|| Print ISSN: 2589-7837 || Online ISSN: 2581-3935 ||
International Journal of Medical Science and Diagnosis Research (IJMSDR)
Available Online at www.ijmsdr.com
PubMed (National Library of Medicine ID: 101738824)
Original Research Article
Volume 4, Issue 4; April: 2020; Page No. 01-04

BMI & FAMILY HISTORY – PREDICTORS OF HYPERTENSION

Dr Jaswinder Kaur[1], Dr Lily Walia[2], DR Manjeet Kaur Narula[3]
[1]M.D. Physiology, Associate Professor, Dept. of Physiology, M.M. Medical College & Hospital, Kumarhatti Solan, H.P.
[2]M.D. Physiology, Prof & Head, Dept. of Physiology, M.M. Medical College & Hospital, Mullana, Ambala, Haryana.
[3]M.D. Physiology, Ex Professor, Dept. of Physiology, DMC & H, Ludhiana, Punjab.
Conflicts of Interest: Nil
Corresponding author: Dr Jaswinder Kaur

Abstract:
Background: Hypertension in new era is due to sedentary lifestyle, altered feeding habits, stress & decreased physical activity. Genetic factors have been assumed to be important in genesis of hypertension. BMI is an important factor that leads to development of early hypertension. In present study we will see the correlation of family history of hypertension and BMI in development of early hypertension.
Aims and Objectives: Evaluating the correlation of family history of hypertension and BMI in development of early hypertension.
Methodology: Two groups with 20 subjects each, one having family history of hypertensions and another without any such history were examined and their blood pressure, heart rate and BMI were recorded and analysed.
Observations: Systolic blood pressure and BMI were significantly more in subjects with family history of hypertension.
Conclusion: It was concluded that both family history and BMI can be regarded as predictors of hypertension.
Keywords: Hypertension, BMI, family history, systolic blood pressure.

INTRODUCTION

The prevalence of hypertension has significantly increased among children and adolescents in recent years, and hypertension have affected 20 to 30% of the population worldwide [1–3].The most common cause of having hypertension in new era is sedentary lifestyle, altered feeding habits, stress, decrease physical activity. Genetic factors have been assumed to be important in genesis of hypertension [4]. Hypertension represent a polygenic disorder in which a single or combination of gene act in concert with environmental exposure to contribute only the modest effect of blood pressure [5] thus family history of hypertension is a known risk factor of developing early hypertension in offsprings. Family history (FH) is an important marker of genetic factors, it is often used as an alternative indicator to study the relationship between genetic factors and diseases [6–9]. Stamler et al found that parental history of hypertension was associated with twice the risk of development of hypertension. Men with family history of hypertension are at higher risk of developing hypertension.[10] BMI a ratio of height to weight is an indicator of leaness. Studies have shown that having high BMI is also an important factor that leads to development of early hypertension. Prevalence of elevated blood pressure is associated with the presence of obesity in adolescents and young adults.{11} Studies have proven the genetic susceptibility to complex traits and disorders including blood pressure levels, essential hypertension and obesity are transmitted in recessive manners.[12] Body mass index (BMI) is a comprehensive indicator of the outcome of acquired lifestyle, and closely related to the occurrence of hypertension [13–17]A re-view of meta-analytic studies has shown that general obesity is measured by BMI, central and abdominal obesity is measured by anthropometric indictor such as waist circumference or waist-to-hip ratio, and obesity is associated with a risk of hypertension and cardiovascular disease mortality [18].

MATERIAL AND METHODS

Present study was conducted in Department of Physiology, Dayanand Medical College, Ludhiana. Healthy volunteers between age group of 18-30 years were taken as subjects for the study. Brief history along with general and systemic examination was done. Brief history of hypertension was taken from the medical prescription of the parents and anti hypertensive drugs prescribed to them.
They were divided into two groups:
Group 1 (Test group) - with family history of hypertension (20 subjects)
Group 2 (Control group) - without family history of hypertension (20 subjects)

1. **Inclusion criteria:** Age: 18-30 years, Gender: Either, Non-smoker
2. **Exclusion criteria:** Any acute illness, Smokers, Alcoholics, Involvement in physical training program, any recent illness during past 2 weeks
3. **Testing protocol:** Subjects were called to the department at 9 AM and were given 10 min to acclimatize during which subject was informed about procedure.

