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Original Article

The Association Vitamin D and Left Ventricular Hypertrophy in Metabolic Syndrome Patients

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Abstract

Background : Vitamin D deficiency is common, especially in people with metabolic syndrome. This condition increases the risk of cardiovascular problems, including left ventricular hypertrophy (LVH). While the connection between metabolic syndrome and LVH is well-documented, it is still unclear whether vitamin D deficiency alone contributes to the development of LVH in these patients. The aims of this study was to the association between vitamin D levels and LVH in patients with metabolic syndrome.
Methods : A cross-sectional study was conducted with 38 patients diagnosed with metabolic syndrome in Kariadi Hospital, Semarang. Serum vitamin D levels were measured using the ELISA method, and LVH was diagnosed via echocardiography. Patients were categorized into normal and LVH groups. Vitamin D levels were classified as sufficient, insufficient, or deficient. The relationship between vitamin D levels, metabolic syndrome components, and LVH was analyzed.
Results : Our findings revealed no significant difference in vitamin D levels between patients with LVH and those without it (mean 19.98 ± 5.59 ng/mL for the LVH group vs. 20.91 ± 6.56 ng/mL for the normal group, p=0.65). However, patients with LVH had a significantly higher number of metabolic syndrome components compared to those without LVH (p=0.044).
Conclusion : While no direct association was found between vitamin D levels and LVH, the cumulative burden of metabolic syndrome components plays a significant role in the development of LVH. Future research should explore larger populations to investigate the therapeutic potential of vitamin D in cardiovascular outcomes.

Keywords : Cardiovascular Disease, Left Ventricular Hypertrophy, Metabolic Syndrome, Vitamin D

INTRODUCTION

Vitamin D deficiency has become a major health problem worldwide, as studies reveal high prevalence rates,[1,2] commonly in conditions where sun exposure and nutritional supplies of vitamin D are repeatedly low.[3] This deficiency is especially pronounced in individuals with metabolic syndrome, a cluster of interrelated disorders characterized by central obesity, insulin resistance, dyslipidemia, and hypertension, who exhibit lower circulating vitamin D levels[4] and impaired vitamin D synthesis.[5–7]

Metabolic syndrome is a well-known contributor to the development of cardiovascular diseases, particularly left ventricular hypertrophy (LVH), a structural cardiac abnormality that significantly increases the risk of heart failure.[8–10] While vitamin D deficiency has been linked to the progression of LVH in hypertensive patients,[11–12] its specific role in metabolic syndrome remains unclear. Addressing this knowledge gap is essential for understanding the interplay between metabolic syndrome, vitamin D status, and LVH.

Based on these considerations, we hypothesize that lower levels of vitamin D in patients with metabolic syndrome may be associated with an increased risk of LVH. This study aims to explore the relationship between vitamin D deficiency and the incidence of LVH in patients with metabolic syndrome. We thereby can examine how vitamin D might influence mechanisms of cardiovascular remodeling and thus perhaps provide new perspectives on therapeutic approaches aimed at alleviating left ventricular hypertrophy in this vulnerable demographic.

METHODS

This observational study utilized a cross-sectional design to examine 38 patients with metabolic syndrome, consisting of 19 patients with left ventricular hypertrophy (LVH) and 19 patients with normal cardiac function, selected through purposive sampling from the inpatient and outpatient wards of Kariadi Hospital Semarang on July and October 2022. Inclusion criteria include (1) being ≥18 years old; (2) having fulfilled the criteria for Metabolic Syndrome based on National Cholesterol Education Program Expert Panel and Adult Treatment Panel III (NCEP ATP III)[13] and (3) being willing to take part in this research. Exclusion criteria included : (1) pregnancy, (2) patients receiving vitamin D therapy, (3) patients with severe infection or sepsis, (4) malabsorption syndrome, (5) patients receiving phenytoin or phenobarbital therapy, (6) chronic liver disease, (7) patients who had undergone total thyroidectomy, (8) chronic renal failure, and (9) malignancies. The level of vitamin D in the serum was assessed using the ELISA method from blood samples provided by the patients. Vitamin D is categorized as sufficient for more than 30 ng/ml, insufficient at 20 to 30 ng/mL, and deficient if less than 20 ng/m. The left ventricular geometry profile was classified as normal or left ventricular hypertrophy (LVH). LVH was defined as Increased left ventricle mass (LVMI) >115 g/m^2 for males and >95 g/m^2 for females as described by the American society of Echocardiography.[14]

