

Association Of ABO Blood Groups With Scavenging Enzymes In Patients With Essential Hypertension

Dr. Shalini Arora¹, Dr. Mushir Ahamad², Dr. Pawan .A. Kulkarni³, Dr. Shrawan Kumar⁴

¹Assistant Professor, Department of Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur.

²Professor and Head, Department of Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur.

³Associate Professor, Department of Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur.

⁴Professor and Head, Department of General Medicine, Rama Medical College Hospital & Research Centre, Kanpur.

Corresponding author: Dr. Shalini Arora

Assistant Professor, Department of Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur.

Email:arorashalini37@gmail.com

Abstract

A case control study was undertaken to find a possible relationship of A,B,AB and O blood groups with scavenging enzymes superoxide dismutase ,catalase and antioxidant glutathione in patients with essential hypertension in the age group of 45 to 65 year and compared with normal healthy controls of the same age group.Thiobarbituric acid test was used for the estimation of lipid peroxide in plasma.Haemolysate was used for the assay of superoxide dismutase , catalase activity and glutathione concentration .The plasma lipid peroxide levels were increased significantly in all the hypertensive patients predominantly in group A and B. Concurrently significant decrease in the activities of scavenging enzymes and glutathione levels were found in these group patients. Accounting all the data together it is suggested that in comparison to blood group O, patients with blood group A, B and AB, possess specific blood group substances that may interfere in inducing required amount of superoxide dismutase and catalase with rapid depletion of red blood cell glutathione to counteract with free radical generation resulting in oxidative stress in them.

Key words: Blood groups, Essential hypertension, Oxidative stress

Introduction

Blood groups are genetically transmitted glycoconjugate structures with antigenic (agglutinin) properties located within red blood cells. **Abdulganiyu, (2016)** .They tend to provoke antigen-antibody reaction in presence of a specific antibody (agglutinin) in plasma. The ABO system is the most common and important antigenic system in red blood cells and has great relevance for blood transfusion in humans. Apart from their matching role in blood transfusion these specific antigens are known to play their important role in regulation of various biochemical and immunological reactions in the body.**Calafell (2008)**.The historical evidence

revealing association between different blood groups and the presence of hypertension in a large population are available. **Maxwell & Maxwell,(1955), Keslout & Vanhouten(1974), Galegzi & Gualandri (1975) , Miller et al (1979) , Kark & Friedlander (1984) , Nemesure et al (2006)**.Scientists recognized that there is inherited predisposition to arterial hypertension. **Broadbent & Broadbent (1897),Albutt (1915) ,Platt (1947)**. Free radicals are placed as important biochemical intermediates and have been implicated in a very large number in biological consequences. In several human and animal models, a link between oxidative stress and hypertension has been established.**Griending et al (2021)**. To

prevent an overload of free radicals the human body utilizes sophisticated complex defense system of, scavenging enzymes; superoxide-dismutase(SOD), catalase (CAT), reduced glutathione (GSH), produced at cellular level which decreases the concentration of the harmful oxidants in the tissues by functioning as free radicals scavengers and they are found to be preventive for the pathophysiological and metabolic disorder including cardiovascular diseases; atherosclerosis, hypertension .**Fukai and Ushio-Fukai (2011)**. Human body in its innate intelligence has strategically placed these scavenging enzymes in the cells to provide us protection and dysregulation of these enzymes results in oxidative stress which is the pathogenic outcome of oxidant over production that supercedes the cellular antioxidant capacity leading to vascular abnormalities, endothelial dysfunction and hypertension .**Touz (2004), Ulker et al (2003)**.Our blood group and also the enzyme activities are genetically dependent.It is therefore possible that there may be variation in various individuals. The possibility that the scavenging enzymes are preventive against the onset of hypertension and therefore in different blood group individuals the activity of these enzymes may vary. The present study has therefore been undertaken to investigate the possible relationship of different blood groups (A, B, AB and O) with scavenging enzymes (SOD and CAT), reduced glutathione and lipid peroxides in hypertensive patients.

Materials and Methods

The study was performed on 120 subjects divided into normal healthy n=60 and hypertensive n=60. These were again divided into four groups based on their blood groups A, B, AB, and O of healthy and hypertensive and containing n=15 subjects in each group. Patients attending medical OPD of General Medicine of Rama Medical College, Hospital & Research Centre were selected for the study. Some patients were outdoor patients whereas others were indoor patients of the hospital. According to the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7), Hypertension can be defined as a persistent elevation of systemic arterial blood pressure. The diagnosis of hypertension is made when the blood pressure is greater or equal

to 140 mmHg and or 90 mmHg on two or more occasions (JNC7) **Chobanian et al (2003)**. The study consists of the anthropometric assessment that includes age, body mass index (BMI) systolic and diastolic blood pressure (SBP&DBP) pulse rate (PR) and oxidant-antioxidant assay which include estimation of lipid peroxides, assay of superoxide dismutase (SOD) ,catalase (CAT) activity and estimation of reduced glutathione(GSH) .

Inclusion criteria- Diagnosed cases of essential hypertension reported to be suffering from 1-5 years were taken for the study. Hypertension in some of the patients was reported as a hereditary disease. All the patients were males in the age group of 45-65 years. Age was recorded for the information provided by the respondents. Individuals with systolic blood pressure 120mmHg (up to 122 mmHg) and diastolic blood pressure below or equal to 80 mmHg respectively were considered as normal healthy. Patients with systolic blood pressure (SBP) ≥ 140 mmHg and diastolic blood pressure (DBP) ≥ 90 mmHg were considered as patients with essential hypertension.

