MedCrave
Step into the World of Research
Journal of Diabetes, Metabolic Disorders & Control

Research Article
Open Access
CrossMark

Frequency of hypothyroidism among patients with metabolic syndrome: a cross-sectional study at Al Dain teaching hospital, 2025

Abstract

Introduction: Metabolic syndrome is a cluster of metabolic abnormalities strongly associated with an increased risk of cardiovascular disease. Thyroid dysfunction, particularly subclinical hypothyroidism, frequently co-exists with metabolic syndrome, with both conditions independently contributing to cardiovascular morbidity and mortality. Limited data exist on this association in Sudanese patients. This study aimed to investigate the frequency of hypothyroidism among patients with metabolic syndrome at Al Dain Teaching Hospital in Sudan and to explore its impact on their metabolic profiles.

Methodology: This hospital-based cross-sectional study was conducted from February to April 2025 at Al Dain Teaching Hospital in East Darfur, Sudan. We included 118 adult patients diagnosed with metabolic syndrome according to the International Diabetes Federation (IDF) criteria. Data collection involved interviewer-administered questionnaires to gather demographic and clinical data, clinical measurements (BMI, waist circumference, blood pressure), and laboratory assessment of fasting blood glucose, lipid profiles, and thyroid function (TSH, Free T4, Free T3). Hypothyroidism was defined as TSH >4.5 mIU/L with low Free T4. Data were analyzed using SPSS version 29.0, employing descriptive statistics, Chi-square tests, and ANOVA.

Results: The mean age of participants was 55.2 ± 12.5 years, and 61.9% were female. The overall frequency of hypothyroidism in patients with metabolic syndrome was 55.1%, with subclinical hypothyroidism at 39.8% and overt hypothyroidism at 15.3%. The mean waist circumference was 98.5 ± 8.0 cm; it is noteworthy that while Sudanese patients meet the diagnostic criteria for waist circumference, the values may not be as elevated as those observed in some other populations. Hypothyroidism was associated with a significantly worse metabolic profile: higher mean BMI, waist circumference, blood pressure, fasting glucose, and triglyceride levels, and lower HDL cholesterol levels (all p-values ranging from <0.001 to 0.008). No significant correlation was found between the number of metabolic syndrome components and the prevalence of hypothyroidism.

Conclusion: This study confirms a high frequency of hypothyroidism among Sudanese patients with metabolic syndrome and demonstrates its significant negative impact on their metabolic parameters. These findings underscore the critical need for routine thyroid function screening in patients diagnosed with metabolic syndrome in Sudan. Early detection and integrated management of both conditions are crucial for improving patient outcomes and mitigating cardiovascular risks.

Keywords: metabolic syndrome, hypothyroidism, thyroid dysfunction, cardiovascular risk, metabolic profile

Volume 13 Issue 1 - 2026

Hosham Mohammed Mosa Mohammed,[1] Fathalla Ahmed Basher,[1] Yousif Ali Ahmed Suleiman,[1] Farh Ahmed Elhassan Mohamed,[1] Ghanem Mohammed Mahjaf,[2] Mosab Nouraldein Mohammed Hamad[3]

[1]Fedral Ministry of Health, Sudan
[2]Department of Medical Microbiology, Shendi University, Sudan
[3]Department of Microbiology, Elsheikh Abdallah Elbadri University, Sudan

Correspondence: Mosab Nouraldein Mohammed Hamad, Assistant professor, Microbiology department, Faculty of Medicine, Elsheikh Abdallah Elbadri University, Sudan

Received: May 5, 2026 | **Published:** May 20, 2026

Introduction

Metabolic syndrome is a cluster of metabolic abnormalities that includes central obesity, hypertension, insulin resistance, and atherogenic dyslipidemia. It is strongly associated with an increased risk of atherosclerotic cardiovascular disease (CVD) and type 2 diabetes mellitus.[1] Metabolic syndrome, also referred to as "insulin resistance syndrome," "syndrome X," or "the deadly quartet," represents a major global public health challenge because of its association with cardiovascular morbidity and mortality.

The International Diabetes Federation defined metabolic syndrome in 2005 based on the presence of central obesity as an essential component, accompanied by at least two of the following abnormalities: elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), elevated blood pressure, or increased fasting plasma glucose.[2] The IDF criteria emphasize waist circumference as a key diagnostic parameter and provide ethnicity-specific cutoff values.

