Combined vitamin K2, vitexin, and vitamin D3 (K2VD3) intake in patients with peripheral artery disease

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SOUHRN

Cíl: Tato observační studie zkoumala terapeutickou účinnost bioaktivních látek (vitamin K_2, vitexin a vitamin K_2VD_3) při zmírňování intermitentní klaudikace u pacientů s ischemickou chorobou dolních končetin (ICHDK).
Metody: Pacienti s ICHDK stadia IIb při optimální farmakoterapii byli léčeni úpravou životosprávy a podáváním K_2VD_3 po dobu 12 měsíců.
Výsledky: Průměrná vzdálenost překonaná bez bolesti, kterou pacienti uváděli při zařazení do studie jako 123 metrů (95% CI 17–297), se prodloužila na 376 metrů po 2 měsících (95% CI 226–527), 794 metrů po 4 měsících (95% CI 517–1 070), 2 645 metrů po 6 měsících (95% CI 1 511–3 780) a 2 659 metrů po 12 měsících (95% CI 1 529–3 789) ($p < 0,001$ pro každý interval ve srovnání s předchozí a s výchozí hodnotou). Zvýšil se i index kotník-paže z 0,60 (95% CI 0,22–0,87) na 0,62 po 6 měsících (95% CI 0,55–0,79) a na 0,75 po 12 měsících (95% CI 0,65–0,90) ($p < 0,01$ pouze pro výchozí hodnotu vs. 12 měsíců).
Závěr: U pacientů s ICHDK může užívání kombinace K_2VD_3 přispět ke zmírnění intermitentní klaudikace. Statisticky významné prodloužení průměrné vzdálenosti překonané bez bolesti a zvýšení indexu kotník-paže po 12 měsících studie ukazují na terapeutickou účinnost popsané kombinace bioaktivních látek. Z našich zjištění lze usuzovat, že kombinace K_2VD_3 nejenže zvyšuje tělesnou výkonnost, ale zlepšuje i zdraví cév, což je naprosto zásadní aspekt péče o pacienty s ICHDK. Hodnocení účinnosti kombinace si vyžádá další randomizované studie (jak spolu s úpravou životosprávy, tak bez ní), aby bylo možno přesně určit potenciální úlohu kombinace K_2VD_3 v klinické praxi.

ABSTRACT

Objective: This observational study investigates therapeutic efficacy of bioactive compounds (vitexin, vitamin K2, and vitamin D3, K2VD3) in alleviating claudicatio intermittens in patients with peripheral artery disease (PAD).
Methods: Patients with PAD stage IIb under best medical cure were treated with life-style therapy and K2VD3 for 12 months.
Results: Mean pain-free walking distance, described at baseline as 123 meters (95% CI 17–297), increase as follow: 376 (95% CI 226–527), 794 (95% CI 517–1070), 2 645 (95% CI 1511–3780), and 2 659 (95% CI 1529–3789) meters after 2, 4, 6 and 12 months, respectively (p <0.001 for each interval if compared with the previous one and the baseline). Moreover, ankle-brachial index also increased, from 0.60 (95% CI 0.22–0.87), to 0.62 after 6 months (95% CI 0.55–0.79) and 0.75 after 12 months (95% CI 0.65–0.90) (p <0.01 only for baseline vs 12 months).
Conclusion: K2VD3 can contribute to alleviating claudicatio intermittens in PAD patients. The significant increases in mean pain-free walking distance and ankle-brachial index observed over the 12-month study period support the therapeutic efficacy of this bioactive compound combination. These findings suggest that K2VD3 not only enhances physical performance but also improves vascular health, thereby addressing a critical need in PAD management. Further studies are needed to assess its efficacy in randomized trials, both with and without concomitant lifestyle interventions, to fully elucidate its potential role in clinical practice.

Keywords:
Peripheral artery disease
Vitamin D3
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Introduction

Peripheral arterial disease (PAD) of the lower limbs commonly presents with a hallmark symptom known as claudicatio intermittens, described as pain triggered by walking that progressively impairs mobility and significantly reduces quality of life.[1,2] Conservative management in PAD patients, as recommended by the American College of Cardiology and associated societies, centers on risk factor modification and structured exercise therapy.[3] Key components include antiplatelet therapy (typically single antiplatelet agents), high-intensity statin therapy for lipid lowering, antihypertensive therapy, diabetes management, and absolute smoking cessation.[4] These interventions are justified by the high risk of cardiovascular morbidity and mortality in PAD, and their proven benefit in reducing these risks.

