Original Article

Assessment of Interleukin-1 Alpha, Interleukin-1 Beta, and Oxidative Stress in Patients with Atherosclerotic Cardiovascular Disease in Thi-Qar, Iraq

Fatima Raheem Hassan[1] and Hattf Bazool Farhood[2]

ABSTRACT

Objective: To investigate the changes in interleukin-1 alpha, interleukin-1 beta, malondialdehyde and oxidized low-density lipoprotein in serum of elderly and non-elderly patients with atherosclerotic cardiovascular disease

Study Design: Cross-sectional study

Place and Duration of Study: This study was conducted at the Al-Nasiriya Heart Center and the Biochemistry Laboratory of the College of Science, University of Thi-Qar, from 1st October 2024 to 31st March 2025.

Methodology: 120 samples were enrolled. Sixty patients with atherosclerotic cardiovascular disease were divided by age into two groups: thirty elderly patients (aged 60-79) and thirty non-elderly patients (aged 20- 59), in addition to sixty individuals from the same age groups were divided as a control groups (30 elderly/ 30 non-elderly).

Results: The significant increases ($P \leq 0.05$) in oxidized low-density lipoprotein, malondialdehyde, interleukin-1 alpha, and interleukin-1β concentrations in elderly patients compared to non-elderly patients. In healthy individuals, significant increases in malondialdehyde and interleukin-1 alpha levels were observed only in the elderly group. Moreover, both the elderly and non-elderly patient groups showed significantly higher levels of all the studied parameters compared to their corresponding control groups ($P \leq 0.05$).

Conclusion: Atherosclerotic cardiovascular disease is associated with increased oxidative stress and inflammatory activity, with these abnormalities being more pronounced in elderly patients. The aging may amplify oxidative and inflammatory processes involved in atherosclerotic cardiovascular disease.

Key Words: Elderly, Interluken-1 alpha, Interluken-1 beta, Oxidative stress.

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INTRODUCTION

Disruption of Oxidative balance and activation of inflammatory pathways accelerate atherosclerosis and exacerbate cardiovascular diseases (CVD). According to the oxidative stress theory of aging, the accumulation of reactive oxygen species (ROS) worsens with age, and these species are a double-edged sword; at physiological levels, they maintain cellular homeostasis while excess ROS or weakened antioxidant defenses lead to oxidative stress (OS) associated with vascular wall damage[1]. To detect OS in biological systems (human blood and tissue samples), one of the most widely used biomarkers is Malondialdehyde (MDA). It's a by-product of lipid peroxidation, and is formed when unsaturated fatty acids are oxidized by ROS[2]. Such reactive species are involved in the oxidation of low-density lipoprotein (LDL), leading to the formation of oxidized LDL (OxLDL), which is not only a biomarker of OS but also plays a crucial role in the early stages of atherosclerosis development. Macrophages engulf OxLDL via specific receptors, leading to the formation of foam cells and then plaques[3]. When these plaques rupture, arterial blockage, ischemia, and necrosis of heart tissue occur, as in the case of myocardial infarction. This cellular injury leads to an inflammatory response and promotes the release of several pro-inflammatory cytokines[4], such as Interleukin-1alpha (IL-1α) and Interleukin-1beta (IL-1β). IL-1α is a pro-inflammatory cytokine, and its precursor is found in most cell types under normal

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conditions. When cells are damaged or die, it is released as an alarm signal, triggering a local inflammatory response[5].

Interleukin-1β is a secreted inflammatory cytokine produced by immune cells, particularly macrophages. It is initially synthesized as an inactive precursor (pro-IL-1β), then converted to its active form upon activation of the inflammasome complex (NLRP3), which activates the caspase-1 enzyme. It contributes to vascular inflammation by stimulating the production of other cytokines and increasing the expression of cell adhesion molecules, thus contributing to the development of atherosclerotic plaques[6,7]. Most previous studies have focused on assessing biomarkers of OS or inflammatory mediators separately; therefore, data regarding the interaction between markers of OS and markers of inflammation in cardiovascular patients remain limited, particularly when comparing elderly patients (≥ 60 years) with non-elderly patients (<60 years), as well as with age-matched control groups. Accordingly, this research aimed to assess the levels of OS markers (MDA and OxLDL) and pro-inflammatory cytokines (IL-1α and IL-β) in patients and compare them to age-matched control groups.

