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Investigation of oxidative DNA damage levels in patients diagnosed with coronary artery disease using coronary angiography

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Abstract

Coronary artery disease (CAD) is strongly associated with increased oxidative stress, and 8-hydroxydeoxyguanosine (8-OHdG) is a key biomarker reflecting oxidative deoxyribonucleic acid (DNA) damage; however, its clinical relevance in angiographically confirmed CAD patients remains insufficiently defined. This study aims to investigate 8-OHdG levels, which indicate oxidative DNA damage, in patients with CAD diagnosed by coronary angiography and to evaluate the relationship between the presence of CAD and oxidative DNA damage. This prospective study included a total of 100 individuals: 50 individuals diagnosed with CAD based on coronary angiography results (Group 1) and 50 healthy individuals with normal coronary arteries (Group 2). Plasma 8-OHdG levels were measured from venous blood samples taken from both groups using the ELISA method. The groups were similar in terms of age, gender, height, weight, body surface area, and ejection fraction percentage ($p>0.05$). The 8-OHdG level was significantly higher in the CAD group (0.42±0.06 ng/mL), and this increase was statistically significant when compared to the control group (0.25±0.05 ng/mL; $p < 0.001$). This finding indicates that oxidative DNA damage is markedly increased in CAD. Patients in the CAD group were classified angiographically according to vessel involvement, with multivessel disease being predominantly observed. The significantly elevated levels of 8-OHdG in coronary artery disease patients support the notion that oxidative DNA damage plays an important role in CAD pathogenesis. 8-OHdG may be a potential molecular biomarker for CAD. Further investigation of this relationship in larger, multicentre studies is recommended.

Keywords: Coronary artery disease, oxidative stress, 8-OHdG, oxidative DNA damage, biomarker, coronary angiography

Introduction

Coronary artery disease (CAD) is one of the most common and high-mortality pathologies of the cardiovascular system today, creating a significant health burden in terms of morbidity and mortality, particularly in developed countries [1]. This disease, characterised by the development of atherosclerotic plaque, involves a series of complex mechanisms such as endothelial dysfunction, lipid accumulation, inflammation, and vascular wall remodelling. In these processes, reactive oxygen species (ROS) production increases, and oxidative stress causes damage at both the cellular and molecular levels in the microenvironment. For example, the presence of ROS may reduce nitric oxide bioavailability, impair vascular tone, and increase inflammatory activation. All of these factors may contribute to the progression of atherosclerosis [2].

Oxidative damage to Deoxyribonucleic Acid (DNA) constitutes an important part of this scenario. In particular, 8-hydroxydeoxyguanosine (8-OHdG), one of the products resulting from the oxidative modification of guanine, is recognised as a reliable biomarker of DNA damage that can occur in the cell nucleus and mitochondria. In this context, high levels of 8-OHdG may reflect insufficient DNA repair mechanisms or excessive oxidative stress, and may be associated with adverse outcomes for cell life and function. The literature reports increased 8-OHdG levels in individuals with cardiovascular disease; however, most studies have been limited in scope, and systematic investigation of this marker, particularly in patient groups with a definitive diagnosis of CAD confirmed by coronary angiography, is not yet widespread [3,4].

In recent years, considerable attention has been directed toward the identification of molecular biomarkers capable of reflecting the intricate pathophysiological processes underlying CAD, with

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particular emphasis on oxidative stress-mediated mechanisms. Oxidative DNA damage has emerged as a critical indicator, providing a direct link between cellular dysfunction, endothelial impairment, and the progression of atherosclerotic lesions. The assessment of 8-OHdG levels enables the quantitative evaluation of oxidative damage and can provide information on both local vascular changes and systemic oxidative load. By integrating such molecular biomarkers into clinical research, it may be possible to shed light on the severity of the disease at a mechanistic level, improve risk classification, and potentially inform the development of targeted therapeutic interventions aimed at mitigating oxidative damage and its downstream effects.

One of the causes that increase oxidative stress is diabetes, a metabolic condition. This condition has significant effects, particularly on the cardiovascular system. Specifically, it has been reported that 8-OHdG levels measured in leukocyte mitochondrial DNA in patients with type 2 diabetes show significant correlations with CAD presence, degree of coronary artery involvement, and inflammatory markers [5]. These findings suggest that oxidative damage caused by diabetes is closely related to cardiovascular risk. However, it has also been noted that there is no significant relationship between 8-OHdG levels and CAD [6,7]. This suggests that the parameters may show heterogeneity depending on the measurement method, sample material (plasma, urine, cellular DNA), group characteristics, and confounding factors.