The metabolic syndrome status of each patient was evaluated by six components modified from NCEP ATP III[11] (1) Waist circumference > 102 cm for male or > 88 cm for female, (2) serum triglyceride >150 mg/dL, (3) HDL <40 mg/dL for males and < 50 mg/dL for females, (4) blood pressure > 130 / 85 mmHg, (5) fasting glucose > 110 mg/dL and additional (6) obesity. Obesity was taken to be present if BMI exceeded 25 kg/m^2. For each patient, anthropometric measurements, vitamin D levels, and other laboratory tests were collected after the subjects were categorized as having either normal or left ventricular hypertrophy (LVH).

This research has been getting informed consent from patients and the Ethical Clearance of Health Research Ethics Committee of the Faculty of Medicine Diponegoro University and has received permission from the Director of the Kariadi Hospital. The number of ethical approvals was 1193/EC/KEPK-RKDK/2022. To reduce selection biases, information biases, and confounding, inclusion and exclusion were strictly imposed for each subject by at least two examiners. All echocardiographic measurements were done by an internal medicine specialist and cardiovascular consultant, blinded to the results of the other tests.

RESULTS

The characteristics of the patient data we collected from the outpatient ward of Kariadi Hospital from July to October 2022 are presented in Table 1. We observed a trend of decreasing vitamin D levels in patients with LVH, with average level of 20.91 ± 6.56 ng/mL in those with normal LV function and 19.98 ± 5.59 ng/mL in those with LVH, although this difference was not statistically significant (Fig. 1A). A comparison of the observed frequencies of vitamin D deficiency among patients with and without LVH revealed no significant deviation from the expected frequencies (Fig. 1B).

We investigated whether vitamin D influences the occurrence of metabolic syndrome. Our data showed that the level of vitamin D varies between group of metabolic syndromes, 22.10 ± 0.50 ng/mL in patients with two components, 19.30 ± 2.67 ng/mL with three components, 21.76 ± 8.57 ng/mL with four components, 19.97 ± 5.23 ng/mL with five components, and 18.03 ± 4.67 ng/mL with six components. However, these differences were not statistically significant either within or between groups (Fig 1C). Additionally, the observed frequencies of vitamin D deficiency among patients with metabolic syndrome did not differ significantly (Fig 1D).

Our analysis showed a statistically significant association between the number of metabolic syndrome components and the presence of LVH (Fig. 2A) (Mann Whitney, $p = 0.044$). Patients with LVH showed a similar median number of metabolic syndrome components compared to those normal (median = four components in both normal and the LVH group. However, the distribution of metabolic syndrome components skewed from two to five metabolic syndrome components in normal group toward higher values of three to six metabolic syndrome components in the LVH group.

To evaluate the contribution of individual metabolic syndrome factors to the occurrence of LVH, we analyzed six components of metabolic syndrome. Our

TABLE 1
Characteristics of Experimental Subject

Patient characteristics	N	Frequency (%)	Mean ± SD / Median (min-max)
Age			54.42 ± 10.41
<60 years	28	73.68	
>60 years	10	26.32	
Gender			
Male	30	78.95	
Female	8	21.05	
Vitamin D levels			20.44 ± 6.19
Normal	1	2.63	
Insufficiency	17	44.74	
Deficiency	20	52.63	
Left ventricle hypertrophy status			
Normal	19	50.00	
LVH	19	50.00	
BMI			28.5 (18–52)
Waist circumference			99.53 ± 14.53
HDL			34.50 (11–120)
Triglyceride			168.50 (72–677)
Obesity status			
BMI < 25	10	26.32	
BMI ≥ 25	28	73.68	
Waist circumference status			
< 102 cm (male) or < 88 cm (female)	18	47.34	
≥ 102 cm (male) or ≥ 88 cm (female)	20	52.63	
Diabetes status			
No	16	42.11	
Yes	22	57.89	
Hypertension status			
No	6	15.79	
Yes	32	84.21	

TABLE 1
Continued....