METHODOLOGY

This cross-sectional study was conducted at Al-Nasiriya Heart Center and the Biochemistry Laboratory of the College of Science, University of Thi-Qar from 1st October 2024 to 31st March 2025. One hundred and twenty individuals were included (60 patients and 60 controls). They were stratified into two age groups: 30 elderly patients (60-79 years) and 30 non-elderly patients (20–59 years). The same age classification was applied to the control groups. Individuals with thyroid disease, cancers, pregnant women, chronic kidney disease, liver disease and diabetes were excluded. About 5 mL of blood samples were collected in gel tubes and left to coagulate. They were then centrifuged at 3000 rpm for ten minutes, and the serum was separated and stored at -20°C. Serum levels of IL-1α, IL-1β, and OxLDL were measured using enzyme-linked immunosorbent assay (ELISA), while MDA was determined by the TBARS method. All data analyses were carried out using SPSS-25, and t-tests were used to perform comparative analyses.

RESULTS

A highly significant (p≤0.05) difference in age was observed between the studied groups (Table No. 1). Table No. 2 and Figure No. 1 showed that MDA levels were significantly elevated in elderly patients compared with elderly controls and in nonelderly patients compared with their corresponding controls (P≤0.05). In addition, MDA concentrations in elderly patients were significantly higher compared with nonelderly patients, and a similar pattern was observed among controls (P≤0.05).

The OxLDL results indicated a significant increase in all patient groups compared to the control groups (p≤0.05). A significant increase in OxLDL levels was also observed in elderly patients compared to non-elderly patients (p≤0.05). However, in the control groups (elderly and non-elderly) there was no significant (p≤0.05) difference (Table No. 3, Figure No.2).

Table No. 4 and Figure Nos. 3 and 4 showed a significant increase in both IL-1α and IL-1β was observed in all patients compared with their respective controls (P≤0.05). Also, a significant difference was found between elderly and nonelderly patients in both markers (P≤0.05). Among controls, IL-1β levels didn't differ between elderly and non-elderly subjects, whereas IL-1α levels showed a significant difference between the two groups (P≤0.05).

Table No. 1: Demographic and clinical characteristics of the studied groups

Group	No.	Male/Female	Age (years)
Elderly patients	30	15/15	65.70±4.24
Non-elderly patients	30	18/12	48.27±8.92
Elderly control	30	15/15	63.40±4.26
Non-elderly control	30	14/16	42.60±9.57

P<0.001

Table No. 2: Serum MDA values across groups

Groups	Patients	Controls	P value
Elderly	4.06±0.27	1.89±0.27	<0.001
Non-elderly	3.28±0.37	1.64±0.29	<0.001

Table No. 3: Serum OxLDL values across groups

Groups	Patients	Controls	P value
Elderly	4.30±0.55	2.47±0.51	<0.001
Non-elderly	3.19±0.46	2.32±0.50	<0.001

Table No. 4: Serum IL-1α and IL-1β values across groups

Groups	IL-1α (Pg/ml)			IL-1β (Pg/ml)		
	Patients	Controls	P value	Patients	Controls	P value
Elderly	35.36±5.79	24.63±5.51	<0.001	31.14±5.31	20.74±3.65	P<0.001
Non-elderly	31.21±4.28	20.44±5.75	<0.001	26.98±4.37	19.41±4.52	P<0.001

