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Evaluation of sclerostin and oxidative stress markers in coronary artery disease patients

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ABSTRACT

Coronary Artery Disease (CAD) is a heart condition caused by narrowed or blocked coronary arteries. Sclerostin is recognized for reducing bone formation, but new data suggests it may also affect vascular health. The link between sclerostin and CAD is complicated. This study used sclerostin as a marker for CAD in stable coronary disease and examined its relationship to oxidative stress. This study involved 160 people: 80 stable coronary heart disease patients and 80 controls. Patients had significantly lower sclerostin levels (71.256 pg/ml) compared to the control group (98.426 pg/ml) ($p < 0.001$). Additionally, patients had higher oxidative stress (Myeloperoxidase, Malondialdehyde) and lower antioxidant defenses (arylesterase, albumin) compared to the control group. Also, sclerostin strongly positively correlates with arylesterase activity and albumin. Sclerostin levels negatively affect myeloperoxidase activity and MDA concentration. In conclusion, sclerostin may be important role in CAD and can be used to track its progression. The negative relationships between sclerostin and oxidative stress suggest that increased sclerostin levels reduce oxidative load. Sclerostin may protect or regulate oxidative stress-mediated vascular damage based on this inverse association.

1. Introduction

Coronary artery blockage results in an insufficient blood supply to the heart, which causes the symptoms of coronary heart disease CHD [1]. The most frequent sign of CAD is MI, which is brought on by the degradation of the coronary artery endothelium or the disruption of a susceptible atherosclerotic CHD is one of the fatal chronic diseases that lead to the death of millions of people annually in different parts of the world [2], this illness encompasses a broad range of clinical disorders, including as myocardial infarction (MI), unstable angina, stable angina, and sudden death [3]. Important complications are heart failure and arrhythmia [4]. A coronary artery blockage results in an insufficient blood supply to the heart plaque [5]. A coagulation cascade is started when the atherosclerotic plaque ruptures and releases thrombogenic materials [6]. An increase in cardiac biomarkers indicates the permanent necrosis of myocardial cells that ultimately results from MI [7].

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The pathogenesis of CHD is complex and still poorly understood [2]. Low bone mass and high vascular calcification burden are frequently found in elderly patients, but the underlying mechanism is not clear yet [8-10]. As a novel regulator of bone resorption, sclerostin is also expressed in atherosclerosis, but its role is controversial [11]. Sclerostin is an important bone remodeling protein but may also play a role in cardiovascular disease. In preclinical models, sclerostin has been reported to protect against abdominal aortic aneurysm and atherosclerosis formation and prevent cardiac rupture [12,13]. Sclerostin is recognized to be an endogenous inhibitor of the Wnt pathway; therefore, it is foreseeable that the Wnt inhibitory function of sclerostin is important in a tissue such as the heart [14]. Moreover, Wnt is involved in various aspects of heart diseases [15]; thus, the function of sclerostin in the heart in Wnt-independent ways would also be very interesting and beneficial to investigate. By damaging blood arteries and promoting atherosclerosis and plaque development, oxidative stress contributes to CHD [16]. This disorder causes cell damage and vascular wall inflammation due to the body's inability to neutralize reactive oxygen species (ROS) [17]. Endothelial dysfunction, inflammation, and vascular remodeling cause atherosclerosis, plaque, and thrombosis. Overproduction of ROS accelerates these processes [18]. ROS from vascular cells worsen cardiovascular illnesses like ischemic heart disease, atherosclerosis, and hypertension by lipid peroxidation and diminished nitric oxide generation [19]. Insufficient antioxidant capacity causes endothelium and smooth muscle dysfunction, inflammatory signal activation, and mitochondria-mediated death [20]. However, mild oxidative stress can improve cardiovascular health by modifying vascular tone and endothelial function [16]. Oxidative stress markers such malondialdehyde can improve coronary artery disease risk assessment and treatment [21]. MDA is the final product of polyunsaturated fatty acid peroxidation in cells. The innate immune system's heme-containing peroxidase myeloperoxidase (MOP) (EC 1.11.1.7) also causes oxidative stress. Hypohalous acids like hypochlorous acid destroy bacteria and fungus [22]. Besides its role in antimicrobial defense, MPO has been associated to autoimmune diseases and atherosclerotic coronary artery disease (CAD), suggesting it may be a biomarker or therapeutic target for these conditions. Arylesterase (EC 3.1.1.2) serves as a principal defense against free radicals and oxidative stress associated with coronary heart disease [23]. it has a protective effect on LDL: Even though it rides on HDL, PON1/arylesterase indirectly protects LDL particles. Albumin's major thiol group at Cys-34 scavenges reactive oxygen species and inhibits NADPH oxidase activity in vascular smooth muscle cells, resulting in antioxidant, anti-inflammatory, and antithrombotic effects [24]. Low serum albumin (hypoalbuminemia) is associated with worse CAD severity and worse cardiovascular outcomes in epidemiological and clinical studies. The aim of this study was to explore a new perspective of sclerostin by using its level as marker for diagnosing CHD at stable coronary disease and find the correlation of sclerostin concentration and oxidative stress environment.

