Al-Ameed Journal for Medical Research and Health Sciences

Volume 3 | Issue 1 Article 6

2025

Troponin Level as an Early Warning Marker of Subclinical-Clinical Atherosclerosis Complications in Patients with Diabetes and Dyslipidemia

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Recommended Citation

Khudair, Zeena Hussien; Al-Fatlawi, Abeer Cheaid Yousif; and Al-Haidari, Ahmed Qasim Jaafar (2025) "Troponin Level as an Early Warning Marker of Subclinical-Clinical Atherosclerosis Complications in Patients with Diabetes and Dyslipidemia," *Al-Ameed Journal for Medical Research and Health Sciences*: Vol. 3 : Iss. 1 , Article 6.
Available at: https://doi.org/10.61631/3005-3188.1025

ORIGINAL ARTICLE

Troponin Level as an Early Warning Marker of Subclinical-clinical Atherosclerosis Complications in Patients With Diabetes and Dyslipidemia

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Abstract

Background: **Atherosclerosis is a long-term condition where fatty deposits (plaques) build up inside the arteries, causing them to narrow and harden, this reduces blood flow and can lead to serious health problems like heart attacks and strokes.**

Objectives: **Found on the relationship elevated troponin levels in cardiovascular complications particularly atherosclerosis in patients with both diabetes and dyslipidemia.**

Materials and methods: **Cross section study includes (90) males with range of age (20->60) mean of age (33.00 ± 6.131) were used in the present study divided into (45) patients with clinical coronary atherosclerosis mean of age (34.500 ± 4.949) and (45) patients with sub-clinical coronary atherosclerosis mean of age (32.250 ± 7.228). The serum Troponin, insulin, fasting blood glucose calculated by using an Enzyme-Linked Immunosorbent Assay (ELISA), HbA1C measurement by spectrophotometer.**

Result: **The result found a highly significant increase ($p \leq 0.01$) in Troponin for clinical atherosclerosis patients (2303.471 ± 2297.513), compared to the subclinical atherosclerosis group (54.426 ± 35.728) also significant increase ($p \leq 0.01$) in Random glucose, Insulin, HbA1C, Lipid profile revealed significant increase ($p \leq 0.01$) in (TC,TG, VLDL, LDL.) in clinical atherosclerosis as compared to subclinical atherosclerosis.**

Conclusion: **Elevated troponin levels show strong potential as early biomarkers for subclinical cardiovascular complications, including atherosclerosis, in patients with coexisting diabetes and dyslipidemia.**

Keywords: Troponin, Atherosclerosis, Diabetes mellitus

Introduction

Atherosclerosis is a chronic and progressive disease characterized by the thickening and hardening of arterial walls due to the accumulation of lipids, inflammatory cells, and fibrous elements (Beverly & Budoff, 2020). The development of atherosclerosis is a complex, multi-step process. It starts with dysfunction of the endothelial lining of blood vessels, which facilitates the accumulation of lipids within the arterial wall, this lipid buildup triggers an inflammatory response, attracting immune cells and promoting further damage, over time, these changes lead to the formation of atherosclerotic plaques. As plaques mature, they may become unstable and prone to rupture, increasing the risk of serious cardiovascular events such as heart attacks and strokes (Kim, 2024; Zhao et al., 2024). Diabetes Mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia due to defects in insulin secretion, insulin action, or both, the main types include Type 1, Type 2, and gestational diabetes, with risk factors such as genetic predisposition, obesity, age, and lifestyle choices contributing to its rising incidence (Selby & Taal, 2020; Yameny, 2024). Diabetes accelerates atherosclerosis through multiple mechanisms endothelial dysfunction, dyslipidemia,

Received 29 May 2025; revised 30 May 2025; accepted 13 August 2025.
Available online 3 October 2025

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https://doi.org/10.61631/3005-3188.1025

inflammation, and a pro-thrombotic state, this makes cardiovascular disease the leading cause of death in people with diabetes (Al-Fatlawi, 2022a, 2022b; Zhao et al., 2024) (see Figs. 1 and 2).

