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Maternal or paternal history: Which one plays more important role in developing hypertension?

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Original Article

Abstract

BACKGROUND: Hypertension (HTN) is one of the most common non-communicable diseases (NCDs), which in 2017 accounted for 1.65% of all deaths, and 0.66% of disability-adjusted life years (DALYs). About 25% of the adult population are hypertensive in Iran. Prevalence of HTN is significantly higher in those with a family history of HTN. This study compares the impact of paternal and maternal history of HTN on the risk of HTN development.

METHODS: This cross-sectional study was conducted among 2107 adults of 18-84 years old residing in Isfahan, Iran, from August 2015 to March 2016. Blood pressure (BP) measurement standards were taken from World Health Organization (WHO) guidelines. We measured BP in the right arm for three times at 1-minute intervals and considered the mean of second and third measurements. Other data were collected by questionnaire.

RESULTS: Prevalence of HTN was higher in participants whose mother or both parents were hypertensive ($P < 0.001$). Diastolic BP (DBP) was affected by every side of parental history ($P < 0.001$), while systolic BP(SBP) was affected when both parents were hypertensive ($P < 0.001$). As a result, maternal family history increased the odds of HTN by 1.9 times [95% confidence interval (CI): 1.35-2.65] and both maternal and paternal history increased it by 3.1 times (95% CI: 2.01-4.78) compared to those with no family history. However, paternal history was not significantly related to the odds of HTN.

CONCLUSION: Our study results demonstrate that maternal history of HTN doubles the odds of HTN. Besides, if both parents are hypertensive, it will be tripled.

Keywords: Hypertension; Blood Pressure; Medical Family History

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Introduction

Hypertension (HTN) is one of the most common non-communicable diseases (NCDs), which in 2017 accounted for 1.65% of all deaths, and 0.66% of disability-adjusted life years (DALYs). These figures in Iran are 4.51% and 1.43%, respectively. HTN in 2017 was the 4th cause of all death in Iran and in 1990 was the 16th.[1] About 25% of the adult population are hypertensive in Iran.[2,3] On average, 1 in 3 adults in developing countries is hypertensive[4] and global HTN disparities are large and increasing.[5] HTN is the leading modifiable risk factor for cardiovascular disease (CVD) and premature death worldwide. Reductions in risk factors are recommended for the prevention and control of HTN[6] and blood pressure (BP) lowering significantly reduces the cardiovascular risk.[7]

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Significant risk factors for HTN are age, sex, central obesity, alcohol consumption, smoking, socioeconomic status (SES), dyslipidemia, education level, and family history of HTN.[8-11] Researchers have been interested in this subject since 1979.[12] Prevalence of HTN is significantly higher in those with a family history of HTN.[13] Moreover, pediatric healthcare practitioners may not use family history as a screening tool for assessing the future risk of obesity and HTN but instead, gather this information after these chronic conditions have developed, making it difficult to implement preventative or screening strategies based on familial risk.[14]

Besides, it seems necessary to determine the inheritance pattern of HTN for each population, to provide better prevention for at-risk people. Therefore, this study, which is part of IMPROVE-CARE study,[15] aimed to evaluate and compare the impact of paternal and maternal history of HTN on the risk of HTN development.

Materials and Methods

Study design and participants: This cross-sectional study that consisted of the first phase of the IMPROVE-CARE study was conducted on adults of 18-84 years residing in Isfahan, Iran, from August 2015 to March 2016. This study was the second stage of the IMPROVE-CARE study which was conducted in four stages during 2014-2016, and in this stage, the status of prevalence, awareness, treatment and control, and HTN risk factors were assessed. The sample size was 2107. The subjects were selected through multi-stage random cluster sampling and the households were randomly selected in proportion to the population covered by the relevant health centers. More details about the design and sampling have been presented elsewhere.[15,16]

Data collection, variables, and tools: The data were collected by a validated and reliable researcher-developed questionnaire including demographic characteristics and clinical information.

