Duration of labor among women with hypertensive disorders of pregnancy; A Swedish register cohort study

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ABSTRACT

Objective: Preeclampsia is a severe obstetric complication affecting 2–8% of pregnancies.
There is a common belief that women with preeclampsia experience a shorter duration of labor, where it is thought that increased inflammation that occurs with the disease facilitates labor. However, little evidence exists to support or refute this. Thus, we undertook a register-based cohort study investigating the association between hypertensive disorders of pregnancy and labor duration.
Study design: This was a Swedish register-based cohort study of nulliparous women with spontaneous or induced onset of labor at >34 weeks of gestation with a singleton fetus in cephalic presentation. Information of duration of labor was retrieved from electronic birth records and compared between women with hypertensive disorders and normotensives pregnancies. Data was represented as mean adjusted difference in hours (adjusted for pre-gestational disorders, maternal characteristics and mode of delivery) and adjusted hazard ratios (aHR), with an event defined as vaginal birth and women with intrapartum caesarean section censored. An aHR >1 indicated shorter duration of labor.
Results: Among 101,531 women, 5548 (5.5%) developed a hypertensive disorder of pregnancy. The overall mean duration of labor was 9.43 (SD 5.32) hours. Women with hypertensive disorders experienced a shorter duration of labor compared to normotensive women, with an adjusted mean difference of -0.68 h (95% CI -0.90, -0.47) for gestational hypertension and -1.53 h (95% CI -1.72, -1.35) for preeclampsia. This corresponded to an aHR of 1.05 (95% CI 1.01, 1.10) and 1.12 (95% CI 1.08, 1.17), respectively. However, when we confined the analysis to those who labored spontaneously, the presence of hypertensive disorders did not alter duration of labor (aHR 0.98, 95% CI 0.95,1.01). Only women who were induced and also had hypertensive disorders experienced a shorter duration of labor (aHR 1.07, 95% CI 1.04,1.09).
Conclusions: Hypertensive disorders did not alter labor duration among women with spontaneous onset of labor, however an association was observed among women who were induced.

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Introduction

Preeclampsia is a maternal hypertensive disorder affecting 2–8% of pregnant women [1]. It has been proposed that women who develop hypertensive disorders of pregnancy, including preeclampsia, experience a shorter duration of labor. However, little evidence exists to support or refute this, with prior studies showing conflicting results [2–4].

Pregnancy and labor are associated with inflammation and as pregnancy continues, an increased inflammatory response occurs [5,6]. Labor, both at term and preterm gestations, is also associated with inflammation in the myometrium, cervix, fetal membranes and peripheral blood [7–9].

Preeclampsia is also considered a state of excessive circulating inflammation and the development of the disease is thought to follow a two-stage disease process. During the first stage, impaired placentation early in pregnancy results in placental hypo-perfusion,

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ischemia/hypoxia and placental insufficiency [10]. This leads to excessive release of placental factors into maternal circulation, triggering the symptomatic second stage of disease. The target organ of this second stage appears to be the maternal endothelium, with exaggerated maternal endothelial activation and generalized hyper-inflammation [11]. This inflammation is characterized by oxidative stress, an altered angiogenic balance and increased pro-inflammatory cytokines [12].

Given preeclampsia and labor are both associated with inflammation, it is plausible that preeclampsia may exacerbate the inflammatory process of labor and consequently reduce the duration of labor. Thus, we set out to investigate the association between hypertensive disorders of pregnancy and labor duration in a population-based cohort of Swedish nulliparous women.

Methods

We performed a Swedish population register-based cohort study using data obtained from the Swedish Pregnancy Register [13]. The Swedish Pregnancy Register was established in 2013 as a National quality register which has been investigated, validated and presented in detail previously [14,15]. It comprises prospectively collected self-reported demographic, reproductive and maternal health data, as well as information on pregnancy and labor outcomes for both mother and newborn.

146,881 nulliparous women with a singleton pregnancy with spontaneous or induced onset of labor from July 2013 to January 2017 were identified in the register. Hence parous women, multiple pregnancies, stillbirths or women with pre-labor caesarean section were not included. Further, women with either gestational length <34 weeks (n = 2186), breech presentation (n = 3006), unknown fetal presentation (n = 7214), missing parity data (n = 581) or without information about duration of labor (n = 40,180) were excluded, resulting in a study population of 101,531 women.

Information on maternal demographics including age at delivery, height and weight recorded at first antenatal visit was extracted from the Swedish Pregnancy Register and body mass index (BMI) was calculated. Country of birth was classified as Sweden, other Nordic countries, other western countries (Europe, North America, Australia and New Zealand) or non-western countries. Years of education were categorized to ≤12 or >12 years. Smoking during early pregnancy (yes or no) was recorded at the first antenatal visit. Information on pre-gestational disorders were extracted from predefined check boxes in the electronic birth records and/or diagnostic codes recorded by obstetricians according to the International Classification of Diseases, Tenth Version (ICD-10): pre-gestational hypertension (I10, O10), pre-gestational diabetes (O240, O241, E10-14) and systemic lupus erythematosus (M32).