Patient characteristics	N	Frequency (%)	Mean ± SD / Median (min-max)
HDL status			
≥ 40 mg/dL (male) or ≥ 50 mg/dL (female)	9	23.68	
< 40 mg/dL (male) or < 50 mg/dL (female)	29	76.32	
Triglyceride status			
< 150 mg/dl	13	34.21	
≥ 150 mg/dl	25	65.79	

Normally distributed data is shown as Mean ± SD, while Median (min–max) is used for data with abnormal distribution.

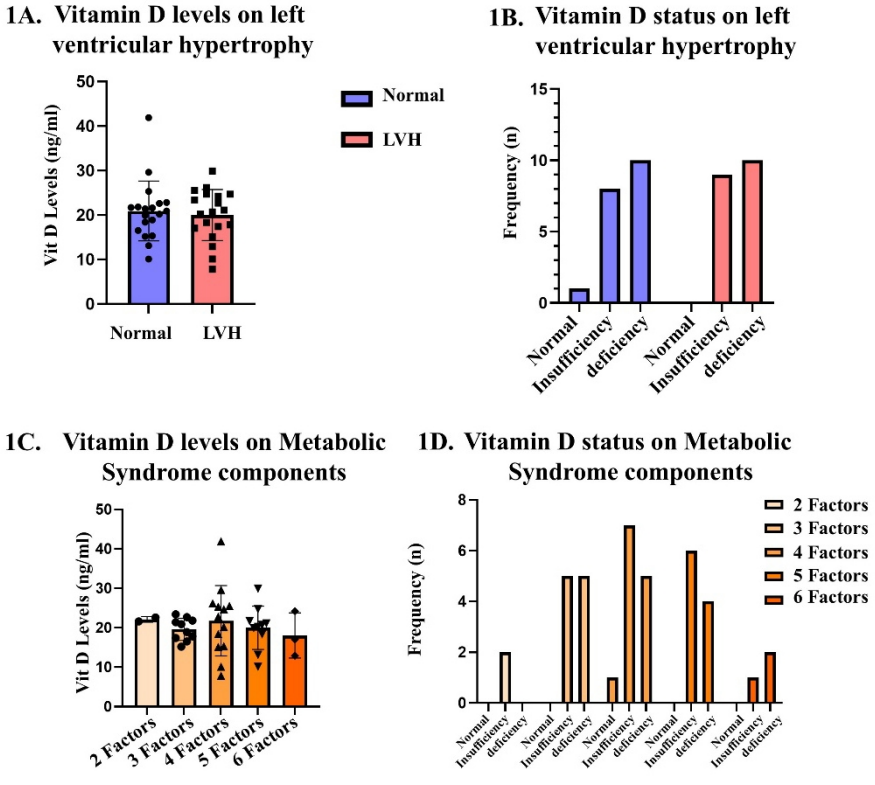

Figure 1. Influence of Vitamin D Levels and Status on Left Ventricular Hypertrophy. **(1A)** Comparison between serum vitamin D levels between individuals with normal left ventricular structure and those with left ventricular hypertrophy (LVH). The data are presented as the mean values with error bars indicating the standard error of the mean. The analysis shows no significant difference (ns) in vitamin D levels between the two groups (independent t-test, p= 0.65). **(1B)** Chi square frequency data of different vitamin D status categories (Normal, Insufficiency, Deficiency) in individuals with normal heart structure (blue bars) and those with LVH (red bars) shows non-significant result (chi square, p=0.58). **(1C)** Comparison between serum vitamin D levels between individuals with the number of metabolic syndromes. The analysis shows no significant difference in vitamin D levels between the two groups (one way ANOVA, p= 0.85) **(1D)** Chi square frequency data of different vitamin D status categories (Normal, Insufficiency, Deficiency) in individuals with metabolic syndrome shows non-significant result (chi square, p=0.81).

