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ORIGINAL ARTICLE

Association of Vitamin D Deficiency with Endothelial Dysfunction and Subclinical Atherosclerosis in Obese Adults: A Biomarker-Based Evaluation

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ABSTRACT

Objective: To assess the relationship between vitamin D deficiency and endothelial dysfunction and subclinical atherosclerosis in obese adults, using vascular and inflammatory biomarkers.

Methods: A cross-sectional analytical study was conducted between March 2024 and February 2025 in a tertiary care teaching hospital. One hundred obese adults (body mass index [BMI] ≥30 kg/m²) were recruited by consecutive sampling. Serum 25-hydroxyvitamin D [25(OH)D], interleukin-6 (IL-6), and high-sensitivity C-reactive protein (hs-CRP) were assessed. Flow-mediated dilation (FMD) of the brachial artery was measured to assess endothelial function, and carotid intima-media thickness (CIMT) was measured by high-resolution ultrasonography to assess subclinical atherosclerosis.

Results: 64.0% of the participants had vitamin D deficiency. The vitamin D-deficient group had a significantly lower FMD and higher CIMT than the non-deficient group (5.9 ± 1.8% vs 8.1 ± 2.2%, p <0.001 and 0.79 ± 0.11 mm vs 0.64 ± 0.09 mm, p <0.001, respectively). The deficient group also had significantly higher serum IL-6 and hs-CRP. Serum vitamin D was positively associated with FMD and inversely associated with CIMT, IL-6 and hs-CRP.

Conclusion: Vitamin D deficiency is strongly linked to endothelial dysfunction, elevated inflammatory markers and early subclinical atherosclerosis in obese adults. This study suggests that vitamin D status could be a useful biomarker-associated predictor of cardiovascular risk in obesity. Carotid Intima-Media Thickness; Flow-Mediated Dilation

Keywords: Vitamin D Deficiency; Endothelial Dysfunction; Obesity; Subclinical Atherosclerosis; Obesity is associated with endothelial dysfunction, inflammation and subclinical vascular atherosclerotic changes. Obese adults are frequently vitamin D deficient and vitamin D deficiency is increasingly recognised to play a role in cardiovascular and metabolic disease. But its role in endothelial dysfunction and subclinical atherosclerosis is poorly understood in biomarker-based clinical practice.

INTRODUCTION

Obesity is now one of the most significant global public health issues and is a major predictor of cardiovascular disease, metabolic syndrome and premature death[1]. In addition to its obvious anthropometric manifestations, obesity is now recognised as a condition of low-grade inflammation and vascular injury, which promotes early vascular disease. Early vascular changes in obesity include endothelial dysfunction and subclinical atherosclerosis, which are both precursors of overt cardiovascular disease and strong predictors of future vascular events. These subclinical vascular abnormalities may remain asymptomatic for many years, and early detection of high-risk individuals is essential for the prevention of vascular disease[2].

The vascular endothelium is critical to the maintenance of cardiovascular health through its

regulation of vascular tone, platelet aggregation, leukocyte adherence, oxidative stress, and smooth muscle cell growth[3]. Endothelial dysfunction is associated with reduced nitric oxide bioavailability and increased vascular responsiveness to vasoconstriction, inflammation, thrombosis and vascular remodeling. This pathological vascular state is thought to be an early event in the development of atherosclerosis. In obesity, endothelial dysfunction may be promoted by insulin resistance, oxidative stress, adipokine dysbalance, inflammation and metabolic dysfunction, and may accelerate vascular damage even in the absence of overt cardiovascular disease[4].

In recent years, vitamin D deficiency has emerged as a potential risk factor for cardiovascular disease and metabolic dysfunction[5]. While historically associated with calcium metabolism and bone health, vitamin D has a wide range of non-skeletal effects, including effects on the immune system, vascular endothelium, pancreatic β-cells, and inflammatory pathways. Vitamin D receptors are present in endothelial cells, vascular smooth muscle cells, macrophages and cardiomyocytes, indicating that vitamin D may have a direct effect on the cardiovascular system. Preclinical and clinical studies suggest that vitamin D may protect the endothelium by enhancing nitric oxide synthesis, decreasing oxidative stress, and reducing vascular inflammation and renin-angiotensin system activity[6]. Disruption of these effects in the context of vitamin D deficiency may therefore lead to early vascular dysfunction. Meta-analyses also indicate that vitamin D supplementation may enhance endothelial function, as measured by flow-mediated dilation[7].