Limitations of current treatments include incomplete symptom relief, adverse effects (e.g., bleeding with antithrombotic, heart failure risk with cilostazol), and suboptimal implementation of guideline-directed therapies. Many patients continue to experience functional impairment and pain despite optimal management, highlighting the need for improved therapies and care delivery.

Despite the abovementioned, the best medical therapy could be considered, several compounds can be also considered as useful pharmacological supports in these patients.

Hawthorn (*Crataegus oxyacantha*), rich in pharmacologically active fractions such as vitexin, flavonoids, and oligomeric procyanidins, has been studied for its potential cardiovascular effects, including antioxidant, anti-inflammatory, lipid-lowering, vasodilatory, and endothelial-protective properties, which are mechanistically relevant to vascular pathologies such as atherosclerosis and endothelial dysfunction.[5,6] Preclinical and small clinical studies suggest that hawthorn extracts may improve endothelial function, reduce lipid accumulation, and attenuate inflammatory processes in vascular tissue.[5–7]

Vitamin K2 (menaquinone-7), although less well-known than vitamin K1, plays a pivotal role in vascular protection. Unlike K1, which is involved in blood coagulation, K2 activates matrix Gla protein (MGP) – a vitamin K-dependent inhibitor of arterial calcification – through a process called carboxylation.[8] MGP is synthesized by vascular smooth muscle cells and chondrocytes and prevents calcium deposition in arterial walls. In advanced atherosclerosis, K2 levels are 20–50 times lower than in healthy arteries, leading to inactive MGP and increased vascular calcification. Long-term intake of K2 has been shown to reduce aortic calcification and improve arterial elasticity.[9] The Rotterdam study, involving over 4,800 participants, confirmed that K2 is significantly more effective than K1 in lowering cardiovascular morbidity and mortality.[10]

Vitamin D3 (cholecalciferol) affects vascular health through several mechanisms. It modulates endothelial function, inhibits vascular smooth muscle cell proliferation, suppresses inflammation, and downregulates the renin-angiotensin-aldosterone system, all of which are implicated in atherogenesis and vascular remodeling. Vitamin D receptors are expressed in endothelial and vascular smooth muscle cells, and the active metabolite 1,25-dihydroxyvitamin D regulates genes involved in cell proliferation, apoptosis, oxidative stress, and matrix homeostasis, contributing to vascular integrity and anti-atherosclerotic effects.[11]

Table 1 summarizes K2VD3 characteristics and effects on cardiovascular system.

Building on this scientific foundation, the present observational study aims to evaluate the clinical efficacy of a combination of vitamin K2, vitexin, and vitamin D3 (K2VD3) in improving the symptoms of claudicatio intermittens associated with PAD.

Table 1 – Vitamin K2, vitamin D3, and vitexin effects as complementary interventions in the management of peripheral artery disease (PAD) (see references as supplementary material)

Vitamin K2 (menaquinone): There is growing mechanistic and clinical interest in vitamin K2 for vascular health, particularly due to its role in activating matrix Gla protein, an inhibitor of vascular calcification. Recent clinical studies, including a 2025 trial, demonstrate that one year of MK-7 supplementation (180 µg daily) in post-menopausal women with low vitamin K status significantly reduced vascular stiffness and improved blood pressure, especially in those with high baseline arterial stiffness, supporting further investigation in populations at risk for PAD. Systematic reviews and meta-analyses also suggest vitamin K supplementation reduces vascular calcification, though effects on vascular stiffness and clinical endpoints remain less certain, warranting larger trials.
Vitamin D3: Large cross-sectional analyses, such as those from NHANES, demonstrate that individuals in the lowest quartile of serum 25-hydroxyvitamin D have a significantly higher prevalence of PAD compared to those in the highest quartile, even after adjustment for confounders. Meta-analyses confirm that PAD patients have lower mean vitamin D levels than controls, and both vitamin D deficiency (<20 ng/mL) and insufficiency (20–30 ng/mL) are associated with higher odds of PAD. Prospective cohort data, such as from the ARIC study, indicate that deficient vitamin D status is linked to a 25–49% increased risk of incident PAD after multivariable adjustment, with the association present in both black and white populations. The relationship appears dose-dependent, with lower vitamin D levels correlating with higher PAD risk. This association is also observed in specific populations, such as patients with type 2 diabetes, where lower vitamin D levels are independently associated with increased PAD prevalence.
Vitexin: Preclinical and translational studies highlight vitexin, a dietary flavonoid, as a promising agent for vascular protection. Recent research shows vitexin inhibits endothelial inflammation and atherogenesis in animal models by targeting APEX1 and activating Nrf2 signalling, both relevant to the pathophysiology of PAD.

