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Factors associated with prehypertension in young adults between 20 and 25 years of age

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Acronyms
HT: hypertension
PHT: prehypertension
BP: blood pressure
BMI: body mass index

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ABSTRACT

Introduction: Hypertension is a major health problem worldwide. Prehypertension is a category that has been little studied in young adults.

Objective: To determine the factors associated with prehypertension in young adults between 20-25 years of age.

Method: A descriptive cross-sectional study was conducted in a universe consisting of 257 second-year medical students. A total of 134 young adults, between 20-25 years of age, were selected by simple random sampling in the academic year 2009-2010.

Results: The prevalence of prehypertension was 27.6 %. Males (51.5 %) and white skin subjects (59.7 %) were the most affected. Home environment, a personal history of low birth weight (OR=2.3; p=0.179) and gestational age less than 37 weeks (OR= 2.5; p=0.187) did not influence the possibility of having prehipertensives figures in the subjects of this sample.

Conclusions: The high body mass index (OR=34.1; p<0.001), family history of hypertension (OR=12, p<0.01) and family obesity (χ^2=11.19, p=0.001) were the factors most strongly associated with prehypertension in these young people.

Key words: Hypertension, Prehypertension, Young adults, Risk factors

Factores asociados a la prehipertensión arterial en jóvenes de 20 a 25 años de edad

RESUMEN

Introducción: La hipertensión arterial constituye un importante problema de salud a nivel mundial. La prehipertensión es una categoría que se ha estudiado poco en los jóvenes.

Objetivo: Determinar los factores asociados a la prehipertensión arterial en jóvenes entre 20 a 25 años de edad.

Método: Se realizó un estudio descriptivo y transversal a un universo constituido por 257 estudiantes de segundo año de medicina. Por muestreo aleatorio simple se escogieron 134 jóvenes entre 20 a 25 años de edad, correspondientes al curso académico 2009-2010.

Resultados: Se observó una prevalencia de prehipertensión arterial de 27,6 %. Los del sexo masculino (51,5 %) y de color de piel blanca (59,7 %) fueron los más afectados. El ambiente familiar, el antecedente personal de bajo peso al nacer (OR=2,3; p=0.179) y la edad gestacional menor de 37 semanas (OR=2,5; p=0.187) no influyeron en la posibilidad de presentar cifras prehipertensivas en los jóvenes de esta muestra.

Conclusiones: El índice de masa corporal elevado (OR=34,1; p<0.001), los antecedentes familiares de hipertensión arterial (OR=12; p<0.01) y la obesidad familiar (χ^2= 11,19; p=0.001), fueron los factores más fuertemente asociados a la prehipertensión arterial en estos jóvenes.

Palabras clave: Hipertensión arterial, Prehipertensión, Adultos jóvenes, Factores de riesgo

INTRODUCTION

According to reports from the World Health Organization[1], chronic noncommunicable diseases are the leading cause of death worldwide. In this regard, there are forecasts that claim that between 2010 and 2020 the number of deaths from these diseases will increase in 15%, that is, approximately 44 million people. Contrary to popular opinion, many of the deaths from these diseases are occurring in poor and developing countries[2].

Hypertension (HT) alone caused more than 7 million deaths worldwide in 2010[2,3]. Hence it has become a serious health problem everywhere, not only because of its prevalence, affecting up to one third of world population[1], but as a risk factor directly related to diseases in other systems that may lead to ischemic heart disease, heart failure, cerebrovascular disease and chronic renal failure, among others[3-5].

In 1980, there were some 600 million hypertensive people worldwide, but this figure rose dramatically in 2008, reaching one billion people[4]. It is striking its prevalence in certain African regions (46 %), but it also affects 30 % of the US population[5], 40 million people in Japan[6], and in European countries such as Spain, it affects 30 % of the population[7].

Many Latin American countries are currently in a stage of epidemiological transition, due to various circumstances, with an increase in the prevalence of HT. Central America has also been affected. In Costa Rica hypertensive disease has a prevalence of 36.7 %, in both sexes, Guatemala 32.3 %, Nicaragua 34.3 %, and El Salvador 31.9 %[1].

In 2005, the prevalence of hypertensive patients diagnosed by the Primary Health Care System in Cuba was between 28-32% of the total population, that is, about two million people[8], and the estimated prevalence rate could be 202.7 per 1 000 inhabitants in 2010. However, for ages between 20 and 24 years the rate was 88.9 per 1 000 inhabitants[8].