Understanding the molecular mechanisms underlying CAD is crucial for gaining deeper insight into the disease process. By examining markers of oxidative DNA damage, such as 8-OHdG, researchers can explore how cellular stress contributes to vascular injury and atherosclerotic progression. This perspective not only enhances the comprehension of disease biology but also highlights the importance of integrating molecular assessments into studies of CAD, offering a more detailed view of how systemic and cellular processes interact in the development and progression of the disease.

Material and Methods

This study was designed as a prospective investigation to systematically evaluate patients undergoing coronary angiography. Patients and volunteer participants were included in the study between August 2025 and September 2025, and all participants were included consecutively.

Ethical Dimension of the Research

The conduct of this investigation was authorised by the relevant institutional bodies, including the Harran University Clinical Research Ethics Committee, which reviewed and approved the study protocol (Date: 14.07.2025; Approval No: HRÜ/25.13.17). All procedures involving human participants were designed and implemented in accordance with the ethical guidelines and fundamental principles set forth in the Declaration of Helsinki. Prior to their enrolment, each participant received detailed information regarding the purpose and procedures of the research, after which written informed consent was obtained to ensure voluntary and ethically sound participation.

Research Population

The research population consisted of patients who underwent coronary angiography and were diagnosed with CAD, as well as patients with normal coronary arteries. Within this population, the relationship between the presence of CAD and oxidative DNA damage (8-OHdG) levels was examined comparatively.

Research Groups

Patients included in the study were divided into two groups based on angiography results:

- Group 1 (CAD Group): Composed of patients diagnosed with CAD. The CAD diagnosis was based on coronary angiography findings. Patients were classified according to the number of vessels showing ≥50% luminal stenosis (1 vessel, 2 vessels, 3 vessels, 4 vessels, 5 vessels).
- Group 2 (Control Group): Consisted of patients with normal coronary vessels who did not receive a CAD diagnosis.

8-OHdG levels were measured in peripheral blood samples from both groups, and a comparative analysis was performed between the groups.

Inclusion and Exclusion Criteria

The study included individuals who underwent coronary angiography and were diagnosed with CAD or found to have normal coronary arteries, and who voluntarily agreed to participate. Individuals with a history of myocardial infarction, chronic renal failure, liver disease or active infection, autoimmune or inflammatory disease, those using antioxidant supplements or who were smokers, and those who declined to participate in the study were excluded. However, patients with common cardiovascular comorbidities such as diabetes mellitus, hypertension, and obesity were not excluded from the study in order to reflect real-world clinical conditions.

Measurements

In both groups, venous blood samples were taken and 8-OHdG levels, an indicator of oxidative DNA damage, were measured. Measurements were performed using the ELISA method. The data obtained were statistically compared between groups and the relationship between the presence of CAD and 8-OHdG levels was evaluated.

Individuals who underwent coronary angiography and were diagnosed with CAD or found to have normal coronary arteries, and who voluntarily agreed to participate in the study, had blood samples collected in anticoagulant-containing sterile biochemistry tubes as part of routine procedures. The samples were centrifuged at 4,000 rpm for 10 minutes, and the plasma fraction was transferred to Eppendorf tubes and stored at -80°C until the day of the study. On the day of the study, all samples

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were removed from -80°C, thawed at room temperature, and 8-OHdG measurements were performed.

Statistical Analyses

Statistical analyses were performed using IBM SPSS Statistics version 25® (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation. The normality of data distribution was assessed using the Kolmogorov–Smirnov test, considering that each study group included more than 30 participants. For comparisons between groups, the Student's t-test was applied to normally distributed variables, while the Mann–Whitney U test was used for variables that did not follow a normal distribution. A two-tailed p value of <0.05 was considered statistically significant.

Result

As presented in Table 1, the demographic characteristics of the study participants were carefully compared between the two groups. Group 1 included patients who had been diagnosed with CAD, while Group 2 comprised healthy control subjects with no evidence of CAD. Variables such as age, height, weight, body surface area and ejection fraction percentage were assessed for both groups, along with gender distribution, to ensure baseline comparability. Statistical analysis revealed no significant differences between the groups for any of these parameters (p>0.05), indicating that the groups were well-matched and that potential confounding effects related to these demographic factors were minimized. Furthermore, within the CAD group, patients were stratified according to the number of obstructed coronary vessels identified during angiographic evaluation, allowing for more detailed analyses of disease severity and its potential association with measured molecular markers. This classification provides a structured framework for exploring how the extent of vascular involvement may correlate with biochemical and oxidative stress parameters.

As shown in Table 2, the 8-OHdG levels of Group 1 with CAD were compared with those of the healthy control group (Group 2). The mean 8-OHdG level in Group 1 (0.42±0.06 ng/mL) was significantly higher than that in the control group (0.25±0.05 ng/mL) (Z= -8.232, p<0.001). This finding indicates that oxidative DNA damage is significantly increased in individuals with CAD (Figure 1).

