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Changes over-time in blood pressure of women with preeclampsia compared to those with normotensive pregnancies: A 15 year population-based cohort study

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ABSTRACT

Objectives: To estimate the incidence of hypertension and the trend of systolic and diastolic blood pressure changes and relating factors influencing women with and without prior preeclampsia (PE).
Study design: This prospective population based study included a total of 3022 eligible women (355 with PE and 2667 non-PE) recruited from participants of the Tehran Lipid and Glucose Study (TLGS) who were assessed for progression to subsequent hypertension over 15-year follow up. Pooled logistic regression model was utilized to estimate odds ratio (OR) of hypertension. The generalized estimating equation (GEE) was used to evaluate the trend of changes in hypertension parameters over time.
Results: At the end of follow-ups, 109 women (30.7%) in the PE group and 575 (21.5%) in the non-PE group had hypertension. The total cumulative incident rate of hypertension was 34/1000 person-years for PE groups and 22/1000 person years for non-PE groups ($P < 0.001$). Pooled logistic regression analysis showed that compared to non-PE women, OR of hypertension progression in women with PE was 3.70 after adjustment for age, body mass index (BMI), parity, triglycerides (TG) and high-density lipoprotein (HDL-C) (P-value < 0.001). Based on GEE analysis, mean changes of systolic and diastolic blood pressure in PE women increased by 4.66 and 2.55 mmHg, respectively, compared to the non-PE group, after adjustment for age, and BMI at baseline ($P < 0.001$), although the interaction term (follow-up year × PE) was not statistically significant.
Conclusion: This study demonstrated increased chances of developing hypertension among women with prior PE, particularly in those who develop additional risk factors in their later life, compared to the non-PE women. While the trajectory of blood pressure change over time is similar between women with and without pre-eclampsia, women with a history of preeclampsia consistently have higher levels of blood pressure.

1. Introduction

Preeclampsia (PE), considered a serious condition in pregnancy, is characterized by new-onset hypertension (HTN) [blood pressure (BP) ≥ 140/90 mmHg] and proteinuria of at least 300 mg in 24-hours after 20 weeks gestation. This disease, one of the most important causes of maternal mortality ratio (MMR) and morbidities (acute and long-term), which globally complicates approximately 3–5% of pregnancies [1]. In Iran, the prevalence of PE is estimated at approximately 5% of pregnancies [2].

Pregnancy is associated with insulin resistance, hyperlipidemia [3] hypercoagulability [4], inflammation [5], and a hyper-dynamic circulation [6]. In women with PE these changes are often exaggerated [7]. While HTN and proteinuria associated with PE generally resolve soon after delivery [8], several studies have been demonstrated that PE increases risk of future cardiovascular (CVD) disease in affected women [8–12]. One cohort study reported a five-fold increase in HTN rates in the first five years after a preeclamptic pregnancy [12].

Although the underlying mechanisms of progression of PE to chronic HTN in later life are not well understood, it seems that it could be because of a shared etiology or due to long-term metabolic and vascular damage that occurred during PE [9].

Previous studies show a vascular endothelial dysfunction in pregnant women with PE [13]. In addition, long-term vascular endothelial

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impairment has also been detected in women with a history of PE [14].

Although several studies have assessed later risk of HTN in women with previous gestational hypertensive disorders, they had mostly small sample sizes [10,11,15–19], inappropriate comparison groups [10,11,20,21], or relatively short follow ups [12,15,19,22,23]. In addition, most studies were not adjusted for other risk factors such as age, BMI and time [16,21,24–26], were not population based [8,11,15–19,27,28], or had retrospective methodology [11,16,18,24–26,28]. Despite these limitations the short term consequences of PE and its risk for developing subsequent HTN are relatively well known, while trend of BP changes over time is still a controversial and debatable issue [8,29,30]. This information can help clinicians to make timely decisions regarding the post-pregnancy follow-up and diagnosis of HTN.

In this longitudinal study, we aimed to estimate the incidence of HTN and its influencing factors in women with and without prior PE history, and to simultaneously investigate the trend of systolic and diastolic blood pressure changes over time in both groups.

