Journal of Pioneering Medical Sciences
Received: March 24, 2026 | Accepted: May 01, 2026 | Published: June 05, 2026
Volume 15, Issue 05, Pages 11-15

DOI https://doi.org/10.47310/jpms2026150502

Comparative Analysis of the Levels of Matrix Metalloproteinase 9, Renal and Lipid Profile between Hypertensive and Normotensive Individuals in South Indian Population

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Abstract: Background/Aim: Hypertension is a prevalent chronic medical condition where the force of blood against the artery walls is high exceeding 130/80 mmHg. The vessel wall has collagen and matrix content in it. Thus the MMPs plays a role in the regulation and remodelling of the vessel wall architecture. The early stage of hypertension is regulated by MMP 9 leading to arterial stiffness and debilitation. The current research is delving into the complex interaction between MMP-9 and various inflammatory markers to uncover the fundamental mechanisms behind hypertension and discover novel avenues for more efficient treatment strategies. This study seeks to assess the levels of plasma MMP-9, serum lipid, liver and renal profile in individuals with primary hypertension, aiming to explore potential associations between these biomarkers. **Methods:** Fifty hypertensive individuals aged between 40 to 60 years from rural areas from south Indian population were included in this study. A control group comprised of twenty-five normotensive individuals matched for age and gender was included in the study. Under aseptic conditions, 5 ml of venous blood samples were collected in three different test tubes (EDTA, oxalate/citrate, and plain tubes), while urine samples were obtained in a fasting state. List of parameters analysed includes complete hematogram, Plasma Sugar, serumUrea and creatinine, serum Uric acid, serum Lipid profile, liver function test -LFT (SGOT, SGPT), Serum sodium, potassium, chloride, Serum MMP9 and Urinary excretion of albumin. **Results:** SPSS15 software has been used for Statistical analysis. There was significantly higher level of total cholesterol, TGL, LDL-C in hypertensive subjects compared to control. There was no significant change in serum HDL-C. The levels of serum MMP 9 was significantly higher in hypertensive subjects compared to controls. Urine albumin was within the normal range. **Conclusion:** Thus our study concludes that the MMP 9 level is elevated in hypertensive individuals and is also associated with other inflammatory markers like lipids, urea, creatinine and blood glucose levels.

Key Words: Health, Disease, cardiovascular disease, Primary hypertension, LFT, MMP9

INTRODUCTION

Hypertension, a prevalent chronic medical condition affecting a substantial proportion of the global populace, is characterized by elevated levels of blood pressure [1]. The primary (essential) and secondary types are the main classifications of hypertension, with primary hypertension representing the majority of cases. In contrast, secondary hypertension is linked to identifiable factors such as chronic kidney disease or endocrine disorders. Various factors including genetic predisposition, lifestyle preferences, and environmental factors influence the development of hypertension [2]. Lifestyle adjustments like weight control, physical activity, and dietary changes are pivotal in the prevention and treatment of hypertension. Complications arising from hypertension can result in severe health consequences like atherosclerosis, coronary artery disease, stroke, and heart failure [3]. Matrix metalloproteinase is a group of zinc dependent endopeptidases. It consists of 23 members and are classified as either secreted types or membrane bound MMPs. Among the secreted group, the

Among the secreted group, the gelatine binding MMPs includes MMP 2 and MMP 9. The MMPs plays a crucial role during both various physiological and pathological process. The vessel wall has collagen and matrix content in it. Thus the MMPs plays a role in the regulation and remodelling of the vessel wall architecture. The early stage of hypertension is regulated by MMP 9 leading to arterial stiffness and debilitation [4].