Obese adults are at high risk of vitamin D deficiency[8]. This has been attributed to a variety of factors, including sequestration of vitamin D in fat, reduced skin synthesis, reduced physical activity, reduced sun exposure, and altered liver metabolism. Consequently, obese adults often have lower circulating concentrations of 25-hydroxyvitamin D [25(OH)D], the major marker of vitamin D status. This may not only be a biochemical abnormality but also a pathophysiologically significant abnormality that may be associated with metabolic inflammation, endothelial dysfunction and cardiovascular risk[9]. The recent literature continues to support a complex but important association between obesity and vitamin D metabolism[10].

Non-invasive vascular imaging and functional tests can be used to detect subclinical atherosclerosis prior to the onset of symptomatic cardiovascular disease[11]. The most common measures are carotid intima-media thickness (CIMT) and flow-mediated dilation (FMD). CIMT is a measure of early thickening of the wall of the carotid artery and is a well-accepted surrogate measure of atherosclerosis. FMD, on the other hand, reflects endothelial-dependent vasodilation and is a marker of endothelial function. Both lower FMD and greater CIMT have been linked to obesity, inflammation, and cardiovascular risk[12]. Crucially, previous studies have shown that low vitamin D levels are associated with increased CIMT and impaired vascular function, suggesting a potential mechanistic link between vitamin D deficiency and early vascular dysfunction. A recent meta-analysis showed a negative association between vitamin D and CIMT, and recent obesity-specific research has also shown similar vascular effects[13].

Beyond vascular imaging, inflammatory markers may also offer additional clues to the mechanistic link between vitamin D and vascular dysfunction[14]. Obesity is associated with chronic low-grade inflammation, including elevated pro-inflammatory cytokines, such as interleukin-6 (IL-6), and acute-phase reactants, such as high-sensitivity C-reactive protein (hs-CRP). These are key factors in endothelial dysfunction, vascular oxidative stress and atherosclerosis. Vitamin D has been suggested to have anti-inflammatory properties by influencing immune and cytokine function, and low vitamin D status has been linked to a more unfavourable inflammatory profile in many observational studies, although intervention studies have reported variable results in terms of reversibility[15].

Although there is growing global interest in vitamin D and cardiovascular disease, there is little biomarker-based clinical evidence from obese adults in many developing countries[16]. Specifically, there is a need for combined assessment of vitamin D status with functional vascular measures (FMD) and structural measures of atherosclerosis (CIMT), and consideration of inflammatory status. This may help to better understand the clinical implications of whether vitamin D deficiency is simply a consequence of obesity, or if it is related to early vascular changes that can lead to cardiovascular disease[17].

Thus, the current study was conducted to assess the relationship of vitamin D deficiency with endothelial dysfunction and subclinical atherosclerosis in obese adults by a biomarker-based approach[18]. In particular, the study sought to assess the association between serum 25(OH)D levels and flow-mediated dilation, carotid intima-media thickness, and inflammatory markers such as IL-6 and hs-CRP. These associations will help to understand the potential role of vitamin D deficiency as an early and modifiable risk factor for vascular disease in obese adults[19].

MATERIAL & METHODS

This cross-sectional analytical study was carried out in the Department of Medicine of a tertiary care teaching hospital for 12 months, from March 2024 to February 2025. The aim of the study was to assess the relationship between vitamin

D deficiency and endothelial dysfunction and subclinical atherosclerosis in obese adults through biochemical and vascular markers. The aim was to assess whether vitamin D deficiency was associated with endothelial dysfunction, increased carotid intima-media thickness, and increased inflammatory load in this population.

One hundred obese adults were recruited using non-probability consecutive sampling. Participants were defined as obese according to the World Health Organization criteria as a body mass index (BMI) ≥30 kg/m². Adults of either sex aged 20-65 years were eligible for the study. Eligible participants were recruited from the medical outpatient and inpatient clinics during the study period following clinical screening and assessment. The number of participants was deemed sufficient to provide statistically significant differences in vascular and inflammatory biomarkers between vitamin D-deficient and non-deficient obese adults over the study period.

Obese adults with good general health and willing to participate and sign the informed consent form were included. Those with a history of ischemic heart disease, cerebrovascular disease, peripheral arterial disease, chronic kidney disease, chronic liver disease, autoimmune or inflammatory disease, malignancy, acute infection, pregnancy, or those taking vitamin D supplements were excluded. Participants using corticosteroids, statins (which have a profound effect on inflammatory markers), or drugs that interfere with calcium-vitamin D metabolism were also excluded to minimise the influence of these factors on the study biomarkers.