2. Materials And Methods

2.1 Study Design

The study design is a case-control which was included 160 individuals that distribute to two groups: 80 patients, 49 males and 31 females with stable coronary heart disease, their aged ranged between 46-74 years , and 80 sample which are apparently healthy, 47 males and 33 females as a control group with 46-70 years, the patients' samples were collected from Shar hospital in Erbil city and heart center in Mosul city from March to August 2024, after diagnoses of the cases were confirmed by cardiologist who depend on electrocardiogram and coronary angiogram which were carried in the above-mentioned hospitals. The sample collection was limited to patients with stable coronary heart disease only and not suffering from diabetes or any other chronic disease. The above patient and control groups were divided into subgroups according to demographic data, comprising age and sex. Serum was collected from patients and controls individuals by collect blood samples from them and centrifuged for 10 minutes at 2500 rpm [25].

2.2 Ethics Review

The study adhered to all legal and ethical standards and requirements. Approval was obtained from the Ministry of Health/Nineveh Health Directorate, Mosul, Iraq (Protocol Number: 18187). Written informed consent was obtained from all participants, and the consent forms were signed on may13, 2024.

2.3 Methods

Sclerostin concentration was measured in serum by using Enzyme linked immunosorbent assay (ELISA) kit from Sunlong company (China), The activity of myeloperoxidase was evaluated using the colorimetric method described by [26] using O-dianisidine as substrate, The methodology of Allwsh and Jasim was utilized to assess arylesterase activity [27]. The enzyme was utilized to catalyze the decomposition of the substrate, phenyl acetate, into phenol and acetic acid. Albumin was determined in serum by bromocresol green method using BIOLABO kit. In addition, the improved Guidet and Shah method measures serum MDA, Thiobarbituric acid (TBA) and malondialdehyde (MDA) react to produce a colored product that quantifies lipid peroxidation at 532 nm [28].

2.4 Statistical Analysis

By using SPSS program (version 28), mean and standard deviation were found to compare between the main two groups; control and patient and t-test was used to find the significant change of mean value, in addition ANOVA test was use for comparing between subgroups, in addition Pearson test was used to find the correlation between sclerostin and the biochemical cardiac parameters. In addition, ROC was drawn by using this program for sclerostin.

3. Results And Discussion

The recent study comprised 80 patients with stable coronary heart disease and 80 healthy individuals as control group, the average of control age group was 52.55 years which almost match with the average age of patient group which was 55.75 years. The result was shown a highly significant decrease of sclerostin concentration in the serum of patient group (71.256 ± 15.535 pg/ml) comparing with control group (98.426 ± 11.508 pg/ml) at level ($p < 0.001$) as shown in fig.1.