2. Materials and methods

2.1. Subjects

We used data from the Tehran Lipid and Glucose Study (TLGS) [31], an ongoing prospective cohort from a geographically defined population in Tehran, initiated in 1998 to assess the prevalence and risk factors of atherosclerosis.

A multi-stage stratified cluster random sampling technique was applied to select 15, 005 individuals aged ≥3 years from urban district 13 of Tehran. A random sample of the households under coverage of the district's three healthcare centers, was selected to achieve a distribution similar to the original population. In each household, all members above the age of 3 years were recruited. A number of 10, 368 adults (4397 men and 5971 women), aged ≥20 years participated in TLGS [32]. All of the participants were visited at TLGS clinic between February 1999 and August 2001. After signing written informed consent forms, the participants interviewed, were assessed for outcomes by trained examiners at each follow-up; they had follow-up visits every 3 years and demographic, anthropometric, reproductive and metabolic features and general physical examinations as well as laboratory measurements were documented. To date TLGS has completed five phases at 3-year intervals (phase 1: 1999–2001, phase 2: 2002–2005, phase 3: 2005–2008, phase 4: 2008–2011 and phase 5: 2011–2014). Current data are available for five phases, including baseline and four follow-ups.

We examined all women, aged 20–50 years with at least one a pregnancy at the beginning of the study (n = 3858) and excluded those with HTN at baseline (n = 478) and those with no follow up visits (n = 358). Of the remaining 3022 women, 355 had history of PE and 2667 had none. All participants were present at baseline of the study and had at least one follow-up visit; otherwise they were considered as lost to follow-up cases. As shown in the Supplementary Fig. 1, 21 and 328 women were lost to follow-ups for PE and non-PE groups, respectively.

We included all pregnant women with and without PE, regardless of term or preterm termination of pregnancy. For those women who could not recall their pregnancy complications, or were not sure about having both hypertension and proteinuria or a definite diagnosis of PE, we assessed their summary from hospital records.

2.2. Ethics considerations

The current study was approved by the medical ethics committee of the Research Institute for Endocrine Sciences. Code: IR.SBMU.RIES.REC.1394.125.

2.3. Measurements

All subjects were interviewed for their history of pregnancies, and to obtain medical, obstetrics and family histories using pretested questionnaires. At the time of data collection, women were asked about their history of preeclampsia, based on a self-reporting questionnaire at each follow-up, details of which have been previously published [33,34]. Clinical and anthropometric measurements were assessed by trained examiners at each follow-up, details of which have been previously published [33]. In summary, weight was measured when they were minimally clothed using a digital scale (Seca 707, Seca GmbH) and rounded to the nearest 100 g. Height was measured without shoes in the standing position with shoulders in normal alignment, using a tape measure. Waist circumference was measured with an unstretched tape measure at the level of umbilicus, without any pressure to the body surface and recorded to the nearest 0.1 cm. Hip circumference was measured at the level of anterior superior iliac spine without any pressure to the body surface. Body mass index (BMI) was calculated as weight in kilograms (kg) divided by height squared (m2). SBP and DBP were measured twice on the right arm with the subject in a seated position with a standard mercury sphygmomanometer after the subject sat for 15 min; the mean of these 2 measurements was recorded.

Triglyceride (TG) levels were assayed using glycerol phosphate. Total cholesterol (TC) was determined using the enzymatic colorimetric method with cholesterol esterase and cholesterol oxidase. The level of high-density lipoprotein cholesterol (HDL-C) was measured after precipitation of the apolipoprotein B (apo B)-containing lipoproteins with phosphotungistic acid. To calculate LDL-C, a modified Friedewald equation was used [35]. Analyses were performed using related kits (Pars Azmon Inc., Tehran, Iran) and a Selecta 2 autoanalyzer (Vital Scientific, Spankeren, Netherlands). Intra-assay and inter-assay coefficients of variations for TG, TC, HDL-C, and LDL-C were less than 2.1, 1.9, 3, and 3%, respectively. All laboratory evaluations were performed at the TLGS research center.