Participants were asked to sign a written consent form and answered questions about their demographic details, including gender, age, years of education, smoking, body mass index (BMI), and history of diabetes and elevated serum lipids.[15,16]

Measurement standards: Given the fact that BP has physiological fluctuations over time,[17,18] it was essential to consider the intra-subject variation and it was minimized by using a properly-calibrated digital automatic BP monitor and applying the World Health Organization (WHO) standards for taking BP, which ensure that a patient's BP is measured in a stable condition. Intra-observer and inter-observer variations were also contemplated and overcome by working with trained observers who were expert in taking BP, using the same tool for all participants, and considering the numbers displayed on the tool's monitor screen instead of traditional method that uses the observer's hearing not to be affected by random errors. Furthermore, BP measurement standards were taken from WHO guidelines to increase accuracy.[19,20] The participants were sitting for a while and were relaxed in a quiet environment for five minutes. We measured BP in the right arm for three times at 1-minute intervals and considered the mean of second and third measurements. Participant was considered as a case of HTN if she or he had the mean systolic BP (SBP) $\geq$ 140 mmHg and/or the mean diastolic BP (DBP) $\geq$ 90 mmHg or if he or she reported having been diagnosed with HTN and taking antihypertensive medications. The participants' height was measured in centimeters in a standing position using a non-elastic measuring tape mounted on the wall and calibrated with a metal measuring tape. Their weight was measured in kilograms using a digital scale, without shoes, and wearing lightweight clothing. The prevalence of pharmacological treatment, control of HTN, and awareness was determined according to definitions presented by Gee et al.[21]

Our data collection tools used in this study included a digital arm BP monitor (Microlife, Widnau, Switzerland), which was compared to a mercury sphygmomanometer and calibrated every 3 months.

In order to maximize the reliability of the data, BP readings were taken by trained healthcare practitioners rather than being self-reported by patients.

The inclusion criteria were being 18 years old and over as well as living in Isfahan. The exclusion criteria were having chronic diseases, including kidney failure and cancer, pregnancy for female participants, and fasting or engaging in a weight gain or weight loss diet at the time of the study.

Ethical considerations: The study was ethically approved under the code of IR.MUI.MED.REC.1398.059. The study objectives were explained to each of the subjects and written consent was obtained from them. The participants' personal information remained confidential.

Statistical analysis: Data were shown as mean $\pm$ standard deviation (SD) for quantitative variables and frequency (percentage) for qualitative variables. One-way analysis of variance (ANOVA) was used to compare quantitative variables among

groups of family history of HTN and chi-square test was performed for qualitative ones. Wherever the test was significant, Bonferroni post-hoc test was performed to determine the different groups. Crude and adjusted logistic regression models were used to evaluate association between family history of HTN and developing it. Data were analyzed using SPSS software (version 16, SPSS Inc., Chicago, IL, USA). P-value < 0.05 was considered as significant.

Results

We enrolled 2107 participants of adult residents of Isfahan aged 18 to 84, including 1017 women (48.3%) and 1090 men (51.7%).

Table 1 demonstrates the gender-specific distribution of HTN with respect to the parental history. Table 1 also presents risk factors and demographic data regarding HTN in the participants.

As shown in table 1, participants whose both parents were hypertensive had higher BMI (P < 0.001) and participants whose mother or both parents were hypertensive (not statistically different between these two groups) were more susceptible to dyslipidemia (P < 0.001).

The prevalence of HTN was not statistically different either between the groups with maternal history and both maternal and paternal history or between the groups with paternal history and negative history. However, the prevalence of HTN was higher in participants whose mother or both parents were hypertensive (P < 0.001).

It is also apparent from table 1 that if both parents were hypertensive, SBP was significantly higher in offspring (P < 0.001). However, SBP in participants with a negative family history or positive maternal or positive paternal history was not statistically different. On the other hand, every side of family history including maternal history, paternal history, and both affected DBP in the same statistical way (P < 0.001). Furthermore, mean arterial pressure (MAP) in participants with a positive paternal and maternal history was remarkably higher, compared to negative, maternal, or paternal history (P < 0.001).

Table 2 reports that according to crude analysis, maternal family history increased the odds of HTN by 1.7 times [95% confidence interval (CI): 1.30-2.21] compared to those with no family history. In addition, both maternal and paternal history increased it by 2.7 times (95% CI: 1.90-3.82) compared to those with no family history.

