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Original Research Article.

Evaluation of Mannose Binding Lectin Levels in Hypertensive Patients of Tertiary Care Teaching Hospital in Central India

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ABSTRACT

Background: The present study was conducted for evaluating Mannose Binding Lectin levels in hypertensive patients.

Materials & Methods: The study included 100 hypertension cases and 100 controls who met the inclusion requirements. All subjects had 5 mL of blood drawn into serum tubes after an overnight fast. After letting the blood clot for 15 minutes at 3000 RPM, the serum was centrifuged out. For the mannose binding lectin test, 0.5 mL of serum had to be stored at - 20°C. ELISA technique was used for evaluating the serum mannose binding lectin levels. All the results were recorded on a Microsoft excel sheet followed by statistical analysis using SPSS software.

Results: The mean Mannose Binding Lectin (MBL) in Cases was more (912.56 ± 43.51) as compared to Controls (612.18 ± 21.43) shows statistically significant. (By Un-paired T test; p>0.05). The above table shows the association of type (NYHA) of hypertension and MBL among cases. The mean MBL in Stage II was more (968.39 ± 46.41) as compared to Stage I (856.13 ± 40.56) shows statistically significant. (By Un-paired T test; p>0.05)

Conclusion: The present study concludes that, high MBL levels are associated with an increased risk of coronary artery disease and are high in the serum prior to the development of hypertensive symptoms.

Key words: Mannose Binding lectin, Hypertension.

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INTRODUCTION

According to the Joint National Committee (JNC) 8 hypertensive recommendations, "hypertension is defined as blood pressure that is greater than 140 mmHg or 90 mmHg at the diastolic level".[1] Primary, Essential, or Idiopathic hypertension refers to cases of arterial hypertension in which the underlying cause cannot be identified. Arterial pressure is controlled by a variety of systems, including the adrenergic system (both peripheral and central), the kidneys, hormones, and blood vessels. Multiple genes contribute to the intricate integration of these systems. Patients with essential hypertension have had a number of abnormalities described, with some of these being held responsible for the majority of the hypertension.[1]

Heart and blood vessel disorders such as atherosclerosis, arteriosclerosis, coronary artery disease, heart failure, stroke, and peripheral artery disease are together referred to as cardiovascular disease (CVD). According to the World Health Organisation, approximately 17.9 million deaths annually are caused by cardiovascular disease. (WHO, 2018) Genetics, bad lifestyle choices like smoking and eating, and underlying medical disorders like high blood pressure, diabetes, and cholesterol are just a few of the many possible causes of heart disease. Along with genetics, other CVD risk factors include age, gender, and family history. The high incidence of cardiovascular disease and cardiac deaths in India is largely attributable to the widespread use of risk factors such as hypertension, diabetes, tobacco use, and poor dietary habits. Overall, the burden of cardiac deaths in India is high, necessitating immediate action from policymakers and healthcare providers to develop and implement efficient methods of detecting and treating cardiovascular disease.[2,3]

A 2018 study published in the Indian Heart Journal found that 28% of all deaths in India are caused by cardiovascular disease (CVD), with an estimated age-standardized prevalence of 272 deaths per 100,000 people in India.[4]

The innate immune system contains a protein called mannose-binding lectin (MBL). MBL has opsonic activity and may activate complement via the lectin route in conjunction with MBL-

associated serine proteases (MASPs). MBL may contribute to the formation of atherosclerotic plaques as a result of its association with innate immunity and atherogenesis.[5,6]

Mannan-binding lectin is created by "hepatocytes and released into the bloodstream as an oligomeric form. The matching mRNA has been found in the bone marrow, foetal lung, small intestine, and testes". MBL2, a gene on chromosome 10 (10q11.2), regulates its production in a fashion analogous to that of acute phase proteins. Serum MBL levels typically range from 400 ng / mL to 800 ng / mL. MBL deficiency is defined as serum levels of less than 100 ng/mL.[7] Hence; the present study was conducted with the aim of evaluating Mannose Binding Lectin levels in hypertensive patients.

MATERIALS AND METHODS

The present study was conducted in the department of biochemistry and medicine of tertiary care centre of central India with the aim of evaluating Mannose Binding Lectin levels in hypertensive patients.

Complete demographic and clinical details of all the patients were obtained. Simple random sampling was used as a sampling method. Every day, searches for admissions of hypertension cases in hospital wards and intensive care units were made. The study included patients who had been given the diagnosis of hypertension by a doctor. One control was chosen for each scenario. The age and sex of the patients and controls were matched by ±5 year's age class interval. The study included 100 hypertension cases and 100 controls who met the inclusion requirements. All subjects had 5 mL of blood drawn into serum tubes after an overnight fast. After letting the blood clot for 15 minutes at 3000 RPM, the serum was centrifuged out. For the mannose binding lectin test, 0.5 mL of serum had to be stored at -20°C. ELISA technique was used for evaluating the serum mannose binding lectin levels. All the results were recorded on a Microsoft excel sheet followed by statistical analysis using SPSS software.

