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Original Research Paper

Assessment of monocytes chemoattractant protein -1 as a biomarker in females serum of hypertensive patients with and without *Entamoeba histolytica*

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Abstract:
The current study deals with ninety (90) subjects as patients sixty (60)and thirty(30)as control.Samples were collected from suspected patients attended to Al-Sadr Medical City and Al-Furat Al-Awsat Teaching Hospital in Najaf province and also private laboratories. The serum were given from venous at (3-5)milliliter.Systolic and diastolic blood pressure were measured by Sphygmomanometer and lipid profile by AU480 Chemistry Analyzer, and biomarker Monocytes chemoattractant protein (MCP-1) was measured by ELISA technique. Women hypertensive patients were divided into sex sub groups according to types of hypertension (hypertensive only, hypertensive with hyperlipidemia and hypertensive with *Entamoeba histolytica*) ,also according to age, duration of disease, diagnosis, body mass index , and according to family history . **Results:** The current results revealed a significantly increase($p<0.0001$) in monocytes chemoattractant protein (MCP-1) in women hypertension patients group in compare with control group ,biomarker revealed significant increase ($p<0.0001$) in age (50-59) years than other ages , in types of hypertensive sub groups also indicated significant increase($p<0.0001$) in hypertension with hyperlipidemia in compare with another groups,in duration of disease(1-5) years showed a significant increase ($p<0.0001$) than(6-10) years , new diagnosis women patients were significant increase ($p<0.0001$) than treated ,obese women were highly significant ($p<0.0001$) than over weight and normal weight and familial hypertensive women were highly significant increase($p<0.0001$) than nonfamilial hypertensive. The correlation of biomarker was showed significant positive correlation between all subgroups and both systolic and diastolic blood pressure. **Conclusion:** The present study concluded that Monocytes chemoattractant protein (MCP-1) biomarker considered as prognostic for prediction of hypertension in women.

Keywords: Hypertension ,*Entamoeba histolytica*, Hyperlipidemia, MCP-1, Familial hypertensive

1.Introduction

Hypertension, also known as high blood pressure, is the most prevalent chronic illness and the primary cause of disability and early mortality worldwide. It is caused by blood pressing against the body's artery walls. Blood

pressure is written as two numbers. The first (systolic) number represents the blood vessel pressure during a heartbeat or contraction. The second (diastolic) number represents beats. the blood vessel pressure during the pauses between heartbeats [1-2]. There are two types of hypertension: essential and secondary. An increase in blood pressure that raises the risk of cerebral, cardiac, and renal events but has no known cause is known as essential hypertension. Increased systemic blood pressure with a known cause is referred to as secondary hypertension [3]. Heart failure, stroke, and coronary heart disease are among the cardiovascular disease (CVD) events for which hypertension is linked to an increased risk of death [4]. Over one billion people worldwide suffer from hypertension, and that number is rising, making the condition a major global public health concern [5]. High blood pressure, or hypertension, comes in four different stages. You are classified as having stage 1 hypertension if your diastolic blood pressure is between 90 and 99 or your systolic blood pressure is between 140 and 159. Stage 2 or mild hypertension is defined as a systolic blood pressure of 140/90 or a diastolic blood pressure of 159/99. Stage 3 or moderate hypertension is defined as a systolic blood pressure of 160/100 or a diastolic blood pressure of 179/109. 180/110 or higher is considered stage 4 or severe hypertension [6]. Most cases of hypertension are caused by idiopathic hypertension, also known as essential hypertension. It has long been thought that eating more salt increases the chance of getting high blood pressure. The patient's genetic susceptibility to salt response is one of the factors associated with the development of essential hypertension; between 50% and 60% of patients are salt sensitive and thus have a tendency to develop hypertension. [7].