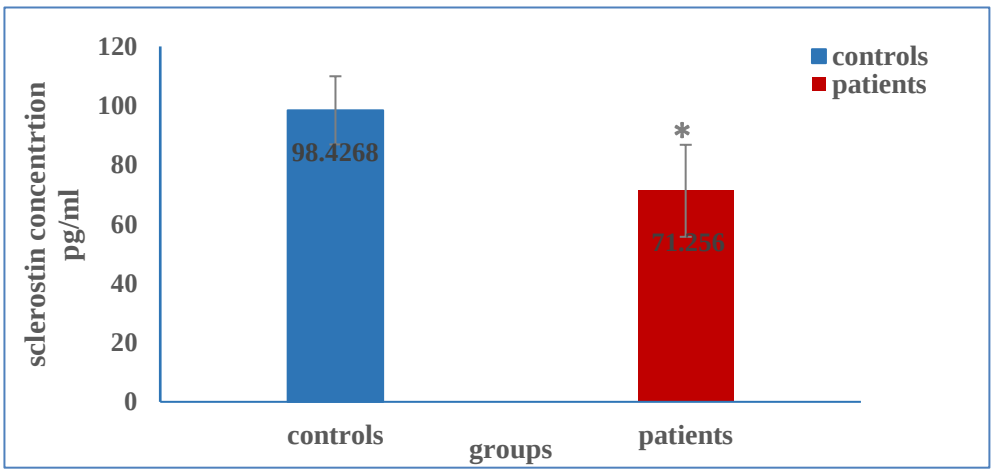

Fig. 1. Sclerostin concentration in coronary heart patients and controls groups
*Significant at the level $p<0.001$

A receiver operating characteristic (ROC) analysis illustrated in fig. 2 and table, depending on the value of area under curves (AUC) (0.941, $P \leq 0.0001$ for sclerostin), Patients at risk for coronary heart disease could be identified with a sensitivity of 0.85 and a specificity of 0.975 when their serum sclerostin concentration was lower than 88.325 pg/ml, which is the cut-off value. ROC elicited that sclerostin concentration had excellent diagnostic performance for stable coronary heart disease.

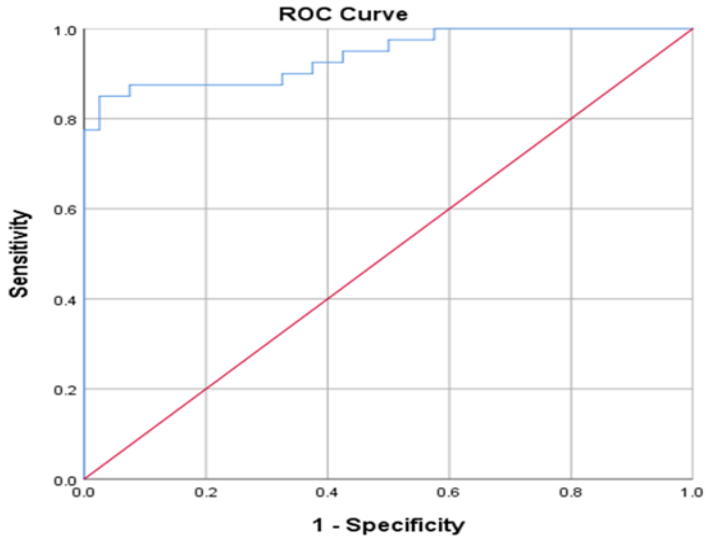

Fig. 2. Sclerostin's receiver operating characteristics (ROC) curve for CHD prediction

Table 1. Sclerostin's receiver operating characteristics (ROC) curve for CHD prediction

Area under the curve	Std. Error [a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval Lower Bound	Asymptotic 95% Confidence Interval Upper Bound	Cut off value	Sensitivity	Specificity
0.941	0.026	0.000	0.891	0.992	88.325	0.85	0.975

a. Under the nonparametric assumption
b. Null hypothesis: true area = 0.5

The major result findings, as shown in table 2, are: Arylesterase activity is significantly lower in CAD patients' group than in controls group while MPO activity is significantly higher in CAD patients. MDA (malondialdehyde), a marker of lipid peroxidation/oxidative stress, is significantly elevated in CAD patients. All with strong statistical significance (P≤0.001), indicating robust differences in this study. However, with statistical significance (P≤0.01), albumin concentration significantly decreases in patients group comparing with control group as shown in table 2.