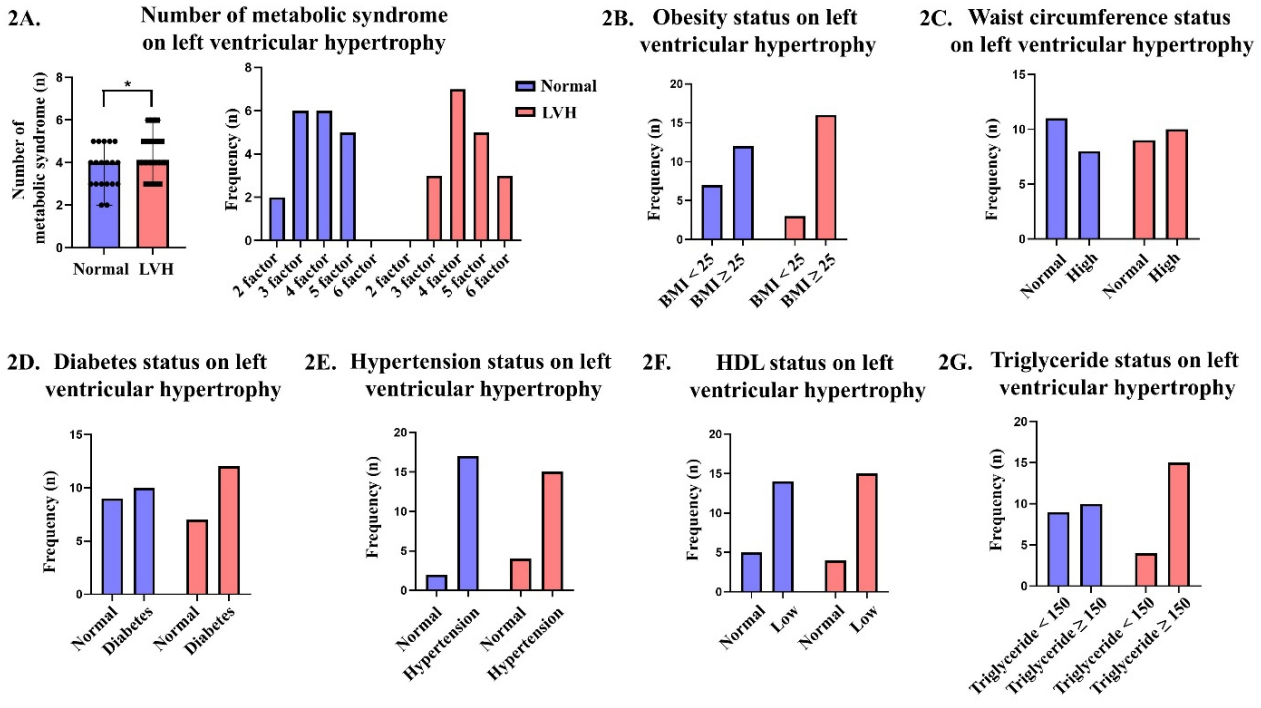

Figure 2. Influence of Metabolic Syndrome Components on Left Ventricular Hypertrophy. **(2A)** Left panel compares the number of metabolic syndrome components between individuals with normal heart structure and those with LVH. The data are shown as the median number of factors with error bars representing max – min value. (Mann Whitney, $p = 0.04$ for 1-tailed, $p = 0.09$ for 2-tailed). The right panel further breaks down the distribution, showing how many people have 2, 3, 4, 5, or 6 factors of metabolic syndrome in both groups. Frequency of individuals with **(2B)** normal BMI and obesity group with BMI ≥25 (chi square, $p = 0.14$), **(2C)** normal and high waist circumferences (> 102 cm for males or > 88 cm for females) (chi square, $p = 0.52$), **(2D)** diabetes status (chi square, $p = 0.51$), **(2E)** hypertension status (chi square, $p = 0.37$), **(2F)** Normal versus low HDL status (< 40 mg/dL for males and < 50 mg/dL for females) (chi square, $p = 0.70$), and **(2G)** Triglyceride status (chi square, $p = 0.09$) in both normal and LVH group.

