International Journal of Research in Medical Sciences
Shrivastav C et al. Int J Res Med Sci. 2019 Apr;7(4):1252-1256
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20191334

Original Research Article

A correlative study of body mass index with oxidative stress parameters (serum uric acid and serum malondialdihyde) in essential hypertension

Chanchal Shrivastav, Paras Arvindbhai Parekh*, G. Indra Kumar

Department of Physiology, Ananta Institute of Medical Sciences and Research Center, Rajsamand, Rajasthan, India

Received: 24 January 2019
Accepted: 09 March 2019

***Correspondence:**
Dr. Paras Arvindbhai Parekh,
E-mail: paras_parekh13@yahoo.com

ABSTRACT

Background: Hypertension is most commonly documented modifiable risk factor for cardiovascular diseases. A growing body of data proposes an appreciated pathogenic role of an elevated serum uric acid in atherosclerosis and cardiovascular disease (CVD). Increased oxidative stress could be involved in the pathogenesis of hypertension. Oxidative stress marker, serum uric acid (SUA) and serum malondialdyhyde level (S. MDA) are affected by both genetic and environmental factors and related to biological factors as gender, age and body mass. So, the aim of the study is to access the association of body mass index (BMI) with oxidative stress parameters in essential hypertension (EHT).
Methods: For the said purpose, this case control study was carried out on a total of 200, age and sex matched 75 hypertensives, 75 prehypertensive and 50 healthy subjects. After diagnosis of cases, SUA was estimated by standard kit method and S. MDA was estimated manually by Buege and Aust method.
Results: This study represents that systolic and diastolic blood pressure were increased with increased BMI in all groups. Oxidative stress marker, SUA and S. MDA level increased significantly with increasing BMI in all groups and were positively correlated.
Conclusions: Our study indicates that monitoring of the blood pressure, SUA and S. MDA at regular interval and maintaining of the oxidative balance would be helpful in preventing the development of hypertension and associated cardio-vascular morbidities.

Keywords: Body mass index, Essential hypertension, Serum malondialdehyde, Serum uric acid

INTRODUCTION

Hypertension is one of the leading causes of the global burden of disease. Rising prevalence of hypertension is a registered public health problem in India as it leads to cardiovascular diseases.[1] The most considerable risk factors for the advancement of hypertension are increased salt intake, obesity, cigarette smoking, lack of physical exercise, genetic factors, stress and strain.[2] Moreover, obesity has various health consequences; it is a major risk factor for the global burden of non-communicable diseases including diabetes, heart diseases, hypertension, stroke and some cancers.[3] Overweight and obesity in children, looks to be a foremost contributor to essential hypertension prevalence in children and adolescents.[4,5] BMI measures the weight in relation to the height and gives a figure of total body fat. Normal body mass index is 20-25kg/m^2. A BMI within 25 and 29.9 is regarded as overweight. A BMI of 30 or more is appreciated as obese.[6] A BMI of less than 25 is the goal for controlling blood pressure. Body mass index is positively and independently linked with morbidity and mortality from

hypertension, cardiovascular disease, type II diabetes mellitus and other chronic diseases.[7] A similar positive relationship between BMI and blood pressure (BP) has also been accounted among Asian populations.[8,9]

A growing body of data proposes an appreciated pathogenic role of an elevated serum uric acid in atherosclerosis and cardiovascular disease, particularly in patients with diabetes mellitus, heart failure and hypertension.[10] Hyperuricaemia is typically specified as increased levels of serum uric acid (>7mg/dl in males and >6mg/dl in females).[11]

In search for a causative factor for essential hypertension, the life style changes and obesity could contribute the increase of oxidative stress markers such as uric acid and lipid peroxidation. A reduction in antioxidant enzymes and increase in oxidants in the hypertensive state have been accounted to raise the production of reactive oxygen species (ROS). ROS can attack polyunsaturated fatty acids in cell membrane phospholipids, resulting in the development of lipid hydroperoxides, a destructive process known as lipid peroxidation which can then break up to many small compounds such as malondialdehyde (MDA). In the present study MDA, was used as a biochemical marker for the assessment of lipid peroxidation.