Table 2. Comparison antioxidant and oxidative stress indicators in control and patients' groups (N=80 of each group)

Parameters	groups	Mean	S.D	P value
Aryl esterase activity (U/L)	control	79.446	9.500	P<0.001
	patients	57.370	12.031	
Myloperoxidase activity (U/L)	control	12.118	1.390	P<0.001
	patients	25.195	3.842	
MDA (µmole/ml)	control	3.960	0.795	P<0.001
	patients	6.596	1.431	
Albumin (g/dl)	control	4.617	0.517	P<0.01
	patients	3.392	0.493	

The correlation analysis between circulating sclerostin concentrations and various antioxidant and oxidative stress indicators in coronary heart patients in this study provides significant insights into the interplay between sclerostin and antioxidant and oxidative stress in coronary heart disease. The data in table 3 show a strong positive correlation between sclerostin and both arylesterase activity ($r = 0.827$, $p \leq 0.01$) and serum albumin concentration ($r = 0.803$, $p \leq 0.01$). This suggests that elevated sclerostin levels are associated with enhanced antioxidant defense capacity. The observed positive

associations may reflect a compensatory response in coronary heart patients, where higher sclerostin secretion is linked with stronger antioxidant activity, potentially to mitigate vascular oxidative stress. Conversely, sclerostin levels correlate negatively and significantly with myeloperoxidase activity ($r = –0.830$, $P \leq 0.01$) and malondialdehyde (MDA) concentration ($r = –0.799$, $P \leq 0.01$). Thus, the negative correlations indicate that higher sclerostin levels are associated with a reduction in oxidative burden. This inverse relationship suggests that sclerostin may play a protective or regulatory role in limiting oxidative stress-mediated vascular injury.

Table 3. Correlation between sclerostin and oxidative stress indicators in coronary heart patients by using Pearson coefficient

Sclerostin concentration (pg/ml)	
Parameters	**Pearson Correlation**
Arylesterase activity (U/L)	0.827**
Myeloperoxidase activity (U/L)	-0.830**
Malondialdehyde concentration (µmole/ml)	-0.799**
Albumin concentration (g/dl)	0.803**

* Correlation is significant at level P≤0.01 level.

The result of this study indicates there was a highly significant decline of serum sclerostin level in patient CAD comparing to normal individuals, the result agrees with Milovanova *et. al.* which emphasized that dropping sclerostin level is a risk factor for cardiovascular complications in end stage renal patients [29], also the recent result agrees with He W *et. al* which documented a decrease level in elderly patients with CAD [30]. The significant decrease of sclerostin level in patient group in this study suggest that sclerostin can be a cause of heart disease, which may be due to low expression of this protein in atherosclerotic plaques [31]. Since sclerostin acts as inhibitor of Wnt pathway [32], which promotes osteogenesis. Dropping sclerostin may enhance Wnt/β-catenin signaling in endothelial cells, promoting plaque instability and atherosclerosis progression., which may lead to calcium accumulation, leading to form harden plaques and arterial stiffness [33] However, this remains controversial and requires more studies.

The reduced arylesterase that is observed in this study agrees with [34] which conclude a decreased PON-1 arylesterase activity in patients suffering from CAD. As arylesterase is one of the primary functions of the PON1 protein, it is one activity of the enzyme Paraoxonase-1 (PON1) [35]. PON1 associates with HDL and has antioxidant functions, including hydrolyzing oxidized lipids, preventing LDL oxidation. Lower PON1/arylesterase activity has been observed in CAD [34]. Mechanistically, reduced PON1/arylesterase may lead to less protection against oxidative modification of lipids, HDL dysfunction, increased LDL oxidation, more foam cell formation, and progression of atherosclerosis. In order that, the significant drop of arylesterase in patients' group of this study; that suggests markedly compromised antioxidant defense.

Myeloperoxidase is an enzyme released primarily from activated neutrophils and some monocytes; it produces reactive oxidants such as hypochlorous acid, contributes to oxidative stress, inflammation, promotes LDL oxidation, endothelial dysfunction. Elevated MPO has been linked to increased risk and severity of CAD [36].