data showed an increased proportion of patients with a BMI ≥ 25 kg/m² in the LVH group, whereas individuals with a BMI < 25 kg/m² were more prevalent in the normal ventricular function group (Fig. 2B). A similar trend was observed for waist circumference. Patients with a normal waist circumference were more commonly found in the normal group, while those with a high waist circumference (> 102 cm for males or > 88 cm for females) were more frequent in the LVH group (Fig. 2C).

Additionally, diabetes was observed at a higher frequency in the LVH group compared to the normal group (Fig. 2D). However, we didn't find any differences in hypertension status between normal and LVH groups (Fig. 2E). Similarly, HDL levels (Low if < 40 mg/dL for males and < 50 mg/dL for females) did not differ between normal or LVH group (Fig. 2F) Conversely, patients with triglyceride levels ≥ 150 mg/dL were more prevalent in the LVH group (Fig. 2G). Despite these trends, none of the individual metabolic syndrome factors were statistically significantly associated with LVH.

DISCUSSION

Vitamin D deficiency is a prevalent health concern, particularly among individuals with metabolic syndrome. In our study, we hypothesized that lower vitamin D levels in patients with metabolic syndrome might be associated with an increased risk of LVH. However, our findings indicate that low vitamin D levels do not significantly contribute to the development of LVH in these patients. Additionally, vitamin D levels were not associated with the number of positive metabolic syndrome components. Notably, our data revealed that an increased number of metabolic syndrome components is a significant risk factor for LVH, emphasizing the critical role of metabolic syndrome severity in cardiac remodeling.

Although our initial hypothesis suggested a potential link between low vitamin D levels and the incidence of LVH, our results did not show a statistically significant association between the two. The mean serum vitamin D level in patients with LVH was not significantly different from those without LVH.

Moreover, our data indicates that low vitamin D doesn't have any effect on causing metabolic syndrome. Indicating that there is no association of vitamin D levels with LVH or metabolic syndrome. This aligns with several previous studies where vitamin D deficiency alone did not directly correlate with structural cardiac despite its known role in cardiovascular health.[15,16] Our study reveals that nearly all patients with metabolic syndrome exhibited vitamin D levels below the sufficient threshold. This finding is consistent with prior studies that associate metabolic syndrome with reduced vitamin D levels due to factors such as obesity, insulin resistance, and inflammation, all of which are prevalent in this population.[4,17,18]

Vitamin D has been postulated to influence LVH through several mechanisms, including the regulation of calcium homeostasis, modulation of the reninangiotensinaldosterone system (RAAS), and direct effects on cardiomyocytes. Experimental studies have demonstrated that vitamin D metabolites can act on cardiomyocytes, endothelial cells, and smooth vascular muscle cells, suggesting a role in cardiovascular health.[19] Specifically, vitamin D deficiency has been associated with increased activity of the RAAS, leading to hypertension and subsequent cardiac hypertrophy.[20] Moreover, the presence of vitamin D receptors in left ventricular cardiomyocytes indicates that vitamin D may directly affect cardiac structure and function.[21] Despite these theoretical mechanisms, our study did not find a significant association between vitamin D levels and LVH, suggesting that vitamin D's role in cardiac remodeling may be more complex or indirect than previously thought.

The relationship between metabolic syndrome and LVH is well-supported by the literature,[8,9,22–24] and our findings reinforce the understanding that an increased number of metabolic syndrome components elevates the risk of LVH. Obesity, as a key component, plays a significant role in this process through both mechanical and metabolic mechanisms.[8,10,22] Additionally, adipose tissue in obesity is known to secrete pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), contributing to systemic inflammation and oxidative stress.[25] Although our data doesn't show direct evaluation of lipid and inflammatory markers, our study indicates that obesity increases the trend of LVH.