3.1 Blood pressure and Heart rate was recorded at rest. Systolic pressure was taken as korotkoff phase

1(appearance of sounds) and diastolic blood pressure was taken as korotkoff phase V (disappearance of sound) (19).

3.2 MAP: It was calculated using formula DBP+1/3PP

3.3 Anthropometric measures: Height was measured to the nearest 0.1 cm, while the subject was standing in erect position with bare feet on flat floor against a vertical scale and with heels touching the wall and head straight. Body Weight was measured using Bathroom weighing scale, while the subject was minimally clothed and without shoes, standing motionless on a weighting scale and it was recorded nearest to 0.1 Kg.[20]

3.4 Body mass index: BMI was calculated using the Quetlet's index. Revised WHO Criteria for Asian Indians was used for the categorization of BMI.[21]

4. **Statistical analysis**: The data thus obtained was analyzed using student t test.

RESULTS

It was observed that SBP was significantly higher in subjects with a family history of hypertension as compared to the control group.

Table 1: Distribution according to SBP

SBP	Test Group	Control Group
Mean	119.80	108.00
SD	12.34	13.99
SEM	2.76	3.13

The two-tailed P value equals 0.0125. By conventional criteria, this difference is considered to be statistically significant.

There was no statistically significant difference in DBP in subjects of both the groups, although it was slightly higher in subjects of test group.

Table 2: Distribution according to DBP

DBP	Test Group	Control Group
Mean	76.00	74.10
SD	6.42	10.43
SEM	1.44	2.33

The two-tailed P value equals 0.5437. By conventional criteria, this difference is considered to be not statistically significant.

BMI in subjects of test group was found to be significantly higher as compared to that of control group.

Table 3: Distribution according to BMI

BMI	Test Group	Control Group
Mean	29.0350	26.2485
SD	2.5432	3.4556
SEM	0.5687	0.7727

The two-tailed P value equals 0.0030. By conventional criteria, this difference is considered to be very statistically significant.

Overall it was observed that subjects in test group had a higher SBP and BMI as compared to the subjects in control group. DBP and MAP were also on higher side in test group as compared to the control group but the difference was not statistically significant.

Table 4: Comparison of mean, SD and p-values of parameters between test and control group

t test	Test group		Control group		p- value
	Mean	SD	Mean	SD	
BMI	29.0350	2.5432	26.2485	3.4556	0.003
SBP	119.8	12.34	108.0	13.99	0.0125
DBP	76.0	6.42	74.10	10.43	0.5437
MAP	90.2665	7.6088	80.4	10.7954	0.1682

The p value of SBP (p value-0.003) and of BMI (p value-0.0125) is statistically significant where as for DBP (p value-0.5437) and MAP (p value-0.1682) are not statistically significant.

DISCUSSION

Our study proved the influence of parental hypertension on the increased adiposity on their offspring by showing significant correlation between BMI and systolic blood pressure between offspring of hypertensive parents and normotensive parents. BMI of group-I individuals was found to be significant higher when compared with Group II individuals with a p-value of 0.0030 (<0.05). Increased BMI either overweight or obesity induces physiological changes which promotes vasoconstriction and sodium retention resulting in elevated blood pressure. Increased levels of leptin, free fatty acids and insulin in obesity augments the sympathetic activity in a mutual way resulting in vasoconstriction, which is magnified by obesity-induced insulin resistance and endothelial dysfunction .Increased renal tubular reabsorption of sodium occurs by an enhanced renal sympathetic nerve activity, the direct effect of insulin, hyperactivity of the renin-angiotensin system and probably by an alteration of intrarenal physical forces.[22*] Similar findings of significant increase in systolic blood pressure and BMI in off springs of hypertensive parents were found by Syed et al and Uehara Y et al in their study.[12,23*] .A cross sectional study in united states reported a significantly higher risk of elevated BP in participants with high BMI[24*]. In another study, BMI was significantly associated with an increased risk of prehypertension [25]; The current study showed the importance of the interactions of different anthropometric indicators of obesity in assessing the risk of hypertension; overweight/obesity can assess cardiovascular risk in children and adolescents[26, 27].