The province of Villa Clara, which ranks fifth in the country in terms of prevalence of the disease, showed a rate of 217.3 per 1 000 inhabitants[9].

The seventh report on high blood pressure[10] proposed the concept of prehypertension (PHT). And since its classification, numerous studies have assessed its actual role in the development of HT itself, and its influence on cardiovascular disease.

The prevalence of PHT in the US is estimated at 28% (women 23%, men 40 %), accounting for about 70 million people in this country, and is more prevalent in those under 60 years of age than in those over 60 (34 *vs.* 24%), in which HT is more common[11].

The study of young adults in search of factors associated with PHT allows the early detection and gives the possibility of implementing early preventive actions. Placing them into this category highlights the increased risk and the consequent possibility of developing HT (10% per year). Additionally, it has been shown that the risk of developing a coronary or cerebrovascular syndrome is double in patients with a systolic blood pressure (BP) of 135 mmHg compared to those with 115 mmHg. That is why it is necessary to identify those people with these levels of BP, which were previously considered to be normal, but have been proven to have future implications[5,12,13].

In summary, it could be said that by means of the identification of hypertensive disease in its early stages, such as the prehypertensive stage and its associated factors, it is possible to exert an influence on a prepathogenic state in order to delay its onset, probably for years, and avoid the existence of an important risk factor for other more deadly and disabling diseases[3].

The objective of this study was to determine the prevalence of PHT, and the factors associated with it, in young adults between 20 and 25 years of age.

METHOD

A descriptive cross-sectional study was conducted in a universe consisting of 257 second-year medical students (academic year 2009-2010) at the Dr. Serafín Ruíz de Zárate Ruíz Medical University in Villa Clara, Cuba.

Sampling

Initially, a random cluster sampling was conducted among 9 groups of second-year medical students, selecting 5 groups (N=257), in a proportional way. Subsequently, 134 young adults between 20 and 25 years of age were selected by simple random sampling.

Informed consent was obtained from all participants. Their BP was measured in three occasions. According to the criteria established in the seventh report[10], two categories were defined: Normotensive (< 120 and < 80 mmHg) and PHT (systolic blood pressure from 120 to 140 mmHg and diastolic blood pressure from 80 to 90 mmHg). Each student filled out an epidemiological questionnaire in order to determine the factors associated with PHT. Their anthropometric measurements for weight and height were taken in order to determine the body mass index (BMI), using the formula of weight in kilograms divided by the square of height in meters.

Measurement of BP

The procedure was explained to the subject. Then, after a 15-minute rest, and making sure that he/she had not eaten, smoked or done exercise for at least 30 minutes before measurement, the subject sat on a sturdy chair with back support, and with the bare right arm flexed at heart level and resting on a table, the BP measurement was taken. A previously calibrated aneroid sphygmomanometer was used. It had an inflatable cuff that covered two thirds of the arm length and its circumference. The cuff was inflated to 20 mmHg above the pressure at which radial pulse is blocked, and was slowly deflated. The pressure at which the first Korotkoff sound was heard was recorded as the systolic pressure; and the pressure at which the Korotkoff sounds disappeared was recorded as the diastolic pressure.

Definition of variables

PHT: Subjects whose systolic BP levels were equal to or greater than 120, and less than 140 mmHg, and with a diastolic BP between 80 and 90 mmHg[10].

HT: Systolic/diastolic BP levels greater than or equal to 140/90 mmHg in 3 or more occasions[10].

Home environment (good, fair and poor): Depending on the individual's perception of the home environment in terms of quarrels and conflicts among family members.

Birth weight: Normal weight ≥ 2 500 and low birth weight < 2 500 grams.

Gestational age at birth: Full term ≥ 37 weeks and preterm <37 weeks.

Information processing

Quantitative and qualitative variables were used. The former were summarized by absolute numbers and percentages. Factors associated with PHT were analyzed, first in a univariate way by calculating the odds ratio (OR), with 95 % confidence intervals, when they included a unit they were not considered as risk. The risk was also recalculated by the method of Mantel-Haenszel, in case there were variables with a certain degree of confusion. Then a multivariate analysis was held using a simple logistic regression to determine the factors associated with the fact of having prehypertensive BP levels. The model was evaluated by the statistical significance associated with Chi-square of Hosmer and Lemeshow test, if it was greater than

0.05, the null hypothesis that the model fitted the data was not rejected.