Exclusion criteria- Patients with diabetes, congestive heart failure/heart block, malignant clinically significant hematological disease, anemic patients, infectious diseases, Rh negative blood group patients, female hypertensives, cigarette smokers, alcoholics and those who are in current use of any medication including dietary supplements were excluded from the study. **Ethical Consideration** - The study process was started after obtaining ethical approval from the institutional ethical committee. All the patients who fulfill the necessary criteria were subjected to a detailed history and thorough clinical examination, data were collected. Those patients who fulfilled the criteria for inclusion in the study were fully reassured and explained about the procedure of the study. BMI was calculated by dividing weight in kg and height in m²: kg/m². According to the proposed criteria of the World health organization (WHO) .BMI was categorized as underweight (<18.5 kg/m²), normal weight (18.5-24.9 kg/m²), over -weight (<25-29.9kg/m²) and obese (≥ 30 kg/m²). The blood group was determined in all the patients separately by the use of monoclonal agglutinating antibodies .**Dacie and Lewis (2001)**.Thiobarbituric acid (TBA) test was used

to estimate lipid peroxide. **Ohkawa et al (1979)**. Superoxide dismutase (SOD) activity in hemolysate was determined by using spectrophotometric method. **McCord and Fridovich (1969)**. Catalase activity was determined using spectrophotometric method of **Aebi Aebi (1984)**. Total serum protein was estimated by Lowry method. **Lowry et al (1951)**) The concentration of GSH was determined in hemolysate by the method of Ernst Beutler. **Beutler et al (1963)**.

Statistical Analysis-

Mean and standard deviation (S.D) was calculated separately for all the groups and compared with normal healthy control groups. Unpaired student's t-test was performed to find the source of variation for the normally distributed parameters. $P < 0.05$ was considered as significant, $P < 0.001$ highly significant.

Results

The results presented in Table 1-4 and Figure 1-4 shows the anthropometric parameters of healthy and hypertensive male patients of blood group A, B, AB and O of age group (45-65) years mean age; 57.86 ± 4.45 , 52.93 ± 4.92 , 55.46 ± 3.83 and 50.33 ± 2.89 respectively.

The analysis of data shows significant increase in the body mass index (BMI) in group A (15.04 %; $P < 0.001$), B (12.95%; $P < 0.05$), AB (9.72%; $P < 0.05$) and O (8.28%; $P < 0.05$) hypertensives compared with healthy men. While comparing BMI of hypertensive subjects between groups there was mild non-significant change in group B (2.11%), AB (3.25%), O (3.62%), compared to group A.

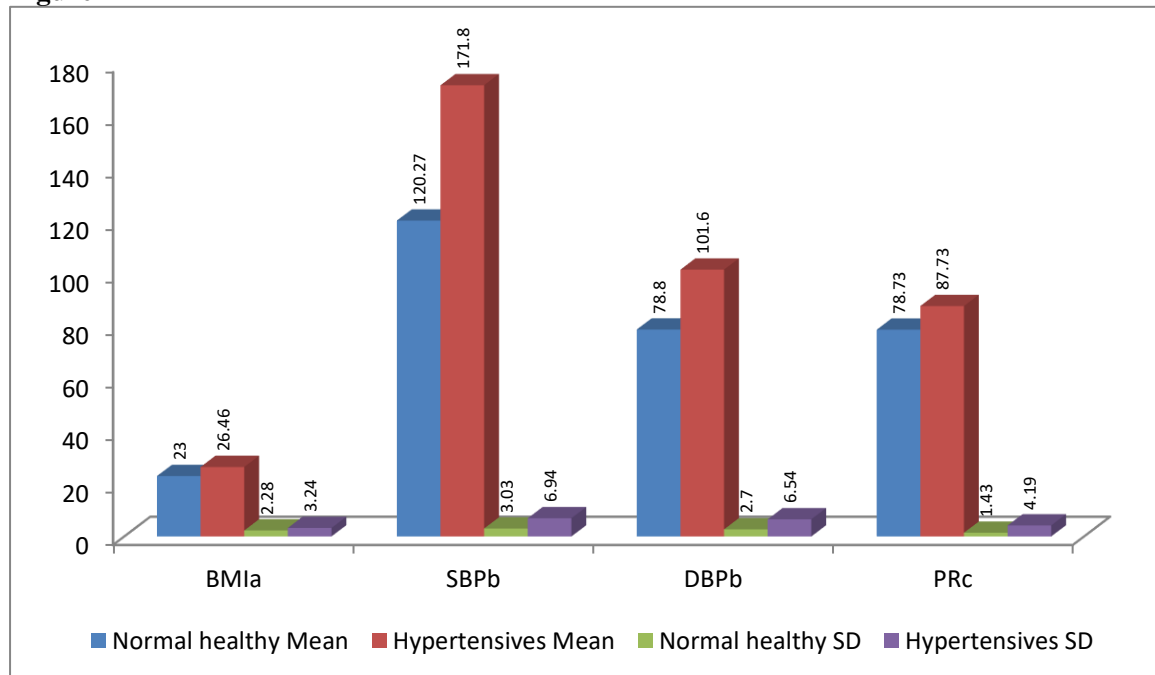
When systolic and diastolic blood pressures (SBP and DBP) were recorded significant increase of SBP in group A (42.84%; $P < 0.001$), B (36.40%; $P < 0.001$), AB (32.84%; $P < 0.001$) and O (31.60%; $P < 0.05$) was found when compared with healthy controls of respective groups. While comparing SBP of hypertensive subjects between groups significant decrease has been observed in group B (4.34%; $P < 0.05$), AB (5.04; $P < 0.05$) and O (7.25%; $P < 0.001$) hypertensives as compared to group A. The DBP was found to be increased significantly ($P < 0.001$) in all the hypertensive patients compared to normal healthy control. The increase in group A (28.93%), B (21.98%) AB (25.01) and O (23.09%) have been observed. When the DBP of group A hypertensives was compared with the DBP of group B, AB and O non significant decrease in group B (3.86%) and AB (2.95%) has been observed except group O (4.85% ; $P < 0.05$).

