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Serum Level of Monocyte Chemoattractant Protein-1 in Arterial Hypertension Patients and Their Association with Disease-Related Factors

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ABSTRACT

Background: *Arterial hypertension is a prevalent long-term cardiovascular condition that contributes significantly to disease and mortality around the world. may represent a critical biomarker of vascular inflammation in patients with arterial hypertension.* ***Methods:*** *A case-control study involved 90 men participants, 60 of whom had arterial hypertension and 30 of whom were healthy controls. The level of monocyte chemoattractant protein-1 (MCP-1) were assessed using an enzyme-linked immunosorbent assay. Additionally, individuals with hypertension were assessed based on their age, body mass index, lipid profile, length of illness, status of treatment, and family history of hypertension.* ***Results:*** *the presented study showed significant increase in level of MCP-1 in patients with arterial hypertension compared to healthy controls. Patients with longer disease duration, obesity, hyperlipidemia, and a positive family history of hypertension were shown to have higher MCP-1 levels. Furthermore, MCP-1 levels were increased in newly diagnosed patients than in treated patients, and all differences were statistically significant ($p < 0.0001$). Conclusion : Overall, our findings suggest that elevated serum MCP-1 levels are associated with arterial hypertension useful inflammatory biomarker for evaluating disease severity and monitoring hypertension in men patients and may serve as a potential biomarker of vascular inflammation and cardiovascular risk.*

Keywords: *Cardiovascular disease , Hypertension , Hyperlipidemia, Inflammatory biomarker , Monocyte chemoattractant protein-1 , Obesity.*

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INTRODUCTION

Arterial Hypertension is a clinical condition characterized by a persistently elevation of blood pressure within the vascular system and is regarded as one of the main risk factors for adverse cardiovascular events (1,2). Furthermore, hypertension is closly associated with cardiovascular disease (CVD), microvascular complications (MVC), and atherosclerotic cardiovascular disease (ASCVD) (3). Hypertensive heart disease remains a main cause of CVD morbidity and mortality on a global scale, necessitating the continuous development of evidence-based management strategies (4).

At the global level, hypertension affects about one to third of people with aged 30–79 years, and its frequency remains to increase due to several factors such as population ageing and modifiable lifestyle-related risk factors(unhealthy diets, high sodium intake, low potassium intake, physical inactivity, obesity, and excessive alcohol consumption) (5),(6). The epidemiology of hypertensive heart disease varies among different countries and regions. These differences are influenced by socioeconomic situations, access to healthcare facilities, and public health systems quality (7).

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From a pathophysiological perspective, genetic predisposition and environmental factors interact to induce structural and functional alterations in the arterial wall through dysregulation of sodium and water homeostasis, activation of the renin–angiotensin–aldosterone system (RAAS), endothelial dysfunction, and vascular smooth muscle remodeling. These processes, further amplified by transcriptional and epigenetic modifications, promote chronic vascular inflammation and arterial remodeling, ultimately leading to the development and persistence of arterial hypertension (8). While chemokine-induced recruitment of peripheral leukocytes to tissues is a crucial step in the initiation and maintenance of inflammatory responses, monocytes from the bloodstream must migrate across the vascular endothelium for routine immunological surveillance of tissues and in response to inflammation (9).

MCP-1, also known as CCL-2, is produced by a variety of cell types and plays a central role in recruiting monocytes and macrophages to sites of tissue injury, thereby amplifying local inflammation and contributing to tissue damage. (10) (11). MCP-1 exerts its biological effects by binding to the seven-transmembrane G protein-coupled receptor CCR-2, triggering intracellular signaling pathways that promote monocyte migration and accumulation at sites of inflammation. This MCP-1/CCR-2 signaling axis plays a central role in the pathogenesis of a wide range of chronic inflammatory diseases (12).

