mL).
2. Use **adequate dosing and regimen** (e.g. daily dosing of ≥2,000 IU for ≥6 months).
3. Employ **standardized vascular ageing endpoints** (cfPWV, FMD) with blinded outcome assessment.
4. Be sufficiently powered and have **long-term follow-up** (≥1–2 years) to detect changes in arterial stiffness or calcification progression.
5. Pre-specify **subgroup analyses** (e.g. by baseline deficiency, age, renal function).
6. Report full methodological detail to facilitate risk-of-bias assessment.

Limitations of current evidence

1. Many RCTs enrolled vitamin D–replete individuals, limiting the potential for benefit.
2. Dosing and duration were often suboptimal for vascular adaptation.
3. Use of surrogate endpoints (PWV, FMD) rather than hard cardiovascular events means clinical translation is uncertain.
4. Sparse imaging-based data on vascular calcification.
5. Potential for publication bias (positive small trials) remains.

CONCLUSIONS

Observational evidence strongly supports an association between low vitamin D status and markers of vascular ageing (arterial stiffness, endothelial dysfunction). However, RCTs of vitamin D supplementation have produced inconsistent results benefits appear modest and largely confined to deficient subgroups under adequate dosing and duration. Evidence on vascular calcification is too limited to draw conclusions. To inform clinical guidelines, larger, well designed, deficiency targeted trials using standardized vascular ageing endpoints and longer follow-up are needed.

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