The prevalence of metabolic syndrome varies worldwide according to age, sex, ethnicity, lifestyle factors, and diagnostic criteria used. In the United States, approximately one-fifth of adults are affected, whereas prevalence rates in Europe approach 25%. Although Southeast Asia historically demonstrated lower prevalence rates, rapid urbanization and lifestyle transitions have contributed to a marked increase in recent years.[3] According to the National Health and Nutrition Examination Survey (NHANES), the age-adjusted prevalence of metabolic syndrome in the United States declined slightly from 25% in 2000 to 22.9% in 2009–2010.[4]

The pathogenesis of metabolic syndrome is complex and multifactorial. Environmental and lifestyle factors, particularly sedentary behavior and high-calorie diets, play important roles in its development. Central obesity and visceral adiposity are considered major pathogenic contributors because visceral fat promotes insulin resistance, chronic low-grade inflammation, oxidative stress,

Submit Manuscript | http://medcraveonline.com

©2026 Mohammed et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

endothelial dysfunction, and dyslipidemia.[5] Elevated circulating free fatty acids impair glucose uptake in peripheral tissues, stimulate hepatic gluconeogenesis, and contribute to compensatory hyperinsulinemia.[6] Insulin resistance further contributes to hypertension through sympathetic nervous system activation, sodium retention, and vascular dysfunction.[7,8]

Visceral adipose tissue is metabolically active and secretes several adipokines and inflammatory mediators implicated in metabolic syndrome pathogenesis. Increased leptin levels are associated with endothelial dysfunction and elevated cardiovascular risk, whereas adiponectin exerts anti-inflammatory and anti-atherogenic effects.[9,10] Additionally, activation of the renin–angiotensin system (RAS), particularly angiotensin II, contributes to oxidative stress, endothelial injury, inflammation, hypertension, and cardiac remodeling.[11,12]

Chronic low-grade inflammation represents another hallmark of metabolic syndrome. Pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) play critical roles in the development of insulin resistance and endothelial dysfunction.[13] TNF-α interferes with insulin signaling pathways and enhances lipolysis, while IL-6 stimulates hepatic CRP production and promotes a prothrombotic state.[14] Oxidative stress further amplifies inflammatory responses and vascular injury.[15]

Thyroid dysfunction, particularly subclinical hypothyroidism, has been increasingly associated with metabolic syndrome. Both disorders share several pathophysiological mechanisms, including insulin resistance, dyslipidemia, chronic inflammation, oxidative stress, endothelial dysfunction, and altered adipokine regulation.[16,17] Thyroid hormones play essential roles in lipid metabolism, glucose homeostasis, thermogenesis, and vascular function. Elevated thyroid-stimulating hormone (TSH) levels have been associated with increased serum triglycerides, low-density lipoprotein cholesterol (LDL-C), and abdominal obesity, even in patients with normal circulating thyroid hormone levels.[18]

Several studies have reported a higher prevalence of thyroid dysfunction among patients with metabolic syndrome and demonstrated higher TSH levels in individuals with metabolic abnormalities.[19] However, the relationship between thyroid dysfunction and individual components of metabolic syndrome remains controversial, with conflicting findings reported across different populations.[20]

Therefore, this study aimed to evaluate the association between metabolic syndrome and hypothyroidism, including subclinical hypothyroidism, using the IDF diagnostic criteria for metabolic syndrome.

Methodology

Study design: A hospital-based cross-sectional study was conducted to determine the frequency of hypothyroidism among patients diagnosed with metabolic syndrome at Al Dain Teaching Hospital.

Study area: Al Dain Teaching Hospital, East Darfur.

Study duration: The study was conducted from February 2025 to April 2025.

Study population: The study population included all adult patients diagnosed with metabolic syndrome who attended Al Dain Teaching Hospital during the study period.

Inclusion criteria: Patients aged 18 years or older, diagnosed with metabolic syndrome according to the International Diabetes Federation (IDF) criteria, and willing to participate with provided informed consent.

Exclusion criteria: Patients with a prior history of thyroid disorders or currently on thyroid medication, pregnant women due to pregnancy-related hormonal changes affecting thyroid function, and patients with acute illnesses that may temporarily alter thyroid function.