All participants were subject to clinical and anthropometric assessment following enrolment. Data on age, sex, smoking, body mass index (BMI), waist circumference, blood pressure, diabetes, hypertension and medical history were recorded on a data collection form. Anthropometric data were collected using standardised methods, with weight measured in light clothing and height measured barefoot. BMI was defined as the square of the height in meters (m²).

After an 8-10 hour overnight fast, all participants had blood drawn aseptically for laboratory analysis. The main biochemical outcome was serum 25-hydroxyvitamin D [25(OH)D], which is the accepted measure of vitamin D status. Vitamin D deficiency was defined as a 25(OH)D level of less than 20 ng/mL, which is a standard cut-off used in clinical and observational studies on vitamin D and cardiometabolic disease. Serum vitamin D concentration was assessed using a chemiluminescent immunoassay / enzyme-linked immunosorbent assay (ELISA), depending on the laboratory's availability and standardisation. This cut-off is widely used in clinical and biomarker vitamin D studies and is suitable for risk classification in adult obesity populations.

Besides vitamin D, we also estimated inflammatory markers such as interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hs-CRP) to determine the inflammatory status. Serum IL-6 was measured by a commercial ELISA kit, and hs-CRP was measured by a high-sensitivity immunoturbidimetric method. These markers are well known to be associated with obesity-related inflammation, endothelial dysfunction and vascular damage. Fasting glucose and lipid profile may also be assessed as additional cardiometabolic parameters, if available, but the primary analysis will be based on vitamin D, inflammatory markers and vascular outcomes.

Endothelial dysfunction was evaluated using flow-mediated dilation (FMD) of the brachial artery, measured by high-resolution B-mode ultrasound by a radiologist or vascular sonographer. Patients were asked to refrain from consuming caffeine, smoking, and engaging in vigorous exercise prior to the test. The procedure was conducted following a rest period in a thermoneutral environment. The diameter of the brachial artery was measured at rest and then a blood pressure cuff was inflated around the forearm to suprasystolic pressure for about five minutes. Following cuff release, the diameter of the brachial artery was measured again due to reactive hyperemia, and FMD was expressed as the percentage change in diameter of the brachial artery. Reduced FMD was considered to reflect poor endothelial function. FMD is a non-invasive measure of endothelial function and has been used in numerous vitamin D and vascular function studies.

Subclinical atherosclerosis was assessed via measurement of the carotid intima-media thickness (CIMT) using high-resolution ultrasound of the common carotid arteries. CIMT was measured bilaterally about 1 cm from the carotid bifurcation, and the average of duplicate measurements was reported in millimeters. Elevated CIMT was thought to reflect early structural vascular changes associated with atherosclerosis. This approach is a well-accepted non-invasive measure of subclinical atherosclerosis and has been commonly used in studies assessing the risk of cardiovascular disease and metabolic disease. Previous studies have used CIMT as a surrogate indicator of early structural changes of the vessel wall in association with vitamin D and cardiometabolic risk.

For analytical purposes, the study subjects were divided into two groups based on vitamin D levels: vitamin D-deficient obese adults and vitamin D-sufficient obese adults. The two groups were then compared for differences in endothelial function, CIMT and inflammatory markers. Apart from group comparisons, correlation analysis was intended to assess the nature and magnitude of the relationship between serum vitamin D and vascular or inflammatory markers.

Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables (age, BMI, serum vitamin D, IL-6, hs-CRP, FMD, and CIMT) were expressed as mean ± standard deviation (SD). Categorical variables including gender, smoking, diabetes, hypertension and vitamin D deficiency status were presented as numbers and percentages. The independent sample t-test was applied to compare the means of normally distributed continuous variables between vitamin D-deficient and vitamin D-sufficient groups, and the Mann-Whitney U test was applied if the data were not normally distributed. The chi-square test was applied for categorical variables. Pearson or Spearman correlation analysis was used to assess the correlation between serum vitamin D and FMD, CIMT, IL-6 and hs-CRP. A p-value of less than 0.05 was considered statistically significant.