A clinical study reported that MCP-1 concentrations vary with the severity of hypertension, supporting its potential role as a biomarker reflecting different stages of the disease.(13) This case–control study included

METHODS

Totally 90 men participants, comprising 60 patients with arterial hypertension and 30 healthy controls were included in this case control study. Men participants were exclusively enrolled to reduce biological variability and potential confounding factors. Hypertension was diagnosed according to established criteria : a systolic blood pressure ≥140 mmHg and/or a diastolic blood pressure ≥90 mmHg on two separate occasions (14). Patients were further categorized by age (30–39, 40–49, and 50–59 years), disease duration (1–5 and 6–10 years), treatment status (newly diagnosed or receiving antihypertensive therapy), body mass index (normal weight, overweight, and obesity), and family history of hypertension.

All samples were collected from participant attending Al Sader Teaching Hospital \ Al-Najaf city .

Blood Pressure Assessment and Serum MCP-1 Determination

Both of Systolic blood pressure and diastolic blood pressure were measured using a calibrated mercury sphygmomanometer (MDF® 800, MDF Instruments, USA) following standard clinical procedures. Hypertension was diagnosed according to the predefined study criteria.

Three milliliter of venous blood was collected in gel tube from each participant. The collected blood samples were allowed to clot at room temperature for five minutes and were subsequently centrifuged at 3000 rpm for 5 minutes. The separated serum was carefully transferred into labeled Eppendorf tubes and stored at −20°C until analysis of MCP-1 . Serum MCP-1 concentrations were determined using a commercially available enzyme-linked immunosorbent assay kit (Elabscience Biotechnology Inc., Wuhan, China), according to the manufacturer's instructions.

For the assessment of lipid status, patients were categorized into two groups: hypertension alone and hypertension with hyperlipidemia. Hyperlipidemia was identified based on documented clinical diagnoses and information obtained from the participants' medical records; lipid profile parameters were not measured as part of the present study.

ETHICAL APPROVAL

Ethical approval was obtained from Jaber Ibn Hayyan University of Medical and Pharmaceutical Sciences, and informed consent was obtained from all participants.

Statistical Analysis

In presented study ,The data were analyzed using the Statistical Package for the Social Sciences (SPSS; IBM Corp.) and Microsoft Excel (Microsoft Corporation). Statistical analyses were performed to evaluate the study variables, and a $p < 0.05$ was considered to indicate statistical significance.

RESULTS

In the present study, the patient group showed a significant increase ($p < 0.0001$) in the serum concentration of MCP-1 compared with the healthy control group. The mean MCP-1 level in the hypertension patient group was (222.97±3.25), whereas it was (165.4±3.98) in the control group (Figure 1). These findings indicate a marked elevation of MCP-1 levels in patients compared with healthy individuals.

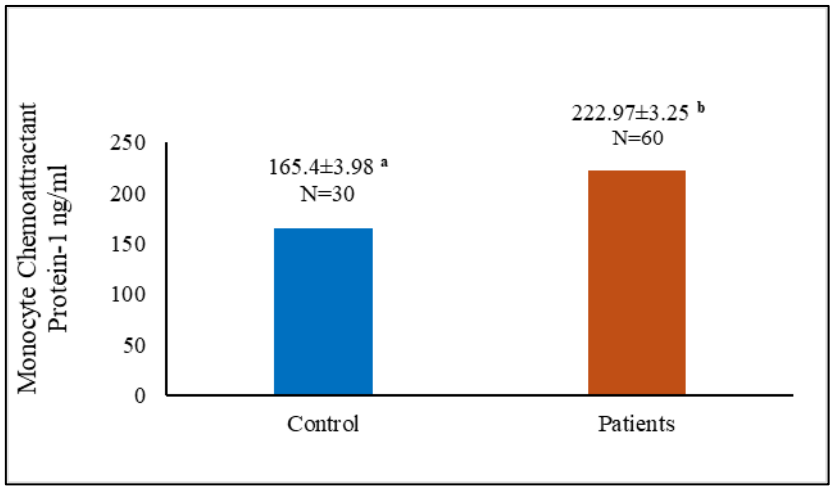

Figure 1 : Comparison of serum MCP-1 levels between men hypertensive patients and control group.