The table is remarkable in the way adjusted by age and gender and reveals that maternal family history doubled the odds of HTN (95% CI: 1.45-2.78) compared to those with no family history. Likewise, both maternal and paternal history increased it by 3.43 times (95% CI: 2.26-5.23) compared to those with no family history.

In the model adjusted by age, gender, smoking, and BMI, maternal family history increased the odds of HTN by 1.91 times (95% CI: 1.37-2.67) compared to those with no family history, and both maternal and paternal history increased it by 3.11 times (95% CI: 2.03-4.77) compared to those with no family history.

In the other analysis, age, gender, BMI, smoking, diabetes, and dyslipidemia were adjusted. As a result, maternal family history increased the odds of HTN by 1.9 times (95% CI: 1.35-2.65) compared to those with no family history, and both maternal and paternal history increased it by 3.1 times (95% CI: 2.01-4.78) compared to those with no family history.

Table 1. The relationship between hypertension (HTN) development and family history of participants based on parental history

Offspring variable	No history (n = 1088)	Father (n = 272)	Mother (n = 550)	Both (n = 197)	Total (n = 2107)	P
Sex (female)*	491 (45.1)[3]	129 (47.4)	295 (53.6)[1]	102 (51.8)	1017 (48.3)	0.009
Age (year)**	38.34 ± 16.94[3,4]	36.97 ± 13.69[3,4]	41.91 ± 14.29[1,2]	43.02 ± 13.89[1,2]	39.53 ± 15.74	< 0.001
BMI (kg/m^2)**	26.06 ± 4.61[3,4]	26.72 ± 4.89[4]	27.46 ± 4.72[1]	28.39 ± 4.68[1,2]	26.74 ± 4.75	< 0.001
Smoking*	204 (18.9)	46 (17.0)	79 (14.4)	33 (16.8)	362 (17.3)	0.168
Diabetes*	89 (8.2)	20 (7.4)	56 (10.2)	22 (11.2)	187 (8.9)	0.276
Dyslipidemia*	148 (13.6)[3,4]	31 (11.4)[3,4]	115 (20.9)[1,2]	45 (22.8)[1,2]	339 (16.1)	< 0.001
HTN*	152 (14.0)[3,4]	33 (12.1)[3,4]	119 (21.6)[1,2]	60 (30.5)[1,2]	364 (17.3)	< 0.001
SBP (mmHg)**	116.83 ± 15.68[4]	117.23 ± 13.98[4]	118.60 ± 16.07[4]	121.34 ± 17.55[1,2,3]	117.77 ± 15.81	0.001
DBP (mmHg)**	70.99 ± 9.87[2,3,4]	72.82 ± 9.76	73.43 ± 10.53	74.73 ± 10.73	72.21 ± 10.20	< 0.001
MAP (mmHg)**	93.91 ± 11.89[3,4]	95.03 ± 11.17[4]	96.01 ± 12.48[1]	98.03 ± 13.36[1,2]	94.99 ± 12.17	< 0.001

*Data are presented as frequency (%); chi-square test was used.
**Data are presented as mean ± standard deviation (SD); one-way analysis of variance (ANOVA) was performed.
[1,2,3,4]Corresponding to group (or groups) that are significantly different based on Bonferroni post-hoc test
BMI: Body mass index; HTN: Hypertension; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure

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Table 2. The odds ratio (OR) of developing hypertension (HTN) based on family history

Models*	Family history	OR (95% CI)	P
Crude	None	1	
	Paternal	0.85 (0.56-1.27)	0.429
	Maternal	1.70 (1.30-2.21)	< 0.001
	Both	2.69 (1.90-3.82)	< 0.001
Model 1	None	1	
	Paternal	1.32 (0.82-2.12)	0.246
	Maternal	2.00 (1.45-2.78)	< 0.001
	Both	3.43 (2.26-5.23)	< 0.001
Model 2	None	1	
	Paternal	1.28 (0.80-2.05)	0.302
	Maternal	1.91 (1.37-2.67)	< 0.001
	Both	3.11 (2.03-4.77)	< 0.001
Model 3	None	1	
	Paternal	1.27 (0.79-2.05)	0.316
	Maternal	1.89 (1.35-2.65)	< 0.001
	Both	3.10 (2.01-4.78)	< 0.001

*Data adjusted in Model 1 with age and gender, Model 2: further adjusted with age, gender, body mass index (BMI), and smoking, and Model 3: further adjusted with age, gender, BMI, smoking, diabetes, and dyslipidemia
OR: Odds ratio; CI: Confidence interval

Discussion

Our study results demonstrate that parental history of HTN is one of the most important risk factors of having high BP. Maternal history plays a considerable role and increases the odds of upgrading the stage of HTN by 1.89 times. The effect will be more prominent when both parents are hypertensive; this increases the odds by 3.1 times.