Table-1 Anthropometric parameters of normal healthy and hypertensive patients of blood group A. (Mean age 57.86 ± 4.45)

Parameters	Normal healthy	Hypertensives	Percent increase
BMI ^a	23.00±2.28	26.46±3.24*	15.04
SBP ^b	120.27±3.03	171.80±6.94*	42.84
DBP ^b	78.80±2.70	101.60±6.54*	28.93
PR ^c	78.73±1.43	87.73±4.19*	11.43

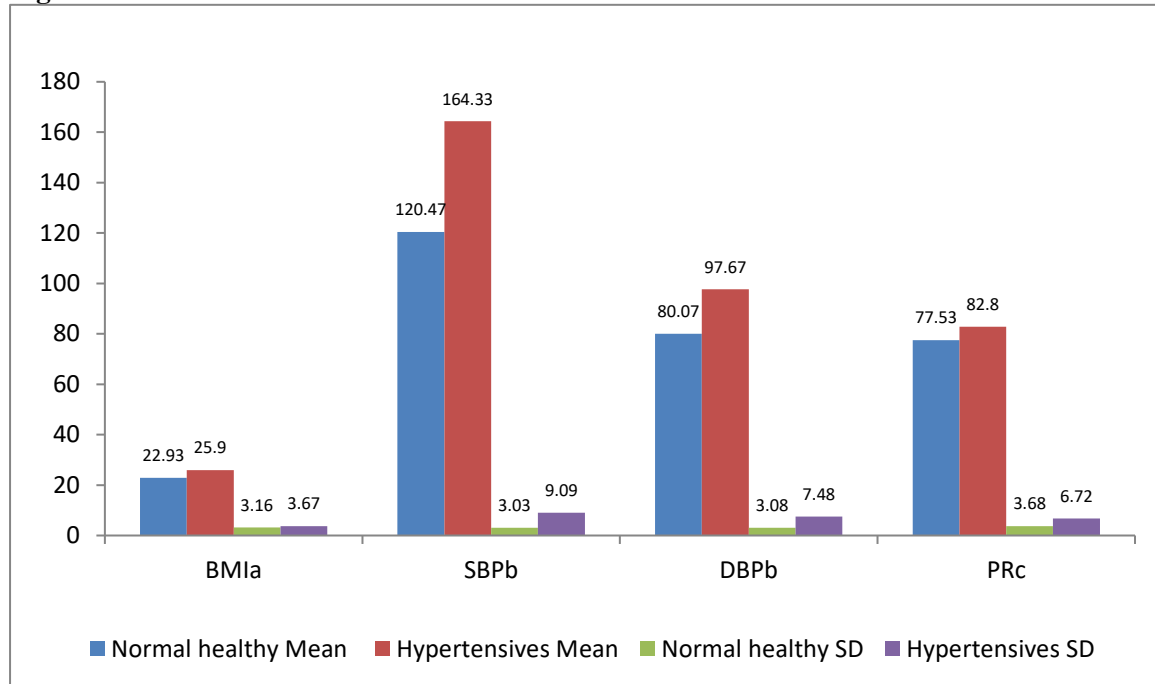
Units a = kg/m²; b = mmHg; c = beats /minute. Values are expressed as mean \pm S.D of 15

subjects compared to normal healthy control. * $P < 0.001$.

Figure-1**Table-2 Anthropometric parameters of normal healthy and hypertensive patients of blood group B. (Mean age 52.93± 4.92 year)**

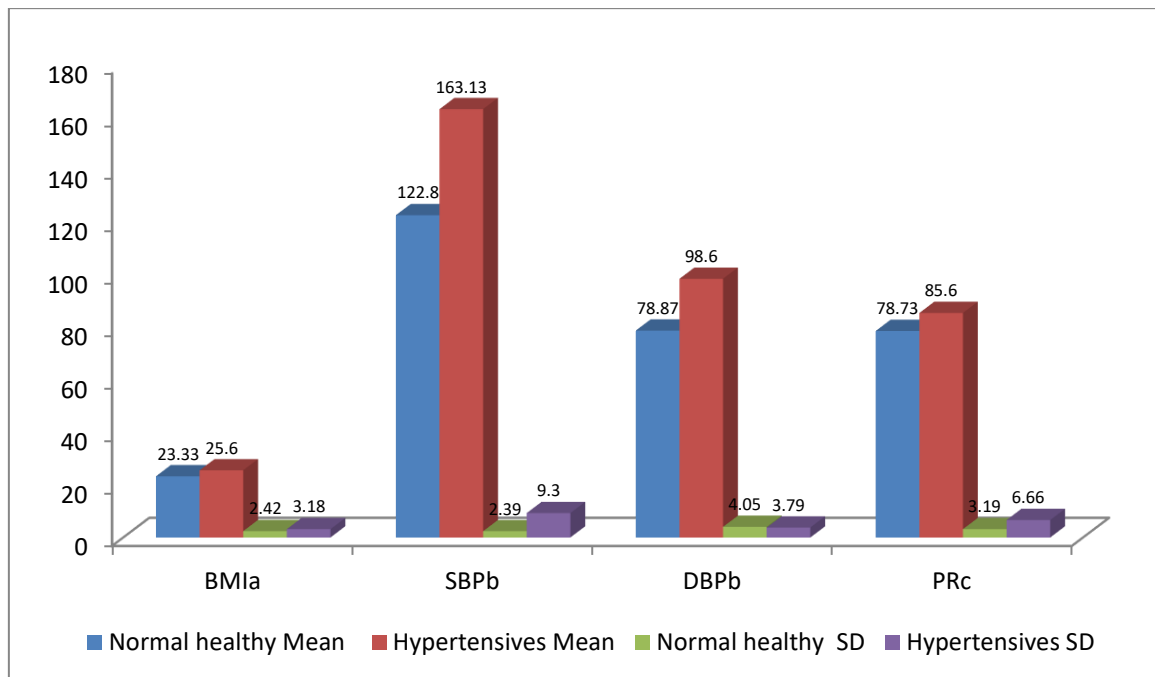
Parameters	Normal healthy	Hypertensives	Percent increase
BMI ^a	22.93±3.16	25.90±3.67*	12.95
SBP ^b	120.47±3.03	164.33±9.09**	36.40
DBP ^b	80.07±3.08	97.67±7.48**	21.98
PR ^c	77.53±3.68	82.80±6.72*	6.79

Units a = kg/m²; b = mmHg; c = beats /minute.
 Values are expressed as mean ±S.D of 15 subjects compared to normal healthy control.
 *P< 0.05, **P<0.001.