***Different letters indicate significant differences ($p < 0.0001$), while the same letters indicate nonsignificant differences.**

As shown in Figure 2, the concentration of monocyte chemoattractant protein-1 (MCP-1) demonstrated a progressive increase across age groups. The mean MCP-1 level was lowest in individuals aged 30–39 years (193.25±0.83), followed by those aged 40–49 years (222.27±2.66), and reached the highest level in the 50–59 years group (250.56±1.12). This upward trend was statistically significant ($p < 0.0001$), indicating an age-associated elevation in MCP-1 levels.

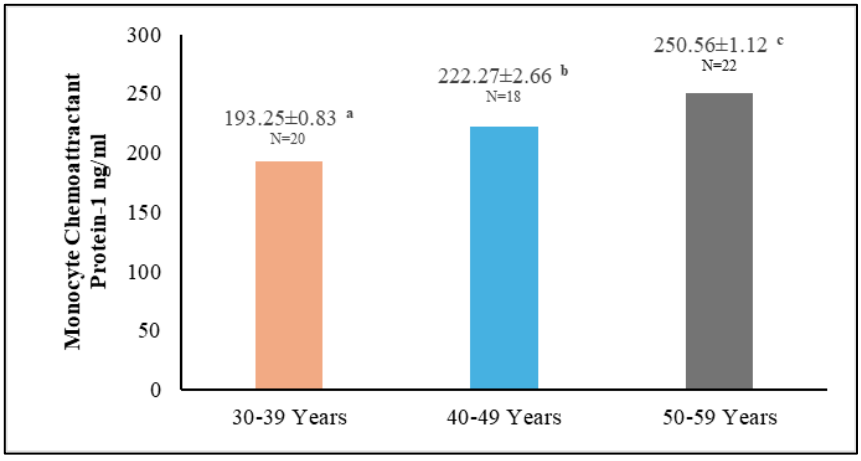

Figure 2 : Association between age and serum MCP-1 concentration in men hypertensive patients.

***Different letters indicate significant differences ($p < 0.0001$), while the same letters indicate nonsignificant differences.**

The current investigation showed a significant increase ($p < 0.0001$) in the MCP-1 concentration in hypertension patients accompanied by hyperlipidemia (242.11±2.13) and patients with hypertension only (196.16±1.43) in comparison to health control group(165.40±3.98). Furthermore, patients with hypertension accompanied by hyperlipidemia exhibited significantly higher MCP-1 levels ($p < 0.0001$) than patients with hypertension only (Figure 3).

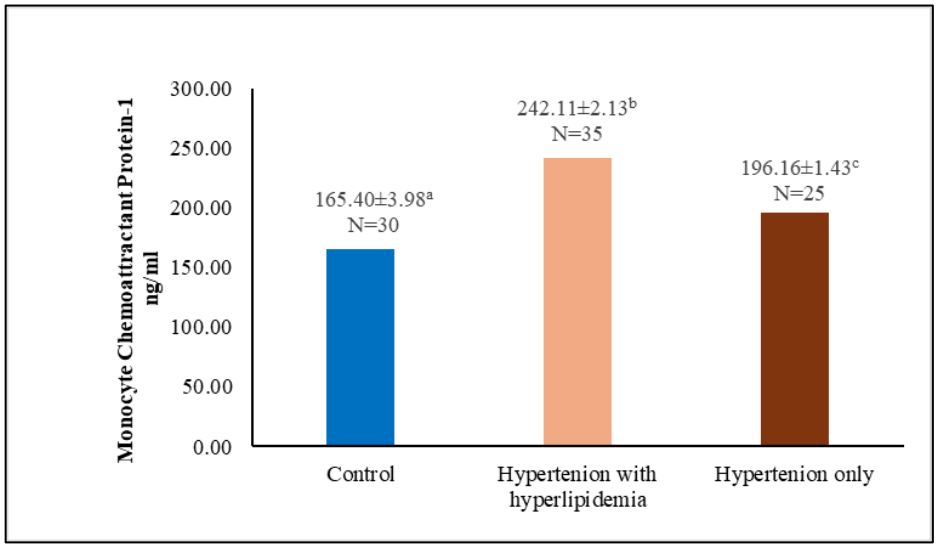

Figure 3 : Comparison of serum MCP-1 levels among hypertensive patients with and without hyperlipidemia and control group.