Materials and methods

The study included all male and female subjects who presented at the Vascular and Diagnostic Angiology outpatient clinic and underwent arterial lower limb Duplex ultrasound scan analysis between January 1, 2023, and November 30, 2024. Medical history was assessed to evaluate the presence of walking pain, considering also the Pain-Free Walking Distance (PFWD), defined as the distan-

ce walked without pain. All patients diagnosed with PAD at stage IIb according to the Leriche–Fontaine classification were enrolled and underwent an initial examination (T0), which included a detailed medical history focusing on present and past medical conditions, with particular attention to risk factors such as diabetes mellitus, hypertension, dyslipidaemia, cardiopathies, ischemic heart disease, and atrial fibrillation. Following the collection of clinical data, each participant underwent ankle brachial index (ABI) calculation for both limbs. All enrolled patients were on best medical therapy (e.g. Cardioaspirin 100 mg and Atorvastatin 40 mg). All subjects received a daily therapy consisting of Vitamin K2 90 mg + Vitexina 300 mg + Vitamin D3 25 μg (K2VD3), administered as one tablet twice daily for 12 months. Moreover, a dedicated physical activity program was released for each enrolled patient.

PFWD was measured for each patient using a treadmill test at 2 months (T1), 4 months (T2), 6 months (T3), and 12 months (T4). Additionally, the ABI index was also assessed at T3 and T4.

The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines and checklist for cohort studies were used as reporting standard recommendations.[12] As per retrospective nature, approval for this specific study was waived by local Institutional Review Board. Data were treated according to the National Policy in the matter of the Privacy Act on retrospective analysis of anonymized data.

Statistical analysis

Clinical data were recorded and tabulated in a Microsoft Excel (Microsoft Corp., Redmond, WA, USA) spreadsheet; statistical analysis was performed with JMP 16.0 (SAS Institute Inc., Cary, NC). Missing data were reported during data extraction and flagged as such (–). Categorical/nominal variables were presented using frequencies and percentages, while continuous variables by mean (μ) ± standard deviation (SD), or median with interquartile range (IQR) and ranges, according to data distribution.

The Friedman test, a non-parametric statistical method, was employed to evaluate modifications in Pain-Free Walking Distance (PFWD) and Ankle-Brachial Index (ABI) across the multiple follow-up time points (T1, T2, T3, and T4). This test was chosen due to the repeated measures design of our study, where the same subjects were assessed at different intervals, making it suitable for analysing changes in these variables without assuming a normal distribution.

To assess the significance of differences in PFWD and ABI measurements over time, we applied the Friedman test to determine whether there were statistically significant changes among the related groups. Given the potential for increased Type I error due to multiple comparisons across the four time points, we implemented the Bonferroni correction method. This adjustment involved dividing the significance level ($\alpha = 0.05$) by the number of comparisons (i.e., the number of time points). As a result, a corrected significance level of $\alpha = 0.0125$ was used to evaluate the *p*-values obtained from the Friedman test.

If the Friedman test indicated significant differences, post-hoc analysis using the Wilcoxon signed-rank test was conducted to compare specific time points while controlling for multiple comparisons using the Bonferroni adjustment. This approach ensured that we accurately interpreted the significance of changes in PFWD and ABI, maintaining the integrity of our statistical conclusions.

Results

During the study period 64 patients were enrolled. **Table 2** summarizes demographic characteristics.

PFWD showed a progressive increase over the course of the treatment steps. At the baseline (T0) mean PFWD was 123 meters (95% CI 17–297). It increased during the follow-up, reaching 376 (95% CI 226–527) meters at T1, 794 (95% CI 517–1 070) meters at T2, 2 645 (95% CI 1 511–3 780) meters at T3 and 2 659 (95% CI 1 529–3 789) meters at T4 (p <0.001 for each interval if compared with the previous one and the baseline), as described in **Figure 1** and **Table 3**. Moreover, ABI also increased, from 0.60 (95% CI 0.22–0.87) at the baseline, to 0.62 after 6 months (95% CI 0.55–0.79) and 0.75 after 12 months (95% CI 0.65–0.90) (p