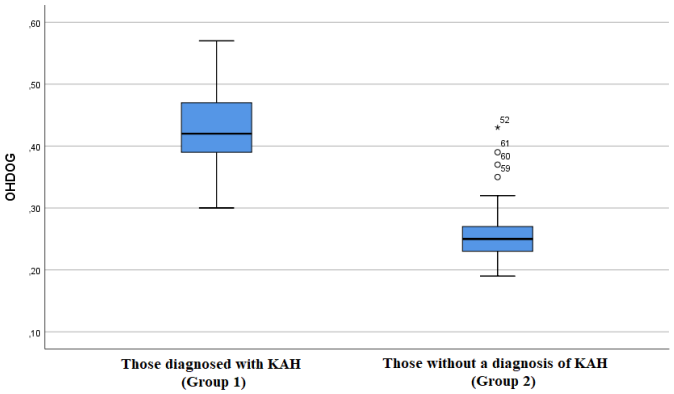

Figure 1. Distribution of 8-OHdG Levels Between Study Groups

Table 1. Demographic characteristics of the study population

Variable	CAD Group (Group 1) (n=50)	Healthy Group (Grup 2) (n=50)	Z / T	P
Age (years) (Mean±SD)	62.80±8.89	61,24±10.02	-0.846	0.398[a]
Height (cm) (Mean±SD)	168.04±10.05	167.82±8.63	-0.376	0.707[a]
Weight (kg) (Mean±SD)	79.10±13.52	74.62±17.31	0.1442	0.181[b]
BSA (m²) (Mean±SD)	1.86±0.167	1.83±0.192	0.935	0.506[b]
EF % (Mean±SD)	51.76±7.01	49.50±8.87	-1.431	0.152a
Female, n (%)	24 (48.0%)	21 (42.0%)	0.598	0.305[b]
Male, n (%)	26 (52.0%)	29 (58.0%)		
Diagnosis n (%)				
2-vessel disease	5 (10%)			
3-vessel disease	24 (48%)			
4-vessel disease	16 (32%)			
5-vessel disease	5 (10%)			

[a]: Mann- Whitney U Testi, [b]: Student-t test, Mean±SD: Mean±Standard Deviation, n: Frequency, %: Percent

Table 2. Comparison of Oxidative DNA Damage (8-OHdG) Levels in Groups

Variable		CAD Group (Group 1) (n=50)	Healthy Group (Grup 2) (n=50)	Z	P
8-OHdG (ng/mL)	Mean±SD	0.42 ± 0.06	0.25 ± 0.05	-8.232	0.000[a]
	Mean rank	74.36	23.64		

a: Mann-Whitney U, Mean±SD: Mean±Standard Deviation, 8-OHdG: 8-hydroxydeoxyguanosine

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Discussion

In this study, 8-OHdG levels were found to be significantly higher in patients diagnosed with CAD via coronary angiography compared to the healthy group without CAD disease identified by coronary angiography. This finding supports the notion that the presence of CAD increases oxidative DNA damage and reinforces the role of ROS in the pathogenesis. Elevated 8-OHdG levels may indicate that cellular DNA repair mechanisms are inadequate or that excessive oxidative stress is present, reflecting the molecular effects of CAD.

Similar results to those of our study have also been reported in the literature [4,8-11]. Xiang et al. demonstrated that serum 8-OHdG levels are associated with the presence of CAD and the severity of the disease [4]. Furthermore, Di Minno et al.'s systematic review highlighted that increased levels of 8-OHdG were detected in individuals with CAD and indicated that this is a reliable biomarker of oxidative stress [3]. Our study, consistent with the literature, provides an important contribution in terms of the systematic investigation of 8-OHdG levels in a group of patients with a definitive diagnosis of CAD based on coronary angiography.

The findings of our study provide further evidence supporting the pivotal role of oxidative DNA damage in the development and progression of CAD. ROS have been shown to induce endothelial dysfunction, promote pro-inflammatory signaling, and accelerate the formation and destabilization of atherosclerotic plaques, all of which contribute to the complex pathophysiology of CAD [2]. By quantifying 8-OHdG, a reliable biomarker of oxidative DNA injury, our research demonstrates that these molecular perturbations can be detected and monitored in a clinical setting. The observed elevation of 8-OHdG in patients with CAD suggests that oxidative DNA damage may not only reflect local vascular injury but also serve as an indicator of systemic oxidative stress, potentially influencing multiple cellular pathways involved in disease progression. These results underscore the translational potential of oxidative DNA markers, emphasizing their utility in both mechanistic studies and as prospective tools for risk stratification, early detection, and monitoring of therapeutic responses in CAD patients.