***Different letters indicate significant differences (p < 0.0001), while the same letters indicate nonsignificant differences**

The results shown in Figure (4) indicate a significant increase (p < 0.0001) in serum MCP-1 level in patients with 6–10 years of hypertension compared with those with 1–5 years of hypertension. The mean MCP-1 concentration was (201.05 ±2.17) in patients with 1–5 years of hypertension and increased to (246.40±1.66) in patients with 6–10 years of hypertension. These findings indicate that prolonged duration of hypertension is associated with a progressive elevation in serum MCP-1 levels.

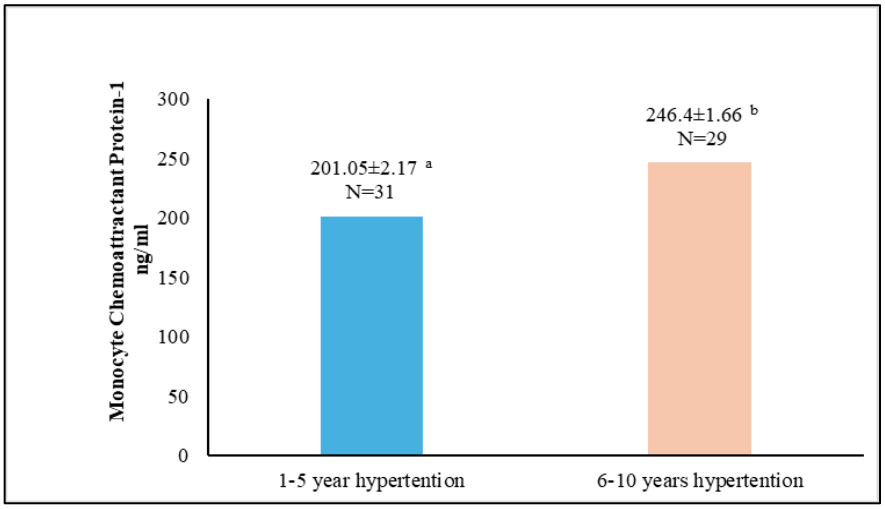

Figure 4 : Comparison of serum MCP-1 levels in men hypertensive patients according to duration of hypertension

***Different letters indicate significant differences (p < 0.0001), while the same letters indicate nonsignificant differences.**

The data of the present study revealed a significant increase (p < 0.0001) in the serum level of MCP-1 in newly diagnosed patients (248.83±1.37) compared with treated patients (204.50±2.53), Figure (5).