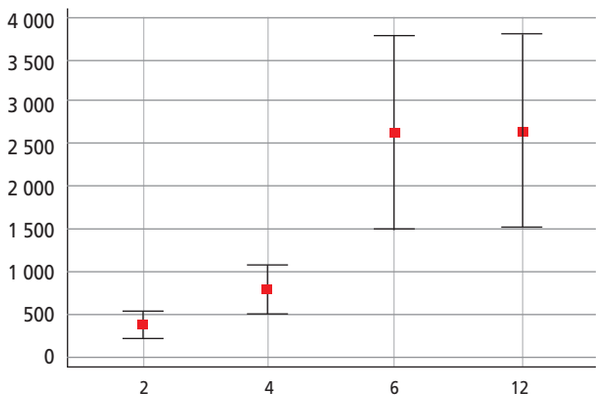

Fig. 1 – Pain-free walking distance measured in meters during follow-up period (p <0.001 for each interval if compared with the previous one and the baseline, 123 meters [95% CI 17–297]).

Table 2 – Baseline characteristics

Condition	Percentage	N
Total patients on oral anticoagulants (DOAC)	13%	8
Age	79 ± 14	
Female	34.4%	22
Smoking	51%	33
Hypertension	70%	45
Hypercholesterolemia	46%	29
Diabetes mellitus	35%	22
Carotid lesions	70%	45
Myocardial infarction (IMA)	12%	8
Atrial fibrillation	9%	6
Total	**100%**	**64**

Table 3 – This table summarizes the changes in pain-free walking distance (PFWD) and ankle-braxial index (ABI) at each assessment interval. The values indicate the mean measurements along with their corresponding 95% confidence intervals (CIs), highlighting the improvements observed over the 12-month study period

Time point	PFWD (meters)	95% Confidence interval (CI)	ABI	95% Confidence interval (CI)
Baseline (T0)	123	17–297	0.60	0.22–0.87
2 months (T1)	376	226–527	–	–
4 months (T2)	794	517–1070	–	–
6 months (T3)	2 645	1511–3780	0.62	0.55–0.79
12 months (T4)	2 659	1529–3789	0.75	0.65–0.90

<0.01 only for baseline vs 12 months). No adverse effect was observed during the K2VD3 treatment.

Discussion

The results of this observational study highlight the potential benefits of the combined supplementation of vitamin K2, vitexin, and vitamin D3 (K2VD3) in managing claudicatio intermittens among patients with PAD. The significant increase in PFWD. PFWD observed at various follow-up time points indicates that K2VD3 may enhance physical performance and overall quality of life in these patients, supporting the notion that additional pharmacological interventions can complement established medical therapies.

Several considerations arise from these findings. First, the progressive improvement in PFWD underscores the importance of both pharmacological and lifestyle interventions in PAD management. While traditional therapies focus on risk factor modification and structured exercise, the addition of bioactive compounds like K2VD3 could provide a synergistic effect, enhancing vasodilation, reducing inflammation, and improving endothelial function. This is particularly relevant in the context of PAD, where vascular health is paramount for mitigating symptoms and preventing disease progression.

Moreover, the increase in ABI further corroborates the vascular benefits of K2VD3. The ABI is a critical indicator of peripheral arterial health, and its improvement suggests a possible reduction in vascular calcification and enhancement of arterial elasticity, mechanisms that are consistent with the known roles of vitamin K2 in activating matrix Gla protein. This aspect is especially significant given the association between lower K2 levels and increased vascular calcification in advanced atherosclerosis.

The rationale under that study derives from the role of K2VD3 in vascular homeostasis. Vitamin K2 is hypothesized to improve vascular health primarily through its role as a cofactor for the γ-carboxylation of MGP, a potent inhibitor of vascular calcification. Adequate vitamin K2 status enables the activation of MGP, which in turn inhibits the deposition of calcium in the arterial wall, potentially reducing vascular calcification and stabilizing plaque composition.[13] This mechanistic rationale is supported by animal studies and observational data suggesting an association between higher vitamin K2 intake and reduced vascular calcification and cardiovascular risk, as well as by evidence that vitamin K antagonists accelerate vascular calcification by inhibiting MGP activation.[14,15]

Vitexin might be expected to improve vascular health, particularly in the context of arterial plaque composition or atherosclerosis, because it exhibits multiple mechanisms that target key processes in atherogenesis, including endothelial inflammation, oxidative stress, and lipid metabolism. Unlike vitamin K2, which primarily acts through inhibition of vascular calcification, vitexin directly suppresses endothelial inflammation by inhibiting APEX1-mediated NF-κB activation and nuclear translocation, thereby reducing proinflammatory gene expression in response to disturbed flow, a critical driver of atherosclerotic plaque vulnerability and progression. In animal models, vitexin administration attenuates flow-induced endothelial inflammation, neointimal formation, and atherosclerosis.[16]