METHODS

The hospital based case control study was carried out on a total of 200 sex matched subjects of 20-50 years old, in department of Physiology. All subjects were broadly divided in to three groups according to JNC7 criteria.[12] Control group: 50 subjects with normal blood pressure (SBP= 90-119 mmHg, DBP= 60-79mmHg) PreHT group: 75 cases of prehypertension (SBP= 120-139mmHg, DBP= 80-89mmHg) HT group: 75 cases of newly diagnosed cases of essential hypertension (SBP= 140-159mmHg, DBP= 90-99mmHg). The subjects with gout, diabetes mellitus, gestational hypertension and/or secondary hypertension, smokers, alcohol consumers and patient using medication for hypertension were excluded from the study. After obtaining a written voluntary informed consent from all the subjects, data were collected in the detailed proforma along with requisite physical examination. After diagnosis, blood sample (5ml) was drawn after an overnight fast (12hrs) by venous puncture and serum was used for biochemical analysis. S. Uric acid (SUA) level, Serum Malondialdehyde (MDA) was estimated by using commercially available reagents or kits.[13,14]

The data was analyzed by using statistical package of social science (SPSS) version 16. Significance testing of difference for mean±SD of three groups was done by analysis of variance test (ANOVA). BMI wise comparisons of various parameters were assessed by student t-test. The correlations of BMI with SUA and MDA were assessed by Pearson coefficient of correlation. A p-value of <0.05 was used to establish statistical significance.

RESULTS

Body mass index, SBP and DBP were significantly high (p<0.0001) in hypertensive group as compared to pre-hypertensive and control group (Table 1).

Table 1: Characteristics of study population among different groups (Mean±SD).

Variables	Control	Pre HT Group	HT Group	ANOVA P Value
Age (Yrs.)	37.46±8.09	35.84±6.5	40.25±7.71	<0.001
BMI (Kg/m^2)	21.89±1.47	24.27±2.6	27.34±2.77	<0.0001
SBP (mmHg)	114.06±16.77	134.00±5.1	160.04±11.49	<0.0001
DBP (mmHg)	74.66±6.23	86.45±2.93	92.00±10.15	<0.0001

Table 2: BMI wise variations of SBP among different groups (Mean±SD).

BMI group	Control			Pre HT group			HT Group		
	Mean	SD	N	Mean	SD	N	Mean	SD	N
<25	113.63±6.30		40	135.23±4.49		26	173.33±5.77		3
≥25	115.80±4.47		10	138.35±5.33		49	179.49±11.36		72
T value	1.25			2.68			1.72		
P value	NS*			<0.01			NS		

In prehypertensive group, systolic blood pressure was significantly increased (p<0.01) in overweight and obese group with BMI ≥25, while this association was not significant in hypertensive group (Table 2).

Mean±SD value of DBP was increased with increased BMI level. This difference was statistically significant in prehypertensive (p<0.0001) and control group (p<0.01) but it was insignificant in hypertensive group (p>0.05) (Table 3).

Table 3: BMI wise variations of DBP among different groups (Mean±SD).

BMI group	Control			Pre HT group			HT Group		
	Mean	SD	N	Mean	SD	N	Mean	SD	N
<25	73.83±6.53		40	85.77±3.46		26	93.33±5.77		3
≥25	78.00±3.27		10	88.82±2.58		49	91.94±10.72		72
T value	2.85			3.95			0.39		
P value	<0.01			<0.0001			NS		

Table 4: BMI wise variations of S. uric acid level among different groups (Mean±SD).

BMI group	Control			Pre HT group			HT Group		
	Mean	SD	N	Mean	SD	N	Mean	SD	N
<25	4.84±0.88		40	5.56±0.97		26	5.43±0.21		3
≥25	5.20±0.82		10	6.06±0.89		49	6.57±0.63		72
T value	1.22			2.19			3.10		
P value	NS			<0.05			<0.005		

Table 5: BMI wise variations of S. MDA level among different groups (Mean±SD).