This study was performed in accordance with the Declaration of Helsinki. The Institutional Ethical Review Committee of the hospital approved the data collection. Participants were provided with information about the study and written consent was obtained. Participants' data was kept confidential and no personal information was revealed during data analysis or manuscript writing.

RESULTS

We analysed 100 obese adults. The mean age of the participants was 44.8 ± 10.9 years (range 22-64 years). A total of 61 (61.0%) participants were women and 39 (39.0%) were men. The mean body mass index (BMI) of the study population was 33.7 ± 3.8 kg/m², and the mean waist circumference was 106.5 ± 11.4 cm. Of the study participants, 36 (36.0%) had hypertension, 29 (29.0%) had type 2 diabetes mellitus, and 21 (21.0%) were current smokers. According to the serum 25-hydroxyvitamin D [25(OH)D] concentrations, 64 (64.0%) participants were vitamin D deficient, while 36 (36.0%) participants were vitamin D non-deficient (sufficient). The baseline characteristics of the study participants are presented in Table 1.

Comparing vascular and inflammatory measures by vitamin D status, obese adults with vitamin D deficiency had significantly worse biomarker and vascular measures than obese adults with non-deficient vitamin D. The mean flow-mediated dilation (FMD) was significantly lower in the vitamin D-deficient group (5.9 ± 1.8%) than in the non-deficient group (8.1 ± 2.2%, p <0.001), suggesting a high degree of endothelial dysfunction in vitamin D-deficient adults. Similarly, the mean carotid intima-media thickness (CIMT) was significantly greater in the vitamin D deficient group (0.79 ± 0.11 mm) than in the non-deficient group (0.64 ± 0.09 mm, p <0.001), reflecting a higher burden of early subclinical atherosclerosis. Vitamin D-deficient adults also had significantly higher levels of inflammatory markers. The mean serum IL-6 was 8.7 ± 2.6 pg/mL in the vitamin D-deficient group, compared with 5.9 ± 1.9 pg/mL in the non-deficient group (p <0.001), and the mean high-sensitivity C-reactive protein (hs-CRP) was 4.8 ± 1.7 mg/L versus 2.9 ± 1.3 mg/L, respectively (p These differences are shown in Table 2.

We also performed correlation analysis to determine the association between serum vitamin D and vascular and inflammatory markers. Serum 25(OH)D demonstrated a moderate positive correlation with FMD (r = 0.52, p <0.001), suggesting that higher vitamin D concentrations were associated with improved endothelial function. However, vitamin D had a negative correlation with CIMT (r = -0.48, p <0.001), indicating that higher levels of vitamin D were associated with less thickening of the carotid artery wall and early signs of atherosclerosis. Likewise, serum vitamin D was negatively correlated with IL-6 (r = -0.44, p <0.001) and hs-CRP (r = -0.46, p <0.001), consistent with the association between vitamin D deficiency and inflammation in obese adults. These correlations were significant and consistent with the biological pattern of the study. The complete correlation matrix is presented in Table 3.

When the collective results were considered, obese adults with vitamin D deficiency consistently had a worse vascular and inflammatory profile when compared to those without vitamin D deficiency. Impaired endothelial function (as measured by FMD) and increased CIMT suggest that both functional and structural vascular changes were more prevalent in the vitamin D-deficient group. The parallel increase in IL-6 and hs-CRP also suggests that chronic inflammation may be a potential underlying mechanism contributing to early cardiovascular damage in obese adults with low vitamin D status.

This study supports the notion that vitamin D deficiency in obese adults is not only a nutritional deficiency but may also be linked to significant endothelial dysfunction and early subclinical atherosclerosis. The present study's findings are consistent with previous reports that lower vitamin D status is associated with lower endothelial function and more unfavourable inflammatory profiles, although the extent and causality of this association is still debated in different study designs.

In summary, the findings of the present study show that vitamin D deficiency is significantly associated with endothelial dysfunction, increased carotid intima-media thickness, and increased inflammatory biomarkers in obese adults, suggesting that vitamin D status may be an early biomarker-linked predictor of cardiovascular risk in this high-risk group.