Sedentary lifestyles and high calorie intake can result in increased adiposity, which has been linked to a higher risk of worsening insulin resistance. Insulin resistance has been linked to increased vascular oxidative stress, inflammation, and endothelial dysfunction, which is characterized by decreased vascular nitric oxide bioactivity. These factors all contribute to vascular stiffness, which in turn causes a persistent elevation of blood pressure and the promotion of CVD[8]. Numerous pathophysiological mechanisms have been demonstrated to be present in both obesity and obesity-induced hypertension. Obesity is a global pandemic that contributes to the hypertensive state and cardiovascular morbidity, increasing hospitalizations, events, and healthcare system costs [9]. Elevated blood pressure and structural and functional cardiac and vascular abnormalities that further harm target organs (heart, kidneys, brain, and vessels) and result in early morbidity and death are the hallmarks of hypertension [10]. Impaired vasodilatation is also linked to hypertension. It has long been known that hypertension reduces NO signaling and endothelium-dependent vasodilatation. The mechanisms underlying endothelium-dependent vasodilatation in larger conduit arteries, branch arteries, and resistance arterioles differ fundamentally, as was recently reviewed [11]. The main function of the renin-angiotensin-aldosterone system (RAAS) is to regulate the concentration of sodium and potassium ions in bodily fluids, which is essential for controlling the amount of blood in circulation [12]. The hormone system known as the renin-angiotensin-aldosterone system (RAAS) controls blood pressure and fluid/electrolyte balance. Blood pressure rises when angiotensin II (Ang II) binds to the AngII type 1 (AT1) receptor on vascular smooth muscle cells and tubules, causing vasoconstriction and sodium reabsorption, respectively [13].

Millions of people worldwide suffer from dyslipidemia, a public health issue that raises the risk of cardiovascular disease, the world's leading cause of death. Genetic and environmental factors influence the epidemiology of dyslipidemia, which varies by region, age, sex, and ethnicity [14]. An abnormal rise in the levels of one or more plasma lipids and lipoproteins in the blood is known as hyperlipidemia, a complex lipid metabolism disorder [15]. Hypertension and lipid disorders, particularly hypercholesterolemia (an abnormally high plasma low-density lipoprotein (LDL) concentration, the most atherogenic lipid fraction), are two of the most important cardiovascular risk factors. [16]. Elevated serum levels of total cholesterol (TC), triglycerides (T), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein (VLDL) are all linked to an increased risk of developing hypertension (HTN). Dyslipidemia is common in hypertensive patients. Consequently, the contributing factors to hypertension interact with one another, making it a multifactorial disease [17]. Dyslipidemia and hypertension frequently coexist in clinical practice. There is growing evidence that hypertension and dyslipidemia interact pathophysiologically through oxidative stress,

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proinflammatory activities, renin-angiotensin-aldosterone system (RAAS) stimulation, and endothelium dysfunction [18].