Troponin is a cardiac-specific protein complex released into the bloodstream when heart muscle cells (cardiomyocytes) are damaged (Jaffe et al., 2006). In the atherosclerosis, elevated troponin levels (Fig. 2) often indicate myocardial injury due to reduced blood flow caused by narrowed or blocked coronary arteries, troponin serves as a highly sensitive and specific biomarker for diagnosing acute coronary syndromes (ACS), particularly myocardial infarction, its detection plays a critical role in assessing the severity and progression of atherosclerotic cardiovascular disease (Oemrawsingh et al., 2016).

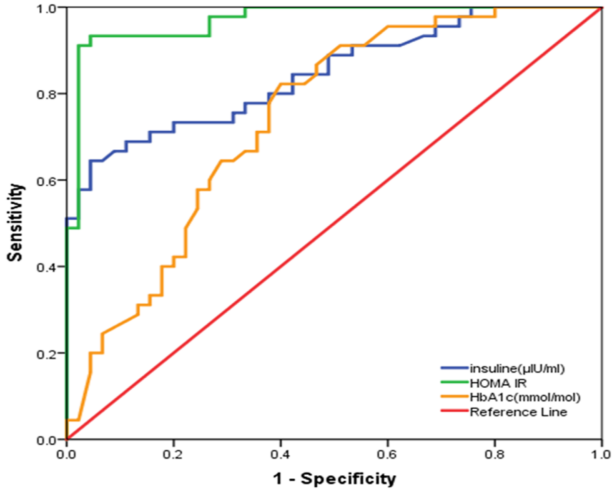

Fig. 1. Receiver Operating Characteristic curve analysis of glucose parameters predictive accuracy for atherosclerosis detection.

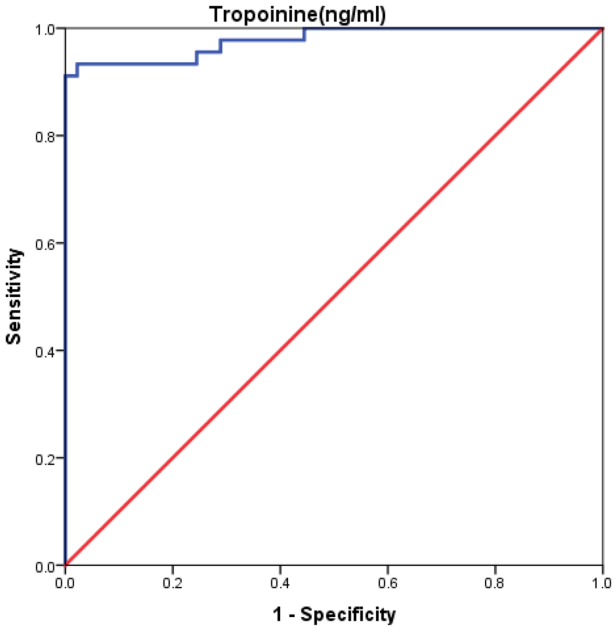

Fig. 2. ROC curve analysis of Troponin predictive accuracy for atherosclerosis detection.

The aim of study to elevated the level of troponin in both subclinical and clinical in atherosclerosis patients.

Material and methods

A Cross-sectional study includes (90) males with range of age (20->60) mean of age (33.00 ± 6.131) were used in the present study divided into (45) patients with clinical coronary atherosclerosis mean of age (34.500 ± 4.949) and (45) patients with sub-clinical coronary atherosclerosis mean of age (32.250 ± 7.228). was conducted to evaluate the clinical and sub-clinical coronary atherosclerosis males related with diabetes type 2 in Karbala, Iraq. Diabetes can be classified into three main categories: non-diabetes (normal), prediabetes, and diabetes (Xiong et al., 2025). The data was collected and recorded from September 2024 to December 2024 at the Imam Hussein Karbala Center for Heart Diseases and Surgery Teaching Hospital. Diseases were diagnosed through laboratory tests, monitoring of some clinical symptoms, and all patients diagnostic by conventional coronary angiography or CT scan by cardiologist in addition to collecting information for each group according to the questionnaire. The exclusion of this study includes Female, Patients with a history of an endocrinal disease, cancers, liver disease, nephropathy, alcohol drinking. The inclusion male patients have clinical or sub-clinical coronary atherosclerosis.