Ethical considerations

The study was approved by the ethics committees of the hospital and the Medical University. Ethical research principles were followed, respecting the subjects' autonomy and self-determination. All subjects signed the informed consent form.

RESULTS

Of the 134 students included in the sample, 27.6 % (37) showed BP levels within the range of PHT. The rest showed BP levels within normal parameters.

In general, male subjects predominated (51.5%), as well as those with white skin (59.7 %). Among the 37 prehypertensive young adults, 62.2% were male and 56.8 % had white skin (**Table 1**). Females (52.6 %) and white skin color (60.8 %) predominated among normotensive subjects.

No statistically significant difference ($p > 0.05$) was found in the two variables among normotensive subjects.

With regard to home environment, 91.0 % of the subjects included in the study felt that it was good (**Table 2**). A similar result was found by dividing them into prehypertensive (91.9%) and normotensive subjects (90.7%).

Among those diagnosed with PHT, there was a predominace of those with a BMI ≥ 25 (67.6%), a birth weight ≥ 2500 grams (91.9 %) and a gestational age ≥ 37 weeks (83.8%). Consequently, the probability of having prehypertensive BP levels was much higher for those who had a BMI ≥ 25 (OR = 4.1), and it was not related to prematurity or low birth weight, with confidence intervals for the respective OR that included a unit (**Figure 1**).

Among the prehypertensive subjects, 70.3 % had a family history of hypertension and 56.8 % of obesity; however, family histories of heart disease (16.2%) and

Table 1. Distribution of prehypertensive subjects, according to sex and skin color.

Sex and skin color	Prehypertensive				Total	
	Yes		No			
	Nº	%	Nº	%	Nº	%
Sex*						
Male	23	62,2	46	47,4	69	51,5
Female	14	37,8	51	52,6	65	48,5
Color of skin**						
White	21	56,8	59	60,8	80	59,7
Non white	16	43,2	38	39,2	54	40,3
Total	37	100	97	100	134	100

Source: Questionnaire
*p=0.176 **p=0.697

Table 2. Distribution of prehypertensive subjects, according to home environment and personal history.

Variable	Prehypertensive				Total	
	Yes		No			
	Nº	%	Nº	%	Nº	%
Home environment						
Good	34	91,9	88	90,7	122	91,0
Fair	2	5,4	8	8,2	10	7,5
Poor	1	2,7	1	1.0	2	1,5
BMI (kg/m^2)						
≥25	25	67,6	4	4,1	29	21,7
<25	12	32,4	93	95,9	105	78,3
OR=34,1; IC 95% (LL-UL): 10,3-112,9; p<0.001						
Birth weight (g)						
< 2500	3	8,1	5	5,2	8	6,0
≥ 2500	34	91,9	92	94,8	126	94,0
OR=2,3; IC 95% (LL-UL): 0,7-8,2; p=0.179						
Gestational age at birth (weeks)						
< 37	6	16,2	7	7,2	13	9,7
≥ 37	31	83,8	90	92,8	121	90,3
OR=2,5; IC 95% (LL-UL): 0,8-8,0; p=0.187						

Source: Questionnaire
LL: Lower limit, UL: Upper limit

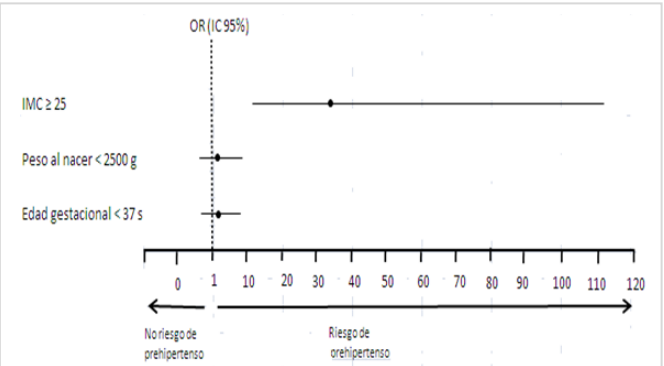

Figure 1. Odds ratio for prehypertension, according to personal history.