Additionally, vitexin activates the Nrf2 pathway by disrupting Keap1-Nrf2 interaction, leading to upregulation of antioxidant defences and further suppression of vascular inflammation, as demonstrated in both in vitro and in vivo models of low-grade vascular inflammation.[17] Vitexin also reduces high-fat diet-induced vascular inflammation and improves lipid profiles, in part by modulating gut microbiota and inhibiting TMAO-mediated RNA m6A modification, which is implicated in vascular inflammation and atherogenesis.[18] These pleiotropic effects – anti-inflammatory, antioxidant, and lipid-lowering – distinguish vitexin from vitamin K2 and provide a mechanistic rationale for its potential benefit in improving arterial plaque composition and reducing atherosclerotic risk.

Vitamin D3 is thought to improve vascular health and influence arterial plaque formation primarily through its effects on vascular smooth muscle cells, endothelial function, and inflammation.[19] Vitamin D3 modulates endothelial nitric oxide synthesis, reduces oxidative stress, and suppresses pro-inflammatory cytokine production via inhibition of the NF-κB pathway, all of which contribute to improved endothelial function and reduced atherogenesis.[13,19–22] Experimental studies show that vitamin D3 inhibits foam cell formation in both macrophages and vascular smooth muscle cells by promoting autophagy and cholesterol efflux, thereby reducing lipid accumulation within plaques and potentially stabilizing them.[21] Observational data consistently associate low vitamin D levels with increased carotid intima-media thickness, higher prevalence of carotid plaques, and greater arterial stiffness.[23]

Limitations

This observational study has several limitations that must be acknowledged. Firstly, the lack of a control group limits the ability to attribute improvements in PFWD and ABI solely to the K2VD3 intervention, as changes may also result from lifestyle modifications or natural disease progression. Additionally, the study's reliance on self-reported measures of walking distance introduces potential bias, as participants may overestimate their capabilities. The sample size, while providing some insights, may not be sufficient to generalize findings across a broader population of patients with PAD. Long-term adherence to the lifestyle therapy and K2VD3 supplementation was not monitored, which could influence the outcomes. Finally, the observational nature of the study means that confounding variables could not be controlled, necessitating further randomized controlled trials to validate these findings and establish causality.

Future research directions

To validate our findings and better understand the efficacy of K2VD3, we recommend the following future research directions:

- Randomized controlled trials (RCTs): Conducting RCTs with appropriate control groups is essential to establish causality. This design would allow for direct comparisons between patients receiving K2VD3 and those receiving a placebo or standard treatment, thereby providing clearer insights into the intervention's effectiveness.
- Long-term effects: Longitudinal studies that assess the long-term effects of K2VD3 supplementation on PFWD, ABI, and overall vascular health are crucial. Understanding the sustainability of benefits over extended periods can inform clinical recommendations and patient management strategies.
- Mechanistic studies: Future research should explore the underlying mechanisms by which K2VD3 affects vascular health. Investigating the biochemical pathways and physiological processes involved can enhance our understanding of how these compounds work individually and synergistically.
- Diverse populations: It would be also beneficial to investigate the effects of K2VD3 in diverse populations, including different age groups, genders, and ethnic backgrounds, to determine if the observed benefits are consistent across various demographics.
- Combination therapies: Exploring the efficacy of K2VD3 in combination with other therapeutic interventions, such as structured exercise programs or dietary modifications, could provide insights into integrated approaches for managing PAD.

By addressing these limitations and pursuing these research directions, we can gain a deeper understanding of the potential role of K2VD3 in the management of peripheral artery disease and improve clinical practice in this area.

Conclusion

This observational study provides promising evidence that the combination of vitamin K2, vitexin, and vitamin D3 (K2VD3) can significantly improve PFWD and ABI in patients with claudicatio intermittens due to PAD when used alongside best medical therapy and a structured exercise program. The observed enhancements in vascular function suggest that K2VD3 may play a vital role in promoting vascular health and mitigating the effects of PAD. However, due to the study's limitations, including the absence of a control group and potential biases in self-reported measures, these findings should be interpreted with caution. Future randomized controlled trials and long-term follow-up studies are essential to confirm the efficacy of K2VD3, explore its mechanisms of action further, and establish its role in the comprehensive management of PAD.

