

Immune signatures in primary hypertension: The roles of IL-10, IgM, and IgG

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Abstract

Hypertension (HTN) is the leading modifiable risk factor of cardiovascular diseases. Implicated immunological mechanisms are of great interest nowadays. This study aimed to assess the role of B cells by measuring serum immunoglobulin (IgG) and IgM levels, regulatory T cell levels, and interleukin (IL)-10 levels in hypertensive patients. This observational case-control study included 100 subjects. Sixty newly diagnosed untreated HTN patients were subdivided into three equal groups based on HTN severity. Another twenty patients controlled on their antihypertensive medications over the past three months were enrolled. Patients were compared to twenty apparently healthy subjects. Serum IgG, IgM, and IL-10 were measured by enzyme-linked immunosorbent assay (ELISA), along with complete labs, including complete blood count, erythrocyte sedimentation rate, S-creatinine, C-reactive protein, lipid profile, glycated hemoglobin, glomerular filtration rate, fundus examination, echocardiography, and carotid duplex, which were performed for all participants. IL10 levels were significantly lower among patients (54.84 ± 9.91 pg/ml) compared to the control group (91.12 ± 12.24 pg/ml) ($p < 0.001$). The lowest level was among the mild HTN group. Serum IgG and IgM levels were not significantly higher in patients than in the healthy group. ($p = 0.71$ and 0.31 , respectively). Significant correlation was detected in the moderate HTN group; IL10 was negatively correlated with right common carotid artery intima media thickness (RTCCA IMT) ($r = -0.44$, $p = 0.05$). In conclusion, IL10 is significantly lower among patients with HTN. Dysregulation of immune mechanisms may be associated with HTN. Further studies are needed to reveal nature of this relationship.

Keywords: Immunoglobulin, T regulatory cells, Interleukin, Hypertension

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Introduction

Hypertension (HTN) is the primary risk factor for cardiovascular diseases on a global scale,

resulting in 9.4 million fatalities annually and contributing to 7% of global disability-adjusted life years.¹ Nowadays, it is a growing health problem affecting even younger age groups

than before, causing an increase in cardiovascular disease (CVD) morbidity and even mortality rates worldwide.² The cause of essential hypertension is not well understood, and its pathogenesis is intricate. Disruption of the parasympathetic and sympathetic nervous systems, the endothelin system, the renin angiotensin-aldosterone system (RAAS), genetic predisposition, and environmental factors, either together or separately, can trigger an increase in blood pressure (BP). This rise in BP, along with the prohypertensive stimuli, leads to cardiovascular damage.³⁻⁴

The method by which the immune system is activated remains tentative. Yet, it has been assumed that the initial increase in BP and/or stimuli that promote high BP can cause damage to the cardiovascular system, leading to the creation of damage-associated molecular patterns (DAMPs).^{5,6}

B and T lymphocytes are part of the adaptive immune system and can be considered the leaders of the immune response, directing either pro- or anti-inflammatory responses.⁷ Regulatory T cells (Treg) play a pivotal role in maintaining self-tolerance and immunological balance. They can control both adaptive and innate immune responses to alloantigens, autoantigens, tumor antigens, and infection pathogens.⁸ Treg cells exert suppressive effects by releasing anti-inflammatory cytokines such as interleukin (IL)-10, IL-35, and TGF β ,⁹ which help prevent cardiovascular damage.¹⁰ Hence, a reduction in Treg function may contribute to the development of hypertension in humans.¹¹

Immunoglobulins (IgGs) are a group of proteins that exhibit antibody function, which is vital to the adaptive immune system. They include IgA, IgE, IgD, IgG, and IgM, which collectively make up about 20% of the proteins found in plasma. IgG is the predominant Ig; it constitutes about 80% of the total Igs. It serves as the primary antibody in the secondary humoral immune response.¹²

Serum Ig levels are commonly measured in clinical practice as they offer crucial insights into the humoral immune state. In addition, Igs play significant roles in several inflammatory conditions, such as atherosclerosis.^{13,14}

The aim of this work was to measure levels of serum IgG, IgM, and IL10 in hypertensive patients and explore possible relationship with its complications.

Materials and Methods

This observational case-control work had been performed on one hundred participants. Patients with diabetes, impairment of renal functions, ischemic heart disease, systemic inflammation or infections, solid organ tumors or malignancies, and any endocrine disease were excluded.