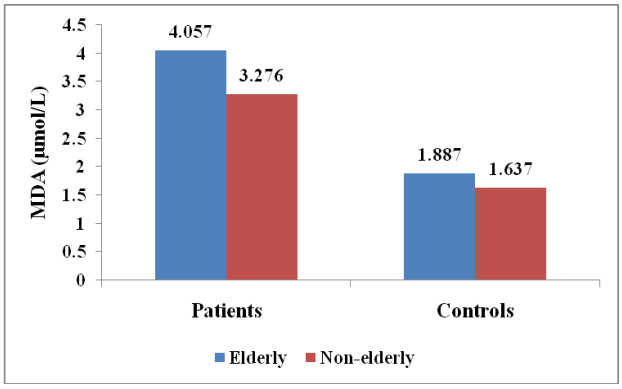


Figure No. 1: Serum MDA levels across groups

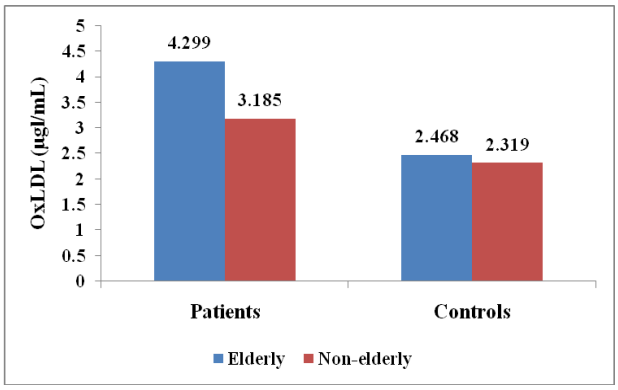


Figure No. 2: Serum OxLDL levels across groups

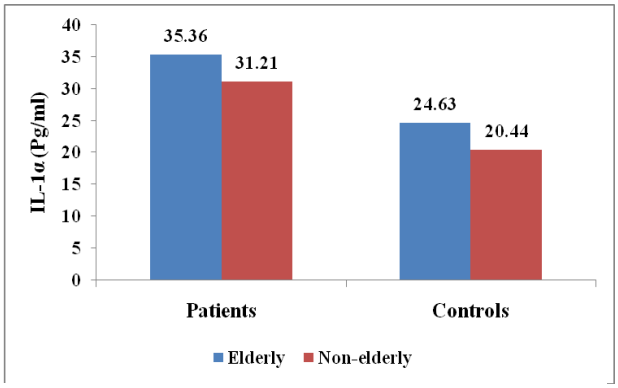


Figure No. 3: Serum IL-1α levels across groups

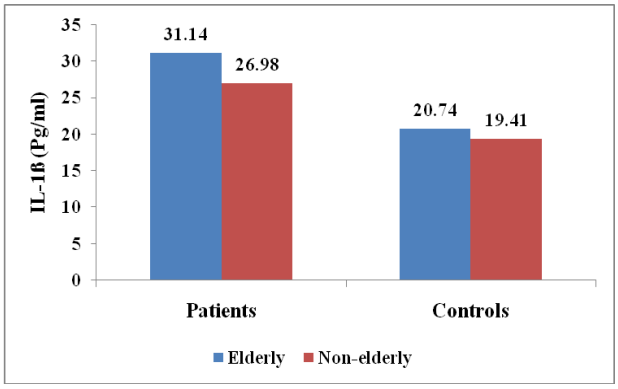


Figure No. 4: Serum IL-1β levels across groups

DISCUSSION

The results of this study showed significantly higher MDA levels in patients compared to control groups, which agrees with recent studies that showed a significant increase in this indicator in patients with MI[8,9]. A cross-sectional study also showed that MDA levels gradually increased with disease severity, supporting the possible role of OS in the development of these diseases[10]. OS contributes to endothelial dysfunction, promotes the inflammatory response, and accelerates atherosclerosis, thereby worsening CVD. During this process, excessive levels of ROS damage cellular macromolecules including lipids, proteins, and DNA, and reduce nitric oxide bioavailability, leading to vasoconstriction and the development of pro-inflammatory and pro-coagulant states within the vasculature[10,11]. Furthermore, the current study showed significantly higher MDA levels in the elderly than in the non-elderly across both patient and control groups ($P<0.05$). This age-related increase can be explained by the synergistic relationship between aging and OS. Aging increases cellular susceptibility to oxidative damage and disrupts the balance of antioxidant systems, promoting lipid peroxidation and increasing the production of its end products, including MDA[12]. Therefore, elevated MDA levels may reflect increased OS associated with both disease progression and aging.