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Authors' contributions
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Informed consent
Informed consent has been obtained from participants, for both clinical and scientific purpose.

References

1. Gornik HL, Aronow HD, Goodney PP, et al. 2024 ACC/AHA/AACVPR/APMA/ABC/SCAI/SVM/SVN/SVS/SIR/VESS Guideline for the Management of Lower Extremity Peripheral Artery Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation 2025;151:e918.
2. Polonsky TS, McDermott MM. Lower Extremity Peripheral Artery Disease Without Chronic Limb-Threatening Ischemia: A Review. JAMA 2021;325:2188–2198.
3. Kansal A, Davis AM, Rymer JA. Management of Peripheral Artery Disease. JAMA 2025;334:444–445.
4. Kullo IJ, Rooke TW. Clinical practice. Peripheral artery disease. N Engl J Med 2016;374:861–871.
5. Lu M, Zhang L, Pan J, et al. Advances in the study of the vascular protective effects and molecular mechanisms of hawthorn (Crataegus anamesa Sarg.) extracts in cardiovascular diseases. Food Funct 2023;14:5870–5890.
6. Wu M, Liu L, Xing Y, et al. Roles and Mechanisms of Hawthorn and Its Extracts on Atherosclerosis: A Review. Front Pharmacol 2020;11:118.
7. Wang J, Xiong X, Feng B. Effect of crataegus usage in cardiovascular disease prevention: an evidence-based approach. Evid Based Complement Alternat Med 2013;2013:149363.
8. Lees JS, Chapman FA, Witham MD, et al. Vitamin K status, supplementation and vascular disease: a systematic review and meta-analysis. Heart 2019;105:938–945.
9. Tsugawa N. Cardiovascular Diseases and Fat Soluble Vitamins: Vitamin D and Vitamin K. J Nutr Sci Vitaminol (Tokyo). 2015;61:S170–S172.

10. Geleijnse JM, Vermeer C, Grobbee DE, et al. Dietary intake of menaquinone is associated with a reduced risk of coronary heart disease: the Rotterdam Study. J Nutr 2004;134:3100–3105.
11. Latic N, Erben RG. Vitamin D and Cardiovascular Disease, with Emphasis on Hypertension, Atherosclerosis, and Heart Failure. Int J Mol Sci 2020;21:6483.
12. Cuschieri S. The STROBE guidelines. Saudi J Anaesth 2019;13:S31–S34.
13. Norman PE, Powell JT. Vitamin D and cardiovascular disease. Circ Res 2014;114:379–393.
14. Shea MK, Berkner KL, Ferland G, et al. Perspective: Evidence before Enthusiasm – A Critical Review of the Potential Cardiovascular Benefits of Vitamin K. Adv Nutr 2021;12:632–646.
15. Shioi A, Morioka T, Shoji T, et al. The Inhibitory Roles of Vitamin K in Progression of Vascular Calcification. Nutrients 2020;12:E583.
16. Zhao CR, Yang FF, Cui Q, et al. Vitexin inhibits APEX1 to counteract the flow-induced endothelial inflammation. Proc Natl Acad Sci U S A. 2021;118:e2115158118.
17. Gan T, Xing Q, Li N, et al. Protective Effect of Vitexin Against IL-17-Induced Vascular Endothelial Inflammation Through Keap1/Nrf2-Dependent Signaling Pathway. Mol Nutr Food Res 2024;68:e2300331.
18. Li W, Deng Z, Xiao S, et al. Protective effect of vitexin against high fat-induced vascular endothelial inflammation through inhibiting trimethylamine N-oxide-mediated RNA m6A modification. Food Funct 2024;15:6988–7002.
19. Verhave G, Siegert CE. Role of vitamin D in cardiovascular disease. Neth J Med 2010;68:113–118.
20. Legarth C, Grimm D, Krüger M, et al. Potential Beneficial Effects of Vitamin D in Coronary Artery Disease. Nutrients 2019;12:99.
21. Rimondi E, Marcuzzi A, Casciano F, et al. Role of vitamin D in the pathogenesis of atheromatosis. Nutr Metab Cardiovasc Dis 2021;31:344–353.
22. Kim DH, Meza CA, Clarke H. Vitamin D and Endothelial Function. Nutrients 2020;12:575.
23. Winckler K, Tarnow L, Lundby-Christensen L, et al.; CIMT trial group. Vitamin D, carotid intima-media thickness and bone structure in patients with type 2 diabetes. Endocr Connect 2015;4:128–135.