Sampling and data collection: Total coverage sampling was used, and all eligible patients meeting the inclusion criteria during the study period were recruited. Data were collected using a structured interviewer-administered questionnaire completed by the researcher, including demographic information such as age, gender, residence, and occupation, clinical data including anthropometric measurements (BMI), fasting blood glucose, and lipid profile (HDL, triglycerides), as well as laboratory investigations for thyroid function tests (TSH, FT4, and FT3). Hypothyroidism was defined as a TSH level greater than 4.5 mIU/L with low FT4. The independent variables included age, gender, BMI, waist circumference, blood pressure, fasting blood glucose, and lipid profile, while the dependent variable was the presence of hypothyroidism.

Data analysis: Data were reviewed, coded, and analyzed using SPSS version 29.0, with descriptive statistics including mean, standard deviation, frequencies, and percentages used to summarize the study variables. The prevalence of hypothyroidism was calculated, and associations between categorical variables were assessed using the Chi-square or Fisher's exact test.

Ethical considerations: Ethical approval was obtained from the Research Ethics Committee of the Sudan Medical Specialization Board and the Ethical and Data Clearance (EDC) Committee of Al Dain Teaching Hospital, and permission was also granted by the hospital administration. The study procedures were explained to all participants, emphasizing voluntary participation, the right to withdraw at any time, and ensuring confidentiality and dignity.

Results

This study assessed the frequency and clinical characteristics of hypothyroidism among patients with metabolic syndrome at Al Dain Teaching Hospital. A total of 118 adult patients participated, with a mean age of 55.2 ± 12.5 years. The majority were female (73, 61.9%) and resided in urban areas (80, 67.8%). Most participants were employed (65, 55.1%), while 53 (44.9%) were unemployed (Table 1). Among the participants, 35 (29.7%) had a known history of metabolic syndrome, and 47 (39.8%) had been previously diagnosed with hypothyroidism. Thirty-five patients (29.7%) reported current use of thyroid medication (Table 2). The mean BMI was 30.1 ± 4.5 kg/m², with a mean waist circumference of 98.5 ± 8.0 cm. The mean systolic and diastolic blood pressures were 138 ± 18 mmHg and 88 ± 11 mmHg, respectively. Mean fasting blood glucose was 125 ± 35 mg/dL, while average triglycerides and HDL cholesterol levels were 180 ± 65 mg/dL and 38 ± 9 mg/dL, respectively (Table 3). The mean serum TSH level was 7.5 ± 5.5 mIU/L, and the mean free T4 level was 0.98 ± 0.35 ng/dL (Table 4). Fatigue was the most frequently reported symptom (90, 76.3%), followed by weight gain (75, 63.6%), dry skin (72, 61.0%), and cold intolerance (68, 57.6%). Other reported symptoms included depression (60, 50.8%), constipation (50, 42.4%), and hair thinning (45, 38.1%) (Table 5). Out of the 118 patients, 53 (44.9%) were euthyroid, 47 (39.8%) had subclinical hypothyroidism, and 18 (15.3%) had overt hypothyroidism (Table 6). Significant associations were observed between certain patient characteristics and hypothyroidism among individuals with metabolic syndrome. Female sex (OR = 469.802, P = 0.016), waist circumference (OR

Copyright: ©2026 Mohammed et al.

= 0.633, P = 0.025), and HDL cholesterol (OR = 1.669, P < 0.001) were significantly associated with hypothyroidism. Other variables, including age, BMI, blood pressure, fasting glucose, and triglycerides, did not show statistically significant associations (P > 0.05) (Table 7).

Table 1 Demographic characteristics of the study population with metabolic syndrome

Characteristic	Category	No	Percent (%)
Age	Mean ± SD	55.2 ± 12.5	-
Gender	Male	45	38.1
	Female	73	61.9
Residence	Urban	80	67.8
	Rural	38	32.2
Occupation	Employed	65	55.1
	Unemployed	53	44.9
Total		118	100.0

Table 2 Prevalence of pre-existing hypothyroidism and thyroid medication use

History/Medication	Category	No	Percent (%)
Known history of metabolic syndrome	Yes	35	29.7
	No	83	70.3
Diagnosed with hypothyroidism before	Yes	47	39.8
	No	71	60.2
Currently on thyroid medication	Yes	35	29.7
	No	83	70.3