Human amoebiasis is caused by the protozoan parasite *Entamoeba histolytica*. After malaria, this is the second most common cause of parasite-related mortality. According to estimates of the global amoebiasis burden, 500 million people were infected with the parasite, and 10% of those people had invasive amoebiasis [19]. Fedor Lochsch initially described *E. histolytica* in St. Petersburg, Russia, in 1875. He provided a thorough description of intestinal amebiasis, and Fritz Schaudinn initially used the species name E. histolytica in 1903 [20]. Invasive amebiasis caused by *E. histolytica* is more prevalent in developing nations. Amebas can spread directly from person to person through fecal-oral contact in endemic areas due to a number of factors, such as ignorance, poverty, overcrowding, contaminated and insufficient water supplies, and poor sanitation [21]. *E. histolytica* has a straightforward life cycle that consists of just two stages: either an invasive trophozoite or an infectious cyst. After the infectious cyst is consumed, transmission takes place. Fecal contamination of hands, food, or water is the most common cause of this. Excystation to trophozoites takes place after ingestion, and the liberated trophozoites move to the large intestine where they multiply by binary fission to create additional cysts. The trophozoite may infiltrate the intestinal epithelium, travel via the hepatic portal circulation to extra-intestinal locations like the liver, or spread hematogenously to distant locations like the brain and lungs. In addition to developing years after infection, symptoms can appear weeks after ingestion [22–23]. A range of cells, including lymphocytes, monocytes/macrophages, smooth muscle cells, epithelial cells, endothelial cells, and fibroblasts, produce chemokines, which include the monocyte chemotactic proteins (MCP), which are small chemoattractant proteins of 70–130 amino acids [24]. By influencing monocyte/macrophage migration, the quantity of monocytes and T lymphocytes, and osmosis, monocyte chemotactic protein 1 (MCP-1) is directly linked to monocyte recruitment in inflammatory and infectious states [25–26]. The CC chemokine family includes MCP-1 (monocyte chemoattractant protein-1), also referred to as Chemokine (CC-motif) ligand 2 (CCL2). It is essential to the inflammatory process because it draws in or increases the expression of other inflammatory factors and cells [27]. The pathophysiology of hypertension, atherosclerosis, and other cardiovascular diseases is significantly influenced by vascular wall inflammation. By controlling the release of inflammatory chemokines like monocyte chemoattractant protein-1 (MCP-1), angiotensin II (Ang II) causes vascular damage [28].

2. Methodology

Number of patients was (60) and control was (30) ,the patients group divided according to :

Hypertension only (N=25) , hypertension with hyperlipidemia (N= 20) , hypertension with infected *E .histolytica* (N=15), age (30_39) N=20 , (40_49) N=19 , (50_59) N=21, body mass index (normal weight N= 15) (Over weight N=20) (Obese N=25) , familial hypertension or not (Family N=30) (Non_ Family N= 30) , diagnosis (New diagnosis N= 25) (Treated N=35) and duration of disease (1_5 years N= 29) (6_10 years N=31). When blood pressure is measured twice on different days and the systolic and diastolic readings are ≥140 mmHg and ≥90 mmHg, respectively, hypertension is diagnosed [29].

A lipid profile test was performed by using patients serum which was measured using a device AU480 Chemistry Analyzer and the values were found to be higher than normal [30].

Cholesterol more than 200 mg /dL

Triglycried more than 150 mg/dL

High density lipoprotein for women more than or equal 60 mg/dL

Low density lipoprotein more than or equal 130 mg/dL

Patients infected with *E. histolytica* are diagnosed by microscopic examination of stool , the presence of cysts and trophozoites in the freshly voided stool specimens confirms the infection, with symptoms . After confirming the diagnosis , blood sample is taken for immunological testing. Blood samples were collected by drawing 3-5 ml of venous blood using a syringe and placing it in a gel tube at room temperature for 5-10 minutes to coagulate. The blood was then separated by

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centrifugation at 3000 RPM for 5-10 minutes. The separated serum was collected in an Eppendorf tube, labeled with the patient's information, placed in a special rack, and stored -20ºC .

3.Results and discussion

Table 1: Mean of blood pressure and lipid profile in women hypertension patients.

Parmeter	Patients	Control
Systolic pressure mm/Hg	15 ± 0.20447	12 ± 0.0
Diastolic pressure mm/Hg	10.47 ± 0.17052	8 ±0.0
Triglycerides mg/dL	323±12.7525	172.8±9.20743
Cholesterol mg/dL	360.45±26.39711	124.65±3.37114
High density lipoprotein mg/dL	39.3±1.19670	53.6±0.53998
Low density lipoprotein mg/dL	161.05±3.32967	87.65±1.21238

Table 1 showed significant increase mean of blood pressure and lipid profile in women hypertension patients compare with control group.

The results of figure (1) revealed significant increase ($p<0.0001$) in Monocytes Chemoattractant Proteins -1 in women hypertensive patients in compare with control group, also figure (3) showed significant increase in hypertensive sub group hypertensive only , hypertensive with hyperlipidemia and hypertensive with *Entamoeba histolytica* .