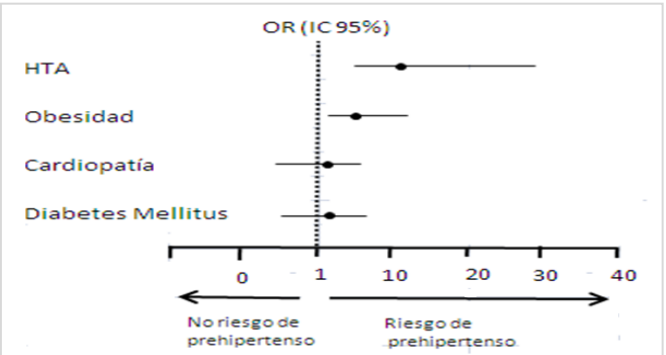

Figure 2. Odds ratio for prehypertension, according to family history.

Table 3. Distribution of prehypertensive subjects, according to family history.

Family hystory	Prehypertension				Total	
	Yes		No			
	Nº	%	Nº	%	Nº	%
HT						
Yes	26	70,3	16	16,5	42	31,3
No	11	29,7	81	83,5	92	68,7
OR=12; IC 95% (LL-UL): 4,9-29; p<0.01						
Obesity						
Yes	21	56,8	14	15,5	35	26,1
No	16	43,2	83	85,5	99	73,9
OR=5,2; IC 95% (LL-UL): 2,2-12,1; p<0.01						
Heart disease						
Yes	6	16,2	10	10,3	16	11,9
No	31	83,8	87	89,7	118	88,1
OR=1,7; IC 95% (LL-UL): 0,5-5; p=0.377						
Diabetes						
Yes	5	13,5	7	7,2	12	9
No	32	86,5	90	92,8	122	91
OR=2; IC 95% (LL-UL): 0,6-6,8; p=0.311						

Source: Questionnaire
LL: Lower limit, UL: Upper limit

diabetes (13.5 %) were not representative (**Table 3 and Figure 2**), and did not constitute an important risk for PHT. HT and obesity did represent significant risks to have prehypertensive BP levels, with an OR of 12 (4.9 to 29) and 5.2 (2.2 to 12.1), respectively.

Familial obesity was associated with the possibility of having PHT, because 56.8% of these subjects had this background. However, obesity in first-degree relatives functions as a confounding variable, since it is also associated with the BMI in the subjects, and 61.6% of those with BMI ≥ 25 had, in turn, obese family members (**Table 4**). This relationship with PHT and the BMI of the subjects could modify the real risk of being prehypertensive. To be able to determine the clear influence of this variable, it was decided to adjust for the risk and control the variable with Mantel-Haenszel statistics (**Table 5**). It was also possible to determine that, when stratifying the variable family obesity, the association between BMI and PHT was highly significant, by obtaining a Mantel-Haenszel common OR of 22.4; with a 95% confidence interval (6.6 to 76.1). However, the previously calculated OR (**Table 2**) showed that the unstratified BMI was 34.1; and it decreased by 12 units after stratification (**Table 5**). Therefore, family obesity functioned as a positive confounder, because when it was not analyzed, it increased the risk, and at the same time, it allowed determining that there was an interaction between family obesity and the BMI of subjects

Table 6 shows the variables that significantly influence PHT, according to the saturated model that includes the interaction between BMI and family obesity. Family HT showed an OR of 83.95 and the BMI an OR of 35.41. Family obesity alone did not represent a

Table 4. Distribution of subjects according to BMI and family obesity.

Family obesity	Body mass index				Total	
	≥25		<25			
	Nº	%	Nº	%	Nº	%
Yes	16	61,6	19	17,6	35	26,1
No	10	38,4	89	82,4	99	73,9
Total	26	100	108	100	134	100

Source: Questionnaire
χ^2= 11,19 p=0.001

Table 5. Distribution of subjects according to prehypertension and BMI, after adjustment for family obesity.

Family obesity	BMI	Prehypertension				Total	
		Yes		No			
		Nº	%	Nº	%	Nº	%
Yes	**≥ 25**	14	61,1	2	14,3	16	39,4
	< 25	7	38,9	12	85,7	19	60,6
	Subtotal	21	100	14	100	35	100
	OR=10,2; IC 95% (LL-UL): 1,7-59,7; p=0.01						
No	**≥ 25**	11	57,9	2	2,4	13	12,9
	< 25	5	42,1	81	97,6	88	87,1
	Subtotal	16	100	83	100	99	100
	OR=55; IC 95% (LL-UL): 10,3-293; p=0.00						
	Total	37	27,6	97	72,4	134	100

Source: Questionnaire
Mantel-Haenszel common OR (95 % CI)=22,4 (6,6 – 76,1)

Table 6. Logistic regression.