In this study, the MPO activity is more than doubled in patients when compared with controls, as shown in table 2, which agrees with [37] who showed MPO median values are significantly higher in stable CAD against angiography-normal controls.

Malondialdehyde is a lipid peroxidation product, often used as a proxy marker of oxidative damage to lipids. Elevated in CAD patients, correlates with severity [38].

The result of elevated MDA concentration in patients group agrees with [39] which conformed that Serum MDA levels significantly increased with the severity of CAD.

The elevation of MDA concentration in this study that shown in table 2 is consistent with more lipid peroxidation, likely driven by higher reactive oxygen species (ROS), MPO, and lower antioxidant enzyme activities such as arylesterase.

The low serum albumin concentration in the patient's group was found in this study agrees with [40], this reduction is associated with increased oxidative stress, inflammation, and platelet aggregation, which contribute to atherosclerosis and plaque instability that underpin CHD [41].
The current study found that stable coronary artery disease (CAD) patients experience increased oxidative stress, including elevated MPO and lipid peroxidation (MDA). Lower arylesterase (PON1) activity and albumin levels weaken antioxidant defenses. Even in stable disease, oxidants exceeding antioxidants cause endothelial dysfunction, LDL/lipid oxidation, and plaque formation and instability. Reduced albumin may worsen damage (reduced free radical binding) and accelerate disease progression. Such processes may explain these findings: Y. Chen et al. [42] describe stable coronary artery disease (CAD) as characterized by continuous low-grade inflammation, where neutrophils, macrophages, and endothelial cells produce myeloperoxidase (MPO) and reactive oxygen species (ROS).
Reduced arylesterase activity can result from genetic variants in PON1, decreased HDL levels, PON1 modification, and oxidative inactivation [43,44].
Lipid peroxidation: ROS interact with polyunsaturated lipids in LDL or cellular membranes, causing MDA and oxidized lipids, worsening inflammation and atherosclerosis [45]. All these elements cause feedback: Oxidized lipoproteins and lipids boost inflammation and ROS generation, which damages antioxidants.

4. Conclusions

Sclerostin may play a crucial role in CAD and may can utilize as a marker for monitoring the progression of this disease. Vascular calcification, a hallmark of advanced CAD, involves osteogenic differentiation of vascular smooth muscle cells (VSMCs). Also, Collectively, the current study indicates that in patients with stable coronary artery disease (CAD): There is an enhanced oxidative stress environment: high MPO, high lipid peroxidation (MDA). Antioxidant defenses are diminished: reduced arylesterase (PON1) activity and decreased albumin levels, which also partially fulfil antioxidant functions. The imbalance (oxidants surpassing antioxidants) certainly contributes to endothelial dysfunction, oxidation of LDL/lipids, and plaque development and/or instability, even in stable illness. The diminished albumin may exacerbate damage (lower binding of free radicals), thereby facilitating disease progression.

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References

[1] U. Ralapanawa and R. Sivakanesan, "Epidemiology and the magnitude of coronary artery disease and acute coronary syndrome: A narrative review," *J. Epidemiol. Glob. Health*, vol. 11, no. 2, pp. 169–177, Jun. 2021, doi: 10.2991/jegh.k.201217.001.

[2] M. Di Cesare *et al*., "The heart of the world," *Global Heart*, vol. 19, no. 1, Art. no. 11, 2024, doi: 10.5334/gh.1288.

[3] S. Mendis *et al*., "World Health Organization definition of myocardial infarction: 2008–09 revision," *Int. J. Epidemiol.*, vol. 40, no. 1, pp. 139–146, 2011, doi: 10.1093/ije/dyq165.

[4] F. Bessière, B. Mondésert, M. A. Chaix, and P. Khairy, "Arrhythmias in adults with congenital heart disease and heart failure," *Heart Rhythm O2*, vol. 2, no. 6, pt. B, pp. 744–753, 2021, doi: 10.1016/j.hroo.2021.10.005.