The many recent studies has been indicated ahigh level of Monocytes Chemoattractant Proteins -1 in hypertensive patients and clarified that a monocytes considered as immune target in arterial hypertensive through infiltrated the vasculature of arterial endothelium [31-32].

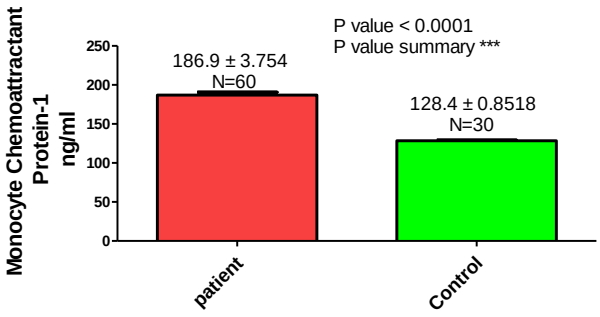

Fig 1: mean of Monocyte chemoattractant protein -1 in hypertensive women patients and control group,,, (*) refer to significant difference ($p<0.0001$)

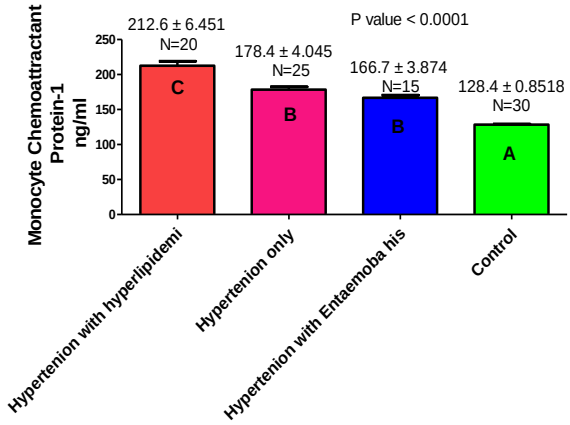

Fig 2: mean of Monocyte chemoattractant protein -1 in hypertensive subgroup .

Different letters refer significant different ($p<0.0001$) and Similar letters refer nonsignificant different

The high level of MCP-1 level in hypertensive patients may be discuss as a role of MCP-1 as inflammation biomarker and reported that deficiency of MCP-1 level may be related with lower level of inflammatory biomarkers such IL-6 and C_ reactive protein (CRP) and these biomarker associated with hypertensive , ischemic stroke and atherosclerosis [33]. The high level of MCP-1 in hypertensive patients with hyperlipidemia may be discuss due to associated between MCP-1 and monocytes on arterial surface and endothelium and smooth muscle may activate or enhanced LDL formation (oxidized) with highly triglyceride and VLDL with accumulated of (ROS) in these patients . MCP-1 level may be fore both hypertensive and hyperlipidemia than hypertensive only and hypertensive with *E*