Participants were assigned to five equal groups based on the 2018 ESC/ESH Guidelines grading of hypertension.¹⁵ Newly diagnosed hypertensives included 60 untreated patients (mild, moderate, and severe), assigned to three equal groups. They were recently diagnosed and had not received therapy for three months. The fourth group enrolled twenty control patients who had been receiving medication for at least three months. The four patient subgroups were compared with 20 apparently healthy individuals, comparable regarding age and sex.

Patients were clinically assessed by detailed medical history-taking and a comprehensive physical examination. Blood pressure was measured by manual Mercury Sphygmomanometer. All patients underwent fundoscopy, two-dimensional and color Doppler echocardiographic examinations, and carotid Doppler.

Laboratory tests

Complete blood picture with differential white blood cell count was made by automated cell counter,¹⁶ Erythrocyte sedimentation rate (ESR): Estimation was done by the Westergren method (mm/hr),¹⁷ C-reactive protein (CRP) by latex agglutination test,¹⁸ Serum creatinine by calorimetric method (mg/dl),¹⁹ Lipid profile was done by colorimetric Method using Roch Reagent Kits,²⁰ HbA1c by immune-turbidimetry,²¹ Glomerular Filtration rate (GFR) calculated by Cockcroft-Gault Formula.²²

Serum IgG assay was done by commercially available ELISA kits Shanghai SunRed Biological

(SRB) Technology Co., Ltd. (Code 201-12-0187). According to supplier instructions, assay was carried out with a detection range of 5-1500mg/dl.

Serum IgM assay was done by SRB Technology Co., Ltd. (Code 201-12-0188). Detection range was 5-1000mg/dl.

IL10 was done by SRB Technology Co., Ltd. (Code 201-12-0103). Reference range was 5-1000mg/dl, as instructed by the manufacturer.

The following investigation aims to assess HTN complications.

Echocardiography

The Transthoracic echocardiogram was conducted utilizing Vivid 5 ultrasound tools (GE Healthcare) equipped with a 2.5 MHz multi-frequency transducer and TDI software. Standard 2-dimensional measurements [end-diastolic and end-systolic dimensions, ventricular septum and posterior wall thickness, left atrial volume index (LAVI), left ventricular mass index (LVMI), left ventricular (LV) outflow tract diameter] including LV ejection fraction (EF) were taken with the patient in the left lateral position.²³

Carotid duplex

Carotid Doppler test was performed to assess early structural vascular changes based on the intima media thickness (IMT). Carotid IMT images were obtained by using Philips Affiniti 50 ultrasound machine equipped with an ultrasonic duplex scanner. The measurements were obtained near the middle of the common carotid artery and the internal carotid artery.²⁴

Fundoscopy

Fundoscopy examination was done for all participants. It proved being accurate at investigating structural microvascular changes in hypertension.^{25,26} Patients were classified into four degrees according to Mitchell–Wong classification.²⁵ Grade 1 was mild, generalized

constriction of retinal arterioles, Grade 2 was definite focal narrowing of retinal arterioles and AV nicking, Grade 3 was add on flame-shaped haemorrhages, cotton-wool spots and hard exudates and the most severe was Grade 4 where papilledema or retinal edema was detected.

Statistical Analysis

Statistical analysis was conducted using SPSS v26 (IBM Inc., Chicago, IL, USA). Quantitative parameters had been displayed as mean $\pm$ standard deviation (SD) and contrasted among all groups employing ANOVA (F) with post hoc test (Tukey). Qualitative parameters had been displayed as frequencies and percentages and had been analyzed employing the Chi-square test. Correlation between variables was done using the Pearson product-moment correlation coefficient. A two-tailed p value < 0.05 was considered statistically significant.

Results

This study comprised 80 patients. Their mean systolic blood pressure was 156.26 $\pm$ 27.5mmHg while diastolic was 94.35 $\pm$ 15.57mmHg. Important laboratory results were shown in table 1. Most relevant findings were: Both absolute neutrophil count (ANC) and CRP level were significantly higher than control group (p : 0.02, <0.001 respectively). Besides, eGFR was significantly lower among cases than control (p <0.001). Regarding fundus examination 18.75% of patients showed abnormal findings while none of the control did. It is obvious too that ECHO parameters were significantly higher (LVEDD, LVEDV) among patients compared to controls (p values 0.02, <0.001 respectively). Besides, carotid intimal thickness (IMT) was significantly higher among patients too (p <0.001). The single most striking observation to emerge from the data was that IL10 was significantly lower among patients (p <0.001), while total IgG and total IgM results were insignificantly higher.

Table 1. Comparison between hypertensive patients and control group.