The involvement of matrix metalloproteinases, especially MMP-9, in the pathophysiology of hypertension impacts vascular restructuring and the formation of atherosclerotic plaques [5]. A comprehensive understanding of the mechanisms underpinning hypertension and its complications is vital for crafting effective management approaches. The various risk factors associated with hypertension includes hyperlipidemia, uric acid, urea creatinine, serum albumin and serum electrolyte levels. Any variations in the levels of these inflammatory markers might influence the cardiovascular pathogenesis [6]. By elucidating the role of MMP-9 in hypertension, this research contributes to enhancing the current comprehension of the pathophysiology of this prevalent ailment. The accumulation of LDL cholesterol in the arteries can lead to a narrowing and hardening of the vascular pathway, increasing the resistance to blood flow and subsequently raising blood pressure. Similarly, higher triglyceride levels can have detrimental effects on the endothelium, the lining of blood vessels, and other regulatory mechanisms that control blood pressure. The renal profile and dyslipidemia are an important risk factor for the cardiovascular morbidity. Additionally, current research is delving into the complex interaction between MMP-9 and various inflammatory markers to uncover the fundamental mechanisms behind hypertension and discover novel avenues for more efficient treatment strategies. This study seeks to assess the levels of plasma MMP-9, serum lipid, liver and renal profile in individuals with primary hypertension, aiming to explore potential associations between these biomarkers.

METHODS

Fifty hypertensive individuals aged between 40 to 60 years from rural areas attending the hypertensive outpatient department and medical ward of Government medical college were recruited for the study. The diagnosis of primary hypertension was established by a general practitioner. Patients diagnosed clinically with essential hypertension (under treatment) and having blood pressure readings exceeding 140/90 were considered eligible for the study. Patients with primary hypertension and under treatment for it for minimum of six months were included in the study. Electrocardiograms were conducted to exclude cardiac ischemia. Hypertensive patients with comorbidities such as diabetes, renal or liver diseases, familial hyperlipoproteinemias, endocrine disorders, smoking, alcoholism, and chronic inflammatory conditions like rheumatoid arthritis were excluded. A control group comprised of twenty-five normotensive individuals matched for age and gender was included in the study. Standardised procedures were followed, providing a calm and ambient setting with the individual seated, arm outstretched and supported, for blood pressure measurement using a manual cuffed sphygmomanometer while auscultating the Korotkoff sounds in the artery.

According to NICE guidelines, individuals were diagnosed with hypertension based on the average of two or more blood pressure readings taken on at least two subsequent occasions, with a gap of one to three weeks following initial screening. The blood pressure readings were measured by two experienced general physicians and if the inter examiner variability exceeded >5mm Hg value, then another physician will examine it.

Blood Collection

Under aseptic conditions, 5 ml of venous blood samples were collected in three different test tubes (EDTA, oxalate/citrate, and plain tubes), while urine samples were obtained in a fasting state. Serum/plasma was separated by centrifugation at 3000rpm at room temperature, and serum was stored at -70 degrees Celsius until analysis. Baseline investigations and special parameters such as MMP-9 was analysed. Baseline investigations were conducted using an autoanalyzer with standard kits, while serum levels of MMP-9 was measured using ELISA. Urine albumin excretion was quantitatively determined by a turbidometric method.

List of parameters analysed includes complete hematogram,Plasma Sugar, serumUrea and creatinine, serum Uric acid ,serum Lipid profile, liver function test - LFT(SGOT,SGPT), Serum sodium , potassium , chloride, Serum MMP9 and Urinary excretion of albumin as per the previous established protocol.

Estimation of Matrix Metalloproteinase-9 (Mmp-9)

MMP-9 in plasma was estimated by using ELISA kit (Booster biological technology co., Ltd., USA)

Procedure

One hundred µl standard or sample, was added to each well and incubated at 37°c for 90 minutes. Then removed the contents from each well and added 100µl of biotinylated anti –human MMP-9 antibody working solution into each well and incubated the plate at 37°C for 60 min , removed the contents and washed the each well 3 times with 300 µl TBS (0.01M) and each time washing buffer stay in the wells for 1 min. Then added 100µl ABC (Avidin-Biotin-Peroxidase complex) working solution and incubated the plate at 37°C for 30 min, and washed the wells 5 times with 300 µl TBS each well. Then added 90µl TMB colour developing reagent and incubated at 37°C for 25-30 min in dark and reaction was stopped by adding 100µl TMB stop solution to each well. Then it was read at 450 nm within 30 minutes and calculated the MMP-9 concentration in the samples by plotting graph with corresponding absorbencies of standards. The plasma values were expressed as ng/ml.