RESULTS

The mean age in Cases was 51.13 ±10.92 years and Controls was 52.38 ±11.48 years. There were 63 (63%) and 62 (62%) male patients among Cases and Controls respectively. It was observed that majority of Cases were with hypertension for 1-5 years (46%) followed by 6-10 years (36%). It was observed that majority of Cases were with Stage I hypertension (54%) followed by Stage II hypertension (46%). The mean Mannose Binding Lectin (MBL) in Cases was more (912.56 ± 43.51) as compared to Controls (612.18 ± 21.43) shows statistically significant. (By Un-paired T test; p>0.05). The above table shows the association of type (NYHA) of hypertension and MBL among cases. The mean MBL in Stage II was more (968.39 ± 46.41) as compared to Stage I (856.13 ± 40.56) shows statistically significant. (By Un-paired T test; p>0.05)

Table 1: Distribution according to type (NYHA) of hypertension among cases:

Type of hypertension	No of patients (N=100)	Percentage
Stage I	54	54.00
Stage II	46	46.00
Total	100	100

Table 2: Mean Mannose Binding Lectin (MBL) among two groups:

MBL	Cases	Controls	P value
Mean	912.56 ± 43.51	612.18 ± 21.43	<0.0001 (S)

(P<0.05 statistically significant)

Table 3: Association of type (NYHA) of hypertension and MBL among cases:

Type of hypertension	MBL levels (Mean ±SD)	P value
Stage I	856.13 ± 40.56	<0.0001 (S)
Stage II	968.39 ± 46.41	

DISCUSSION

Hypertension is both a disease and a major risk factor for other diseases. Population studies show an increasing rate of cardiovascular events such as stroke, myocardial infarction, heart failure, atrial fibrillation and premature mortality, with increasing blood pressure (from systolic blood pressures ≥115 mmHg). This relationship is exponential, and stronger for systolic pressure than for diastolic pressure. Untreated very high (>180/110 mmHg) or rapidly rising blood pressure (such as in eclampsia) can overcome normal microvascular autoregulation. This leads to acute damage in the microcirculation and results in a multisystem clinical syndrome of accelerated or malignant hypertension, or cerebral haemorrhage, which are immediate threats to life.[1] Accelerated or malignant hypertension is now fortunately uncommon. The main consideration in the majority of individuals is the relationship between their blood pressure and subsequent risk of cardiovascular disease.[7-10] The mannose-binding lectin (MBL) is a protein of the innate immune system, belonging to the collectin family, able to deploy a variety of antimicrobial activities. It recognizes and binds various pathogens (including bacteria, viruses, fungi, and parasites), providing protection against the microbial invasion of the host. Although the clinical impact of MBL deficiency and its association to a wide variety of diseases has been extensively studied, the clinical significance of low MBL serum levels in healthy subjects is still debated.[11,12]

In the study, the mean age in Cases was 51.13 ±10.92 years and Controls was 52.38 ±11.48 years with not statistically significant in age distribution in two groups. (By Un-paired T test; p>0.05) Tymen T. Keller et al in "a study on relationship between mannose-binding lectin (MBL) serum levels and the probability of

developing coronary artery disease (CAD). observed mean age among cases was 64 ±8 and among controls was 65 ±8 with no significant difference".[13] More men than women were included in the study by Tymen T. Keller et al "which looked relationship between mannose-binding lectin (MBL) serum levels and the probability of developing coronary artery disease (CAD). This result was consistent with the current investigation".[13] Tymen T. Keller et al in "a study on relationship between mannose-binding lectin (MBL) serum levels and the probability of developing coronary artery disease (CAD) observed no significant difference with respect to family history of hypertension among cases and controls. This finding was in accordance to present study".[13] Hamed Mehri et al in "a study to investigate the serum levels of MBL in patients with CAD observed no significant difference in family history in patients with CAD ($p > 0.05$)".[14] Hamed Mehri et al in "a study to investigate the serum levels of MBL in patients with CAD observed serum level of MBL-2 was 152.5 ± 48 (ng/ml) in the case group and 108 ± 21 (ng/ml) in the control group. Serum levels of MBL were significantly increased in the case group compared to the control group".[14] de Vries B et al in "a study on mannose-binding lectin-pathway observed high-serum MBL-2 levels in men were associated with a higher risk of developing coronary heart disease in future. This association was independent of cardiovascular risk factors".[15]

Multiple mechanisms can contribute to MBL's atherogenic effects. In patients with advanced atherosclerosis, MBL activation of the complement system has been linked to increased cardiovascular risk, suggesting a role for MBL in atherogenesis. Increased complement iC3b deposition in damaged plaques suggests that complement activation contributes to acute coronary syndromes. Endothelial oxidative stress, which is significant for atherogenesis, also activates complement via the lectin complement pathway in human cell cultures. Additionally, anti-MBL monoclonal antibodies in this model suppressed endothelial oxidative stress, which decreased MBL and C3 deposition. MBL, on the other hand, plays a role in complement activation and is a powerful regulator of inflammatory pathways. The recruitment of phagocytes to the subendothelium may be enhanced because MBL has been shown to increase macrophage chemokine production. Recent research also found that MBL can regulate inflammation by binding to leukocytes.[16- 18]

CONCLUSION

The present study concludes that high MBL levels are associated with an increased risk of coronary artery disease and are high in the serum prior to the development of hypertensive symptoms.

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