		HTN patients N=80	Healthy controls N=20	p value
Age(ys)		46.08+/-3.80	46.03+/-3.71	NS
Sex	Male	49(61.3)	13(65)	NS
	Female	31(38.8)	7(35)	
Smoker		40(50)	10(50)	NS
BMI		27.03+/-2.30	26.07+/-1.45	NS
LDL(mg/dl)		123.90+/- 34.74	127.65+/- 46.74	NS
CV risk %		9.30+/-8.17	5.11+/-5.83	0.03
CRP(mg/dl)		2.96+/-0.45	2.15+/-0.37	<0.001
Neutrophil count($10^3/\mu\text{L}$)		6.03+/-2.40	4.64 +/-2.00	0.02
eGFR ml/min		68.07+/-8.06	89.98+/-8.6	<0.001
Total serum IgG mg/dl		1098.20+/-95.40	1068.44+/-354.03	NS
Total serum IgM mg/dl		123.76+/-10.64	120.89+/-13.10	NS
IL10 pg/ml		54.84+/-9.91	91.12+/-12.24	<0.001
LVEDD(cm)		4.87+/-0.49	4.60+/-0.35	0.02
LVEDV (ml)		113.62+/-14.67	101.65+/-7.81	<0.001
LVEF%		69.36+/-7.16	67.84+/-9.47	NS
RCCA IMT		0.71+/-0.07	0.53+/-0.08	<0.001

Student t test, mean $\pm$ SD, CV risk: cardiovascular risk, IL: interleukin, HTN hypertensive, LVEDD: Left ventricular end diastolic diameter, LVEDV: Left ventricular end diastolic volume, LVEF left ventricular ejection fraction, RCCA IMT right common carotid artery intimal thickness. $p > 0.05$ is not significant (NS).

Having presented the general data, now proceed to a more in-depth analysis focusing on comparisons between study groups, where table 2 illustrates comparison of the clinical and laboratory data of the five groups. The most relevant clinical data was that all study groups varied significantly regarding SBP, DBP except controlled patients compared to healthy control. As regard CV risk, it was significantly higher among patients with severe HTN compared to mild, healthy and controlled hypertensive patient groups (p value 0.005).

With respect to laboratory data, patients with severe hypertension (HTN) had the highest mean values of ANC and LDL. Also, ANC was significantly higher among patients with moderate HTN compared to controlled and healthy groups (p values 0.02, 0.03 respectively); and severe HTN compared to mild, controlled hypertensive patients and healthy groups (<0.001). Healthy group subjects had the lowest mean value of CRP, and this was highly significant when compared to the other

groups (p values <0.001). Surprisingly, controlled hypertensive group had the lowest mean value of eGFR, which was significant when compared to healthy control, mild and moderate HTN groups (p values <0.001, <0.001, 0.007 respectively). Regarding lipid profile, LDL was comparable between groups except it was higher among severe compared to mild hypertensive group (p value 0.019).

Table 2 also highlights the key findings in this work, that IL10 varied significantly between study groups (p value <0.001). Healthy controls had the highest mean value (91.12 ± 12.24 pg/ml) and this varied significantly when compared with each of the other hypertensive subgroups ($p < 0.001$). Besides, patients with mild HTN had the lowest mean value (45.99 ± 4.51 pg/ml) and this was significant when compared with severe, controlled and healthy control groups (p value 0.004, <0.001, <0.001 respectively). Nevertheless, there was no significant difference between study groups regarding total serum IgG and IgM levels Figure 1.

Table 2. Comparison of clinical and laboratory data of the studied groups.