A total of 5 mL of venous blood was collected from each fasting patient through venipuncture using a sterile syringe. Each sample was labeled with a unique identification number generated by the laboratory information system. Of the total volume, 2 mL were transferred into an EDTA tube (containing ethylene diamine tetra acetic acid) to prevent coagulation, allowing for the analysis of hematological parameters such as HbA1c and fasting blood glucose. The remaining 3 mL were placed into a plain gel tube to allow clotting for serum separation. These samples were then centrifuged at 4000 revolutions per minute (rpm) for 10 min.

Serum levels of insulin by insulin kit (Elabsciences, Wuhana, China)by using ELISA kit uses the Sandwich-ELISA, HbA1C by using (OSN, Korea) measurement by using automated chemistry analyzer by Cobas intgra 400 plus, Lipid profile kit (Linear Chemicals S.L, Spain) was measured by

using Enzymatic colorimetric method technique, Homeostatic Model Assessment of Insulin Resistance Calculated by following formula:

(HOMA-IR = fasting plasma insulin (μU/ml) × fasting plasma glucose (mg/dl)/405) (Tahapary et al., 2022).

Principle of troponin

Troponin is a regulatory protein associated with the thin filaments in muscle tissue. It is encoded by three distinct genes, each selectively expressed in different muscle types, giving rise to specific isoforms for slow skeletal, fast skeletal, and cardiac muscle. The cardiac-specific isoform, known as cTnI, has a unique amino acid sequence that makes it especially suitable for laboratory detection of acute myocardial infarction—a common complication of atherosclerosis. This uniqueness has enabled the development of monoclonal antibodies that specifically target cTnI without cross-reacting with skeletal muscle troponins. Numerous published studies have confirmed the effectiveness of measuring cTnI in diagnosing cardiac injury.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics software, version 23. The results were presented through descriptive statistics, with the mean and standard deviation calculated for the relevant variables. To determine the statistical significance of the findings, a p-value threshold of 0.05 was applied. The normality of data distribution was assessed using the Shapiro–Wilk test, while the homogeneity of variances was evaluated via Levene's test. To explore relationships between categorical and numerical variables, both chi-square tests and Pearson's correlation analyses were utilized. Comparisons between two independent groups were conducted using the Mann–Whitney U test and the Independent Samples T-Test. For multiple group comparisons, one-way analysis of variance (ANOVA) was employed, followed by Scheffé's post-hoc test to identify significant differences within groups. Additionally, Receiver Operating Characteristic (ROC) curve analysis was carried out to identify optimal cut-off values for distinguishing critical cases among the study parameters. The area under the curve (AUC) was used to assess the predictive accuracy, and Youden's index was applied to determine the most effective cut-off points. Data with p-values less than 0.01 were marked with asterisks.

Result

In the results found a significant increase in the Random glucose, insulin, HOMA IR,HbA1C,C-peptide, troponin in subclinical atherosclerosis patients (119.677 ± 13.521), (2.582 ± 0.238), (0.760 ± 0.091), (7.240 ± 2.747), (40.291 ± 9.174), (54.426 ± 35.728) as compared to clinical atherosclerosis patients (204.484 ± 47.004), (4.175 ± 1.667), (2.137 ± 1.059), (8.893 ± 2.462), (2303.471 ± 2297.513). While on any significant differences in the level of fasting plasma glucose in clinical atherosclerosis (223.629 ± 84.402) compared to Sub-Clinical Atherosclerosis patients (199.222 ± 100.145), as shown in Table 1 and Figure 1.

The results show a significant increase ($p \leq 0.01$) in Cholesterol, Triglyceride, Low Density Lipoprotein, Very Low Density Lipoprotein Levels in sub clinical (197.785 ± 19.577), (208.802 ± 20.866), (96.370 ± 19.841), (41.760 ± 4.173) respectively, as compared to clinical atherosclerosis patients (247.170 ± 28.155), (249.431 ± 20.182), (156.089 ± 27.699), (49.889 ± 4.036) respectively, while significant decrease in level of high density lipoprotein in clinical atherosclerosis patients (41.195 ± 8.828) as compared to sub clinical atherosclerosis patients (59.654 ± 9.309) as in Table 2.

In the results found a significant decrease in cholesterol,triglyceride, low density lipoprotein,

Table 1. Compare serum concentration of different glucose test a, insulin hormone and Troponin in Clinical –Sub clinical atherosclerosis patients.