Pregnancy and labor characteristics included conception via in-vitro fertilization, gestational age at birth, induction of labor, epidural analgesia, fetal presentation at birth (occiput anterior or posterior), mode of delivery (spontaneous vaginal, operative vaginal [vacuum extraction or forceps] or caesarean section) and infant birthweight.

Exposure

Hypertensive disorders of pregnancy were defined as diagnosis of gestational hypertension (ICD-10 code O13) or preeclampsia (ICD-10 code O11, O14-15), recorded by obstetricians in antenatal and/or birth records.

Outcome

Duration of labor was the main outcome measure. This was calculated from patient or midwife reported time of onset of established contractions (day-hour-minute) until the time of delivery (day-hour-minute). The distribution of time was checked visually, values below the 0.5th and above the 99.5th percentile were considered invalid and excluded.

Subgroup analyses

We performed stratified analyses according to onset of labor (spontaneous or induced). Additionally, analyses stratified by categorizing gestational age as preterm (34–36 + 6 weeks of gestation), term (37–41 + 6 weeks of gestation) and post-term (≥42 weeks of gestation) were performed.

Statistical analysis

Characteristics of the population were described according to hypertensive disorders of pregnancy. Hypertensive and normotensive women were compared via bivariate analysis using Pearson's Chi2 test for categorical variables and Student's *t*-test for continuous variables. Mean difference in labor duration by hypertensive disorders of pregnancy compared to normotensive women was calculated with linear regression adjusted for maternal age at birth, height, BMI, country of birth, pre-gestational hypertension, pre-gestational diabetes and mode of birth, and presented as adjusted mean difference in hours with 95% confidence intervals (CI). Cox proportional hazard regression model was used to analyze time to event, where an event was defined as a vaginal delivery. Women with intrapartum caesarean section were censored at the time of delivery, but contributed with time until then. Results were presented as hazard ratios (HR) with 95% CI. Adjusted hazard ratios (aHR) were calculated with maternal age, height, BMI, country of birth, pre-gestational hypertension and pre-gestational diabetes as confounders in the model. Confounders were identified through a theoretical framework and directed acyclic graph (www.dagitty.net) for investigating the total effect of gestational hypertensive disorders on labor duration. Gestational age at birth, birthweight and onset of labor were considered mediators on the causal pathway between exposure and duration of labor and thus, not adjusted for but rather stratified for in sub analyses. To evaluate any clustering effect on reporting onset of contractions among birth units, a mixed model with labor duration under random county factor was performed to calculate an intra class correlation.

Results

Of 101,531 women included in the study, 5548 (5.5%) developed a hypertensive disorder of pregnancy. Compared to normotensive women, those with a hypertensive disorder of pregnancy were older, taller, had a higher BMI, more likely to have pre-gestational disorders (chronic hypertension, diabetes or systemic lupus erythematosus) and to have conceived via in-vitro fertilization. Additionally, women with hypertensive disorders of pregnancy were more likely to have an induction of labor (55.7% vs 14.8%), to give birth prematurely and by caesarean section (Table 1).

Within the cohort, the mean duration of labor was 9.43 (SD 5.32) hours. Women with hypertensive disorders of pregnancy had a mean duration of 8.57 (SD 5.38) hours, corresponding to an adjusted mean difference of -0.75 h (95% CI -0.84, -0.67) compared to normotensive women (mean duration 9.48 h SD 5.31). Investigating gestational hypertension and preeclampsia separately, the mean duration of labor was 8.95 (SD 5.39) hours for women with gestational hypertension and 8.29 (SD 5.35) hours for women who developed preeclampsia, which corresponded to an adjusted

Table 1
Maternal characteristics of study population (N = 101,531).