	Mild HTN N=20	Moderate HTN N=20	Severe HTN N=20	Controlled HTN N=20	Healthy control N=20	<i>p</i> value
Age	45.77± 3.45	46.68±4.51	46.28± 3.62	45.57± 3.75	46.03± 3.71	NS*
Sex	Male	12(60)	11(55)	13 (65)	13 (65)	NS**
	Female	8(40)	9(45)	7(35)	7(35)	
Smoker	9(45)	10(50)	11(55)	10(50)	10(50)	NS**
BMI	26.57±2.68	26.82±2.28	26.85±2.00	27.87±±2.14	26.07±1.45	NS*
SBP	147.85±7.85	166.30±12.21	189.90±12.85	121.00±9.36	115.40±9.59	<0.001*
DBP	92.60±9.17	101.00±9.77	107.75±13.37	76.05±6.76	72.65±7.13	<0.001*
CV risk (%)	7.58±5.78	11.09±11.44	12.80±8.09	5.75±3.89	5.11±5.83	0.005*
Neutrophil count(10 ³ /μL)	5.05±2.42	6.54±1.66	7.97±2.02	4.51±1.89	4.64±2.00	<0.001*
CRP mg/dL	2.85±0.49	3.02±0.28	2.93±0.45	3.03±0.53	2.15±0.37	<0.001*
eGFR ml/min	75.10±7.12	69.47±6.38	65.96±6.31	61.72±6.16	89.98±8.60	<0.001*
HDL mg/dl	36.58±7.29	39.45±10.14	36.14±6.22	36.20±9.63	37.09±6.08	NS*
LDL mg/dl	107.79±33.63	117.17±33.22	144.02±31.18	126.62±32.50	127.65±46.74	0.029*
TG mg/dl	199.14±144.51	183.47±117.75	187.72±119.21	224.80±117.10	226.95±99.69	NS*
HbA1c %	5.34±0.61	5.36 ±0.75	5.55±0.75	5.18±0.71	5.28±0.78	NS*
IgG mg/dl	1090.7±106.7	1102.9±119.4	1095.6±8.5	1103.7±109.6	1068.4±354.0	NS*
IgM mg/dl	126.07±12.04	123.15±9.70	124.77±9.23	121.06±11.47	120.89±13.10	NS*
IL10 pg/ml	45.99±4.51	50.86±6.70	54.58±3.76	67.93±7.04	91.12±12.24	<0.001*

*One Way ANOVA test **Chi square test, mean +/- SD or N(%), BMI : body mass index, CV risk: cardiovascular risk, DBP: diastolic blood pressure, eGFR: estimated glomerular filtration rate, IL : interleukin, SBP: systolic blood pressure.

p > 0.05 is not significant (NS).

ECHO findings are provided in table 3, where LVEDV was highest among both mild and controlled groups and this varied significantly with healthy group (*p* value 0.017, 0.018 respectively). As regards carotid duplex, RTCCA IMT was significantly lower among healthy

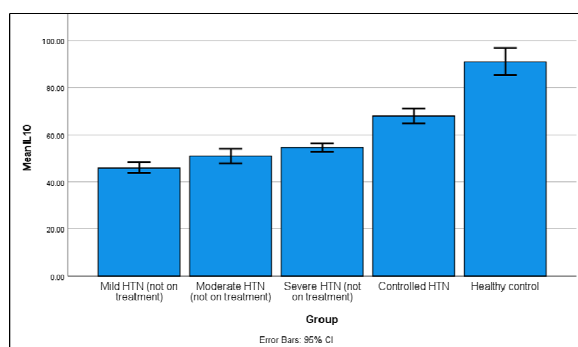
group when compared to each group (*p* value <0.001). Also, the same table shows funduscopy results where all healthy controls had normal fundus examination, unlike patients in the severe hypertensive group where 45% of them had various abnormal findings.

Table 3. Comparison of the studied groups regarding ECHO parameters, carotid and fundus examination.

	Mild HTN N=20	Moderate HTN N=20	Severe HTN N=20	Controlled HTN N=20	Healthy control N=20	p value
LVEDD(cm)	4.93± 0.56	4.84± 0.50	4.92± 0.47	4.79± 0.43	4.60±0.35	NS*
LVEDV (ml)	115.60±13.25	109.48±14.69	113.90±17.00	115.49±13.71	101.65±7.81	0.007*
LVEF%	68.16± 7.39	70.92± 8.52	69.11± 6.34	69.24± 6.43	67.84± 9.47	NS*
RTCCA IMT	0.71± 0.07	0.71± 0.06	0.72± 0.07	0.71± 0.07	0.53± 0.08	<0.001*
Fundoscopy						
Normal	19(95)	18(90)	11(55)	17(85)	20(100)	
Focal	1(5)	1(5)	4(20)	2(10)	0	
Generalized	0	0	2(10)	1(5)	0	0.01**
Hemorrhage	0	0	3(15)	0	0	
Exudate	0	1(5)	0	0	0	

*One Way ANOVA test **Fisher exact test, mean +/- SD or N(%), HTN hypertensive, LVEDD: Left ventricular end diastolic diameter, LVEDV: Left ventricular end diastolic volume, LVEF left ventricular ejection fraction, RTCCA IMT right common carotid artery intimal thickness. $p > 0.05$ is not significant (NS).