RESULTS

SPSS15 software has been used for Statistical analysis. To compare the hypertensives and controls, Man Whitney U test has been applied. To find out the relationship between physiological and biochemical parameters with MMP-9, Pearson correlation coefficient has been applied. The level of significance has been fixed as 5% ($p<0.05$).

Systolic and diastolic increased in hypertensives than control. Body Mass Index showed no significant difference between hypertensives and controls. Pulse rate within normal.

It shows the mean value of WBC, RBC, Hb, platelet count, hematocrit, lymphocyte and neutrophils in hypertension and control. Level of WBC significantly increased in hypertension subjects than the control. Level of RBC significantly decreased in hypertensives than the controls. There was no significant difference of Hb, platelet count, Hematocrit, lymphocyte count neutrophil count between hypertensive subjects and control subjects.

It shows the value of plasma glucose, serum urea, serum creatinine, serum uric acid and serum electrolytes (sodium, potassium, chloride) in hypertensive subjects and control. Serum uric acid and chloride slightly higher in hypertensive subjects than controls. There was no significant difference in blood sugar, urea, creatinine sodium and potassium between hypertensive patients and controls.

Table 4 shows the value of Lipid profile in hypertensives and control subjects. There was significantly higher level of total cholesterol, TGL, LDL-C in hypertensive subjects compared to control. There was no significant change in serum HDL-C (Table 5).

Table 1: Comparison of Physiological Parameters in Hypertensives and Control Subjects

Physiological parameters	Hypertension (N-50)	Control (N-25)	Mann whitny U-test –z value	p-value
AGE (Years)	52.34±6.26	50.68±3.95	1.91	N.S
BMI (Kg/m^2)	24.7±3.53	24.57±3.2	0.87	N.S
BP(systolic)(mmHg)	148.08±16.04	115.96±7.68	6.54	< 0.001
BP (Diastolic) (mmHg)	89.84±9.20	74.48±6.46	6.18	< 0.001
pulse rate/min	81.84±8.44	83±6.59	0.53	N.S

Table 2: Hematological Parameters in Hypertensives and Control Subjects

Hematological parameters	Hypertension (N-50)	Control (N-25)	Mann whitny U-test- z value	p-value
WBC (103cells/µl)	8.53±2.87	7.41±1.02	3.23	0.001
RBC (106ells/µl)	4.40±0.44	4.79±0.54	2.87	0.004
Hb g/dl	11.90±2.09	13.18±1.97	2.53	N.S
Platlets (105cells/µl)	266.08±86.73	260.92±63.16	0.18	N.S
HCT %	34.95±3.81	36.83±3.29	2.37	N.S
LYMPH %	36.46±12.22	36.91±7.31	1.24	N.S
NEUTRO %	56.44±11.97	56.50±7.83	0.55	N.S
MIXED %	7.09±3.59	6.33±2.41	0.53	N.S

Table 3: Biochemical Parameters in Hypertensives and Control Subjects

Biochemical parameters		Hypertension (N-50)	Control (N-25)	Mann whitney U-test- z value	p-value
Plasma Glucose mg/dl		94.94±31.14	100.8±20.79	1.83	N.S
Serum Urea (mg/dl)		30.22±9.49	28.36±4.04	0.9	N.S
Serum Creatinine (mg/dl)		0.83±0.11	0.81±0.11	0.33	N.S
Serum Uric Acid mg/dl		4.38±0.79	4.04±0.67	2.14	0.03
Serum Electrolytes mmol/l	Na	138.64±2.37	138.16±3.10	0.12	N.S
	K	4.17±0.31	4.02±0.34	1.58	NS
	Cl	100.69±3.37	98.08±3.13	2.80	0.005