[5] D. J. Medina-Leyte *et al*., "Endothelial dysfunction, inflammation and coronary artery disease: Potential biomarkers and promising therapeutical approaches," *Int. J. Mol. Sci.*, vol. 22, no. 8, Art. no. 3850, 2021, doi: 10.3390/ijms22083850.

[6] R. H. Olie, P. E. J. van der Meijden, and H. Ten Cate, "The coagulation system in atherothrombosis: Implications for new therapeutic strategies," *Res. Pract. Thromb. Haemost.*, vol. 2, no. 2, pp. 188–198, 2018, doi: 10.1002/rth2.12080.

[7] G. W. Reed, J. E. Rossi, and C. P. Cannon, "Acute myocardial infarction," *Lancet*, vol. 389, pp. 197–210, 2017, doi: 10.1016/S0140-6736(16)30677-8.

[8] S. Y. Lee, C. T. Chao, J. W. Huang, and K. C. Huang, "Vascular calcification as an underrecognized risk factor for frailty in community-dwelling elderly individuals," *J. Am. Heart Assoc.*, vol. 9, no. 18, Art. no. e017308, 2020, doi: 10.1161/JAHA.120.017308.

[9] T.-L. Chuang, M. Koo, and Y.-F. Wang, "Association of bone mineral density and coronary artery calcification in patients with osteopenia and osteoporosis," *Diagnostics*, vol. 10, no. 9, Art. no. 699, 2020, doi: 10.3390/diagnostics10090699.

[10] L. Aaltonen *et al*., "Association between bone mineral metabolism and vascular calcification in end-stage renal disease," *BMC Nephrology*, vol. 23, Art. no. 12, 2022, doi: 10.1186/s12882-021-02652-z.

[11] J. Golledge and S. Thanigaimani, "Role of sclerostin in cardiovascular disease," *Arterioscler. Thromb. Vasc. Biol.*, vol. 42, no. 7, pp. e187–e202, 2022, doi: 10.1161/ATVBAHA.122.317635.

[12] B. Javaheri *et al*., "Sost haploinsufficiency provokes peracute lethal cardiac tamponade without rescuing osteopenia in a mouse model," *Am. J. Pathol.*, vol. 189, pp. 753–761, 2019, doi: 10.1016/j.ajpath.2018.12.007.

[13] A. De Maré *et al*., "Sclerostin protects against vascular calcification development in mice," *J. Bone Miner. Res.*, vol. 37, pp. 687–699, 2022, doi: 10.1002/jbmr.4503.

[14] F. Marini *et al*., "Role of Wnt signaling and sclerostin in bone and as therapeutic targets in skeletal disorders," *Osteoporosis International*, vol. 34, no. 2, pp. 213–238, 2023, doi: 10.1007/s00198-022-06523-7.

[15] S. Foulquier *et al*., "WNT signaling in cardiac and vascular disease," *Pharmacol. Rev.*, vol. 70, no. 1, pp. 68–141, 2018, doi: 10.1124/pr.117.013896.

[16] M. Myszko *et al*., "The dual role of oxidative stress in atherosclerosis and coronary artery disease," *Antioxidants*, vol. 14, no. 3, Art. no. 275, 2025, doi: 10.3390/antiox14030275.

[17] J. Checa and J. M. Aran, "Reactive oxygen species: Drivers of physiological and pathological processes," *J. Inflamm. Res.*, vol. 13, pp. 1057–1073, 2020, doi: 10.2147/JIR.S275595.

[18] X. Yang *et al*., "Regulation of oxidative stress in coronary heart disease by traditional Chinese medicine," *Oxid. Med. Cell. Longev.*, vol. 2019, Art. no. 3231424, 2019, doi: 10.1155/2019/3231424.