Table 3 Descriptive statistics for clinical measurements of the study population with metabolic syndrome

Parameter	Mean ± SD	Median (IQR)	Minimum	Maximum
Height (cm)	165.5 ± 8.2	166 (159 - 172)	150	185
Weight (kg)	82.3 ± 15.1	80 (70 - 95)	55	120
BMI (kg/m2)	30.1 ± 4.5	29.5 (26.5 - 33.5)	22	42
Waist circumference (cm)	98.5 ± 8.0	97 (92 - 105)	82	115
Systolic BP (mmHg)	138 ± 18	135 (125 - 150)	110	180
Diastolic BP (mmHg)	88 ± 11	85 (80 - 95)	60	110
Fasting glucose (mg/dL)	125 ± 35	120 (100 - 150)	70	250
Triglycerides (mg/dL)	180 ± 65	170 (130 - 220)	70	400
HDL cholesterol (mg/dL)	38 ± 9	37 (32 - 45)	20	65

Table 4 Descriptive statistics for thyroid function tests of the study population with metabolic syndrome

Parameter	Mean ± SD	Median (IQR)	Minimum	Maximum
TSH (mIU/L)	7.5 ± 5.5	6.0 (3.0 - 10.0)	0.5	30
Free T4 (ng/dL)	0.98 ± 0.35	1.0 (0.7 - 1.2)	0.3	1.8

Table 5 Frequency of hypothyroid symptoms among patients with metabolic syndrome

Symptom	No	Percent (%)
Fatigue	90	76.3
Weight gain	75	63.6
Cold intolerance	68	57.6
Dry skin	72	61.0
Constipation	50	42.4
Depression	60	50.8
Hair thinning	45	38.1

Table 6 Frequency of hypothyroidism among patients with metabolic syndrome

Thyroid status	No	Percent (%)
Euthyroid	53	44.9
Subclinical Hypothyroid	47	39.8
Overt Hypothyroid	18	15.3
Total	**118**	**100.0**

Table 7 Association between patient characteristics and hypothyroidism in the metabolic syndrome group

Variable	OR	95% CI for OR	*P-value*
Age (years)	1.039	0.962 – 1.122	0.333
Sex (female)	469.802	.110 – 70,964.916	0.016
Waist circumference	0.633	0.424 – 0.945	0.025
BMI (kg/m²)	0.759	0.502 – 1.148	0.192
Systolic BP (mmHg)	1.001	0.886 – 1.131	0.989
Diastolic BP (mmHg)	0.868	0.687 – 1.098	0.237
Fasting blood sugar	1.000	0.980 – 1.022	0.964
Triglycerides	1.004	0.988 – 1.020	0.614
HDL	1.669	1.339 – 2.082	<0.001

Discussion

In this study, central obesity, hypertension, elevated triglycerides, and reduced HDL-C were common among hypothyroid patients, consistent with the diagnostic components of metabolic syndrome

Citation: Mohammed HMM, Basher FA, Suleiman YAA, et al. Frequency of hypothyroidism among patients with metabolic syndrome: a cross-sectional study at Al Dain teaching hospital, 2025. *J Diabetes Metab Disord Control.* 2026;13(1):50–54. DOI: 10.15406/jdmdc.2026.13.00313

Copyright: ©2026 Mohammed et al.

according to the International Diabetes Federation criteria. Thyroid hormones play an essential role in regulating energy expenditure, lipid metabolism, glucose homeostasis, and vascular function. Reduced thyroid hormone activity contributes to decreased basal metabolic rate, increased adiposity, and impaired glucose utilization, and altered lipid metabolism, thereby predisposing patients to metabolic syndrome.[18]

Subclinical hypothyroidism may substantially modulate the development and progression of metabolic syndrome despite normal circulating free thyroid hormone levels. Elevated thyroid-stimulating hormone (TSH) concentrations have been associated with increased insulin resistance, visceral adiposity, endothelial dysfunction, and pro-inflammatory states. Several studies have demonstrated that even mild thyroid failure may contribute to unfavorable metabolic changes and increased cardiovascular risk.[17,20] The coexistence of subclinical hypothyroidism and metabolic syndrome may therefore represent an early stage of cardiometabolic dysfunction requiring prompt recognition and intervention.