Parameters	Subclinical		Clinical		P. value
	Mean	Std. Deviation	Mean	Std. Deviation	
Fasting plasma glucose (mg/dl)	199.222	100.145	223.629	84.402	0.21457
Random glucose (mg/dl)	119.677	13.521	204.484	47.004	0.00005
Insulin (μIU/ml)	2.582	0.238	4.175	1.667	0.00010
HOMA IR	0.760	0.091	2.137	1.059	0.00004
HbA1c (mmol/mol)	7.240	2.747	8.893	2.462	0.00344
C-Peptide (pg/ml)	40.291	9.174	123.614	32.939	0.00007
Troponin (ng/ml)	54.426	35.728	2303.471	2297.513	0.00005

Table 2. Compare serum concentration of lipid profile in subclinical and clinical atherosclerosis patients.

Parameters	Subclinical		Clinical		P. value
	Mean	Std. Deviation	Mean	Std. Deviation	
Total cholesterol (mg/dl)	197.785	19.577	247.170	28.155	0.00005
Total triglycerides (mg/dl)	208.802	20.866	249.431	20.182	0.00007
Low Density lipoprotein (mg/dl)	96.370	19.841	156.089	27.699	0.00010
High density lipoprotein (mg/dl)	59.654	9.309	41.195	8.828	0.00002
Very Low Density Lipoprotein (mg/dl)	41.760	4.173	49.886	4.036	0.00007

very low lipoprotein ($p \leq 0.01$) in sub clinical atherosclerosis patients with in no diabetic, pre diabetic, diabetic (198.964 ± 23.844), (189.877 ± 15.378), (200.854 ± 17.682), (213.597 ± 17.440), (200.355 ± 24.0855), (209.429 ± 21.290), (94.579 ± 24.995), (93.241 ± 17.816), (99.279 ± 16865), (40.071 ± 4.817), (41.886 ± 4.258) as compared to clinical (252.036 ± 20.884), (249.010 ± 29.543), (245.529 ± 29.634), (238.369 ± 32.163), (253.307 ± 22.472), (250.383 ± 16.141), (157.337 ± 19.156), (156.083 ± 31.249), (155.833 ± 28.738), (50.661 ± 4.494), (50.077 ± 3.228), while In the results found a significant increase in HDL ($p \leq 0.01$) in sub clinical atherosclerosis patients with in no diabetic, pre diabetic, diabetic (61.666 ± 8.281), (56.565 ± 9.784), (59.689 ± 9.784) as compared to clinical (47.025 ± 6.061), (42.266 ± 9.687), (39.619 ± 8.688), while as in Table 3.

In the results found a significant decrease ($p \leq 0.01$) in random glucose and c-peptide in, no diabetes, pre diabetic and diabetic and insulin, HHOMA-IR, Troponin in diabetic in sub clinical atherosclerosis patients (116.678 ± 13.350), (124.618 ± 14.638), (0.791 ± 0.087), (40.023 ± 12.081), (41.681 ± 8.209), (1.091 ± 0.141), (0.983 ± 0.116), (53.105 ± 32.255)as compared to clinical (183.001 ± 35.115), (223.577 ± 41.827), (2.383 ± 1.048), (119.324 ± 38.916), (129.894 ± 25.464) (2.626 ± 0.534), (2.847 ± 0.433), (2715.245 ± 2482.232), as in Table 4.

In the Table 5 found that troponin 95 % (CI 0.966–1.000: P-value 0.001: cutoff point:125.000 AUC:97.777 %, with Sensitivity to Specificity 93.333 %–97.437 %; and accuracy: 95.556 %. Also for Insulin 95 % (CI 0.939–1.000: P-value 0.001: cutoff point:2.793: AUC:84.568 %, with Sensitivity to Specificity 71.111 %–84.444 %; and accuracy: 77.778 %. HOM-IR95 % (CI 0.573–1.000: P-value 0.001: cutoff point:0.991: AUC:97.037 %, with Sensitivity to Specificity91.111 %–97.778 %; and accuracy: 94.444 %. HbA1C95 % (CI 0.573–1.000: P-value 0.001: cutoff point:7.350: AUC:68.370 %, with sensitivity to specificity 71.111 %–60.000 %; and accuracy 65.556.