.*histolytica* . The results may be discuss by several mechanism has bean suggested one of these mechanism associated with intestinal aud systemic inflammation due to infection with *E .histolytica* by induced of inflammatory molecules such as IL-6 and TNF-α which may be linked with obesity [34]. Another causes of increase hypertension in *E .histolytica* patients a regulatory markers such as IL-10 also related with inflammation and obesity [35-36-37]. In a study of Fahmi *et al*.(2018) has been indicated a relation between *E .histolytica* infection and diarrhea and chronic kidney disease may have a hypertensive effect by induced inflammatory response by both increase TNF-α also monocytes recruitment to a site of inflammation [38] . In a study of Ahmed *et al*.(2019) has been demonstrated relation between IL-8 level , MCP-1 level and TNF -α in children infected with *E .histolytica* and found that MCP-1 which may related with monocytes attraction to endothelial cells of patients of hypertension with *E .histolytica* with another inflammatory response [39]. The current results indicates significant increase ($p<0.0001$) in MCP-1 levels according to age , in (50_59) years old age group women patients in compare with age group (40_49) years old and (30_39) years old ,also age group (40_49) were significantly increase than (30_39) years old ,MCP-1 level founded significant increase ($p<0.0001$) in obese women hypertensive patients than over weight women hypertensive patients and normal weight, also over weight women hypertensive patients than normal weight women hypertensive patients . The elder ages generally increase in MCP-1 level and positive correlation between older age and MCP-1 and may be related in high inflammation associated with aging also with high body mass index (obesity) in addition to hyperlipidemia with high recruit of monocytes to a site of inflammation in addition to stress also oxidative stress all these factors may play avital role in high level of MCP-1 with age, several recent studies has been found a positive correlation between MCP-1 level and obesity because highly expression of MCP-1 in fat , visceral and subcutaneous tissue in obese patients and MCP-1 level increased lead to general more adipose tissue [40] . The current results founded that significantly increase ($p<0.0001$) in MCP-1 level according to duration of disease (1-5) years than (6-10) years and new diagnosis patients than treated . The results may be explained between treated patients who had received angiotensin II receptors blocker (ARB) lead to diminished CCR2 receptors with of MCP-1 lower associated with reducing blood pressure , which new diagnosis patients who had no receive and type of treatment with MCP-1 higher than treated because highly expression of MCP-1 endothelium[41]. No present studies or previous deal with effect of duration of disease on MCP-1 therefore (1-5) years in hypertensive patients may reflect that some of these patients were new diagnosis than (6-10) years that majority of patients had received a treatment . MCP-1 level showed significant increase in familial hypertension than non familial ,few previous study has been indicated a relation between familial hypertensive and MCP-1 except study of Roque et al .(2002) on mice that showed that angiotensin II may induced gen and protein MCP-1 expression in CCR2 mice [42] . In a studies (Georgakis et al .,2019 ; Georgakis et al .,2019) has been found genetic predisposition of high increase of MCP-1 level associated with hypertension and ischemic stroke [43-44]. The present study showed positive correlation between MCP-1 in all sub group (hypertensive only , hypertensive with hyperlipidemia , hypertensive with *E. histolytica* with both systolic and diastolic pressure respectively. All recent studies proved that MCP-1 in high level considered a predictive and diagnosing biomarker associated with systolic and diastolic pressure positively with several mechanism included high inflammatory process , oxidative stress , dyslipidemia , lipid metabolism and endothelium damage all these mechanism increase biomarker and positively related with systolic and diastolic [45]. while hypertensive with *E. histolytica* positively related with systolic and diastolic because effect of *E. histolytica* on inflammatory process such as cytokines (IL-6, IL-10, IL1β) may be linked or related with endothelium cell constriction [46].

Conclusion

Monocyte chemoattractant protein -1 use for prediction of women hypertensive patients and biomarker use as prognostic to hypertensive disease with hyperlipidemia than hypertension only and hypertensive with E .histolytica .Biomarker link with elder ages than others ages also with new diagnosed patients than treated also with shorter duration of disease than others and link with obesity. Elevation of biomarker in familial hypertensive mean that some genetic causes associated with

hypertensive patients . Relation of systolic and diastolic pressure with biomarker in all groups positively may benefit for uses biomarker as prognostic and treating of hypertension disease .

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Author's Contributions

The principal investigator, **Mariam Abdullah Musa**, carried out the experiments, collected data, performed statistical analysis, and wrote the manuscript. As the academic supervisors, **Professors Dr. Arshad N. AL-Dujaili** and **Saleem Khteer AL-Hadraawy** gave scientific advice, oversaw the design and interpretation of the study, and critically edited the manuscript for intellectual content. Before submitting the final version of the manuscript, both authors reviewed and approved it.

Ethics

The study was conducted under approval by the medical ethics committee at the University of Kufa (2025).

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