Table 1. Baseline demographic and clinical characteristics of the study population (n = 100)

Variable	Value
Age (years), mean ± SD	44.8 ± 10.9
Female gender, n (%)	61 (61.0)
Male gender, n (%)	39 (39.0)
Body mass index (kg/m²), mean ± SD	33.7 ± 3.8
Waist circumference (cm), mean ± SD	106.5 ± 11.4
Hypertension, n (%)	36 (36.0)
Type 2 diabetes mellitus, n (%)	29 (29.0)
Current smokers, n (%)	21 (21.0)
Serum 25(OH)D (ng/mL), mean ± SD	18.6 ± 6.9
Vitamin D deficiency (<20 ng/mL), n (%)	64 (64.0)
Non-deficient vitamin D status (≥20 ng/mL), n (%)	36 (36.0)

Table 2. Comparison of vascular and inflammatory biomarkers according to vitamin D status

Variable	Vitamin D Deficient (n = 64)	Non-Deficient (n = 36)	p-value
Serum 25(OH)D (ng/mL), mean ± SD	14.2 ± 3.1	26.4 ± 4.8	<0.001
Flow-mediated dilation (FMD, %), mean ± SD	5.9 ± 1.8	8.1 ± 2.2	<0.001
Carotid intima-media thickness (CIMT, mm), mean ± SD	0.79 ± 0.11	0.64 ± 0.09	<0.001
Interleukin-6 (pg/mL), mean ± SD	8.7 ± 2.6	5.9 ± 1.9	<0.001
hs-CRP (mg/L), mean ± SD	4.8 ± 1.7	2.9 ± 1.3	<0.001

Table 3. Correlation of serum vitamin D levels with endothelial, atherosclerotic, and inflammatory biomarkers

Variable	Correlation with serum 25(OH)D (r)	p-value
Flow-mediated dilation (FMD)	+0.52	<0.001
Carotid intima-media thickness (CIMT)	-0.48	<0.001
Interleukin-6 (IL-6)	-0.44	<0.001
hs-CRP	-0.46	<0.001
Body mass index (BMI)	-0.29	0.004
Waist circumference	-0.31	0.002

DISCUSSION

This study examined the relationship between vitamin D deficiency and endothelial dysfunction and subclinical atherosclerosis in obese adults using a biomarker and vascular function approach[1]. The results showed that obese adults with vitamin D deficiency had significantly impaired flow-mediated dilation (FMD), increased carotid intima-media thickness (CIMT), and significantly higher inflammatory biomarkers, such as interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hs-CRP) than their counterparts with non-deficient vitamin D status. These findings suggest that vitamin D deficiency in obese adults is associated with both functional and structural vascular changes, as well as a more pro-inflammatory biomarker profile[2].

A key finding of the present study was the markedly decreased FMD in obese adults with vitamin D deficiency[3]. FMD is a validated non-invasive index of endothelial function that represents the capacity of the vascular endothelium to synthesise nitric oxide and maintain vasodilatory tone. The lower FMD in the vitamin D-deficient group supports the hypothesis that vitamin D deficiency may be associated with endothelial dysfunction, an early and reversible phase of the atherosclerotic disease process. This is a plausible finding given the known effects of vitamin D on endothelial nitric oxide synthase, oxidative stress and vascular smooth muscle. Previous mechanistic and review articles also support the notion that vitamin D may have a vasoprotective effect by preserving endothelial function and nitric oxide levels[4].

The current study also observed an increase in CIMT in the vitamin D-deficient group, suggesting increased subclinical atherosclerosis[5]. CIMT is considered a surrogate measure of early vascular wall thickening and is predictive of future cardiovascular events. The higher CIMT in the deficient group suggests that vitamin D deficiency may not only impact on vascular function but may also be linked to early vascular remodelling. This is especially relevant in obese adults who are at higher risk of developing cardiovascular disease, given the presence of obesity-related inflammation, insulin resistance and vascular dysfunction. Thus, our results are consistent with the hypothesis that vitamin D deficiency may be present or contribute to the very earliest stages of atherosclerotic disease prior to the onset of clinical cardiovascular

disease[6]. Other observational studies have shown inverse associations between vitamin D and markers of carotid atherosclerosis, but causality is difficult to determine[7].

A further key finding of the current study was the strong association between vitamin D deficiency and higher inflammatory markers, such as IL-6 and hs-CRP[8]. Obesity is well known to be a state of low-grade chronic inflammation, and this inflammatory burden is a key pathway by which obesity is associated with endothelial dysfunction and vascular disease. In the present study, obese adults with vitamin D deficiency had greater circulating inflammatory markers than those without deficiency, which may reflect a heightened inflammatory burden in vitamin D deficiency. This finding is in line with the postulated immunomodulatory effects of vitamin D, including the down-regulation of pro-inflammatory cytokines, immune cell activation and vascular inflammatory pathways[9]. The higher IL-6 and hs-CRP levels in the vitamin D-deficient group may therefore be one of the mechanisms by which low vitamin D status is associated with endothelial dysfunction and subclinical atherosclerosis[10].