Figure-2**Table -3 Anthropometric parameters of normal healthy and hypertensive patients of blood group AB. (Mean age 55.46± 3.83 year)**

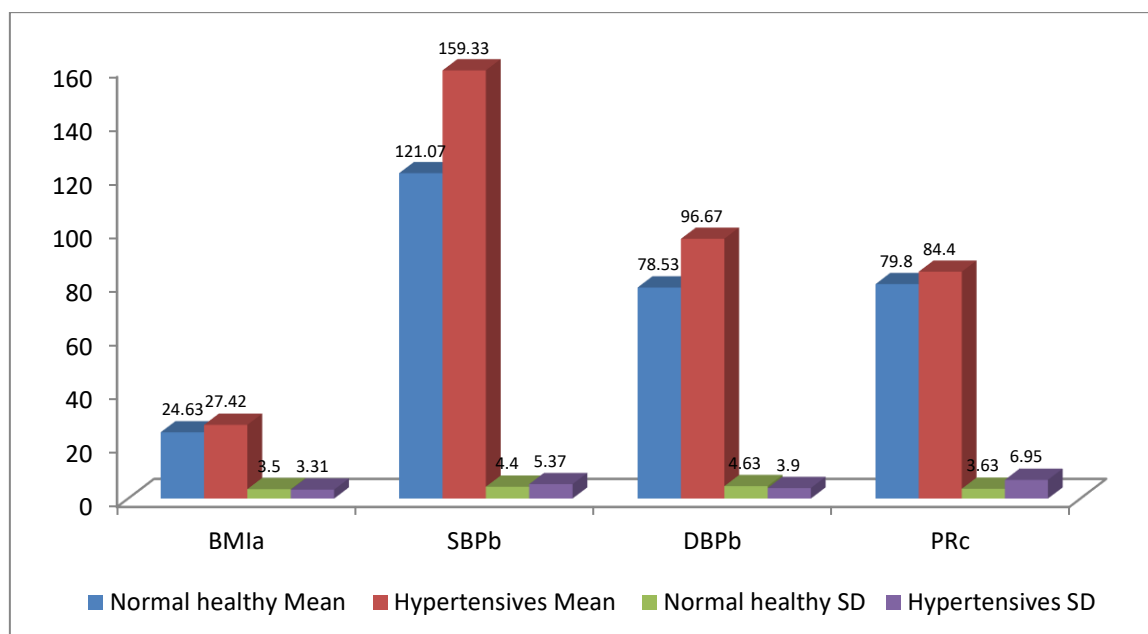
Parameters	Normal healthy	Hypertensives	Percent increase
BMI ^a	23.33±2.42	25.60±3.18*	9.72
SBP ^b	122.80±2.39	163.13±9.30**	32.84
DBP ^b	78.87±4.05	98.60±3.79**	25.01
PR ^c	78.73±3.19	85.60±6.66*	8.72

Units a = kg/m²; b = mmHg; c = beats /minute.
 Values are expressed as mean ±S.D of 15 subjects compared to normal healthy control.
 *P< 0.05, **P<0.001.

Figure-3**Table -4 Anthropometric parameters of normal healthy and hypertensive patients of blood group O. (Mean age 50.33± 2.89 year)**

Parameters	Normal healthy	Hypertensives	Percent increase
BMI ^a	24.63±3.50	27.42±3.31*	11.32
SBP ^b	121.07±4.40	159.33±5.37*	31.60
DBP ^b	78.53±4.63	96.67±3.90**	23.09
PR ^c	79.80±3.63	84.40±6.95*	5.76

Units a = kg/m²; b = mmHg; c = beats /minute.
 Values are expressed as mean ±S.D of 15 subjects compared to normal healthy control.
 *P< 0.05, **P<0.001.

Figure-4

Pulse rate was increased significantly in group A (11.43%; $P < 0.001$), B (6.79%; $P < 0.05$), AB (8.72%; $P < 0.05$) and O (5.76%; $P < 0.05$) respectively compared to healthy controls. When pulse rate, of group A hypertensive was compared with other hypertensive groups non-significant changes in group AB and O has been found however, in group B it was significantly lower (5.61% ; $P < 0.05$)

The level of oxidative stress was simultaneously analyzed in the blood of hypertensive patients and compared with normal healthy controls by means of lipid peroxides and antioxidative enzymes; Superoxide dismutase (SOD), Catalase (CAT) and reduced glutathione (GSH). The results presented in table 5 to 8 and figure 5 to 8 shows significantly ($P < 0.001$) increased levels of lipid peroxides in the hypertensive subject of group A, B, AB and O; 161.62%,

140.10%, 115.28% and 105.10% respectively. Superoxide dismutase (SOD) was significantly decreased in group A (24.93%; $P < 0.05$), B (17.51%; $P < 0.05$), AB (18.85%; $P < 0.05$) and O (17.79%; $P < 0.05$) hypertensives. The activity of catalase was found to be decreased significantly ($P < 0.05$) in group A, B, AB and O hypertensives; 12.79%, 13.37%, 14.83% and 9.96% respectively. Reduced glutathione (GSH) was also decreased significantly in all the hypertensive groups. In group A (28.72%; $P < 0.001$), B (25.68%; $P < 0.05$) AB (26.60%; $P < 0.05$) and O (21.90%; $P < 0.05$) values have been observed. The comparative study of oxidant-antioxidative enzymes between hypertensive groups show non-significantly altered values of lipid peroxides, SOD, CAT and reduced GSH. However there was significant increase in SOD (21.62%; $P < 0.05$) in group O compared to group AB hypertensive.