2.4. Definition

For diagnosis of PE, the international standard criteria were used [36]. In our country, PE diagnosis is as a part of routine prenatal care that is determined based on the standard definition of onset of a BP level ≥140/90 mmHg with proteinuria > 0.3 g/24 h after 20 weeks' gestation. At the time of data collection, women were asked about their history of PE based on a validated self-reporting questionnaire [37]. For patients who have no accurate information about their PE diagnosis, we checked their summary from hospital records.

According to the JNC-VI criteria [38], HTN or high BP was defined as mean SBP ≥ 140 mmHg, mean DBP ≥ 90 mmHg, or current use of anti-hypertensive medicine [39,40].

2.5. Statistical analysis

All continuous variables were checked for normality using the one-sample Kolmogorov–Smirnoff test, and expressed as mean (standard deviation) if variables had a normal distribution, or median with interquartile range (IQ25-75) for variables with skewed distribution. Characteristics of participants at the time of recruitment were compared between the PE and non-PE groups using two independent-sample *t*-test or the equivalence nonparametric Mann-Whitney *U* test. The categorical variables, expressed as percentages, were compared using Pearson's test.

To analyze the person-time incidence rate of HTN the following formula was used:

$$\frac{number\ of\ new\ events\ of\ the\ condition\ (cases)\ in\ the\ study\ time}{sum\ of\ person-time\ (person \times year)\ at\ risk\ in\ the\ study\ participants}$$

We used pooled logistic regression to assess the association between the dichotomous outcome variable (PE) and time dependent covariates as the data was interval censored and time to HTN was not known, and to calculate odds ratios (OR) [41]; these covariates included age, BMI, TG, HDL and parity at baseline. This model treats every interval as a mini follow-up study, pools the observations of all intervals together into one pooled sample and does a logistic regression on the pooled dataset. In order to investigate the secular longitudinal trends of SBP and DBP, we used the generalized estimating equation (GEE). We performed pooled logistic regression based on the adjustment levels (stage 1: age-adjusted, stage 2: age and parity-adjusted, stage 3: age, parity, BMI, TG, HDL-adjusted). The GEE approach, by Liang and Zeger, considers the correlation between repeated measurements [42]. Models for examination of the time trend were fitted separately for PE and non-PE groups and marginal means (age-BMI adjusted) and P values for trend are reported in each group. The GEE models were created with an interaction form (follow-up years × PE) to analyze changes in SBP and DBP across time by PE status. This analysis was performed on baseline data, which had the information required in at least one of four follow-up visits, with the following predictors: Time (follow-up years), PE status, and an interaction term of these two (follow-up years × PE group). This model was adjusted for age, BMI, and baseline status of SBP and DBP.

We also conducted the multivariable analysis with time-dependent cox proportional hazard regression assuming the hypertension occurrence, which happens midway between visits. Statistical analysis was performed using software package STATA (version 12; STATA Inc., College station, TX, USA); significance level was set at $P < 0.05$, and confidence interval (CI) as 95%.

3. Results

Baseline characteristics of PE and non-PE women are presented in table 1. At baseline, women with PE were younger (33.75 ± 7.50 years versus 35.65 ± 7.80 years) and had significantly higher waist circumference (88.13 ± 10.80 cm versus 85.99 ± 10.90 cm), BMI (28.11 ± 4.31 versus 27.25 ± 4.28), SBP (113.97 ± 11.10 mmHg versus 109.75 ± 11.06 mmHg), DBP (76.52 ± 7.75 mmHg versus 73.87 ± 8.0 mmHg) and TG (median: 1.50 mmol/L; interquartile range: 1.03–2.13) versus (1.36 mmol/L; 0.94–1) levels (table 1). The median and interquartile ranges for follow-up years of groups were 9.85 (5.53–12.78) and 11.18 (7.21–13.10), respectively.

Table 1
Baseline characteristics of women with and without history of preeclampsia.