Variables	ET	Wald	p	Exp (B)	IC 95%	
					Lower	Upper
Family HT	1,10	15,99	0.000	83,95	9,58	735,95
BMI	1,30	7,50	0.006	35,41	2,76	454,31
Family Obesity	2,53	0,26	0.610	0,28	0,00	39,09
BMI and family obesity	0,41	5,30	0.021	2,60	1,16	5,85

Hosmer and Lemeshow: χ^2=1,29 p=0.8623

risk, according to this model; and the interaction between these two variables (BMI ≥ 25 and a family history of obesity) had a probability of risk of 2.6 compared to those with BMI < 25 and had no family history of obesity.

DISCUSSION

Since the introduction of the concept of PHT, its characteristics have been studied in many regions in order to determine the number of patients who are included in this new category. With this aim, a Ja-

panese study[14] assessed 12 000 patients of both sexes and found a PHT prevalence of 34.3%. Similar results were found in Taiwan and Korea (31.6 and 34 %, respectively)[15,16]; however, in an research conducted by Cuban internationalists in the state of Tachira, in Venezuela, a slightly lower figure was reported (29.6%)[8], a result that is similar to those found in our study (27.6%).

Without doubt, the importance of PHT is that it is a prerequisite to a worse condition such as HT. Also, compared to normotensive individuals, PHT has a higher cardiovascular risk, as demonstrated in a cohort in the Strong Heart Study[11], where 2 629 PHT patients were followed over 12 years, finding that the BP levels increase cardiovascular risk 1.8 times, independently, and 3.7 and 2.1 times when associated with diabetes mellitus or with impaired glucose tolerance.

Liszka *et al*[17] studied a cohort of 8 986 patients with PHT, and followed them for 18 years with the aim of assessing the risk of cardiovascular events (occurrence of myocardial infarction, cerebrovascular disease and heart failure), showing that these BP levels were independently associated with a 1.32-time increase in cardiovascular events, after adjustment for other variables, including other risk factors.

Moreover, it has been observed that the PHT tends to progress to HT. In this regard, the Framingham Heart Study[18] noted that with values in the range of 120-129/80-84 mmHg, in 4 years, the PHT progressed to HT in 17.6 % of individuals aged 30-64 years, and in 25.5 % of those over 65. However, in the group with BP levels in the range of 130-139/85-89, progression to HT was 37.3 % in individuals less than 65 years of age and 49.5 % in those over 65 years.

Compared with normotensive adults, it is known that prehypertensive subjects also have a higher prevalence of certain risk factors[17]. Most studies agree that the male sex is a risk factor for having PHT. Ganguly *et al*[12] found that the risk increased 2.3 times. An Israeli study[19] found prehypertensive values in 50.6 % of men and 35.9 % women. Ferguson *et al*[20], in Jamaica (n = 1972), reported a 35 % prevalence of PHT in the male population, the same as Toprak *et al*[21].

The mechanisms behind this phenomenon include those related to the known hormonal differences and a higher prevalence of risk factors in men[22]; however, some changes are observed in contemporary women, due to the stress of modern life, including stressful professions, and also because they have been developing toxic unhealthy habits such as smoking, and have increased alcohol consumption. These aspects were considered some years ago as limited to males[23]. All this has implications and involves an increase in the prevalence of women with PHT or HT, as it was demonstrated by Li *et al*[24] in China, who observed that the proportion of PHT between men and women was almost similar.

It is recognized that stress is related to the increase in BP in healthy individuals. Thus it is considered a risk factor for developing PHT, although its complexity and the lack of measures to assess it, together with the fact that not everybody reacts to it in the same way, have limited all available tools[25]. However, it seems clear that the states of psychological stress in work, social and family situations, are the source of many diseases, including HT[26-29].