Correlations were done between IgG, IgM, IL10 and other study parameters for each group. This revealed in mild hypertensive group that only IgM was negatively correlated with LVEDV ($r = -0.52$, p value 0.02). While in moderate HTN group IL10 negatively correlated with RTCCA IMT ($r = -0.44$, p value 0.05). Concerning healthy controls, a significant negative correlation between IgG and RTCCA IMT ($r = -0.46$, p value 0.04), negative correlation between IgM and LVEF% ($r = -0.56$, p value 0.01), and positive correlation between IL10 and LVEDD ($r = 0.48$, p value 0.03) were found. Nonetheless, no significant correlation was found among patients with controlled or severe hypertension.

**Figure 1.** Comparison between study groups regarding IL10.

Discussion

This work aimed to evaluate the role of Immunoglobulins IgG, IgM, and IL-10 to HTN and its complications. On comparing IgG and IgM, they were insignificantly higher among hypertensive patients compared to controls (0.71 and 0.31, respectively). Which is closer to study by Tomaszewski et al.,²⁷ who didn't find any significant difference between control and hypertensive patients. Also, a study led by Chen et al.,²⁸ concluded that B cell and serum immunoglobulins are unlikely to play a causal role in development of hypertension. However, Wang et al.²⁹ found out significantly higher IgG levels and lower IgM levels among hypertensive patients. Differences in disease duration and chronic inflammatory burden may explain this discrepancy. Also, more ancient studies led by Kristensen 1987³⁰ and Suryaprabha et al. 1984³¹ found out that IgG was significantly higher among patients.

Although our results corroborate with them regarding serum IgG, however statistical significance was not reached in our study, this may be attributed to different disease duration where most of our patients were newly diagnosed except those of controlled group (who exhibited the highest IgG level in our study). Though the cause of raised serum IgG in hypertension cases is difficult to explain, but it

might be a sequelae to vascular injury and endothelial damage induced by hypertension.

Interestingly, in 1989, a Swedish study was performed on malignant HTN patients and compared them to non-malignant hypertensive patients and controls. Total IgG was insignificantly higher among malignant hypertensive group; nevertheless, on measuring IgG subclasses, IgG3 was significantly higher among malignant hypertensive group.³² Another Chinese study led by Meng et al. (2021) reported significant alterations in IgG glycosylation patterns among hypertensive patients, suggesting a role for IgG subclass-specific changes in disease pathophysiology.³³ A large population-based European study in 2021 reported significant alterations in IgG glycosylation patterns associated with hypertension.³⁴ This warrants studying IgG subclass levels, not only total serum levels, to discover the possible role of IgG in this disease.

IL-10 is considered one of the most important anti-inflammatory cytokines and is produced by several immune cell populations, including regulatory T cells, where it helps suppress the activity of many proinflammatory cytokines. As expected, IL-10 levels were significantly lower among patients. A significant difference was also observed between the study groups ($p < 0.001$), with healthy controls showing the highest mean value. This finding is consistent with previous reports demonstrating lower circulating IL-10 levels in hypertensive subjects.³⁵ Odewusi and his colleagues,³⁶ that IL10 level was significantly lower among hypertensives relative to controls.

Another interesting Russian study examining IL10 gene polymorphism in hypertensives found a significant decline in the IL10 -627 C/C polymorphism (known to be associated with increased IL-10 expression) in the male hypertensive group.³⁷ In our study it was clear that treated group showed the highest levels among hypertensives, and this again agrees with **the afore** mentioned studies where IL10 was significantly lower among untreated compared to treated hypertensives. Those results may suggest a promising therapeutic role for IL10. What is astonishing about our study, that it was significantly lower among mild

hypertensives compared to severe groups. So perhaps, it is not linked to severity of hypertension.

A possible biological explanation for this paradox is that early-stage (mild) hypertension may represent a phase of inadequate compensatory anti-inflammatory response, in which regulatory T cell activity and IL-10 production remain insufficient to counterbalance emerging proinflammatory stimuli. As the disease progresses to moderate and severe stages, a compensatory upregulation of anti-inflammatory pathways, including increased IL10 production, may occur in response to sustained vascular injury and inflammation. Thus, the relatively higher IL10 levels in more severe stages may reflect a reactive, albeit insufficient, immunoregulatory mechanism rather than true resolution of inflammation.³⁸⁻⁴⁰

One of the significant correlations revealed in our study was in the moderate HTN group: IL10 negatively correlated with RTCCA IMT ($r = -0.44$, $p = 0.05$). This might focus that lower IL10 is associated with atherosclerosis and further complications, however this needs further prospective studies to confirm, as ischemic patients were excluded from this study.