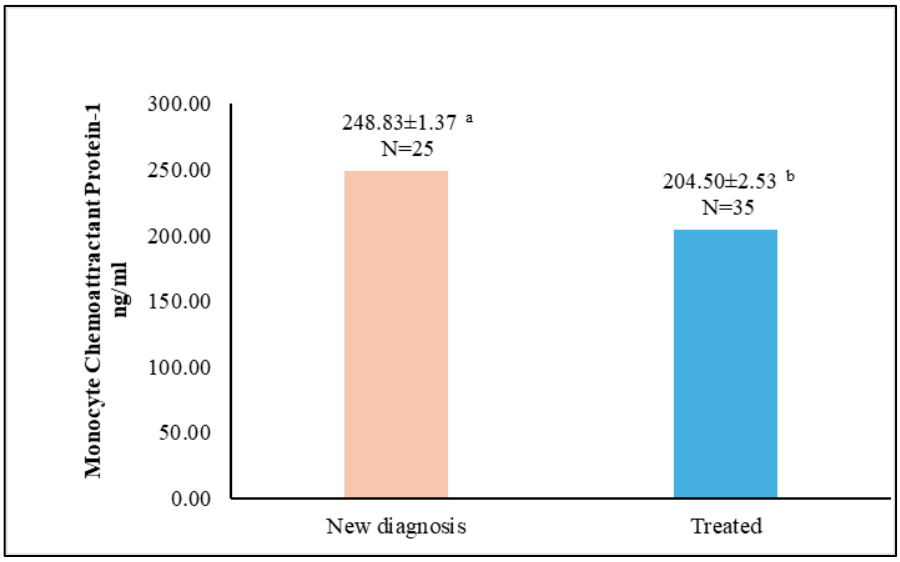

Figure 5 : Comparison of serum MCP-1 levels in newly diagnosed and treated men hypertensive patients

***Different letters indicate significant differences (p < 0.0001), while the same letters indicate nonsignificant differences.**

The results shown in Figure (6) indicated a significant increase (p < 0.0001) in the serum levels of MCP-1 in overweight (214.18±2.90) and obese individuals (248.83±1.37) compared with the normal-weight group (191.59±0.59). The results also showed a significant increase (p < 0.0001) in obese individuals compared with the overweight group. These findings indicate that serum MCP-1 levels increase progressively with increasing body weight.

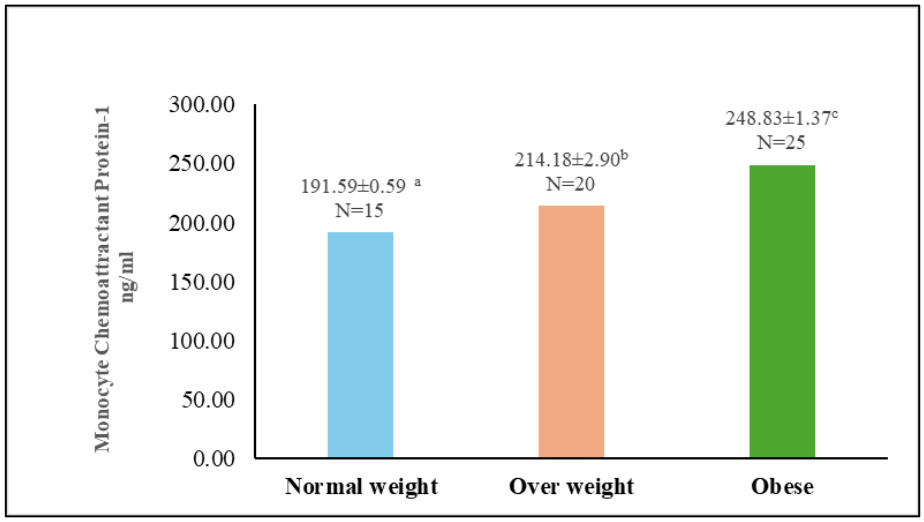

Figure 6 : Comparison of serum MCP-1 levels across body weight categories

***Different letters indicate significant differences (p < 0.0001), while the same letters indicate nonsignificant differences.**

The results obtained from the present study showed a significant increase (p < 0.0001) in serum MCP-1 levels in individuals with a positive family history of hypertension (244.37±1.88) compared with those without a family history (198.51± 1.82) (Figure 7).