Another metabolic syndrome component, an increase of waist circumference, is closely associated with central obesity, insulin resistance and dyslipidemia, which contribute to LVH.[22,26–29] The presence of high triglyceride levels promotes oxidative stress, which can further damage the vascular endothelium,[30,31] thereby increasing vascular resistance and elevating cardiac afterload. As a result, the heart undergoes hypertrophic changes to cope with the increased workload. Although our study did not show statistically significant differences in triglyceride status between LVH and non-LVH patients, the observed trend aligns with the established understanding that hypertriglyceridemia is a contributor to adverse cardiovascular outcomes including LVH.[30–33] Although our data didn't show the association of individual factors of metabolic syndrome to LVH, we could show that combination of these factors is associated with LVH.

A key limitation of our study is its cross-sectional design, which prevents us from establishing causal relationships between vitamin D deficiency, metabolic syndrome components, and LVH. Additionally, we did not include detailed inflammatory markers, lipid profiles, or oxidative stress parameters, which could provide deeper insights. The single-center population limits the generalizability of our findings, and the lack of longitudinal data prevents us from assessing how changes in vitamin D levels or metabolic syndrome severity influence LVH over time. Future studies with diverse cohorts and longitudinal designs are needed to address these gaps.

Given the scope of metabolic syndrome and its profound impact on cardiovascular health, future research should aim to explore the role of vitamin D in larger sample, multi-center study. Investigating vitamin D supplementation as an intervention in metabolic syndrome populations, particularly in those with established cardiovascular disease, could yield insights into its therapeutic potential for preventing or mitigating LVH progression. Additionally, controlling potential confounders and exploring other biochemical markers associated with metabolic syndrome might offer a more comprehensive understanding of its relationship with cardiovascular outcomes.

CONCLUSION

In conclusion, while our study did not find a direct association between vitamin D levels and LVH, we demonstrated that the cumulative burden of metabolic syndrome components plays a significant role in the development of LVH. This highlights the importance of addressing metabolic syndrome holistically in managing cardiovascular risk.

REFERENCES

1. Cashman KD, Dowling KG, Škrabáková Z, Gonzalez-Gross M, Valtueña J, De Henauw S, *et al*. Global differences in vitamin D status and dietary intake: A review of the data. Endocr Connect. 2022;11(1):10–29. https://doi.org/10.1530/EC-21-0282
2. Cashman KD, Sheehy T, O'Neill CM. Is vitamin D deficiency a public health concern for low middle-income countries? A systematic literature review. Eur J Nutr. 2018. https://doi.org/10.1007/s00394-018-1607-3