BMI group	Control			Pre HT group			HT Group		
	Mean	SD	N	Mean	SD	N	Mean	SD	N
<25	1.30±0.20		40	1.39±0.16		26	1.60±0.14		3
≥25	1.29±0.23		10	1.75±0.24		49	2.16±0.41		72
T value	0.13			7.75			5.95		
P value	NS			<0.0001			<0.0001		

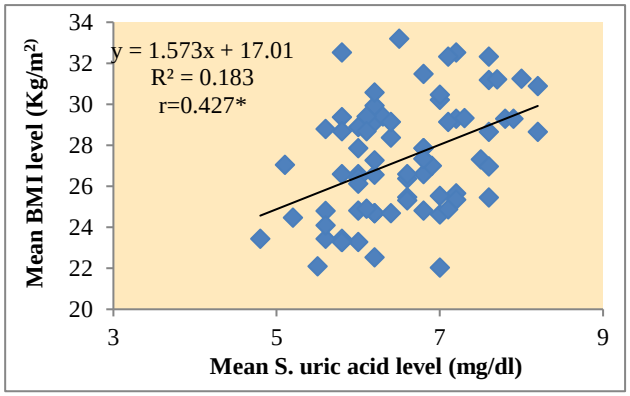

Figure 1: Correlation between s. uric acid levels and BMI.

Serum uric acid was observed to be statistically significant in individual with high BMI in both prehypertensive (p<0.05) and hypertensive group (<0.005) (Table 4).

Significant difference of S.MDA level was observed in overweight and obese individual among prehypertensive and hypertensive group (p<0.0001) (Table 5).

Serum uric acid was statistically significant and positively correlated with BMI (p <0.001) (Figure 1). Significant and positive correlation was observed between S.MDA level and BMI (p<0.001) (Figure 2).

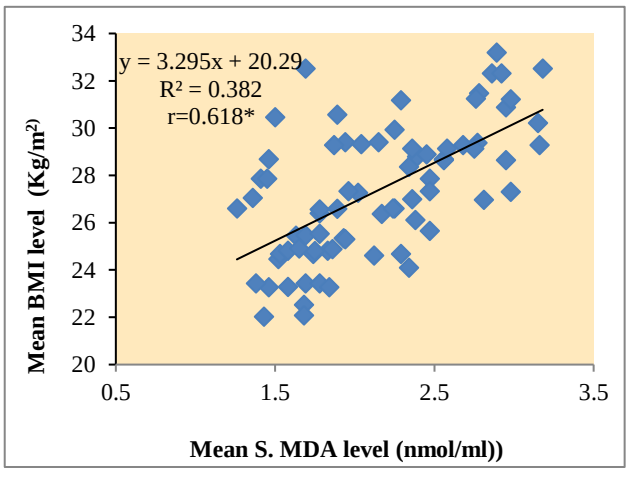

Figure 2: Correlation between S. MDA levels and BMI.

DISCUSSION

The knowledge of the effect of obesity on hypertension is crucial as it is a modifiable risk factor. Overweight and obesity in children, looks to be a foremost contributor to essential hypertension prevalence in children and adolescents.[4,5] Our study showed that, systolic blood pressure was obtained to be increased with increased BMI in hypertensive, prehypertensive and control group but the difference was not statistically significant ($p>0.05$) except in prehypertensive group ($p<0.01$) (Table 2). We also ascertained corresponding results in case of diastolic blood pressure but the difference was statistically significant ($p<0.01$) except in hypertensive group ($p>0.05$) (Table 3). Similarly, Mungreiphy NK et al, studied the association between BMI, blood pressure, age and found that mean value of both the systolic and diastolic blood pressure were increased from underweight to normal and then to overweight and obese category.[15] Similar findings have been described in other studies.[8,16] Overweight or obese subjects were probably to have significantly higher blood pressure than those with normal BMI in all stages, prehypertension, stage I hypertension, and stage II hypertension.

This link between hypertension and increased body weight can be interpreted by the fact that the blood volume increases with excessive body fat and the heart works more laborious to pump the blood through a longer and constricted network of blood vessels.[17] Various mechanisms are tangled in the characteristic haemodynamic model of volume expansion, increased cardiac output and systemic vascular resistance seen in obesity allied hypertension. Sympathetic overactivity selective leptin release, adipokines including leptin, free fatty acids and angiotensin II, RAAS overactivity, reactive oxygen species and NO deficiency, T cell activation and the over activation of endocannabinoid pathway are some of the mechanisms associated with obesity linked hypertension.[18-24] Furthermore, the reduced insulin sensitivity often seen in obesity is directly associated with elevated BP and hypertension.[21]