Discussion

Elevated levels of fasting glucose, insulin, HOMA-IR, and HbA1c in clinical atherosclerosis compared to subclinical atherosclerosis are primarily due to increased insulin resistance and chronic hyperglycemia. Insulin resistance impairs glucose uptake, leading to higher blood glucose

Table 3. Compare serum concentration of lipid profile in Clinical –Sub clinical atherosclerosis patients according to classification of diabetes mellitus groups.

Parameters	Group	Subclinical		Clinical		P. value
		Mean	Std. Deviation	Mean	Std. Deviation	
Total cholesterol (mg/dl)	No diabetes	198.964	23.844	252.036	20.884	0.00014
	Pre diabetes	189.877	15.378	249.010	29.543	0.00003
	Diabetes	200.854	17.682	245.529	29.634	0.00001
Total triglycerides (mg/dl)	No diabetes	213.597	17.440	238.369	32.163	0.03283
	Pre diabetes	200.355	24.085	253.307	22.472	0.00008
	Diabetes	209.429	21.290	250.383	16.141	0.00001
Low Density lipoprotein (mg/dl)	No diabetes	94.579	24.995	157.337	19.156	0.00003
	Pre diabetes	93.241	17.816	156.083	31.249	0.00003
	Diabetes	99.279	16.865	155.833	28.738	0.00001
High density lipoprotein (mg/dl)	No diabetes	61.666	8.281	47.025	6.061	0.00095
	Pre diabetes	56.565	9.877	42.266	9.687	0.00427
	Diabetes	59.689	9.784	39.619	8.688	0.00001
Very Low Density Lipoprotein (mg/dl)	No diabetes	42.719	3.488	47.674	6.433	0.03283
	Pre diabetes	40.071	4.817	50.661	4.494	0.00008
	Diabetes	41.886	4.258	50.077	3.228	0.00001

Table 4. Compare serum concentration of glucose parameters in Clinical –Sub clinical atherosclerosis patients according to classification of diabetes mellitus groups.

Parameters	Group	Subclinical		Clinical		P. value
		Mean	Std. Deviation	Mean	Std. Deviation	
Fasting plasma glucose (mg/dl)	No diabetes	127.667	36.533	230.167	103.438	0.05937
	Pre diabetes	158.000	58.038	162.500	45.140	0.84870
	Diabetes	273.500	99.509	243.355	82.959	0.25509
Random glucose (mg/dl)	No diabetes	115.090	10.264	148.008	23.694	0.01777
	Pre diabetes	116.678	13.350	183.001	35.115	0.00003
	Diabetes	124.618	14.638	223.577	41.827	0.00001
insulin (μIU/ml)	No diabetes	2.569	0.226	3.745	1.687	0.14887
	Pre diabetes	2.594	0.329	4.008	1.866	0.04106
	Diabetes	2.585	0.204	4.321	1.634	0.00001
HOMA IR	No diabetes	0.727	0.059	1.401	0.778	0.08737
	Pre diabetes	0.747	0.123	1.867	1.047	0.00806
	Diabetes	0.791	0.087	2.383	1.048	0.00001
HbA1c (mmol/mol)	No diabetes	4.381	0.783	4.768	0.390	0.26769
	Pre diabetes	6.388	0.395	7.035	0.457	0.00326
	Diabetes	9.810	1.778	10.387	1.497	0.22496
C-Peptide (pg/ml)	No diabetes	38.617	8.568	100.411	47.739	0.02450
	Pre diabetes	40.023	12.081	119.324	38.916	0.00001
	Diabetes	41.681	8.209	129.894	25.464	0.00001
Troponin (ng/ml)	No diabetes	61.380	38.657	1141.667	1145.677	0.06893
	Pre diabetes	46.636	39.598	1806.410	2046.301	0.02363
	Diabetes	53.105	32.255	2715.245	2482.232	0.00000

levels, this metabolic dysfunction is more pronounced in clinical atherosclerosis, where systemic inflammation and endothelial dysfunction further exacerbate glycemic control, these factors collectively contribute to the progression from subclinical to clinical atherosclerosis (Al-Fatlawi, 2022a, 2022b; Scott et al., 2024).