3. Pusparini. Defisiensi vitamin D terhadap penyakit. Indones J Clin Pathol Med Lab. 2014;21(1):90–5. ISSN: 0854-4263
4. Sun HL, Long SR, Fu SX, Chen GY, Wang YJ, Liang R, *et al*. Association between vitamin D levels and the risk of metabolic syndrome in a rural Chinese population. Biomed Environ Sci. 2021;34(4):3303. https://doi.org/10.3967/bes2021.043
5. Argano C, Mirarchi L, Amodeo S, Orlando V, Torres A, Corrao S. The role of vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: State of the art. Int J Mol Sci. 2023;24(20):15485. https://doi.org/10.3390/ijms242015485
6. Talaei A, Mohamadi M, Adgi Z. The effect of vitamin D on insulin resistance in patients with type 2 diabetes. Diabetol Metab Syndr. 2013;5(1):8. https://doi.org/10.1186/1758-5996-5-8.
7. Khademi Z, Hamedi-Shahraki S, Amirkhizi F. Vitamin D insufficiency is associated with inflammation and deregulation of adipokines in patients with metabolic syndrome. BMC Endocr Disord. 2022;22:223. https://doi.org/10.1186/s12902-022-01141-0.
8. Ratto E, Viazzi F, Verzola D, *et al*. Metabolic syndrome is associated with left ventricular dilatation in primary hypertension. J Hum Hypertens. 2016;30(2):158–63. https://doi.org/10.1038/jhh.2015.67.
9. Guerra F, Mancinelli L, Angelini L, Fortunati M, Rappelli A, *et al*. The association of left ventricular hypertrophy with metabolic syndrome is dependent on body mass index in hypertensive overweight or obese patients. PLOS ONE. 2011;6(1):e16630. https://doi.org/10.1371/journal.pone.0016630.
10. Min CY, Gao Y, Jiang YN, *et al*. The additive effect of metabolic syndrome on left ventricular impairment in patients with obstructive coronary artery disease assessed by 3.0 T cardiac magnetic resonance feature tracking. Cardiovasc Diabetol. 2024;23:133. https://doi.org/10.1186/s12933-024-02225-y.
11. Fallo F, Catena C, Camozzi V, Luisetto G, Cosma C, Plebani M, *et al*. Low serum 25-hydroxyvitamin D levels are associated with left ventricular hypertrophy in essential hypertension. Nutr Metab Cardiovasc Dis. 2012;22(10):871–6. https://doi.org/10.1016/j.numecd.2011.06.001.
12. Noor MN, Jamali AA, Rahu HN, Jamali GM, Memon AH, Phull GM. Correlation of vitamin D3 and serum calcium level with left ventricular hypertrophy patients. Prof Med J. 2020;27(6):1143–50. https://doi.org/10.29309/TPMJ/2020.27.06.3697.
13. Alberti KG, Zimmet P, Shaw J. Metabolic syndrome – a new worldwide definition. A consensus statement from the International Diabetes Federation. Diabet Med. 2006;23(5):469–80. https://doi.org/10.1111/j.1464-5491.2006.01858.x.
14. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, *et al*. Recommendations for chamber quantification: A report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18(12):1440–63. https://doi.org/10.1016/j.echo.2005.10.005.
15. Thadhani R, Appelbaum E, Pritchett Y, *et al*. Vitamin D therapy and cardiac structure and function in patients with chronic kidney disease: The PRIMO randomized controlled trial. JAMA. 2012;307(7):674–84. https://doi.org/10.1001/jama.2012.120.
16. Herrero JA, López-Gómez JM, Maduell F, Martín-de Francisco ÁL, Martín-Malo A, Martínez-Castelao A, *et al*. Activation of vitamin D receptors in the optimization of secondary hyperparathyroidism on dialysis. Nefrologia. 2013;33(4):443–622. https://doi.org/10.3265/Nefrologia.pre2013.May.11901.
17. Raposo L, Martins S, Ferreira D, *et al*. Vitamin D, parathyroid hormone and metabolic syndrome – the PORMETS study. BMC Endocr Disord. 2017;17:71. https://doi.org/10.1186/s12902-017-0221-3.
18. Amirkhizi F, Khademi Z, Hamedi-Shahraki S, Rahimlou M. Vitamin D insufficiency and its association with adipokines and atherogenic indices in patients with metabolic syndrome: A case-control study. Front Endocrinol (Lausanne). 2023;14:1080138. https://doi.org/10.3389/fendo.2023.1080138.
19. Al Mheid I, Patel RS, Tangpricha V, Quyyumi AA. Vitamin D and cardiovascular disease: Is the evidence solid? Eur Heart J. 2013;34(48):3691–8. https://doi.org/10.1093/eurheartj/eht166.
20. Nigwekar SU, Bhan I, Thadhani R. Vitamin D, hypertension, left ventricular hypertrophy, and diastolic dysfunction. Curr Cardiovasc Risk Rep. 2011;5:314–22. https://doi.org/10.1007/s12170-011-0175-3.
21. Condoleo V, Pelaia C, Armentaro G, Severini G, Clausi E, Cassano V, *et al*. Role of vitamin D in cardiovascular diseases. Endocrines. 2021;2(4):417–26. https://doi.org/10.3390/endocrines2040037.
22. de Simone G, Devereux RB, Chinali M, Roman MJ, Lee ET, Resnick HE, *et al*. Metabolic syndrome and left ventricular hypertrophy in the prediction of cardiovascular events: The Strong Heart Study. Nutr Metab Cardiovasc Dis. 2009;19(2):98–104. https://doi.org/10.1016/j.numecd.2008.04.001.
23. Drożdż M, Moczulska A, Rudziński A, Drożdż D. Metabolic syndrome as risk factor for left ventricular hypertrophy in children with chronic kidney disease. Front Endocrinol (Lausanne). 2023;14:1215527. https://doi.org/10.3389/fendo.2023.1215527.
24. Papademetriou V, Devereux RB, Narayan P, Wachtell K, Bella JN, Gerdts E, *et al*. Similar effects of isolated systolic and combined hypertension on left ventricular geometry and function: the LIFE Study. Am J Hypertens. 2001;14(8 Pt 1):768–74. https://doi.org/10.1016/s0895-7061(01)01292-4.
25. Liu M, Li X, Sun R, Zeng Y, Chen S, Zhang P. Vitamin D nutritional status and the risk for cardiovascular disease: A review. Exp Ther Med. 2016;11(4):1189–93. https://doi.org/10.3892/etm.2016.3047.
26. Marttila J, Sipola P, Juutilainen A, *et al*. Central obesity is associated with increased left ventricular maximal wall thickness and intrathoracic adipose tissue measured with cardiac magnetic resonance. High Blood Press Cardiovasc Prev. 2024;31:389–99. https://doi.org/10.1007/s40292-024-00659-9.
27. Rodrigues S, Baldo M, Sá Cunha R, *et al*. Anthropometric measures of increased central and overall adiposity in association with echocardiographic left ventricular hypertrophy. Hypertens Res. 2010;33:83–87. https://doi.org/10.1038/hr.2009.188.
28. Zhang X, Li G, Zhang D, Sun Y. Influence of hypertension and global or abdominal obesity on left ventricular hypertrophy: A cross-sectional study. J Clin Hypertens. 2023;1–9. https://doi.org/10.1111/jch.14716.
29. Phanzu BK, Natuhoyila AN, Vita EK, Kabangu MJR, Longo-Mbenza B. Association between insulin resistance and left ventricular hypertrophy in asymptomatic, Black, sub-Saharan African, hypertensive patients: A case-control study. BMC Cardiovasc Disord. 2021;21(1):1.