Several studies have addressed the association between family history and HTN. For instance, one study in Hanzhong in Shaanxi Province, China, assessed the number of first-degree relatives with positive family history and reported a dose-response relationship in the occurrence of HTN.[22] In another study, it was described that there was a significantly linear-trend increase in HTN according to the family history of first-degree relative numbers and family history had a graded association with HTN as well as BP levels among the people with and without HTN.[23] As detailed, the study of Igarashi et al. showed that family history of HTN over two generations with both parents affected was the most important risk factor for the incidence of HTN, and parental history of HTN was a principal component of family history of HTN.[24]

This paper sheds new light on the fact that parental history of HTN, especially maternal history, plays an important role in having high BP. Besides, in case both parents are hypertensive, we should be more cautious because of its triple effect on developing high BP. As a result, these findings highlight the role for maternal genes in developing HTN. As it is not possible to change the genetics and family history and considering the fact that family history is one of the most important non-modifiable risk factors of HTN, we should strongly consider the modifiable risk factors.

According to the WHO, we can prevent the high BP by reducing salt intake (to less than 5 g daily), eating more fruits and vegetables, being physically active on a regular basis, avoiding use of tobacco, reducing alcohol consumption, limiting the intake of foods high in saturated fats, eliminating trans fats in the diet, and managing mental stress. Modifying other risk factors can lead to a decreased risk of HTN among individuals with a family history of HTN.[25]

Based on our findings, parental history of HTN is one of the most important risk factors of being hypertensive, but further studies are needed to strengthen the outcomes. Moreover, the effects of the other sides of family history such as the sibling or the offspring side should be investigated further. It is also suggested to study family history of HTN and other risk factors in healthy individuals to explore factors leading to high BP.

Women with two hypertensive parents and elevated norepinephrine (NE) levels had higher SBP and DBP during sleep/wake periods. In men, the combination of two hypertensive parents and high NE was related only to DBP during waking.[26]

Compared with normotensive offspring of normotensive parents, normotensive offspring of hypertensive parents had increased BP and impaired arterial properties.[27] In consistence with our findings, it was noted in a multicenter study by Lascaux-Lefebvre et al. that maternal-only history was a stronger risk factor in developing HTN compared to paternal-only history after adjusting for sex, age, educational level, sedentary lifestyle, alcohol consumption, BMI, and low-density lipoprotein (LDL) cholesterol level.[28] Previous studies have shown a causal association between mutations in mitochondrial deoxyribonucleic acid (mtDNA) and the development of HTN amongst patients, explained by maternal inheritance of mtDNA.[29,30]

The present study was mainly limited to a single province and was also limited in terms of timeline and study population. Despite measuring BP by WHO guidelines, the results will be more accurate by using ambulatory BP holter monitoring. Future studies are needed with larger sample sizes as well as longer study periods to evaluate the current

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evidence on the hereditary nature of HTN. Similar to any other chronic conditions, HTN develops over a period of many years. Therefore, conducting cohort studies with regular follow-ups would be a plausible strategy to attain robust evidence. Future studies may also benefit from a more comprehensive approach towards establishing hereditary associations in developing predictive models of HTN amongst families by including siblings and extended family members over successive generations. Furthermore, in vitro and in vivo genetic studies should assess a potential causative association between maternal mtDNA mutations and risk of HTN development in the offspring.

Conclusion

It is essential to study the genetics and hereditary basis of HTN so as to design a model of HTN risk assessment. Implementation of this model would help in establishing national prevention strategies to reduce the burden of HTN at an earlier stage and prevent lethal complications including myocardial infarction (MI), cerebrovascular accident (CVA), and renal failure.

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Conflict of Interests

Authors have no conflict of interests.

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