Variables	PE (N = 355)	Non-PE (N = 2667)	P-value[d]
Age[a] (years)	33.75 (7.5)	35.65 (7.8)	< 0.001
Parity[a]	2.78 (1.36)	2.18 (1.26)	< 0.001
BMI[a] (kg/m^2)	28.11 (4.31)	27.25 (4.28)	< 0.001
WC[a] (cm)	88.13 (10.08)	85.99 (10.9)	0.001
HC[a] (cm)	105.39 (9.1)	104.06 (8.5)	0.014
Waist to hip ratio[a]	0.84 (0.07)	0.82 (0.07)	0.013
SBP[a] (mmHg)	113.97 (11.1)	109.75 (11.06)	< 0.001
DBP[a] (mmHg)	76.52 (7.75)	73.87 (8.0)	< 0.001
TG[b] (mmol/L)	1.50 (1.03–2.13)	1.36 (0.94–2)	0.004
TC (mmol/L)[a]	5.20 (1.08)	5.10 (1.04)	0.10
LDL-C (mmol/L)	3.31 (0.93)	3.22 (34.14)	0.11
HDL-C (mmol/L)	1.10 (0.26)	1.15 (0.28)	0.003
Family history of HTN[c] (%)	42 (14.7%)	57 (2.6%)	< 0.001
Smoking[c] (%)	21 (5.9%)	116 (4.4%)	0.18

PE = preeclampsia, Non-PE = Non Preeclampsia, BMI = body mass index, WC = waist circumference, HC = hip circumference, SBP = systolic blood pressure, DBP = diastolic blood pressure, TG = triglycerides, TC = total cholesterol, LDL-C = Low-density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, HTN = hypertension.

[a] Values are presented as mean (SD).

[b] Presented as median (interquartile range).

[c] Data shown as percentage.

[d] Significant differences (P-value < 0.05), analyzed using independent *t*-test for superscripts[a], Mann-Whitney *U* test for superscripts[b], and Pearson's χ^2 test for superscripts[c].

Table 2
Pooled logistic regression for hypertension among women with and without preeclampsia.

Variables[§]		Odds Ratio	P-value	95% confidence interval
PE		3.70	< 0.001	(2.81, 4.84)
Time				
	Follow-up 1	Reference		
	Follow-up 2	1.11	0.38	(0.87, 1.43)
	Follow-up 3	1.43	0.003	(1.13, 1.81)
	Follow-up 4	1.50	0.003	(1.15, 1.96)
Age (year)		1.08	< 0.001	(1.07, 1.10)
BMI (kg/m^2)		1.10	< 0.001	(1.08, 1.12)
Parity		1.04	0.18	(0.98, 1.10)
TG (mmol/L)		1.18	< 0.001	(1.10, 1.27)
HDL (mmol/L)		0.66	0.01	(0.48, 0.91)

PE = preeclampsia, BMI = body mass index, TG = triglycerides, HDL-C = high-density lipoprotein cholesterol.

[§] Covariates (age, BMI, TG, and HDL) are time dependent and parity is considered at baseline visit.

At the end of follow-ups, 109 women (30.7%) in the PE group and 575 (21.5%) in the non-PE group had hypertension. The total cumulative incidence rate of hypertension at the median follow-up time of approximately 11 years was 34/1000 person-years (95% CI: 28/1000, 41/1000) and person-years 22/1000 (95% CI: 20/1000, 24/1000) in PE and non-PE women respectively (p-value < 0.001).

According to the pooled logistic regression analysis, the effects of PE on HTN progression [adjusted for age (p-value < 0.001), BMI (p-value < 0.001), parity (p-value = 0.18), TG (p-value < 0.001) and HDL (p-value = 0.01)] were highly significant (OR: 3.70; P-value < 0.001; 95% CI: 2.81, 4.84), indicating that the odds of HTN progression was 3.70 fold higher in women with previous PE than in non-PE women (Table 2). As the table shows, odds of HTN progression increased over time. Also, for a one-unit increase in BMI, we expect to see about 10% increase in the odds of HTN progression; on the other hand, for a one-unit increase in HDL the odds of HTN progression decreased by 34%. Details of pooled logistic regression, based on the adjustment levels (stage 1: age-adjusted, stage 2: age and parity-adjusted, stage 3: age, parity, BMI, TG, HDL-adjusted) are presented as Supplementary Tables 1–4.