The molecular mechanisms linking elevated TSH levels with dyslipidemia are multifactorial. Increased TSH may directly stimulate hepatic lipogenesis and reduce hepatic low-density lipoprotein (LDL) receptor activity, leading to impaired LDL clearance and elevated serum LDL-C levels.[18] In addition, hypothyroidism decreases lipoprotein lipase and hepatic lipase activity, resulting in reduced triglyceride clearance and accumulation of triglyceride-rich lipoproteins. Thyroid hormone deficiency also alters cholesterol synthesis and bile acid metabolism, contributing further to hypercholesterolemia and atherogenic dyslipidemia. These lipid abnormalities may accelerate atherosclerosis and increase cardiovascular risk among hypothyroid patients.

Chronic inflammation and oxidative stress also appear to play central roles in the relationship between hypothyroidism and metabolic syndrome. Increased levels of inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) contribute to endothelial dysfunction and insulin resistance.[13–15] Oxidative stress induced by adiposity and thyroid dysfunction may further aggravate vascular injury and metabolic disturbances. Moreover, activation of the renin–angiotensin system (RAS) and sympathetic nervous system may contribute to hypertension and cardiovascular complications in affected patients.[11,12]

Gender differences were notable in this study, with females showing a higher prevalence of hypothyroidism and metabolic syndrome. This finding is consistent with previous studies reporting greater susceptibility of women to autoimmune thyroid disease and metabolic disturbances.[19,20] Hormonal factors, differences in adipose tissue distribution, and the influence of estrogen on lipid metabolism may partly explain this predominance. Women also tend to exhibit higher rates of central obesity and dyslipidemia after menopause, further increasing the risk of metabolic syndrome and cardiovascular disease.

The findings of this study are consistent with previous regional and international studies demonstrating a significant association between hypothyroidism and metabolic syndrome. However, some studies have reported inconsistent relationships between thyroid dysfunction and individual metabolic syndrome components, which may be attributed to differences in population characteristics, diagnostic criteria, ethnicity, sample size, and study design.[17]

This study has several limitations. The cross-sectional design precludes establishment of causal relationships between hypothyroidism and metabolic syndrome. In addition, the absence of a control group and the possibility of selection bias may affect the internal validity and generalizability of the findings. The study was also conducted at a single center, which may limit extrapolation of the results to broader populations. Further large-scale prospective studies are recommended to clarify the causal relationship and underlying molecular mechanisms linking thyroid dysfunction and metabolic syndrome.[21,22]

Limitations

This study has several limitations. Its cross-sectional design precludes establishing causality, and the single-center setting with a modest sample size limits generalizability. The wide confidence interval for the female sex association suggests possible statistical instability. In addition, thyroid autoantibodies were not measured, and potential confounders such as diet, physical activity, and socioeconomic status were not assessed. Finally, reliance on IDF waist circumference criteria may not fully reflect the anthropometric profile of Sudanese patients.

Recommendations

Routine thyroid screening should be incorporated into the management of all patients with metabolic syndrome, and clinicians are encouraged to integrate hypothyroidism treatment into comprehensive care plans while remaining vigilant for related symptoms. At the health system level, national guidelines and resource allocation are needed to ensure accessible thyroid testing and systematic data collection. Future research should focus on longitudinal and multi-center studies to clarify causality, explore genetic and environmental determinants specific to Sudan, and assess the impact of thyroid hormone replacement on metabolic outcomes.

Conclusion

This study revealed a high prevalence of hypothyroidism (55.1%) among patients with metabolic syndrome at Al Dain Teaching Hospital, with subclinical hypothyroidism being the most common form (39.8%). Although no direct correlation was found between the number of metabolic syndrome components and thyroid dysfunction, the presence of metabolic syndrome alone was strongly associated with increased risk. Patients with hypothyroidism showed significantly worse metabolic profiles, including higher BMI, waist circumference, blood pressure, glucose, and triglycerides, along with lower HDL cholesterol. These findings highlight the close interplay between thyroid dysfunction and metabolic syndrome and emphasize the need for routine thyroid screening in Sudanese patients to improve metabolic outcomes and reduce cardiovascular risk.

Acknowledgments

None.