https://doi.org/10.1186/s12872-020-01829-y.
30. Yang M, Shangguan Q, Xie G, Sheng G, Yang J. Oxidative stress mediates the association between triglyceride-glucose index and risk of cardiovascular and all-cause mortality in metabolic syndrome: Evidence from a prospective cohort study. Front Endocrinol (Lausanne). 2024;15:1452896. https://doi.org/10.3389/fendo.2024.1452896.
31. Al-Aubaidy HA, Jelinek HF. Oxidative stress and triglycerides as predictors of subclinical atherosclerosis in prediabetes. Redox Rep. 2014;19(2):87–91. https://doi.org/10.1179/1351000213Y.0000000080.
32. Picard F, Bhatt DL, Ducrocq G, Ohman EM, Goto S, Eagle KA, *et al*. Generalizability of the REDUCE-IT trial and cardiovascular outcomes associated with hypertriglyceridemia among patients potentially eligible for icosapent ethyl therapy: An analysis of the REduction of Atherothrombosis for Continued Health (REACH) registry. Int J Cardiol. 2021;340:96–104. https://doi.org/10.1016/j.ijcard.2021.08.031.
33. Zhou H, Ding X, Yang Q, Chen S, Li Y, Zhou X, *et al*. Associations of hypertriglyceridemia onset age with cardiovascular disease and all-cause mortality in adults: A cohort study. J Am Heart Assoc. 2022;11(20):e026632. https://doi.org/10.1161/JAHA.122.026632.