In our study, SBP in test group was more than that in control group (p=0.0074, very statistically significant) .This may be due to augmented sympathetic activity and vagal withdrawal which facilitates the onset of hypertension. This is similar to the finding from a study conducted by GK pal et al, in which they demonstrated the effect of sympathovagal imbalance in off springs of single and both

parent hypertensive. As suggested by the same researcher, both of these findings suggests the influence of increased sympathetic activity, vagal withdrawal and adiposity on the development of hypertension in the offspring of single or both parent hypertensive.[28]

In our study DBP in test group was more than that in control group (p=0.4922) but not statistically significant which in contradictory with the findings of Hassan et al who reported significant increase in diastolic pressure in offspring with hypertensive parents.[29]

MAP was not statistically significant (p=0.1682) in our study where as Alpay Harika et al stated that there mean arterial pressure (MAP) standard deviation scores (SDS) were significantly elevated in children with hypertensive parent.[

Influence of familial hypertension on blood pressure activity and BMI may be attributed to the concept of the variability gene and it could also be due to interactions between different genes directly influencing the regulation of blood pressure response to various physical and psychological stimuli.[31]

Researchers have found significant rise in blood pressure and BMI of children of hypertensive when compared with normotensive parents.[32,33] These studies shows the influence of parental hypertension on blood pressure and BMI, and its occurrence even in the first decade of life. According to Framingham heart study, influence of both paternal and maternal BP levels significantly gets correlated with those of their offspring.[34]. Thus we can conclude from our study that the adults with positive family history of hypertension have higher BMI and SBP and are at risk of developing early hypertension in future. Genetic factors and family history can't be modified but people with such factors can delay the development of hypertension by keeping their weight in control and adopting healthy eating habits and lifestyle.

REFERENCES

1. Rosner B, Cook NR, Daniels S, Falkner B. Childhood blood pressure trendsand risk factors for high blood pressure: the NHANES experience 1988-2008.Hypertension. 2013;62(2):247–54
2. Delles C, Mcbride MW, Graham D, et al. Genetics of hypertension: from experimental animals to humans [J]. Bio Chim Bio physacta. 2010;1802(12):1299–308.
3. Liang YJ, Xi B, Hu YH, Wang C, Liu JT, Yan YK, et al. Trends in blood pressure and hypertension among Chinese children and adolescents: China healthand nutrition surveys 1991–2004. Blood Press. 2011;20(1):45–53
4. Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, et al, editors. Harrison's principles of internal medicine. 15th ed. New York: McGraw Hill;1415.
5. Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, et al, editors. Harrison's principles of internal medicine. 17th ed. New York: McGraw Hill;1549-53.
6. International Consortium for Blood Pressure Genome-Wide Association Studies, Ehret GB, Munroe PB, et al. Genetic variants in novel pathways influence blood pressure and cardiovascular disease risk[J]. Nature. 2011;478(7367):103–9
7. Kato N, Takeuchi F, Tabara Y, et al. Meta-analysis of genome-wide association studies identifies common variants associated with blood pressure variation in East Asians [J]. Nat Genet. 2011;43(6):531–8.
8. Zhang Y, Shen J, He X, et al. A rare variant at the KYNU gene is associated with kynurenines activity and essential hypertension in the Han Chinese population [J]. Circ Cardiovasc Genet. 2011;4(6):687–94.
9. Rafiq S, Anand S, Roberts R. Genome-wide association studies of hypertension: have they been fruitful? [J]. Cardiovasc Transl Res. 2010;3(3):189–96
10. Wang NY, Young JH, Meoni LA, Ford DE, Erlinger TP, Klag MJ. Blood pressure change and risk of hypertension associated with parental hypertension: The John Hopskins Precursors Study. Arch Intern Med 2008;168:643-8
11. Mo R, Omvik P, Lund Johansen P. The Bergen blood Pressure study: offspring of two hypertensive parents have significantly higher blood pressures than offspring of one hypertensive and one normotensive parent. JHypertens.1995;13(12Pt2):1614-7
12. Kazim, S, Salman, M, Zubairi, A, Afzal, A, Ahmad, U, Frossard P. Offsprings of hypertensive parents have higher blood pressure and BMI. J Coll Physicians Surg Pak. 2008;18(1):64-5.
13. Tadic M, Cuspidi C, Vukomanovic V, et al. The association between obesity, blood pressure variability and right ventricular function and mechanics in hypertensive patients [J]. J AM Soc Echo cardiogr. 2016;29(8):802–11.
14. Tadic M, Cuspidi C, Pencic B, et al. The interaction between blood pressure variability, obesity, and left ventricular mechanics: findings from thehypertensive population [J]. J Hypertens. 2016;34(4):772–80.
15. Manimunda SP, Sugunan AP, Benegal V, Balakrishna N, Rao MV, Pesala KS. Association of hypertension with risk factors & hypertension related ehaviour among the aboriginal Nicobarese tribe living in Car Nicobar Island, India. Indian Journal of Medical Research. 2011;133(3):287–93.
16. Kotchen TA. Obesity-related hypertension: epidemiology, pathophysiology and clinical management. Am J Hypertens. 2010;23(11):1170–8.
17. Modesti PA, Agostoni P, Agyemang C, et al. Cardiovascular risk assessment in low-resource settings: a consensus document of the European Society of Hypertension Working Group on hypertension and cardiovascular risk in low resource settings. J Hypertens. 2014;32(5):951–60.
18. Millar S, Perry IJ, Phillips CM. Surrogate measures of adiposity and cardio-metabolic risk–why the uncertainty? A review of recent meta-analytic studies. J Diabetes Metab. 2013;S11-004:1–11
19. Ganong WF. Review Of Medical Physiology.22nd Edition: McGraw Hill:549.
20. Ramya K, Mukundan A. Assessment of pulmonary functions in overweight adults- a case control study. Int J Pharm Bio Sci. 2015;6(4):238-43.
21. Physical status: the use and interpretation of anthropometry. Technical report series no. 854, World Health organisation expert committee. Geneva.1995;427-38.
22. Ramya K, Ganesh KJ, Mukundan A. A study of anthropometric indices between hypertensive and normotensive adults. Int J Cur Res Rev. 2014;06(06):36-43
23. Uehara Y, Shin WS, Watanabe T, Osanai T, Miyazaki M, Kanase H. A hypertensive father, but not hypertensive mother, determines blood pressure in normotensive male offspring through body mass index. Journal of Human Hypertension. 1998;12:441-5.
24. Nafiu OO, Zepeda A, Curcio C, Prasad Y. Association of neck circumference and obesity status with elevated blood pressure in children[J]. J Hum Hypertens. 2014;28:263–8.
25. Guo X, Li Y, Sun G, Yang Y, Zheng L, Zhang X, et al. Prehypertension in children and adolescents: association with body weight and neck circumference. Intern Med. 2012;51(1):23–7.