[19] M. Batty, M. R. Bennett, and E. Yu, “The role of oxidative stress in atherosclerosis,” *Cells*, vol. 11, no. 23, Art. no. 3843, 2022, doi: 10.3390/cells11233843.
[20] B. Pang *et al*., “Vascular endothelial injury-associated diseases: Focus on mitochondrial dysfunction,” *Angiogenesis*, vol. 27, no. 4, pp. 623–639, 2024.
[21] S. Simantiris *et al*., “Perivascular fat: A novel risk factor for coronary artery disease,” *Diagnostics*, vol. 14, no. 16, Art. no. 1830, 2024, doi: 10.3390/diagnostics14161830.
[22] W.-Q. Liu *et al*., “Myeloperoxidase-derived hypochlorous acid promotes endothelial senescence,” *Biochem. Biophys. Res. Commun.*, vol. 467, no. 4, pp. 859–865, 2015, doi: 10.1016/j.bbrc.2015.10.053.
[23] D. Kunachowicz *et al*., “Modulatory effect of lifestyle-related factors on paraoxonase-1 activity,” *Int. J. Environ. Res. Public Health*, vol. 20, no. 4, Art. no. 2813, 2023, doi: 10.3390/ijerph20042813.
[24] D. A. Belinskaia, P. A. Voronina, and N. V. Goncharov, “Integrative role of albumin,” *J. Evol. Biochem. Physiol.*, vol. 57, no. 6, pp. 1419–1448, 2021, doi: 10.1134/S002209302106020X.
[25] M. K. Tuck *et al*., “Standard operating procedures for serum and plasma collection,” *J. Proteome Res.*, vol. 8, no. 1, pp. 113–117, 2009, doi: 10.1021/pr800545q.
[26] P. Kumar *et al*., “NADH-oxidase, NADPH-oxidase and myeloperoxidase activity in visceral leishmaniasis,” *J. Med. Microbiol.*, vol. 51, no. 10, pp. 832–836, 2002.
[27] T. Allwsh and R. Jasim, “Arylesterase activity in atherosclerotic patients,” *Rafidain J. Sci.*, vol. 19, no. 2, pp. 143–157, 2008.
[28] B. Guidet and S. V. Shah, “Enhanced in vivo H_2O_2 generation in renal failure,” *Am. J. Physiol. Renal Physiol.*, vol. 257, no. 3, pp. F440–F445, 1989, doi: 10.1152/ajprenal.1989.257.3.F440.
[29] L. Y. Milovanova *et al*., “FGF-23/sKlotho/sclerostin ratio disturbance in ESRD,” *Ter. Arkh.*, vol. 90, no. 6, pp. 48–54, 2018, doi: 10.26442/terarkh201890648-54.
[30] W. He *et al*., “Serum sclerostin and adverse outcomes after PCI,” *Aging Clin. Exp. Res.*, vol. 32, no. 10, pp. 2065–2072, 2020, doi: 10.1007/s40520-019-01393-2.
[31] F. Burger *et al*., “Reduced sclerostin expression in atherosclerotic plaques,” *J. Am. Heart Assoc.*, vol. 13, 2024, doi: 10.1161/JAHA.123.033038.
[32] F. Marini *et al*., “Role of Wnt signaling and sclerostin,” *Osteoporosis International*, vol. 34, no. 2, pp. 213–238, 2023, doi: 10.1007/s00198-022-06523-7.
[33] J. Ohan *et al*., “Coronary artery calcification,” in *StatPearls*. Treasure Island, FL: StatPearls Publishing, 2025.
[34] M. Zuin *et al*., “Paraoxonase-1 arylesterase activity in coronary artery disease,” *Disease Markers*, vol. 2022, Art. no. 4264314, 2022, doi: 10.1155/2022/4264314.
[35] G. L. Erre *et al*., “Paraoxonase/arylesterase activity and rheumatoid arthritis,” *Antioxidants*, vol. 11, no. 12, Art. no. 2317, 2022, doi: 10.3390/antiox11122317.
[36] C. L. Hawkins and M. J. Davies, “Role of myeloperoxidase in inflammation-induced tissue damage,” *Free Radic. Biol. Med.*, vol. 172, pp. 633–651, 2021, doi: 10.1016/j.freeradbiomed.2021.07.007.
[37] M. Cheng *et al*., “Association of myeloperoxidase with coronary artery disease severity,” *Exp. Ther. Med.*, vol. 20, no. 2, pp. 1532–1540, 2020, doi: 10.3892/etm.2020.8817.