Table -5 Blood levels of lipid peroxide, scavenging enzymes and glutathione in normal healthy & hypertensive patients of blood group A.

Parameters	Normal healthy	Hypertensives	Percent Change
Lipid Peroxide ^a	2.18±1.16	5.71±1.34**	+161.92
SOD ^b	1176.65±263.12	883.27±212.42*	-24.93

CAT ^b	10499.06±1855.15	9156.03±1802.41*	-12.79
GSH ^c	28.58±8.70	20.37±7.97**	-28.72

Unit a = n mol MDA/ml plasma; b = units/g Hb; c=mg/dl. Values are expressed as mean ±S.D of 15 subjects compared with normal healthy controls. *P<0.05; **P<0.001; + = increase, - = decrease

Figure-5

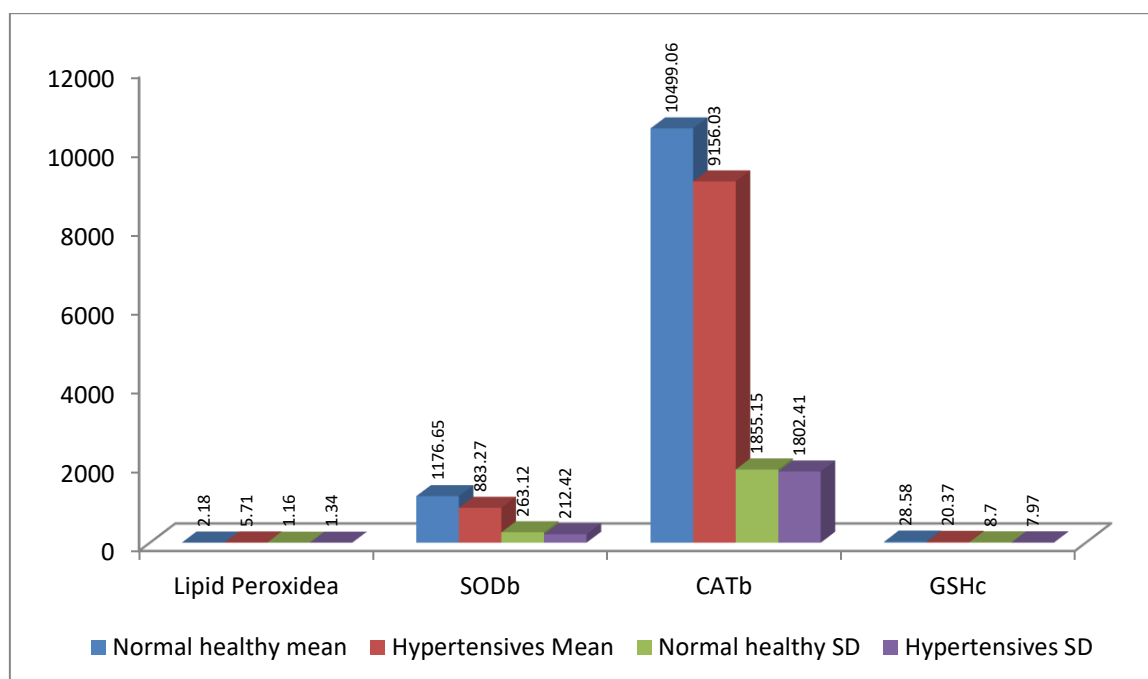


Table -6 Blood levels of lipid peroxide, scavenging enzymes and glutathione in normal healthy & hypertensive patients of blood group B.

Parameters	Normal healthy	Hypertensives	Percent Change
Lipid Peroxide ^a	1.97±1.44	4.73±1.68**	+140.10
SOD ^b	1015.26±170.56	837.46±326.60*	-17.51
CAT ^b	9373.31±875.51	8119.31±2048.76*	-13.37
GSH ^c	27.45±9.16	20.40±5.11*	-25.68

Unit a = n mol MDA/ml plasma; b = units/gHb; c=mg/dl. Values are expressed as mean ±S.D of 15 subjects compared with normal healthy