Based on GEE analysis, mean changes of SBP and DBP were significantly different in PE group, compared with non-PE women, after adjustment for age, BMI at baseline of this variable ($p < 0.001$) (Table 3). Fig. 1A and B illustrate age-BMI adjusted trends of hypertension parameters in PE and non-PE groups, respectively. As figures show, SBP and DBP demonstrated a significant increase in both PE ($P_{trend} < 0.001$) and non-PE ($P_{trend} < 0.001$) groups.

The PE group showed a constantly higher trend for systolic and diastolic blood pressure over time, compared to the non-PE one. The interaction term (follow-up years × PE) was not statistically significant for SBP and DBP, indicating that the mean change is proportional between PE and non-PE women over time, although it was still higher in the PE group (Fig. 1A and B).

According to cox regression, hazard ratio of HTN progression adjusted for age, BMI, parity, TG, and HDL in women with a history of PE was 3.62-fold higher, compared to non-PE (95% CI: 2.70–4.62; P-value < 0.001) (Supplementary Table 5).

4. Discussion

This study demonstrated increased chances of developing HTN in their later life among women with prior PE, compared to non-PE ones;

Table 3
Parameter estimates of the generalized estimating equation (GEE) method in women with and without preeclampsia.

Dependent Variables	Variable	Beta coef.	95% Wald Confidence Interval	P-Value
SBP (mmHg)	PE	4.66	(3.15, 6.17)	< 0.001
	Non-PE	Reference		
	Age (years)	0.44	(0.40, 0.48)	< 0.001
	BMI (kg/m2)	0.60	(0.52, 0.69)	< 0.001
	Follow-up Years	1.41	(1.26, 1.55)	< 0.001
	PE*Follow-up Years	0.07	(−0.35, 0.49)	0.75
	Non-PE*Follow-up Years	Reference		
DBP (mmHg)				
	PE	2.55	(1.49, 3.61)	< 0.001
	Non-PE	Reference		
	Age (years)	0.19	(0.16, 0.22)	< 0.001
	BMI (kg/m^2)	0.43	(0.37, 0.49)	< 0.001
	Follow-up Years	0.92	(0.81, 1.02)	< 0.001
	PE*Follow-up Years	−0.04	(−0.34, 0.26)	0.78
	Non-PE*Follow-up Years	Reference		

PE = preeclampsia, Non-PE = non Preeclampsia, BMI = Body Mass Index, SBP = systolic blood pressure, DBP = diastolic blood pressure.