There is a close relationship between weight gain and an increase in BP. According to the Framingham Heart Study[18], obesity explains 78 and 65% of essential hypertension in men and women, respectively. The link between obesity and other cardiovascular risk factors is insulin resistance, which has also been observed in patients with PHT[30]. People who are overweight or obese, have hyperinsulinemia and insulin resistance. Its production mechanisms (in addition to those related to the insulin receptor) include hyperleptinemia, hypercortisolemia, vascular alterations, hyperreactivity of the sympathetic nervous system and the renin-angiotensin system, and natriuretic peptide activity, all of which explain the gradual increase in BP in the subject with increased BMI[30-33].

A Japanese study[34] found that a BMI ≥ 25 was the determinant most strongly associated with the likelihood of having PHT, moreover, Grotto *et al*[19] showed that BMI was a strong predictor of PHT in over 36 000 young Israelis, with an increase in BP levels per each kg/m^2 of weight gained. Ganguly *et al*[12] were able to show that a BMI ≥ 25 represented a risk of suffering from PHT 2.25 times higher compared to the risk in those with a lower BMI.

This study found that the majority of young prehypertensive adults (67.6%) had a BMI ≥ 25, and when it was analyzed through a logistic regression model, it represented a significant risk to develop PHT, together with a family history of hypertension and family obesity.

The "fetal programming" describes a process in which the intrauterine environment induces changes

that affect the fetus and lead to increased susceptibility to certain diseases for the rest of life. Barker[35] was one of the pioneers in this theory, as he demonstrated this relationship by observing that patients with a history of low birth weight had a BP 5.2 mmHg higher than those without this background, which was associated in turn with a greater likelihood of cardiovascular disease in adulthood. Some authors believe that this relationship is stronger with advancing age[36-38], with a higher impact on males[39]. On the other hand, it has been successfully demonstrated, by means of autopsies, that there are fewer nephrons in patients with a history of low birth weight who, at the same time, had high levels of BP[40]. Similarly, Tian *et al*[41], in China, confirmed the influence of low birth weight on BP levels, which also become a powerful predictor of type 2 diabetes mellitus when associated with abdominal obesity. By contrast, there are studies in young adults which found that 45.9% of those with prehypertensive BP levels did not have the influence of low birth weight or gestational age at birth[42]. Despite variations in the results of different studies, it appears that low birth weight and low gestational age at birth influence the development of hypertensive disease. However, our study failed to demonstrate this relationship, probably because the sample was small, although it is necessary to clarify that these results may be influenced by the characteristics of the prenatal care under the Mother and Child Health Care Program in Cuba, which has achieved significant results in reducing low birth weight in recent years.

A family history of HT significantly predicts the future onset of the disease in members of that family. The strength of prediction also depends on the sex and age of the person at risk; and the more first-degree relatives suffer from it the greater the risk. Genes play an important role in the pathogenesis of hypertensive disease, although the identification of specific genes is still limited. However, several studies[43-45] have related some of them to the progressive increase in BP. Moreover, a research conducted in Australian twins showed that heritability estimates for systolic BP was 19-56 %, and for diastolic, 37-52 %[46].

A study that included 41 pairs of twins, in the town of Chambas, Ciego de Ávila province, Cuba[47], found that the match of HT was 38% in monozygotic twins and 18 % in dizygotic twins, which shows that the more genes are shared the greater the probability of having increased BP.

There is a study in adolescents, also conducted in Villa Clara province[48], which shows similar results to ours. The study found that a) a family history of hypertension was present in 25.3 % of the adolescents in the study; b) most prehypertensive adolescents had a family history of obesity, c) which in turn was related with increased BMI. Therefore, weight gain is influenced by family history due to the interaction of genes and the environment in which these individuals develop.

There is no doubt that the explanation for this phenomenon involves different aspects. Both genetic and environmental aspects are combined. Therefore, it is important to consider the influence of bad eating habits, due to excessive consumption of fat and carbohydrates, in addition to a lack of physical exercise that predisposes to weight gain, overweight and obesity[49], which leads to the appearance of other chronic diseases such as diabetes mellitus and hypertensive disease[50,51].

CONCLUSIONS

The factors associated with PHT in these young adults aged 20 to 25 years included a BMI ≥ 25, a family history of hypertension and family obesity. Home environment, gestational age at birth and low birth weight were not associated with the probability of PHT.

RECOMMENDATIONS

It is necessary to conduct longitudinal studies with a larger number of subjects, in order to determine its progression, cardiovascular risk and the development of future HT.

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