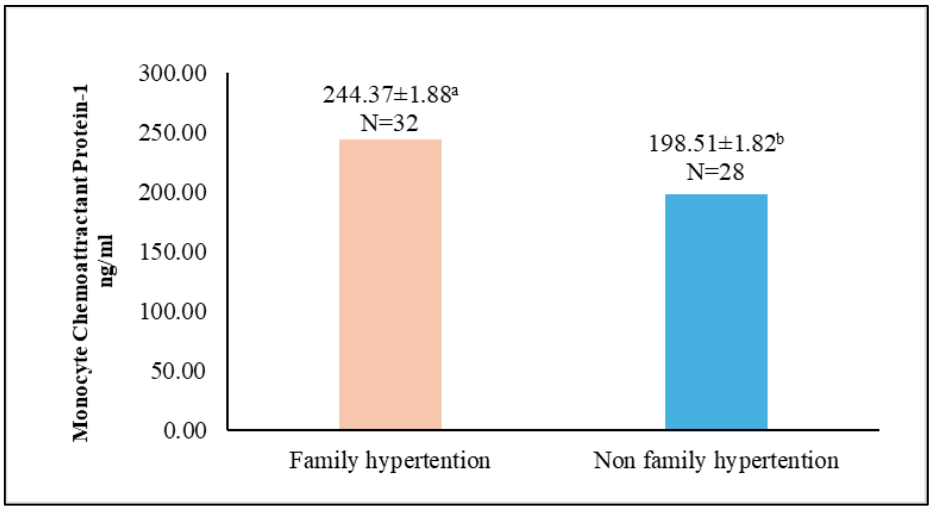

Figure 7 : Comparison of serum MCP-1 levels in hypertensive patients according to family history status

***Different letters indicate significant differences (p < 0.0001), while the same letters indicate nonsignificant differences.**

DISCUSSION

The elevated MCP-1 levels observed in the present study may reflect its important role in the inflammatory mechanisms underlying hypertension. MCP-1 promotes the recruitment of monocytes and macrophages to vascular tissues, contributing to endothelial dysfunction, oxidative stress, vascular remodeling, and arterial stiffness. In addition, MCP-1 facilitates monocyte recruitment into the vascular wall, enhancing atherosclerotic plaque formation and vascular inflammation. These effects may explain the association between increased MCP-1 levels and a higher risk of cardiovascular events, supporting its potential value as a biomarker and therapeutic target in cardiovascular disease(15).

The age-related pattern observed in the present study further supports the role of MCP-1 in hypertension, as its levels increased progressively with advancing age and reached their highest concentrations among patients aged 50–59 years.This finding is consistent with previous studies identifying MCP-1 as an age-dependent inflammatory biomarker associated with biological aging.The increase in MCP-1 with age may be attributed to the combined effects of endothelial dysfunction, vascular stiffness,impaired antioxidant defenses,and enhanced production of pro-inflammatory cytokines, all of which contribute to the chronic inflammatory milieu characteristic of aging and hypertension (16). Moreover, mechanistic evidence suggests that MCP-1, together with TNF-α, amplifies vascular inflammation through Ang II-mediated signaling, thereby accelerating arterial remodeling and vascular stiffness. Persistent elevation of MCP-1 may therefore reflect the progression of endothelial dysfunction and target organ damage, contributing to the increased cardiovascular risk observed in middle-aged and older hypertensive individuals (17).

In the present study, patients with hypertension and hyperlipidemia had significantly higher MCP-1 levels than both hypertensive patients without hyperlipidemia and healthy controls. This finding highlights the close relationship between dyslipidemia and vascular inflammation.Previous studies have shown that oxidized low-density lipoprotein (OxLDL) activates endothelial cells and induces the expression of adhesion molecules on the endothelial surface (18).These adhesion molecules facilitate leukocyte rolling and adhesion to the vascular endothelium, followed by chemokine-mediated migration into the intimal layer (19).Consequently, increased monocyte recruitment into the vascular wall contributes to the initiation and progression of atherosclerotic lesions..

The present study demonstrated that patients with hypertension duration of 6–10 years had significantly higher serum MCP-1 levels than those with a shorter disease duration, suggesting that prolonged hypertension is associated with increased vascular inflammatory activity. The observed elevation in MCP-1 among patients

with a longer history of hypertension may reflect the persistence of the low-grade inflammatory state that characterizes chronic hypertension. Previous studies have indicated that chronic inflammation and oxidative stress play important roles in the development of endothelial dysfunction, vascular remodeling, atherosclerosis, and target organ damage(20). One possible explanation is that prolonged exposure of the vascular wall to elevated blood pressure results in sustained mechanical stress on endothelial cells. This mechanical stimulation promotes endothelial activation and enhances the expression of inflammatory mediators, including MCP-1. Experimental evidence has demonstrated that increased intraluminal pressure can trigger vascular inflammation through mechanosensitive signaling pathways that activate vascular cells(21).