In this research, OxLDL levels were significantly elevated in patients compared to control groups. These results are consistent with recent evidence reported by Rahmanian et al[13] who demonstrated a significant increase in serum OxLDL concentration in patients with MI compared with healthy individuals ($P\leq0.05$). This increase can be attributed to heightened OS and the associated oxidation of LDL molecules, leading to altered biochemical properties and increased vascular damage. OxLDL is a key component in the initiation and progression of atherosclerosis. It contributes to endothelial dysfunction, activates inflammatory responses, and promotes foam cell formation through its uptake by macrophages, ultimately leading to lipid accumulation and the formation and instability of atherosclerotic plaques[14]. A clinical study also showed that elevated OxLDL levels are associated with increased disease severity, with a positive correlation found between OxLDL levels and the number of ischemic events and the number of damaged blood vessels in coronary artery patients, suggesting that it reflects not only the presence of disease but also its severity and the associated oxidative burden[15]. Regarding age, Table No. 3 indicated significant differences in OxLDL levels between elderly and non-elderly patients, while no significant differences were detected between age groups within the control groups.

This may suggest that the effect of aging on OxLDL levels becomes more pronounced in the presence of the disease, as disease-related vascular changes, such as endothelial dysfunction and increased inflammatory activity, promote LDL oxidation and elevated OxLDL levels[16,17]. In healthy individuals, although age-related changes may affect oxidative balance, these changes may not be sufficient to cause a significant increase in OxLDL levels. Therefore, it can be concluded that elevated OxLDL levels are primarily associated with the disease, while aging may act as a contributing factor that exacerbates this increase in the context of the disease.

A significant increase in the levels of both IL-1α and IL-1β was observed in this study, with IL-1α and IL-1β levels being significantly higher in patients compared to control groups. These results agreed with Ibrahim et al[18], who showed a significant increase in IL-1α levels in patients with acute coronary syndrome compared to healthy individuals. Another study conducted on patients with stable angina also demonstrated a significant increase in IL-1β levels compared to the control group, supporting the association of these cytokines with the inflammatory state associated with atherosclerosis and the development of CVD[19]. When analyzing the effect of age within the patient group, the results showed a significant increase in the levels of both IL-1α and IL-1β in elderly patients compared to non-elderly patients. This can be explained by the fact that aging is associated with a chronic, low-grade inflammatory condition known as "inflammaging", which results from increased OS, the accumulation of cellular damage, and increased activity of the innate immune system with advancing age. This inflammatory context contributes to stimulating the immune priming, leading to increased production of inflammatory cytokines in the presence of triggering pathological factors such as atherosclerosis or damage to heart tissue[20]. Elevated levels of IL-1α and IL-1β in elderly patients can also be explained by the functional difference between these two cytokines. IL-1α acts as an alarm cytokine (alarmin) released directly from damaged or necrotic cells 5 , while IL-1β is produced primarily by immune cells following activation of the NLRP3 inflammasome complex[6]. This inflammatory pathway appears to be more active in older adults due to increased age-related inflammatory sensitivity, leading to increased IL-1β production in the presence of disease[20]. In healthy individuals, the age-related difference was limited to IL-1a levels, which were higher in the elderly compared to others. However, IL-1β levels didn't show a significant difference between the two groups. This may be explained by the fact that IL-1α is directly associated with cumulative cellular damage and oxidative stress associated with aging. It can also be released even in the absence of overt disease due to the increased accumulation of senescent cells and their associated senescence-associated secretory phenotype (SASP)[21,22]. Accordingly, the results indicate that elevated IL-1α and IL-1β in patients reflect an active inflammatory response associated with the disease condition, whereas elevated IL-1α only in healthy older adults more likely reflects the effects of aging and cumulative cellular stress than evidence of active pathological inflammation.

CONCLUSION

Patients with atherosclerotic cardiovascular disease showed significantly higher levels of malondialdehyde, oxidized low-density lipoprotein, interleukin-1 alpha, and interleukin-1 beta than healthy controls, with greater elevations generally observed in elderly patients. malondialdehyde and interleukin-1 alpha showed clearer age-related changes, whereas oxidized low-density lipoprotein and interleukin-1 beta were more strongly elevated in diseased elderly individuals. The aging may intensify oxidative stress and inflammatory activity in atherosclerotic cardiovascular disease.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Fatima Raheem Hassan, Hattf Bazool Farhood
Drafting or Revising Critically:	Fatima Raheem Hassan, Hattf Bazool Farhood
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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