Elevated lipid profiles (excluding HDL) are more pronounced in clinical atherosclerosis because the disease is more advanced, with longer and more intense exposure to atherogenic lipids like LDL and triglycerides, clinical stages involve greater inflammation, metabolic disruption, and more harmful lipid particle types, often alongside comorbidities like diabetes or obesity, these factors contribute to higher lipid levels compared to subclinical (early, asymptomatic) atherosclerosis (Ahmadi et al., 2019; Al-Fatlawi, 2022a, 2022b).

Troponin is elevated in atherosclerosis due to myocardial ischemia and subsequent necrosis caused by acute myocardial infarction, the rupture of atherosclerotic plaques leads to thrombus formation in coronary arteries, resulting in reduced blood flow to the heart muscle, this ischemic process, if prolonged, causes damage to cardiac tissue, releasing cardiac troponin into the bloodstream (Oemrawsingh et al., 2016).

In patients with atherosclerosis, glucose parameters are notably higher in those with diabetes and prediabetes compared to nondiabetic individuals due to several interrelated mechanisms, these include Insulin resistance is prevalent in prediabetes and diabetes, leading to elevated blood glucose levels, which contribute to vascular dysfunction, Studies indicate that individuals with prediabetes exhibit a greater atherosclerotic burden

Table 5. Receiver Operation Characteristic (ROC) Curve for evaluation Diagnostic, Accuracy and predictive value for atherosclerosis Biomarkers.

Metrics		Insulin (μIU/ml)	HOMA IR	HbA1c (mmol/mol)	Troponin (ng/ml)
Std. Error		0.041	0.016	0.057	0.014
Asymptotic Sig.		0.002	0.001	0.003	0.006
Asymptotic 95 %	Lower Bound	0.766	0.939	0.573	0.951
Confidence Interval	Upper Bound	0.925	1.000	1.794	1.000
Cutoff Point		2.793	0.991	7.350	125.000
Area Under Curve (AUC)		84.568 %	97.037 %	68.370 %	97.778 %
Sensitivity		71.111 %	91.111 %	71.111 %	93.333 %
Specificity		84.444 %	97.778 %	60.000 %	97.437 %
Accuracy		77.778 %	94.444 %	65.556 %	95.556 %
Positive Predictive Value		82.051 %	97.619 %	64.000 %	97.674 %
Negative Predictive Value		74.510 %	91.667 %	67.500 %	93.617 %

than their nondiabetic counterparts, with significant increases in coronary artery plaque and stenosis severity (Açar et al., 2019; Fuster et al., 2024).

Diabetic dyslipidemia, often referred to as atherogenic dyslipidemia, is characterized by elevated levels of cholesterol and triglycerides along with reduced levels of HDL. To investigate this further, we examined the link between diabetes—another condition closely linked to the progression of atherosclerosis—and lipid abnormalities, developing cardiovascular diseases was markedly recognized (Ali et al., 2015; Peng et al., 2022).

Conclusions

This study has demonstrated an important association between increased serum Troponin levels and risk factors of atherosclerosis such as diabetes mellitus, increased total and LDL cholesterol. Troponine these decreases in metabolic and inflammatory biomarkers indicate that subclinical atherosclerosis represents an earlier, less severe stage of vascular disease, with more preserved physiological function compared to clinical atherosclerosis.

Ethical research approvals

This study was approved by the ethics committee of Department of Clinical Laboratories/College of Applied Medical Sciences /University of Karbala (Ref: CLAMSKU/12). After the Ministry of Health and Environment granted permission to conduct the study, samples were collected and starting the study. The study participants gave their permission to collect socio-demographic data as well as undertake experiments on the selected samples with respecting patient confidentiality.

Authors' contributions

Zeena Z.: Conceptualization, Methodology, Writing-Original Draft. Author B.: Data Curation, Formal Analysis. Author Abeer C. Y. Al-Fatlawi: Investigation, Validation. Author Ahmed Q.J. Al-Haideri: Supervision, Project Administration.

Funding

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

Conflicts of interest

The authors declare that there is no conflict of interest.

References

Açar, B., Ozeke, O., Karakurt, M., Ozen, Y., Özbay, M. B., Unal, S., Karanfil, M., Yayla, C., Cay, S., & Maden, O. (2019). Association of prediabetes with higher coronary atherosclerotic burden among patients with first diagnosed acute coronary syndrome. *Angiology, 70*(2), 174–180.