Table 4: Lipid Parameters in Hypertensives and Control Subjects

Serum Lipid parameters	Hypertension (N-50)	Control (N-25)	Man whitny U-test- z value	p-value
Total.Cholesterol(mg/dl)	197.24±37.52	173.16±20.18	2.74	0.006
TGL (mg/dl)	162.1±66.29	111.2±39.73	3.58	0.001
HDL-C (mg/dl)	42.08±2.99	44.08±5.52	1.17	N.S
LDL –C(mg/dl)	121.96± 38.32	96.12±14.76	2.87	0.004

Table 5: Liver Function Testsparameters in Hypertensives and Control Subjects

Liver function parameters	Hypertension (N-50)	Control (N-25)	Man whitny U-test- z value	p-value
SGOT(IU/l)	24.1±8.48	23.52±6.35	0.53	N.S
SGPT(IU/l)	22.4±9.09	20.16±4.17	0.17	N.S
Total bilirubin(mg/dl)	0.77±0.06	0.8±0.11	1.19	N.S
Direct bilirubin(mg/dl)	0.18±0.04	0.17±0.06	1.23	N.S

Table 6: Serum MMP-9, hsCRP and Urinary Albumin in Study Subjects

Parameters	Hypertension (N-50)	Control (N-25)	Man whitny U-test- z value	p-value
MMP-9(ng/ml)	16.02 ±4.51	10.14 ±4.33	4.59	<0.001
Albumin excretion(mg/l)	22.88±9.88	15.84±6.60	2.52	0.012

Table 6 shows the average value of MMP-9 and urine albumin in hypertensive and controls. There was significantly raised serum MMP-9 in hypertensive subjects in comparison with controls. Urine albumin was within normal range.

DISCUSSION

Hypertension represents a significant risk factor for the onset of coronary heart disease and stroke, with the potential of inducing cardiac enlargement and consequent heart failure [1]. Various lifestyle elements, such as excessive salt consumption, obesity, smoking, and alcohol intake, have been identified as factors that elevate the likelihood of developing hypertension. The relationship between obesity and hypertension has been acknowledged in the scientific literature [7]. Notably, our research revealed no substantial disparity in BMI across the cohorts. The findings underscored that BMI did not exhibit a significant variance among the groups, hinting at other factors exerting a more pronounced impact on hypertension within the study cohort. The discourse section of the paper thoroughly examines the consequences and interpretations of the study outcomes concerning the existing knowledge domain. The investigation primarily concentrated on the repercussions of hypertension on coronary heart disease, stroke, cardiac hypertrophy, heart failure, as well as the associated risk determinants like excessive salt ingestion, obesity, smoking, and alcohol usage. Moreover, the study accentuated the pivotal role of lifestyle adjustments and medication in the management of hypertension and the mitigation of health hazards. This discovery is consistent with prior research highlighting the dominant contribution of non-specific lifestyle and genetic influences to hypertension.

The WBC count within hypertensive individuals fell within the normal range; however, it was notably elevated compared to the controls, possibly indicating a state of low-grade inflammation in hypertensive subjects [8]. On the other hand, the RBC count exhibited a significant difference from the controls but remained within the normal parameters, suggesting a potential decrease in erythropoiesis due to an inflammatory cascade [9]. Blood viscosity plays a critical role in peripheral resistance to blood flow, consequently influencing blood pressure dynamics [10]. Nonetheless, the hematocrit levels, serving as a determinant of whole blood viscosity, did not show any significant distinctions among hypertensive individuals.

Platelets are crucial actors in the progression of atherosclerotic plaques, plaque destabilisation, and atherothrombosis, with mean platelet volume serving as an established indicator of platelet activation and its correlation with cardiovascular ailments being well-documented [11]. Contrary to expectations, our investigation did not unveil any notable discrepancies in platelet count across the various cohorts.