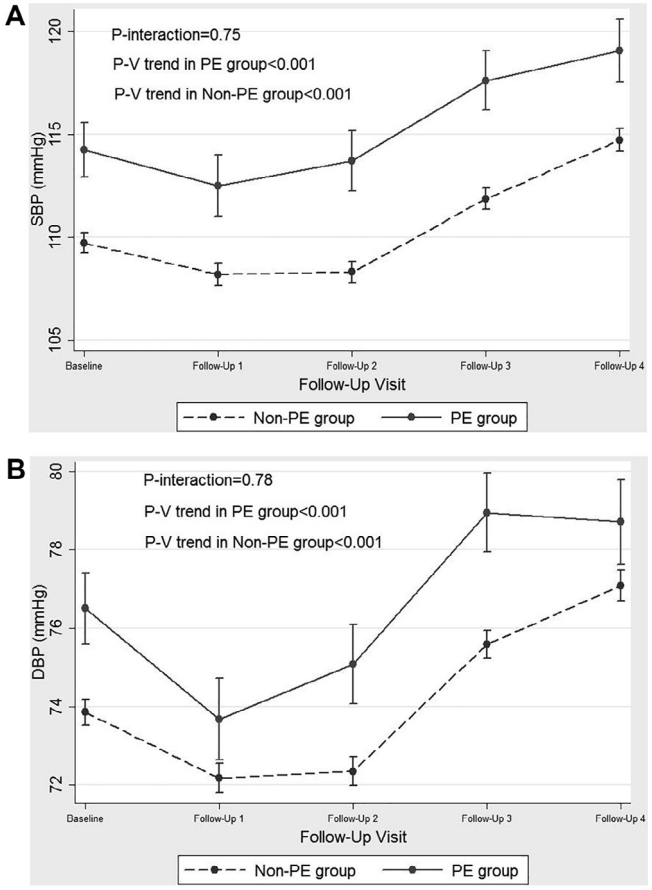

Fig. 1. (A & B). Generalized estimating equation (GEE) measures. Mean changes within follow-ups between PE and Non-PE groups assuming the interaction between time and study groups (adjusted for age, BMI and baseline SBP/DBP). A) SBP (Systolic Blood Pressure) changes, B) DBP (Diastolic Blood Pressure) changes. Non-Preeclampsia: - - - - -, Preeclampsia:—.

the chance further increased in those PE women with additional risk factors including obesity, aging, and dyslipidemia. We observed that the PE group constantly had higher systolic and diastolic blood pressure over time compared to the non-PE one. The interaction term (follow-up years × PE) was not statistically significant for SBP and DBP, indicating that the mean change is proportional between PE and non-PE women over time, although it was still higher in the PE group.

Although numerous studies have demonstrated risk of future HTN in women with a history of PE, it still unclear how this increased risk is affected by aging, obesity, parity, and dyslipidemia. Besides, to the best of our knowledge, it is unclear whether or not this risk is increased with passage of time. In this study, we compared chances of HTN progression between women with and without prior PE, after adjustment for risk factors such as time of follow-up, age, BMI, parity and lipid profiles. Our findings confirmed previous reports that PE is an important risk factor for HTN progression [15,28,43]. We found that, in addition to PE, other risk factors including aging, obesity, and dyslipidemia can influence progression of HTN. Our study revealed that older women are about 8% more likely to develop hypertension, compared to non-PE women and that with each kg/m^2 of increment in BMI, the odds of HTN increases 10%. A prospective cohort study with 10-year follow-up suggested that a reduction in BMI, with a consequent reduction in inflammation, improvement in insulin sensitivity and endothelium dependent vascular function might decrease risk of cardiovascular diseases [28]. Interestingly, our findings showed that with each 1 mmol/L increment in TG, the odds of HTN increases approximately 18%, whereas with HDL, it decreases to 34%, a finding in line with previous data that dyslipidemia is an important risk factor for development of hypertension [44,45].

The present study showed that women with prior PE developed HTN with an adjusted odds ratio 3.70 fold higher than their healthy peers during a median follow-up time of 11 years, results compatible with findings of a meta-analysis conducted by Bellamy et al. (2007), who demonstrated that after an average of 10 years postpartum, women with PE have higher rates of chronic hypertension (relative risk: 3.7; 95% CI: 2.7, 5.1) [9]. Wilson et al. (2003) [46] during a 20-year follow-up study reported an odds of HTN between 1.40 and 2.97 for PE or eclampsia, compared to the control group. Also, Smith et al. (2009) [15] in a cohort study with a one year follow-up after childbirth reported an approximately 2–3 fold higher risk of CVD progression in women with history of PE, compared to their healthy counterparts; they suggested that the risk was greatest for women with severe PE. Similar to our results, a recent cohort study with a mean follow-up ranging between 25 and 35 years demonstrated that women with gestational hypertension or preeclampsia in their first pregnancy had increase rates of HTN compared to those who were normotensive during pregnancy (HRs: 2.8; 95% CI, 2.6–3.0 and 2.2; 95% CI, 2.1–2.3, respectively) [47].

Although major mechanisms for development of HTN after PE have not been completely elucidated, they may be a result of increased plasma concentrations of the endothelial inflammatory markers and long-term metabolic and vascular damage [15,28]. In addition, some risk factors, including hypertensive pregnancy disorders (in particular PE), aging, obesity, and renal disorders may contribute to progression to hypertension following development of endothelial dysfunction [28,43,48]. Moreover, many of the genetic variants implicated in PE are also associated with CVD disease, suggesting that preeclampsia and CVD share genetic risk factors [49]; however it is not completely understood whether the development of HTN in women with hypertensive disorders of pregnancy is due to common predisposing factors or to specific pathophysiological processes of pregnancy [50].