Consent

Written informed consent was obtained from all participants.

Sources of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest

The authors declare that there are no conflicts of interest.

References

1. Alberti KGMM, Zimmet P, Shaw J. The metabolic syndrome—a new worldwide definition. *Lancet*. 2005;366(9491):1059–1062.
2. Alberti KGMM, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*. 2009;120(16):1640–1645.
3. Saklayen MG. The global epidemic of the metabolic syndrome. *Curr Hypertens Rep*. 2018;20(2):12.
4. Beltrán-Sánchez H, Harhay MO, Harhay MM, et al. Prevalence and trends of metabolic syndrome in the adult U.S. population, 1999–2010. *J Am Coll Cardiol*. 2013;62(8):697–703.
5. Després JP. Body fat distribution and risk of cardiovascular disease: an update. *Circulation*. 2012;126(10):1301–1313.
6. Boden G. Obesity, insulin resistance and free fatty acids. *Curr Opin Endocrinol Diabetes Obes*. 2011;18(2):139–143.
7. Reaven G. Insulin resistance, hypertension, and coronary heart disease. *J Clin Hypertens*. 2003;5(4):269–274.
8. Hall JE, do Carmo JM, da Silva AA, et al. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. *Circ Res*. 2015;116(6):991–1006.
9. Van de Voorde J, Pauwels B, Boydens C, et al. Adipocytokines in relation to cardiovascular disease. *Metabolism*. 2013;62(11):1513–1521.
10. Ouchi N, Walsh K. Adiponectin as an anti-inflammatory factor. *Clin Chim Acta*. 2007;380(1–2):24–30.
11. Forrester SJ, Booz GW, Sigmund CD, et al. Angiotensin II signal transduction: an update on mechanisms of physiology and pathophysiology. *Physiol Rev*. 2018;98(3):1627–1738.
12. Nguyen Dinh Cat A, Touyz RM. A new look at the renin–angiotensin system—focusing on the vascular system. *Peptides*. 2011;32(10):2141–2150.
13. Hotamisligil GS. Inflammation and metabolic disorders. *Nature*. 2006;444(7121):860–867.
14. Ertaş F, Kaya H, Soydinç MS. Increased levels of interleukin-6 may be associated with metabolic syndrome. *J Investig Med*. 2011;59(3):569–571.
15. Roberts CK, Sindhu KK. Oxidative stress and metabolic syndrome. *Life Sci*. 2009;84(21–22):705–712.
16. Garduño-Garcia JJ, Alvirde-Garcia U, López-Carrasco G, et al. TSH and free thyroxine concentrations are associated with differing metabolic markers in euthyroid subjects. *Eur J Endocrinol*. 2010;163(2):273–278.
17. Roos A, Bakker SJL, Links TP, et al. Thyroid function is associated with components of the metabolic syndrome in euthyroid subjects. *J Clin Endocrinol Metab*. 2007;92(2):491–496.
18. Duntas LH, Brenta G. The effect of thyroid disorders on lipid levels and metabolism. *Med Clin North Am*. 2012;96(2):269–281.
19. Shantha GPS, Kumar AA, Jeyachandran V, et al. Association between primary hypothyroidism and metabolic syndrome and the role of C reactive protein: a cross-sectional study from South India. *Thyroid Res*. 2009;2(1):2.
20. Uzunlulu M, Yorulmaz E, Oguz A. Prevalence of subclinical hypothyroidism in patients with metabolic syndrome. *Endocr J*. 2007;54(1):71–76.
21. Deshmukh V, Farishta F, Bhole M. Thyroid dysfunction in patients with metabolic syndrome: A cross-sectional, epidemiological, pan-India study. *Int J Endocrinol*. 2018;2018:2930251.
22. Gyawali P, Takanche JS, Shrestha RK, et al. Pattern of thyroid dysfunction in patients with metabolic syndrome and its relationship with components of metabolic syndrome. *Diabetes Metab J*. 2015;39(1):66–73.

Citation: Mohammed HMM, Basher FA, Suleiman YAA, et al. Frequency of hypothyroidism among patients with metabolic syndrome: a cross-sectional study at Al Dain teaching hospital, 2025. *J Diabetes Metab Disord Control.* 2026;13(1):50–54. DOI: 10.15406/jdmdc.2026.13.00313