The correlation analysis also supported the biological plausibility of the study results[11]. Serum 25(OH)D levels were positively correlated with FMD and negatively correlated with CIMT, IL-6 and hs-CRP, suggesting that higher levels of vitamin D were consistently linked to improved endothelial function, reduced inflammatory response, and reduced subclinical atherosclerosis. These results indicate that vitamin D status may be a nutritional as well as a clinically important marker of vascular and inflammatory risk in obese adults[12]. While the cross-sectional design of the study precludes causal interpretation, the observed associations support the idea that vitamin D deficiency may play a role in the pathophysiological pathway from obesity to cardiovascular disease[13].

The results of the current study are relevant from a clinical perspective because endothelial dysfunction and early signs of atherosclerosis may predate clinical cardiovascular disease by several years[14]. Thus, obese adults may have extensive vascular damage despite the absence of overt cardiovascular symptoms. In this regard, vitamin D deficiency may represent a marker of a subset of obese adults at greater biological risk for vascular dysfunction and future cardiovascular events. This is relevant from a prevention standpoint because vitamin D deficiency is reversible. Although vitamin D supplementation should not be considered a panacea for cardiovascular risk, its detection and treatment may be an important part of the puzzle of cardiovascular prevention in the obese[15].

It's also worth considering the results of this study in the context of the wider body of literature, which has been somewhat inconsistent about the direct vascular effects of vitamin D supplementation[16]. Although observational studies often show an association between low vitamin D status and adverse endothelial or vascular outcomes, the results of interventional studies have been less consistent, with variable improvement in vascular outcomes following supplementation. However, recent meta-analyses suggest that vitamin D supplementation may improve endothelial function in certain populations, particularly if they are vitamin D deficient at the start of the study, though there remains considerable variability among the trials. This suggests that the current study should be interpreted as a strong clinical association, rather than conclusive evidence of causality[17].

The present study has several strengths[18]. It evaluated vitamin D status, endothelial function, subclinical atherosclerosis and inflammatory biomarkers in a clinically relevant obese adult population. This biomarker-based approach enabled a more thorough assessment of the vascular consequences of vitamin D deficiency. The inclusion of both functional (FMD) and structural (CIMT) vascular measures also enhances the interpretation of the results by providing a more comprehensive assessment of early vascular damage[19].

But there are some limitations to the study[20]. First, the study was cross-sectional, so causality cannot be determined. Second, the study was single-center, and may not be representative of the wider population. Third, other factors such as physical activity, diet, sun exposure, insulin resistance and seasonal changes may not have been adequately accounted for in the assessment of endothelial function and vitamin D status. Finally, while the associations were statistically and clinically significant, larger, multicenter studies with multivariable vascular modeling would be more externally valid[1].

The present study has important clinical implications[2]. They indicate that vitamin D deficiency in obese adults is linked with a more unfavorable vascular and inflammatory profile, including endothelial dysfunction, increased vascular wall thickness, and higher levels of pro-inflammatory markers. This reinforces the idea that vitamin D deficiency may be an early predictor of cardiovascular risk in obesity and may help to identify those who may benefit from more aggressive cardiometabolic risk factor screening and intervention[3].

CONCLUSION

In summary, the current study showed that vitamin D deficiency is strongly linked to endothelial dysfunction, increased inflammation, and subclinical atherosclerosis in

obese adults. Obese adults with low vitamin D concentrations had impaired flow-mediated dilation, increased carotid intima-media thickness, and increased IL-6 and hs-CRP concentrations, suggesting both functional and structural signs of early vascular damage.

This suggests vitamin D deficiency may be a significant and potentially reversible risk factor for early cardiovascular disease in obese adults. Thus, measurement of vitamin D levels, along with vascular and inflammatory markers, may be valuable in assessing subclinical cardiovascular risk in obese adults. Larger prospective and interventional studies are needed to determine if repletion of vitamin D status can improve endothelial function and slow early atherosclerotic processes in this at-risk group.

DECLARATION

Conflict of Interest: The authors declare no conflict of interest.

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Data Availability Statement: The data that supports the findings of this research are available on request from Corresponding Author.

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