26. Lu X, Shi P, Luo CY, Zhou YF, Yu HT, Guo CY, et al. Prevalence of hypertension in overweight and obese children from a large school-based population in Shanghai [J]. China BMC Public Health. 2013;13:24.

27. Hu YH, Reilly KH, Liang YJ, Xi B, Liu JT, Xu DJ, et al. Increase in body mass index, waist circumference and waist-to-height ratio is associated with high blood pressure in children and adolescents in China [J]. J Int Med Res. 2011; 39(1):23–32.

28. Pal GK, Pal P, Nanda N, Lalitha V, Dutta TK, Adithan G. Sympathovagal imbalance in prehypertensive offspring of two parents versus one parent hypertensive. Int J Hypertension. 2011:1-8.

29. Hassan MO, Al Kharusi W, Ziada A. Blood pressure and its reactivity in the offspring of first cousin hypertensive and first cousin normotensive parents: a preliminary report. J Hum Hypertens. 2001;15:869-72.

30. Harika Alpay, Nihal Özdemir, Elke Wühl & Ahmet Topuzoğlu Ambulatory blood pressure monitoring in healthy children with parental hypertension. Pediatric Nephrology,2009 volume 24, pages155–161(2009)

31. Berg K. Gene-environment interaction; variability gene concept. In: Goldbourt U, de Faire U, Berg K eds. Genetic factors in coronary heart disease. Dortrecht Kluwer Academic Publishers. 1994:153-77.

32. Singhal V, Agal P, Nutan Kamat. The prevalence of elevated blood pressure and the association of obesity in asymptomatic female adolescent offsprings of hypertensive and normotensive parents. J Clin Diagn Res. 2012;Suppl 6(7):1158-61.

33. Kelishadi R, Hashemipour M, Bashardoost N. Blood pressure in children of hypertensive and normotensive parents. Indian pediatrics. 2004(41): 73-7.

34. Havlik RJ, Garrison RJ, Feinleib M, Kannel WB, Castelli WP, McNamara PM. Blood pressure aggregation in families. Am J Epidemiol. 1979;110: 304-12.