controls. *P<0.05; **P<0.001; + = increase, - = decrease

Figure-6

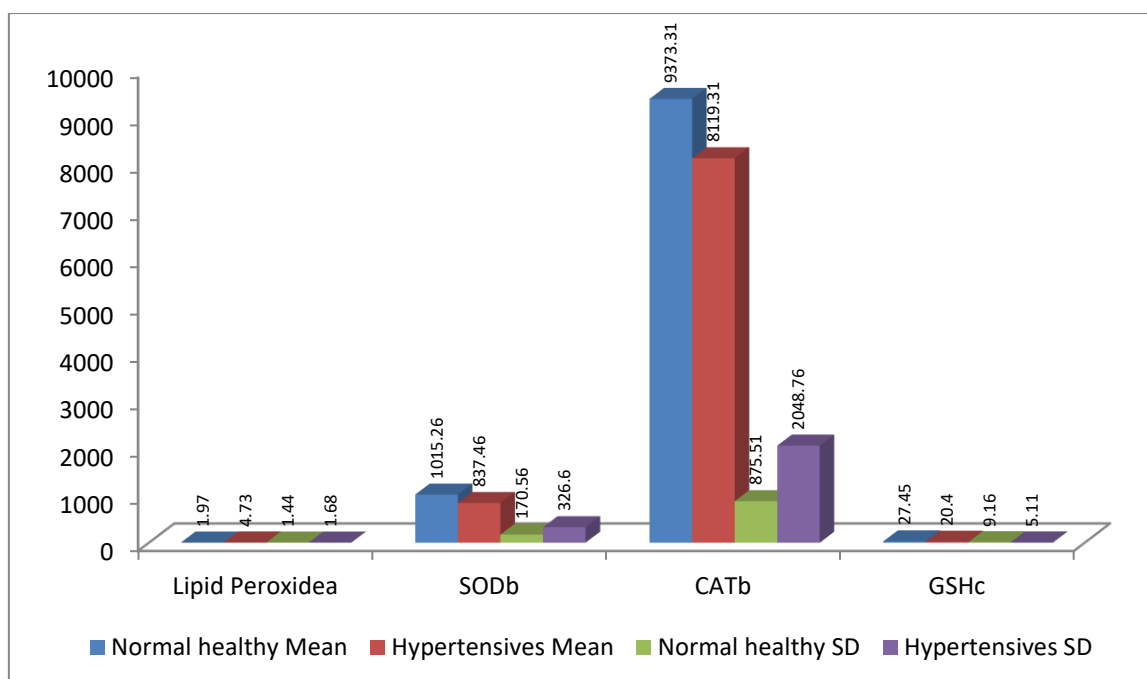
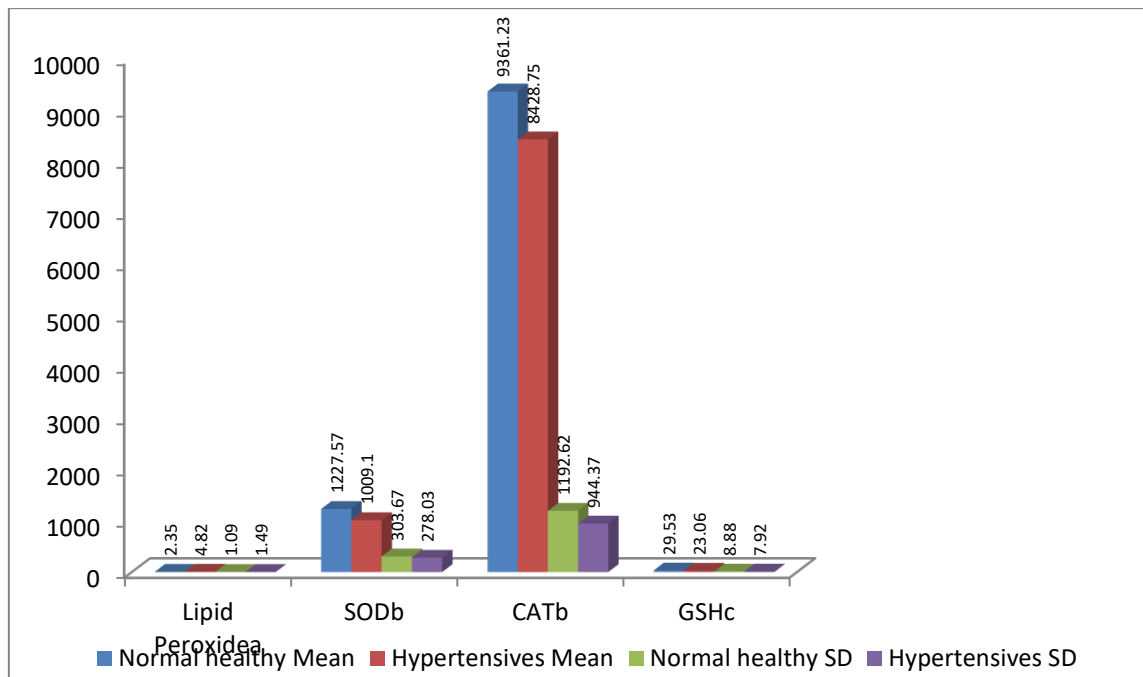


Table -7 Blood levels of lipid peroxide, scavenging enzymes and glutathione in normal healthy & hypertensive patients of blood group AB.

Parameters	Normal healthy	Hypertensives	Percent Change
Lipid Peroxide ^a	2.42±1.45	5.21±1.88**	+115.28
SOD ^b	1022.45±201.57	829.70±183.50*	-18.85
CAT ^b	10183.05±1954.21	8672.12±1766.20*	-14.83
GSH ^c	25.71±3.53	18.87±5.10**	-26.60

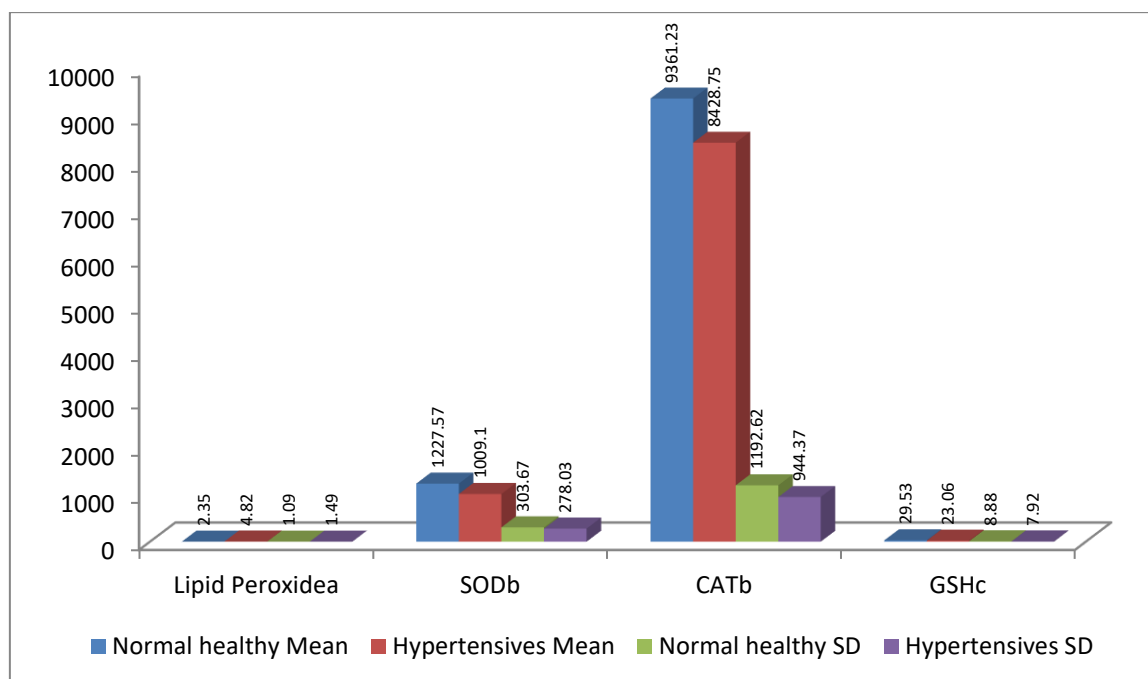
Unit a = n mol MDA/ml plasma; b = units/gHb; c=mg/dl. Values are expressed as mean \pm S.D of 15 subjects compared with normal healthy controls. *P<0.05; **P<0.001; + = increase, - = decrease

Figure-7**Table -8** Blood levels of lipid peroxide, scavenging enzymes and glutathione in normal healthy & hypertensive patients of blood group O.