This study represent that serum uric acid level increases significantly with increasing BMI in hypertensive, prehypertensive and control group (Table 4). Similarly, Jawed S et al, found highly significant difference in serum uric acid level of obese patients as equated to nonobese essential hypertensive patients.[25] In overweight and obese subjects, hyperinsulinemia secondary to insulin resistance may enhance the reabsorption of uric acid and thus leads to the association of hyperuricaemia with hypertension.[26] Uric acid generally has an antioxidant effect; however, uric acid turns in to a strong oxidant in the ambiance of obesity.[27] Experimental studies have accounted that hyperuricaemia accelerates systemic hypertension via stimulation of the renin angiotensin system, and direct access of uric acid into both endothelial and vascular smooth muscle cells, resulting in local suppression of endothelial nitric oxide levels, stimulation of vascular smooth muscle cell proliferation, and activation of vasoactive and inflammatory mediators.[28,29] This finding indicates that the affinity of SUA to blood pressure may at least in part be interrelated by the strong relationship of BMI to both SUA and BP.

Results of our study demonstrated that serum MDA level was significantly increased with increasing BMI level among hypertensive and prehypertensive group ($p<0.0001$) (Table 5). These results were in accordance with Saxena T et al, who noted that MDA level was significantly ($p<0.001$) increased in prehypertensive obese than nonobese as compared with control group.[30] Laboratory tests of overweight subjects (BMI>25) indicate a higher oxidative stress than in subjects with BMI<25.[31] In the present study S. uric acid and S. MDA were significantly and positively correlated with BMI (Figure 1, Figure 2), SBP, total cholesterol (TC) and triglycerides ($p <0.001$). Additionally, the lipid profile is bound to be altered in essential hypertension along with increased oxidative stress.

Obesity drives an oxidative stress by increasing endogenous lipid peroxides products.[32] Inflammation and oxidative stress hastened by obesity may predispose individuals to a higher risk for hypertension. Significant reduction in oxidative stress after dietary confinement and weight loss has been accounted.[33]

CONCLUSION

Obesity is a positive risk factor in the evolution of hypertension, dyslipidemia and insulin resistance. Numerous studies have evidenced the association of over nutrition with hypertension. Obesity can be considered as an autonomous and modifiable risk factor because of its action on blood pressure and cardio-vascular mortality. Serum uric acid and serum malondealdehyde level can be used as oxidative stress marker which helps to detect the risk for development of essential hypertension and its further progression to cardio-vascular diseases. Govern of obesity in the elderly helps in diminution of blood pressure, so emphasis on active lifestyle and a healthy diet are cost efficient measures in enriching the quality of life.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. Comparative Risk Assessment Collaborating Group. Selected major risk factors and global and regional burden of disease. Lancet. 2002;360(9343):1347-60.
2. Williams GH, Braunwald E. Hypertensive vascular disease. In: Harrison's Principles of Internal Medicine. Prentice Hall; 1987.