Ahmadi, A., Argulian, E., Leipsic, J., Newby, D. E., & Narula, J. (2019). From subclinical atherosclerosis to plaque progression and acute coronary events: JACC state-of-the-art review. *Journal of the American College of Cardiology, 74*(12), 1608–1617.

Al-Fatlawi, A. C. Y. (2022a). An evaluation of blood glucose and lipid profile in female hypothyroidism patients in Kerbala province, Iraq. *Biomedicine, 42*(3), 556–560.

Al-Fatlawi, A. C. Y. (2022b). Evaluation of leptin serum concentration in cases of blood transfusion dependent Beta thalassemia and its relationship with thyroid dysfunction. *Biomedicine, 42*(5), 1029–1033.

Ali, F., Jamil, H., Anwar, S. S., & Wajid, N. (2015). Characterization of lipid parameters in diabetic and non-diabetic atherosclerotic patients. *Journal of Geriatric Cardiology: JGC, 12*(1), 37.

Beverly, J. K., & Budoff, M. J. (2020). Atherosclerosis: Pathophysiology of insulin resistance, hyperglycemia, hyperlipidemia, and inflammation. *Journal of Diabetes, 12*(2), 102–104.

Fuster, V., García-Álvarez, A., Devesa, A., Mass, V., Owen, R., Quesada, A., Fuster, J. J., García-Lunar, I., Pocock, S., & Sánchez-González, J. (2024). Influence of subclinical atherosclerosis burden and progression on mortality. *Journal of the American College of Cardiology, 84*(15), 1391–1403.

Jaffe, A. S., Babuin, L., & Apple, F. S. (2006). Biomarkers in acute cardiac disease: The present and the future. *Journal of the American College of Cardiology, 48*(1), 1–11.

Kim, H.-L. (2024). Differences in risk factors for coronary atherosclerosis according to sex. *Journal of Lipid and Atherosclerosis, 13*(2), 97.

Oemrawsingh, R. M., Cheng, J. M., García-García, H. M., Kardys, I., van Schaik, R. H. N., Regar, E., van Geuns, R.-J., Serruys, P. W., Boersma, E., & Akkerhuis, K. M. (2016). High-sensitivity troponin T in relation to coronary plaque characteristics in patients with stable coronary artery disease; results of the ATHEROREMO-IVUS study. *Atherosclerosis, 247*, 135–141.

Peng, X., Huang, J., Zou, H., Peng, B., Xia, S., Dong, K., Sun, N., Tao, J., & Yang, Y. (2022). Roles of plasma leptin and resistin in novel subgroups of type 2 diabetes driven by cluster analysis. *Lipids in Health and Disease, 21*(1), 7.

Scott, D. A., Ponir, C., Shapiro, M. D., & Chevli, P. A. (2024). Associations between insulin resistance indices and subclinical atherosclerosis: A contemporary review. *American Journal of Preventive Cardiology*, Article 100676.

Selby, N. M., & Taal, M. W. (2020). An updated overview of diabetic nephropathy: Diagnosis, prognosis, treatment goals and latest guidelines. *Diabetes, Obesity and Metabolism, 22*, 3–15.

Tahapary, D. L., Pratisthita, L. B., Fitri, N. A., Marcella, C., Wafa, S., Kurniawan, F., Rizka, A., Tarigan, T. J. E., Harbuwono, D. S., & Purnamasari, D. (2022). Challenges in the diagnosis of insulin resistance: Focusing on the role of HOMA-IR and Tryglyceride/glucose index. *Diabetes & Metabolic Syndrome: Clinical Research Reviews, 16*(8), Article 102581.

Xiong, Y.-J., Meng, X.-D., Xu, H.-Z., & Zhu, X.-Y. (2025). Association of frailty index with all-cause and cardiovascular mortality with different diabetic status: NHANES 1999–2018. *Acta Diabetologica, 62*(2), 215–226.

Yameny, A. A. (2024). Diabetes mellitus overview 2024. *Journal of Bioscience and Applied Research, 10*(3), 641–645.

Zhao, N., Yu, X., Zhu, X., Song, Y., Gao, F., Yu, B., & Qu, A. (2024). Diabetes mellitus to accelerated atherosclerosis: Shared cellular and molecular mechanisms in glucose and lipid metabolism. *Journal of Cardiovascular Translational Research, 17*(1), 133–152. https://doi.org/10.1007/s12265-023-10470-x