In our study also serum total cholesterol and triglycerides levels were significantly increased in hypertensive subjects in comparison with control subjects. No significant change in HDL-C was observed in our study. Serum LDL-C values were significantly increased in hypertensive subjects in comparison with the healthy control subjects as reported by previous studies [12,13]. It has been recently reported that Xuezhikang (a cholestin extract containing statin-like components) significantly reduced the arterial stiffness parameters, low-density lipoprotein cholesterol, hs-CRP, and MMP-9 compared to baseline. However, pulse wave velocity changes were significantly associated with the changes of hs-CRP and MMP-9 but not with lipid profile changes [14].

Statin therapy significantly decreases plasma TIMP-1 levels. Random-effects meta-regression analysis indicated that neither treatment duration nor changes in low-density lipoprotein cholesterol levels are associated with changes in plasma MMP-9 levels following statin therapy. The results of the meta-analysis suggested a significant reduction in plasma concentrations of TIMP-1, but not MMP-9 and MMP-3, following statin therapy [15].

Matrix metalloproteinases are implicated in the pathology of vascular diseases [16]. We observed that serum MMP-9 level was significantly higher in patients with hypertension than in controls, which is consistent with previous studies [17]. This elevation could be due to the inflammatory process and vascular remodelling. Increased circulating levels of MMP-9 are associated with an increased incidence of recurrent cardiovascular events in the long term. MMP-9 plays an important role in extracellular matrix remodelling during all phases of atherosclerosis. It also promotes the local destruction of ECM in atheroma, leading to plaque destabilisation and rupture [18]. Studies have shown that various antihypertensive medications reduce serum MMP-9 levels in hypertensive subjects through various mechanisms [19,20].

Moreover, heightened blood pressure is also recognised for stimulating the production of MMP-9. Inflammation reduces endothelial relaxation, potentially due to a decreased ability of the endothelium to produce vasodilatory agents, especially nitric oxide (NO), which consequently elevates blood pressure. Serum levels of MMP-9 are associated with C-reactive protein, interleukin-6, and fibrinogen levels, serving as a diagnostic indicator for individuals at risk of future myocardial infarction [21,22,23,24].

A significant relationship was also noted between the levels of MMP-9, and systolic blood pressure in the present investigation. Additionally, MMP-9 has been linked to the breakdown of extracellular matrix proteins, resulting in vascular restructuring and heightened arterial rigidity, which are crucial elements contributing to hypertension and the advancement of atherosclerosis. Recent studies have emphasised the role of MMP-9 in fostering inflammation within atherosclerotic plaques, intensifying the progression of cardiovascular disease by destabilising these lesions and increasing the chances of plaque rupture. These discoveries emphasise the diverse impact of MMP-9 on cardiovascular well-being, highlighting the necessity for targeted therapeutic strategies to alleviate its adverse effects on vascular function and disease advancement.One promising method to counteract the harmful effects of MMP-9 on cardiovascular health involves the development of specific

inhibitors that can selectively target and inhibit the function of this protease within atherosclerotic plaques. These inhibitors have displayed encouraging outcomes in preclinical trials, showcasing their potential to decrease plaque inflammation and enhance overall vascular stability in CAD patients. Additionally, recent clinical trials have revealed that the use of MMP-9 inhibitors not only diminishes plaque vulnerability but also correlates with improved cardiovascular results and decreased risk of adverse events in patients with coronary artery disease.

CONCLUSION

Thus our study concludes that the MMP 9 level is elevated in hypertensive individuals and is also associated with other inflammatory markers like lipids, urea, creatinine and blood glucose levels. Further studies required in analysing the molecular and genetic regulation of these markers in the etiopathogenesis of hypertension.

Funding

The authors declare no source of funding for the present research paper.

Conflict of Interests

The authors declare that there is no conflict of interest in relation to the present research paper.

Ethics Statement

The study was approved by the institutional ethics committee of Government medical college, Cuddalore (0125/IHEC-RMMCHBio/2019).

Consent

The study participants were all informed about the study and informed consent obtained from all the subjects.

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