[38] M. E. Elsetiha *et al*., "Serum malondialdehyde as predictor for coronary artery disease severity," *Egypt. J. Hosp. Med.*, vol. 95, no. 1, pp. 2206–2211, 2024, doi: 10.21608/ejhm.2024.360963.

[39] H. Asfandiyar *et al*., "Serum malondialdehyde as a biomarker for coronary artery disease," *Cureus*, vol. 16, no. 9, Art. no. e69756, 2024, doi: 10.7759/cureus.69756.

[40] S. Arques, "Serum albumin and cardiovascular disease," *Eur. J. Intern. Med.*, vol. 80, pp. 122–123, 2020.

[41] L. Azouaou *et al*., "Oxidative stress markers in chronic kidney disease," *Arch. Med. Sci. Atheroscler. Dis.*, vol. 9, Art. no. e183, 2024, doi: 10.5114/amsad/192427.

[42] Y. Chen *et al*., "Heterogeneous monocytes in coronary artery disease," *Front. Immunol.*, vol. 16, Art. no. 1428978, 2025, doi: 10.3389/fimmu.2025.1428978.

[43] S. Bassu *et al*., "Paraoxonase and arylesterase activity in psoriatic patients," *Clin. Exp. Med.*, vol. 23, no. 2, pp. 301–311, 2023, doi: 10.1007/s10238-022-00818-z.

[44] M. Vavlukis *et al*., "Paraoxonase 1 gene polymorphisms in atherosclerosis," *Front. Genet.*, vol. 13, Art. no. 966413, 2022, doi: 10.3389/fgene.2022.966413.

[45] K. S. Patil and R. R. Wadekar, "Lipid peroxidation as a signaling mechanism," in *Accenting Lipid Peroxidation*. London, U.K.: IntechOpen, 2021, p. 13, doi: 10.5772/intechopen.99706.

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التأثير المتبادل بين السكليروستين وعلامات الإجهاد التأكسدي في مرض الشريان التاجي المستقر

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معلومات البحث

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سكليروستين، ميلوبيروكسيديز، أريلستريز، مالونديالديهايد، ألبومين، مرض القلب التاجي

الملخص

مرض الشريان التاجي (CAD) هو حالة قلبية ناجمة عن تضيق الشرايين التاجية أو انسدادها. يُعرف السكليروستين بأنه يقلل من تكوين العظام، ولكن تشير البيانات الجديدة إلى أنه قد يؤثر أيضًا على صحة الأوعية الدموية. إن العلاقة بين السكليروستين ومرض الشريان التاجي معقدة. استخدمت هذه الدراسة السكليروستين كعلامة على أمراض القلب التاجية في مرض الشريان التاجي المستقر وفحصت علاقته بالإجهاد التأكسدي. شملت هذه الدراسة 160 شخصًا: 80 مريضًا بمرض القلب التاجي المستقر و80 شخصًا من الضوابط. كان لدى المرضى مستويات سكليروستين أقل بكثير (71.256 بيكوغرام / مل) مقارنة بالمجموعة الضابطة (98.426 بيكوغرام / مل) ($p < 0.001$). بالإضافة إلى ذلك، كان لدى المرضى إجهاد تأكسدي أعلى (ميلوبيروكسيديز، مالونديالدهيد) ودفاعات مضادة للأكسدة أقل (أريلستراز، ألبومين) مقارنة بالمجموعة الضابطة. كذلك، يرتبط السكليروستين ارتباطًا إيجابيًا قويًا بنشاط إنزيم أريلستراز والألبومين. تؤثر مستويات السكليروستين سلبًا على نشاط الميلوبيروكسيداز وتركيز MDA. ختامًا، قد يكون للسكليروستين دورٌ مهم في مرض الشريان التاجي، ويمكن استخدامه لتتبع تطوره. تشير العلاقات السلبية بين السكليروستين والإجهاد التأكسدي إلى أن زيادة مستويات السكليروستين تقلل من الحمل التأكسدي. بناءً على هذا الارتباط العكسي، قد يحمي السكليروستين أو ينظم تلف الأوعية الدموية الناتج عن الإجهاد التأكسدي.

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