3. Steyn K, Damasceno A. Lifestyle and related risk factors for chronic diseases. In: Jamison DT, Feachem RG, Makogoba WM, Bos RE, Baingana KF, Hofman JK, Rogo OK, editor. Disease and mortality in Sub-Saharan Africa. Washington: The World Bank; 2006:247-264.
4. Barlow SE. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;120(4):S164-92.
5. Stabouli S, Papakatsika S, Kotsis V. The role of obesity, salt and exercise on blood pressure in children and adolescents. Expert Rev Cardiovasc Ther. 2011;9(6):753-61.
6. Appropriate body-mass index for Asian population and its implication for policy and intervention strategies. Lancet. 2004;363(9403):157-63.
7. Pi-Sunyer FX. Medical hazards of obesity. Annals Internal Med. 1993;119(7):655-60.
8. Tandon K. Obesity, its distribution pattern and health implications among Khatri population, Ph. D. theses. Department of Anthropology, University of Delhi, Delhi, India; 2006.
9. Gupta R, Prakash H. Assessment of dietary ghee intake with coronary heart disease and risk factor prevalence in rural male. J Indian Med Association. 1995;95:67-9.
10. Norman M, Kaplan. Clinical hypertension. 9th ed. USA: Lippincott and Wilkins; 2006:15-16.
11. Garrod A. Observations on the blood and urine of gout, rheumatism and Bright's disease. Medical Chirurgical Transactions. 1848;31:83.
12. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, et al. Seventh report of the Joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension. 2003;42(6):1206-52.
13. Trinder P. Quantitative determination of uric acid in human serum. J Clin Pathol. 1949;22:246-50.
14. Buege JA, Aust SD. The thiobarbituric acid assay methods. Enzymol. 1978;52:306.
15. Mungreiphy NK, Kapoor S, Sinha R. Association between BMI, blood pressure, and age: study among Tangkhul Naga tribal males of Northeast India. J Anthropology. 2011 Dec 25;2011.
16. Rosmarakis ES, Vergidis PI, Soteriades ES, Paraschakis K, Papastamataki PA, Falagas ME. "Estimates of global production in cardiovascular diseases research. Int J Cardiol. 2005;100(3):443-49.
17. Hoeger W, Hoeger S. Lifetime physical fitness and wellness: A personalized program: Cengage Learning. 2010:324.
18. Mancia G, Bousquet P, Elghozi JL, Esler M, Grassi G, Julius S, et al. The sympathetic nervous system and the metabolic syndrome. J Hypertens. 2007;25(5):909-20.
19. Lambert E, Straznicky N, Schlaich M, Esler M, Dawood T, Hotchkin E, et al. Differing pattern of sympathoexcitation in normal-weight and obesity-related hypertension. Hypertension. 2007;50(5):862-8.
20. Yang R, Barouch LA. Leptin signaling and obesity: cardiovascular consequences. Circ Res. 2007;101(6):545-59.
21. Katagiri H, Yamada T, Oka Y. Adiposity and cardiovascular disorders: disturbance of the regulatory system consisting of humoral and neuronal signals. Circ Res. 2007;101(1):27-39.
22. Wu H, Ghosh S, Perrard XD, Feng L, Garcia GE, Perrard JL, et al. T-cell accumulation and regulated on activation, normal T cell expressed and secreted upregulation in adipose tissue in obesity. Circulation. 2007;115(8):1029-38.
23. Guzik TJ, Hoch NE, Brown KA, McCann LA, Rahman A, Dikalov S, et al. Role of the T cell in the genesis of angiotensin II induced hypertension and vascular dysfunction. J Exp Med. 2007;204(10):2449-60.
24. Grassi G, Quarti-Trevano F, Seravalle G, Arenare F, Brambilla G, Mancia G. Blood pressure lowering effects of rimonabant in obesity-related hypertension. J Neuroendocrinol. 2008;20(1):63-8.
25. Jawed S, Khawaja TF, Sultan MA, Ahmed S. The effect of essential hypertension on serum uric acid level. Biomedica. 2005;21:98-102.
26. Facchini F, Chen YD, Hollenbeck CB, Reaven GM. Relationship between resistance to insulin-mediated glucose uptake, urinary uric acid clearance, and plasma uric acid concentration. JAMA. 1991;266:3008-11.
27. Hayden MR and Tyagi SC. Uric acid: a new look at an old risk marker for cardiovascular disease, metabolic syndrome, and type 2 diabetes mellitus: the urate redox shuttle. Nutr Metab (Lond). 2004;1:10.
28. Mazzali M, Hughes J, Kim YG, Jefferson JA, Kang DH, Gordon KL, et al. Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. Hypertension. 2001;38:1101-6.
29. Johnson RJ, Kang DH, Feig D, Kivlighn S, Kanellis J, Watanabe S, et al. Is there a pathogenetic role for uric acid in hypertension and cardiovascular and renal disease? Hypertension. 2003;41:1183-90.
30. Saxena T, Agarwal BK, Sharma VK, Naz S, Lanke P. Paraoxonase activity in prehypertension and its relation to oxidative stress. Biomed Pharmacol J. 2013;6(2):389-94.
31. Taddei S, Ghiadoni L, Salvetti G, Virdis A, Salvetti A. Obesity and endothelial dysfunction. G Ital Cardiol. 2006;7(11):715-23.
32. Ahmad A, Singhal U, Hossain MM, Islam N, Rizvi I. The role of the endogenous antioxidant enzymes and malondialdehyde in essential hypertension. J Clin Diagn Res. 2013;7(6):987-90.
33. Dandona P, Mohanty P, Ghanim H, Aljada A, Browne R, Hamouda W, et al. The suppressive effect of dietary restriction and weight loss in the obese on the generation of reactive oxygen species by leukocytes, lipid peroxidation, and protein carbonylation. J Clin Endocrinol Metab. 2001;86:355-62.

Cite this article as: Shrivastav C, Parekh PA, Kumar IG. A correlative study of body mass index with oxidative stress parameters (serum uric acid and serum malondialdihyde) in essential hypertension. Int J Res Med Sci 2019;7:1252-6.