In addition, activation of the renin–angiotensin system, particularly angiotensin II, may contribute to this process. Angiotensin II is known to promote oxidative stress and activate both endothelial cells and vascular smooth muscle cells, leading to increased production of chemokines and other inflammatory mediators within the vascular wall. These mechanisms may partly explain the higher MCP-1 concentrations observed in patients with a longer duration of hypertension(22).

The present findings demonstrated significantly higher MCP-1 levels in newly diagnosed hypertensive patients compared with treated patients, suggesting that antihypertensive therapy may attenuate vascular inflammation in addition to lowering blood pressure. This effect is particularly evident with renin–angiotensin system inhibitors, including ACE inhibitors and angiotensin receptor blockers (ARBs), which have been shown to reduce the production of pro-inflammatory mediators such as MCP-1, IL-6, and TNF-α. Furthermore, ARBs suppress inflammatory signaling through inhibition of NF-κB activation and reduction of reactive oxygen species generation. These anti-inflammatory and antioxidant actions contribute to the cardioprotective and renoprotective benefits of these agents beyond their antihypertensive effects (23,24). Therefore, early initiation of antihypertensive treatment may help limit vascular inflammation and reduce the risk of cardiovascular complications.

In addition, the study found a positive association between body weight and MCP-1 levels, with obese individuals demonstrating the highest concentrations. Obesity is recognized as a chronic low-grade inflammatory condition in which adipose tissue, particularly visceral adipose tissue, acts as an active endocrine organ by releasing pro-inflammatory adipokines and chemokines, including MCP-1 (25). Increased adiposity promotes macrophage infiltration, chronic inflammation, insulin resistance, endothelial dysfunction, and oxidative stress, thereby contributing to the development of hypertension and cardiovascular disease (26). Furthermore, weight reduction has been associated with favorable changes in adipokine profiles, highlighting the importance of weight management in reducing obesity-related inflammatory and cardiovascular complications (25).

Another notable finding was the significantly higher MCP-1 levels in hypertensive patients with a positive family history of hypertension compared with those without such a history. This finding may indicate a possible genetic contribution to the observed variation in MCP-1 levels. However, a positive family history alone does not establish the presence of a specific genetic variant or a direct causal relationship, as it may also reflect the combined influence of genetic susceptibility, shared environmental exposures, and lifestyle factors. In this context, previous genetic studies have reported an association between MCP-1 polymorphisms, particularly the MCP-1-2518A/G variant, and susceptibility to vascular disease (26) .

The relationship observed between MCP-1 and Vascular Endothelial Growth Factor (VEGF) may further suggest that inflammatory and genetic mechanisms influence both vascular inflammation and angiogenic activity. In addition to its role in monocyte recruitment, MCP-1/CCR2 signaling may affect endothelial-cell behavior and interact with pathways involved in angiogenesis, whereas VEGF contributes to endothelial function, vascular permeability, and vascular remodeling (27). The interaction between these pathways may partly explain individual differences in vascular dysfunction and disease progression among

patients with hypertension Overall, the present findings suggest that MCP-1 may reflect the inflammatory and vascular burden associated with hypertension and its related risk factors. Its potential role as a biomarker for disease status and cardiovascular risk warrants further investigation.

CONCLUSION

In conclusion, the present study demonstrates that MCP-1 is associated with hypertension and several clinical factors related to disease burden. Higher MCP-1 levels were observed in association with older age, hyperlipidemia, longer disease duration, obesity, positive family history, and the absence of antihypertensive treatment. These findings support the potential role of MCP-1 as a biomarker of disease status and vascular risk in hypertension. Further studies are needed to clarify its prognostic significance and the mechanisms underlying these associations.

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