Although the time course of progressing to HTN after PE is unclear and conflicting, there is some evidence suggesting these changes are detectable as early as one year after delivery [8,15,23,27,29]. Several studies have reported higher SBP and DBP means for women with a history of PE compared with those without PE, in 5-years of follow-up, post-delivery [15,23].

We assessed trend of BP changes within follow-ups after adjustment for age, BMI, and baseline BP levels and noted that the mean of systolic and diastolic blood pressure had an increasing trend in both groups (women with and without prior PE), indicating that aging is an independent and important risk factor for progression to HTN. We noted that increasing trend of diastolic blood pressure by the time is not as much as those observed for systolic blood pressure. This may partly explain the discrepancy in the overtime trends of systolic and diastolic blood pressure in our study participants. In agreement with our results, two population based study showed less prominent changing in diastolic blood pressure over the time and also weaker prediction of CVD death by diastolic blood pressure [51,52].

Our results also revealed that in women with prior PE, mean SBP is 2.73 units (mmHg) higher compared with non-PE women, whereas there was no significant difference in DBP between both groups. However, the interactions of follow-up years × PE were not significant for HTN parameters (SBP and DBP); indicating that per visit changes of SBP and DBP over follow-ups were the same between the two groups.

We also found that with per 1 kg/m^2 increment in BMI, means of SBP and DBP in women with PE compared to their healthy peers significantly increased by approximately 0.28 and 0.22 mmHg, respectively, indicating that BMI is an important predictor for HTN progression.

Our study has several strengths including: Its ongoing design as a prospective population-based study with a long-term follow-up that minimizes selection bias and allows further observations, multiple assessments; the large sample size and low levels of lost to follow-up and performing GEE analysis to assess trend of blood pressure changes with adjustment for important predisposing factors. The present study however also has its limitations. We did not follow the study participants from throughout their pregnancies and relied on their history of PE or non-PE; however, to deal with this limitation we checked their medical documents in cases with controversial data (when the women were not sure of the precise diagnosis of their preeclampsia). Despite reports that a history of hypertensive pregnancy disorders has a sensitivity of approximately 72–80% and specificity of 96–99% [37,53], the validation of the self-reporting questionnaire has not been checked in our population. We have not collected data regarding the time gap between event of PE and first data collection, so this variable has not been adjusted for in our analysis. In addition, accurate information of preeclampsia severity before initiation of the study were not collected in our dataset. Also the risk of development of chronic hypertension after PE may be underestimated in our study as a number of women with PE history could have progressed to chronic hypertension before initiation of the study were excluded. Our results have not been adjusted for family history of hypertension, and lifestyles, as these data were not collected. The participants did not know their specific blood pressures or protein concentrations, as a result of which we were unable to adjust our analysis based on the severity of their PE condition. These limitations should be considered in interpreting our results.

5. Conclusion

This study demonstrated that more women with previous PE, in particular those with additional risk factors, develop HTN than non-PE women in their later life. Although in both groups, over time blood pressure increases, women with prior PE experience higher mean SBP, compared to their healthy peers, nonetheless as time progresses, changes of SBP in women with prior PE do not worsen significantly, compared to their non-PE counterparts. Hence, all women who have prior PE, in particular, those with having other predisposing factors such as obesity should be consider for the evaluation of risk of subsequent HTN. The early diagnosis and management of subsequent hypertensive disorders in these women can potentially prevent the morbidity and mortality from this disease.

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Author contributions

M.A. contributed to the data analysis, manuscript drafting, and critical discussion. F.R.T contribute to study design, execution, data analysis, and critical discussion. M.R contributed to the data analysis and manuscript writing. S.B.G. contributed to the data analysis, and critical discussion. F.A contribute to study design, execution, and critical discussion. All authors have read and approved the final manuscript.

Declaration of interest

The authors have no conflicts of interest to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.preghy.2019.05.007.

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