However, some studies have found no significant association between 8-OHdG levels and CAD, yielding heterogeneous results [6,7]. These differences may be due to measurement methods (plasma, serum, urine, cell DNA), characteristics of the patient population, and confounding factors. In our study, plasma 8-OHdG was measured using the ELISA method, and comprehensive inclusion/exclusion criteria were applied; this enhances the reliability of the data obtained.

In the present study, patients with CAD were categorized according to the number of affected coronary vessels, and the association between 8-OHdG levels and the extent of vascular involvement was evaluated. This classification allowed for a more detailed examination of whether oxidative DNA damage, as indicated by 8-OHdG concentrations, reflects the anatomical severity of the disease. By considering the number of involved vessels, the study provides a framework to explore potential correlations between molecular markers of oxidative stress and the clinical burden of CAD. Future research could build upon these findings by investigating in greater depth the relationship between 8-OHdG levels, disease severity, and long-term clinical outcomes, potentially offering valuable insights into the prognostic significance of oxidative DNA damage in patients with CAD.

In addition, the clinical relevance of 8-OHdG extends far beyond its association with CAD. Numerous studies in the literature have documented strong links between elevated 8-OHdG levels and a wide range of chronic conditions, including type 2 diabetes, hypertension, chronic kidney disease, obesity, and various neurodegenerative disorders. For instance, in individuals with diabetes, increased oxidative injury affecting both mitochondrial and nuclear DNA has been shown to correlate with the onset and progression of vascular complications, underscoring the biomarker's pathophysiological importance [5,12–17]. These findings collectively highlight that 8-OHdG serves not only as a marker of localized oxidative damage but also as a broader indicator of systemic oxidative stress. Consequently, it has gained attention as a promising diagnostic and prognostic biomarker applicable to multiple disease states. Therefore, the elevation detected in patients with CAD may reflect a more widespread oxidative burden within the body rather than being restricted solely to cardiac-specific mechanisms.

Taken together, the biochemical profile observed in our cohort underscores the integral role of oxidative DNA damage in the complex network of molecular events contributing to CAD. The pronounced elevation of 8-OHdG may signify a heightened oxidative milieu capable of modulating endothelial signaling, altering vascular smooth muscle cell behavior, and amplifying inflammatory cascades that collectively drive atherogenic transformation. This interpretation aligns with contemporary evidence suggesting that oxidative stress functions not merely as a by-product of metabolic imbalance but as a dynamic regulator of vascular pathology with direct implications for plaque stability and vascular remodeling. Furthermore, the biomarker's responsiveness to subtle fluctuations in redox homeostasis highlights its potential utility in elucidating pathophysiological trajectories within CAD. When contextualized within existing literature, our findings strengthen the argument that 8-OHdG serves as a sensitive indicator of oxidative perturbation and may reflect a broader disruption of systemic molecular integrity, extending beyond the confines of coronary tissue.

Beyond its role as a biomarker for oxidative stress, 8-OHdG holds potential as a target for therapeutic intervention and risk stratification in CAD management. Monitoring 8-OHdG levels could enable clinicians to identify patients at higher risk of disease

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progression or adverse cardiovascular events, thereby informing personalized treatment strategies. Moreover, interventions aimed at reducing oxidative stress—such as lifestyle modifications, antioxidant therapies, or pharmacological agents—could be evaluated for their efficacy using 8-OHdG as an objective molecular endpoint. Incorporating oxidative DNA damage assessment into routine clinical practice may ultimately enhance early detection, guide preventive measures, and improve patient outcomes, bridging the gap between molecular research and translational cardiovascular medicine.

Limitations

This research is subject to certain constraints. One of the primary limitations is that the study was carried out at a single centre, which may restrict the generalisability of the findings. The exclusion of smokers may limit the generalizability of the findings; however, this approach was chosen to reduce confounding effects related to oxidative DNA damage. Future investigations conducted across multiple institutions, involving broader patient groups and enriched datasets, have the potential to provide more robust and comprehensive evidence.

Conclusion

In this study, it was determined that 8-OHdG levels were significantly higher in patients diagnosed with CAD via coronary angiography compared to healthy controls. This finding indicates that the presence of CAD increases oxidative DNA damage and that significant molecular-level changes occur in the pathogenesis of the disease. The results obtained suggest that 8-OHdG may have potential clinical utility as a molecular biomarker for CAD and may contribute valuable insights into understanding the disease process. Furthermore, these findings indicate that 8-OHdG could be an important biomarker to be investigated for risk stratification and monitoring disease progression in clinical assessments.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
The study received ethical approval from the institutions and from the institutions and the local ethics committee (Harran University Clinical Research Ethics Committee) (Date: 14.07.2025 - Approval no: HRÜ/25.13.17).

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