IL10 is known to exert protective effects on the vascular endothelium through multiple mechanisms, including suppression of proinflammatory cytokines (such as TNF- α and IL-6), inhibition of endothelial cell activation, and reduction of oxidative stress. It also downregulates the expression of adhesion molecules, thereby limiting leukocyte recruitment and vascular inflammation, which are key steps in atherogenesis. Furthermore, IL10 promotes plaque stability by inhibiting macrophage activation and foam cell formation and by enhancing anti-inflammatory signaling within the vascular wall. These combined effects may explain the observed inverse relationship between IL10 levels and carotid intima-media thickness, suggesting that reduced IL10 levels may contribute to endothelial dysfunction and early atherosclerotic changes in hypertensive patients.⁴¹⁻⁴²

CRP was significantly higher in hypertensive study groups compared with healthy controls,

which may indicate chronic subclinical inflammation of the vascular wall.⁴³ This finding is consistent with a previous study demonstrating an association between elevated CRP levels and hypertension, reflecting underlying low-grade systemic inflammation. Also, regression analysis in prior studies has revealed an independent positive association between elevated CRP and the risk of hypertension.⁴⁴ Regarding neutrophil count in this work, we found a significant rise of absolute neutrophil count in hypertensives compared to controls. Notably, this was more pronounced in untreated hypertensive individuals than in both control group and those with controlled hypertension. This was closely related to the increased severity of hypertension between the study group. Our results agreed with Tatsukawa et al.,⁴⁵ who found that an elevated WBCs count, especially ANC, was significantly associated with an increased risk of developing hypertension among Japanese people. Previous studies highlighted the role of neutrophils in oxidative stress. They recruit other inflammatory cells by proinflammatory cytokines, which, in turn, cause vascular damage and further complications.⁴⁶

Regarding ECHO parameters, this study showed that healthy controls had the lowest values of LVEDV, as anticipated, and it was highest among controlled and mild hypertension groups. Although patients were controlled at the time of study yet, this complication is partially reversible only after long term treatment of the disease.

It was now well established that hypertension is a crucial risk factor for atherosclerotic cardiovascular diseases.⁴⁷ Endothelial dysfunction and vascular inflammation are now recognized as central mechanisms in the development of hypertension mediated cardiovascular complications. Carotid intima-media Thickness (cIMT) is widely used as a marker of subclinical atherosclerosis and has been associated with endothelial injury and future cardiovascular event. Previous studies have shown that arterial intima-media thickness (IMT) is a marker of subclinical atherosclerosis, endothelial dysfunction, and cardiovascular adverse

events.⁴⁸⁻⁴⁹ This study showed that hypertensive patients had significantly higher RTCCA IMT values than controls. Moreover, patients with severe HTN had the highest mean RTCCA IMT, while the healthy control group had the lowest ($p < 0.001$). This is consistent with previous studies demonstrating significantly increased carotid IMT in hypertensive patients compared to normotensive controls. This matches the expected progression of HTN and its complications on the vessel wall.^{50,51}

Regarding fundoscopy results, 45% of patients with severe hypertension in this work showed abnormal fundoscopic examination, while all individuals in the control group had normal fundoscopy. Our results are consistent with these previous studies showing that retinal abnormalities become more frequent with increasing hypertension severity and that hypertensive retinopathy reflects hypertension mediated target organ damage.⁵²⁻⁵⁴ Our study also showed no significant correlation between IgG, IgM, IL10, and ECHO parameters or fundoscopy ($p > 0.05$).

In Conclusion, our study showed that IL10, which is a potent anti-inflammatory cytokine, was significantly lower in hypertensive patients than the normal control participants, while serum IgG and IgM were comparable among study groups. This provides new insights to study IL10 as a target for therapy in future to prevent and/or treat hypertension and its complications. And finally, future research is needed to unravel the mysteries of immunological implications in hypertension.

Author Contributions

All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by ZAA, MAYA, HEA, EAAK, and STK. The first draft of the manuscript was written by AMSA, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Declaration of Conflicting Interests

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Ethical approval

The work was performed with approval from the Ethics Committee of Ain Shams University Hospitals, Cairo, Egypt (approval code: FMASU MD2942018).

Informed consent

Informed written consent was obtained from all patients.