Parameters	Normal healthy	Hypertensives	Percent Change
Lipid Peroxide ^a	2.35±1.09	4.82±1.49**	+105.10
SOD ^b	1227.57±303.67	1009.10±278.03*	-17.79
CAT ^b	9361.23±1192.62	8428.75±944.37*	-9.96
GSH ^c	29.53±8.88	23.06±7.92*	-21.90

Unit a = n mol MDA/ml plasma; b = units/gHb; c=mg/dl. Values are expressed as mean \pm S.D of 15 subjects compared with normal healthy controls. *P<0.05; **P<0.001; + = increase, - = decrease

Figure-8



Discussion

In the present study we have observed significant increase in body mass index (BMI) of all the hypertensive patients compared to normal healthy controls. There are so many reports that male gender associated with increased BMI is more susceptible to be hypertensive. **Chakraborty et al (2009)**, **Gelber et al (2007)**, **Joshi et al (2007)** and the reason behind this could be the activation of several presser mechanisms, which may raise the levels of plasma nor adrenaline and adrenaline suggesting a higher sympathetic tone in obese or overweight and this results in increased total blood volume leading to increased cardiac output and increased cystolic free Ca^{2+} levels and reduced intracellular Mg^{2+} levels in the blood cells of these subjects. It is well documented that obesity is closely associated with hypertension and these physiological alternations could be associated with the establishment of this state (Good et al; 2008, **Weidmann et al (1993)**). The earlier study done where author found that B group patients were having high BMI with high prevalence of hypertension. **Bhattacharya et al (2010)**. We have observed that both systolic and diastolic blood pressures were elevated in hypertensive patients and higher values have been found in group A and B. The elevation in blood pressure

could be due to increased generation of reactive oxygen species in particular blood group which in turn can oxidize many other important biomolecules most likely lipids, causing propagation of oxidative process and oxidative stress. This may be true as we have also found profound increase in blood plasma lipid peroxides in group A (162%) and B (140%), positively correlated with SBP and DBP, following diminution of antioxidant enzymes, SOD and catalase. It has been reported that SOD deficiency following an increase in superoxide anion production contributes to a rise in arterial blood pressure **Pedero-Botet et al (2000)**, **Jun et al (1996)**. We have observed an increase in pulse rate following increment in blood pressure of all the hypertensive groups. However, the increase in pulse rate was comparatively higher in blood group A to that of other groups B, AB and O. The reason behind high blood pressure and high pulse rate is abnormal regulation of arterial tone. **Kim et al (1999)**. In the situation of increased blood pressure the arteries experience resistance against the blood flow. To compensate for by the blood supply in the body heart has to beat more per minute. As a result the arterial pressure is persistently higher than normal. It has been suggested that increasing level of obesity is associated with increase in pulse rate and blood pressure. **Martin et al (2003)**. We have observed increased level of

lipid peroxides in hypertensive patients of all the groups (A, B, AB and O) compared to normal healthy controls and the highest level of lipid peroxides was observed in patients of blood group A and B. The increased lipid peroxidation in hypertensives indicates increased oxidative stress in them and this may be due to suppression of antioxidant mechanism (as evident by decreased activities of superoxide dismutase (SOD) and catalase and reduced glutathione levels. The disturbed oxidant-antioxidant balance may in turn can oxidize many other important biomolecules including membrane lipids to initiate the progression of many metabolic alteration in them. The activity of SOD and catalase was found to be suppressed along with the depletion of reduced glutathione.

The possible reasons for the reduction in the activity of scavenging enzymes in hypertensive could be due to difference in the carbohydrate portion of a particular blood group antigen which provides specificity to blood groups. The enzyme N-acetyl galactosamine transferase and galactosyl transferase catalyses the transfer of N-acetyl galactosamine and galactosyl moieties to the basic fucose structures and synthesizes blood group A and B antigen respectively and may interfere in inducing required amount of SOD and catalase resulting in the accumulation of large quantities of peroxidation products. Thus different blood group antigen may possess suppressed activity of these antioxidant enzymes and this could be accompanied by metabolic disturbances which influence reduced GSH concentration suggest that genetic factors might explain some of the variance in the risk of developing the disease. It is suggested that hypertensive patients including blood group A.B.AB having an excess of oxidative stress in comparison to controls may had to use more and more GSH for counteracting an excess of lipid peroxidation in them, that may be the reason of depletion of GSH is more in them. None of the carbohydrate portion is found in the O group may have a selective advantage over other blood groups.

Conclusion

The study concluded that hypertensive patients with blood group A and or B in comparison to blood group O are more susceptible to oxidative

stress due to the interference of carbohydrate moieties present on the red blood cell antigen in inducing the required amount of scavenging enzymes; Superoxide dismutase and catalase to counteract free radical generation and may also failed to protect against depletion of GSH.

Conflict of interest-

The authors have no conflict of interest.

References

- [1] Abdulganiyu. (2016) Distribution of ABO and Rh (D) blood groups and associated traits : a study of the college of nursing and midwifery.MSc Thesis Dissertation,Kogi state Obangede
- [2] Aebi H (1984).Catalase in vitro.*Methods in Enzymology* 105:121-126.
- [3] Albutt TC (1915). Diseases of the Arteries, including Angina Pectoris. pp 164-412 *Macmillan London*.
- [4] Bhattacharya S, Ganaraja B, Bhatt RM . (2010). Correlation between the blood groups, BMI and Pre-hypertension among medical students. *Journal of Chinese Clinical Medicine* 5(2) :2.
- [5] Broadbent WH, Broadbent JFH (1897) Heat Disease, P 80, *Wood, New York*.
- [6] Calafell F,Roubinet A,Ramirez-Soriano,N Et al (2008).Evolutionary dynamics of the human ABO gene.ABO gene *Human Genetics*,124 (2);123-135.
- [7] Chakraborty R,Bose K, Besai S.(2009). Body mass index and blood pressure among adult bengalee male slum dwellas of Kolkata India, *Journal of public Health* 17(5):301-308
- [8] Chobanian ,A.V., Bakris, G.L., Black ,H.R., Cushman, W.C., Green, L.A., et al. (2003) The seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*, Vol. 289, pp. 2560-2571.
- [9] Dacie and Lewis(2001).In : Practical Haematology,9th Ed.,(Churchill Livingstone, UK) p472-475.
- [10] Beutler E, Duran O, Kelly BM(1963) Improved method of determination of blood glutathione. *Journal of Laboratory Clinical Medicine* 61 : 882-88 .

- [11] Fukai T, and Ushiro-Fukai M (2011). Superoxide dismutases: role in redox signaling, antioxidants and redox signaling. *Vascular function and Disease* 15(6):1583-1606.
- [12] Galeazzi L, Gaulandri V (1975). ABO blood group phenotypes and pathogenesis of cardiovascular diseases. Congenital, rheumatic and coronaric heart disease and arterial hypertension. *Giomale Italino di cardiologia Journal* 5(5) : 744-751.
- [13] Gelber RP, Gaziano JN, Manson JE, Bruing JE, Sesso HD. (2007). A Prospective study of body mass index and the risk of developing hypertension in men. *American Journal of Hyper tension* 20(4): 370-377.
- [14] Good D, Morsa SA, Ventura HO, Risin E. (2008). Obesity, hypertension and the heart. *Journal of Cardiometabolic syndrome* 3(3): 168-172.
- [15] Griendling, K.K., Camargo, L.L., Rios, F.J., Alves-Lopes, R. Montezano, A.C., Touyz, R.M (2021). Oxidative stress and Hypertension. *Epub* 128(7);993-1020
- [16] Joshi V, Lim J, Nand Kumar M. (2007). Prevalence and risk factor of undetected elevated blood pressure in an elderly south-east asean population. *Asia Pacific Journal of public Health* 19(2):3-9
- [17] Jun T, Keyan F, Catalaono M. (1996). Increased superoxide anion production in humans: a possible mechanism for the pathogenesis of hypertension. *Journal of Human Hypertension* 10(5): 305-309.
- [18] Kark JD, Friedlander Y (1984). ABO and Rh blood groups and blood pressure in Jerusalem 17 years old. *Human Biology* 56 : 759-769.
- [19] Kesloot H, Van Houte O (1974). An epidemiologic survey of arterial blood pressure in large male population group. *American Journal of Epidemiology* 99 : 14-29.
- [20] Kim YG, Chae JK, Park SH, Chung CH, Hong SP, Cho K K (1986). Serum and erythrocyte lipoperoxides and superoxide dismutase levels in normal persons and hypertensive patients. *The Korean Journal of Internal Medicine* 1(1):113-119.
- [21] Lowry HO, Rosebrough JN, Fari LA, Randall JR (1951). Protein measurement with the Folin Phenol Reagent *Journal of Biological Chemistry* 193 : 265-75 .
- [22] Martins D, Tareen N, Pan D, Noris K. (2003). The relationship between body mass index, blood pressure and pulse rate among normotensive and hypertensive participants in the third national health and nutrition examination (NHANES) . *Cellular and Molecular Biology* 43(8) :1305-1309.
- [23] Maxwell RDH, Maxwell KN (1955). ABO blood groups and hypertension. *British Medical Journal* : 179-180.
- [24] McCord J, Fridovich I (1969) Superoxide dismutase . An enzymatic function for erythrocyte (hemocuprein). *Journal of Biological Chemistry* 244 : 6049-6055
- [25] Miller ZT, Grin EC, Conneally MP, Weinberger HM (1979). Association of blood groups with essential and secondary hypertension. A possible association of the MNS system. *Hypertension by American Heart Association* 1:495-497.
- [26] Nemesure B, Wu SY, Hennis A, Leske MC, Barbados eye study group (2006). Hypertension, type 2 diabetes and blood groups in a population of African ancestry. *Ethnicity and Disease* 16(4): 822-9.
- [27] Ohkawa H, Ohishi N, Yagi K (1979) Assay for Lipid peroxides in animal tissues by thiobarbituric acid reaction. *Analytical Biochemistry* 95 : 351-58
- [28] Pedro Botet J, Covas MI, Martin S, Rubies Prat J (2000). Decreased endogenous antioxidant enzymatic status in essential hypertension. *Journal of Human Hypertension* 14(6): 343-5.
- [29] Platt R (1947). *Quarterly Journal of Medicine* 16 : 111.
- [30] Touz RM (2004). Reactive oxygen species, vascular oxidative stress, and redox signaling in hypertension: What is the clinical significance? *Hypertension* 44(3) : 248-52.
- [31] Ulker Sibel, Master DMC, Mckeown PP, Bayraktulan U (2003). Impaired activities of antioxidant enzymes elicit endothelial dysfunction in spontaneous hypertensive rats despite enhanced vascular nitric oxide generation. *Cardiovascular Research* 23(1) : 33-37.
- [32] Weidmann P, Coustin M, Boehlen L, Shan S. (1993). The Pathogenesis of hypertension in obese subjects. *Drugs* 46(2):197-208.