References

1. Abughazaleh S, Obeidat O, Tarawneh M, et al. (2024). Trends of hypertensive heart disease prevalence and mortality in the United States between 1990–2019. *Curr Probl Cardiol*, 49: 102621.
2. Yang, X., Chen, Y., Zhang, Y et al., 2020. Elevated Blood Pressure in Childhood or Adolescence and Cardiovascular Outcomes in Adulthood: A Systematic Review. *Hypertension*, 75(4), 948-955.
3. Caillon A, Schiffrin EL. (2016). Role of inflammation and immunity in hypertension. *Curr Hypertens Rep*, 18: 21.
4. Rodriguez-Iturbe B, Pons H, Johnson RJ. (2017). Role of the immune system in hypertension. *Physiol Rev*, 97: 1127–1164.
5. Norlander AE, Madhur MS, Harrison DG. (2018). The immunology of hypertension. *J Exp Med*, 215: 21–33.
6. McMaster WG, Kirabo A, Madhur MS, et al. (2015). Inflammation, immunity, and hypertensive end-organ damage. *Circ Res*, 116: 1022–1033.
7. Janeway C, Travers P, Walport M, et al. (2001). Immunobiology: the immune system in health and disease. *Garland*, New York.
8. Vignali DA, Collison LW, Workman CJ. (2008). How regulatory T cells work. *Nat Rev Immunol*, 8: 523–532.
9. Schiffrin EL. (2014). Immune mechanisms in hypertension and vascular injury. *Clin Sci (Lond)*, 126: 267–274.
10. Idris-Khodja N, Mian MO, Paradis P, et al. (2014). Adaptive immunity in hypertension. *Eur Heart J*, 35: 1238–1244.
11. De Ciuceis C, Rossini C, Airò P, et al. (2017). CD4+ T-lymphocytes and microvascular alterations. *Am J Hypertens*, 30: 51–60.
12. Gupta P, Sághy T, Bollmann M, et al. (2024). Immune activation and humoral immunity. *Vaccines*, 12: 479–489.
13. Matsuura E, Kobayashi K, Inoue K, et al. (2005). Immunoglobulin and atherosclerosis. *Clin Rev Allergy Immunol*, 29: 311–319.
14. Tsiantoulas D, Diehl CJ, Witztum JL, et al. (2014). B cells in atherosclerosis. *Circ Res*, 114: 1743–1756.
15. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J*. 1;39(33):3021-3104.
16. Brittin GM, Brecher G, Johnson CA, et al. (1969). Stability of blood in anticoagulants. *Am J Clin Pathol*, 52: 690–694.
17. Burris C, Ashwood E, Burns D 2006 Tietz Textbook of clinical chemistry and Molecular Diagnostics. 4th ed. St. Louis: Elsevier Saunders; 1633: 962-967.
18. Singer JM, Plotz CM, Pader E, et al. (1957). Latex-fixation test for CRP. *Am J Clin Pathol*, 28: (6)611–617.
19. Bowers LD, Wong ET. (1980). Serum creatinine assays. *Clin Chem*, 26: 555–561.
20. Cobas c311 analyzer, operator's manual. Hitachi, Roche Diagnostics international
21. Karl J. (1993). HbA1c immunoturbidimetric assay. *Klin Lab*, 39: 991–996.
22. Cockcroft DW, Gault MH. (1976). Creatinine clearance prediction. *Nephron*, 16(1): 31–41.
23. Modin D, Biering-Sørensen SR, Mogelvang R, et al. (2018). Echocardiography in hypertension. *Hypertension*, 71: 742–751.
24. Cooke S, Nyeche E, Wariboko C. (2022). Carotid duplex in hypertension. *TNHJ*, 22: 190–207.
25. Wong TY, Mitchell P. (2004). Hypertensive retinopathy. *N Engl J Med*, 351: 2310–2317.
26. Gallo A, Diätenbeck T, Giron A, et al. (2021). Retinal vascular remodeling. *Clin Res Cardiol*, 110: 959–970.
27. Tomaszewski M, Zukowska-Szczechowska E, Grzeszczak W. et al (2000). Evaluation of complement component C4 concentration and immunoglobulins IgA, IgG, and IgM in serum of patients with primary essential hypertension. *Pol Arch Med Wewn*, 103(5-6):247-51
28. Chen Y, Dale BL, Alexander MR, et al. (2021). Class switching and high affinity immunoglobulin

- G production by B cells is dispensable for the development of hypertension in mice. *Cardiovasc Res*, 117: 1217–1228.
29. Wang X, Li Y, Li H, et al. (2018). Immunoglobulin levels and hypertension. *J Hum Hypertens*, 32: 212–218.
 30. Kristensen BO. (1978). Immunoglobulins in hypertension. *Acta Med Scand*, 203: 49–54.
 31. Suryaprabha P, Padma T, Rao UB. (1984). IgG levels in hypertension. *Immunol Lett*, 8: 143–145.
 32. Hilme E, Herlitz H, Söderström T, et al. (1989). Immunoglobulin secretion in hypertension. *J Hypertens*, 7: 91–95.
 33. Meng X, Gao X, Yu X, et al. (2021). Glycosylation of immunoglobulin G associates with hypertension in Chinese Muslim ethnic minorities and the Han Chinese. *J Pers Med*, 11: 614.
 34. Kifer D, Novokmet M, Štambuk J, et al. (2021). N-glycosylation of immunoglobulin G predicts incident hypertension. *J Hypertens*, 39: 1–10.
 35. Mohebbati M, Feiz Disfani M, et al. (2014). An imbalance in serum concentrations of inflammatory and anti-inflammatory cytokines in hypertension. *J Am Soc Hypertens*. ;8(9):614–623.
 36. Odewusi O, Osadolor H. (2019). Interleukin-10 levels in hypertension. *J Appl Sci Environ Manage*, 23: 819–824.
 37. Timasheva YR, Nasibullin TR, Imaeva EB, et al. (2015). Inflammatory polymorphisms in hypertension. *Clin Exp Hypertens*, 37: 398–403.
 38. Fu M, Lv M, Guo J, et al. (2025). T-cell regulation in hypertension. *Front Immunol*, 16: 1550206.
 39. Iyer SS, Cheng G. (2012). IL-10 regulation. *Crit Rev Immunol*, 32: 23–63.
 40. Orlova N, Goryaynova S, Khachirova A, et al. (2021). IL-6 and IL-10 in hypertension. *J Hypertens*, 39.
 41. Wang M, Zhou S, Hu Y, et al. (2025). IL-10 and atherosclerosis. *Bioeng Transl Med*, 10: e10717.
 42. Volpatti LR, de Matos SN, Borjas G, et al. (2024). LDL-binding interleukin-10 reduces vascular inflammation in atherosclerotic mice. *bioRxiv*.
 43. Jayedi A, Rahimi K, Bautista LE, et al. (2019). Inflammation markers and risk of developing hypertension: a meta-analysis of cohort studies. *Heart*; 105(9):686–69.
 44. Sesso HD, Buring JE, Rifai N, et al. (2003). C-reactive protein and the risk of developing hypertension. *JAMA*, 290: 2945–2951.
 45. Tatsukawa Y, Wan-Ling Hsu, Michiko Yamada, et al. (2008). White blood cell count, especially neutrophil count, as a predictor of hypertension in a Japanese population. *Hypertension Research*; 31.7: 1391–1397.
 46. Araos P, Figueroa S, Amador CA. (2020). Neutrophils in hypertension. *Int J Mol Sci*, 21.
 47. Korkmaz L, Bektas H, Korkmaz AA, et al. (2012). CIMT and SYNTAX score. *Angiology*, 63: 386–391.
 48. Rios FJ, de Ciuceis C, Georgiopoulos G, et al (2024). Mechanisms of vascular inflammation and potential therapeutic targets: a position paper from the ESH Working Group on Small Arteries. *Hypertension*.; 81(6):1218–1232
 49. Maruhashi T, Higashi Y. et al (2024) Current topic of vascular function in hypertension in 2023–2024. *Hypertens Res*.;47(12):3310–3317.
 50. Mustafa R, Gameraddin M, Gareeballah A, et al. (2020). CIMT in hypertensive patients. *J Curr Res Sci Med*, 6: 84–88.
 51. Naseh G, Fard MM, Kazemi T, et al (2016) A. Comparison of carotid intima-media thickness in hypertensive patients and control group. *J Cardiovasc Echogr*.; 26(2):48–51.
 52. Figueiredo Neto JA, Silva AR, et al (2010). Relationship between the classification of arterial hypertension and funduscopy findings in primary hypertension. *Arq Bras Cardiol*. ;95(2):215–222
 53. Tripathy K, Arsiwalla T, Modi P et al. (2025). Hypertensive retinopathy. *StatPearls*. Updated 2025.
 54. Liew G, Xie J, Nguyen H, et al (2023). Hypertensive retinopathy and cardiovascular disease risk: 6 population-based cohorts meta-analysis. *Int J Cardiol Cardiovasc Risk Prev*.;17:200180.