	Missing n=	Normotensive (n = 95,983)	Hypertensive disorders of pregnancy (n = 5548)	P-value
Age (years), mea n ± SD	25	28.9 ± 4.8	29.4 ± 4.9	<0.001
≥35		10,132 (10.6)	761 (13.7)	<0.001
Maternal height(cm),mea n ± SD	2341	166.5 ± 6.5	166.7 ± 6.4	<0.001
Body mass index (kg/m2), mean ± SD	3907	23.9 ± 4.2	26.3 ± 5.4	<0.001
≥30, n (%)		8204 (8.9)	1155 (21.5)	
Country of birth, n (%)	10,600			<0.001
Nordic		67,138 (78.2)	4402 (87.3)	
Other western		4894 (5.7)	198 (3.9)	
Non-western		13,857 (16.1)	442 (8.8)	
Education (years), n (%)	18,292			0.011
≤12		35,027 (44.5)	2153 (46.5)	
>12		43,579 (55.4)	2480 (53.5)	
Smoking at first antenatal visit, n (%)	11,357	4160 (4.9)	179 (3.6)	<0.001
Pre-gestational disorders, n (%)				<0.001
Hypertension		382 (0.4)	198 (3.6)	
Diabetes		437 (0.5)	107 (1.9)	
SLE		138 (0.1)	12 (0.2)	
In-vitro fertilization, n (%)		5480 (5.7)	399 (7.2)	<0.001
Gestational age at delivery, (weeks), mean ± SD		39.6 ± 1.4	39.2 ± 1.5	<0.001
Preterm delivery (34−36 weeks), n (%)		3049 (3.2)	289 (5.2)	
Post-term delivery (≥ 42 weeks), n (%)		6866 (7.2)	187 (3.4)	
Induction of labor, n (%)		14,169 (14.8)	3090 (55.7)	<0.001
Fetal presentation, n (%)				0.142
Occiput anterior		4617 (4.8)	291 (5.3)	
Occiput posterior		91,366 (95.2)	5257 (94.8)	
Mode of delivery, n (%)				<0.001
Spontaneous vaginal		79,424 (82.8)	4223 (76.1)	
Operative vaginal		11,274 (11.8)	734 (13.2)	
Cesarean section		5285 (5.5)	591 (10.7)	
Epidural analgesia, n (%)		47,348 (49.3)	3167 (57.1)	<0.001
Infant weight (grams),mean ± SD	1914	3,489.9 ± 466.3	3,394.3 ±551.1	<0.001

mean difference of -0.67 h (95% CI −1.00, −0.35) and −0.78 h (95% CI −1.00, −0.56) respectively, when compared to normotensive women (Fig. 1).

Hypertensive disorders of pregnancy were associated with an increased aHR (1.07, 95% CI, 1.05, 1.09) of vaginal birth. To investigate mediation through onset of labor we performed stratified analyses according to spontaneous and induced onsets of labors. Among the 84,272 women with spontaneous onset,
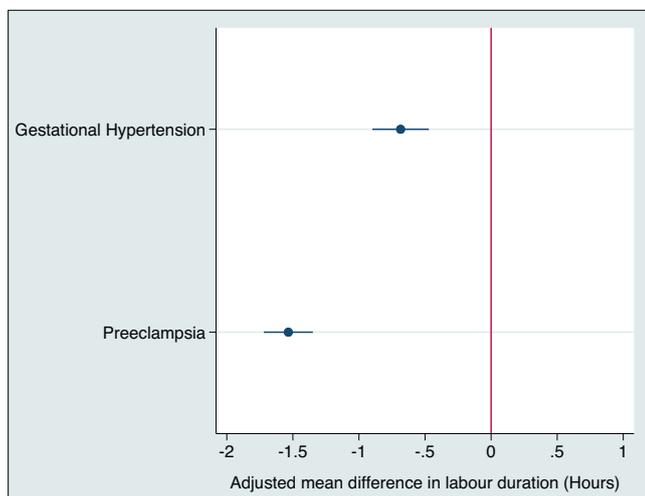
Fig. 1. Mean difference in labor duration by hypertensive disorders of pregnancy compared to normotensive women.
Calculated via linear regression adjusted for maternal age at birth, height, BMI, country of birth, pre-gestational hypertension, pre-gestational diabetes and mode of birth, and presented as adjusted mean difference in hours with 95% confidence intervals.

hypertensive disorders were not associated with an altered hazard ratio of vaginal birth compared to normotensive women. Whilst, among induced women, hypertensive disorders were associated with a shorter duration of labor, gestational hypertension aHR 1.14 (95% CI 1.14,1.24) and pre-eclampsia aHR 1.12 (95% CI 1.07,1.18) (Table 2).

Among women who delivered preterm (34−36 + 6 weeks of gestation) or post-term (≥42 weeks of gestation) no association between hypertensive disorders of pregnancy and duration of labor was found (Table 3). However, among women who delivered at term (37–41 + 6 gestational weeks), women with hypertensive disorders had an increased hazard ratio of vaginal birth compared to normotensive women (Table 3).

Further stratifying for both gestational age at birth and mode of delivery, women with hypertensive disease and spontaneous onset had similar labor duration at any gestational age compared with normotensive women (data not shown). Among those who were induced, hypertensive disorders were not associated with altered duration of labor among preterm or post-term. However, among induced women delivering at term (n = 11,417), gestational hypertensive disorders were associated with a higher chance of vaginal delivery at each time point during labor (aHR 1.03 (95% CI 1.00, 1.05) compared to normotensive women (Supplemental Table 1).

Discussion

In this register-based cohort study, hypertensive disorders of pregnancy did not alter labor duration among women with spontaneous onset of labor. However, among induced women, both gestational hypertension and preeclampsia were associated with a shorter duration of labor; aHR of 1.14 (95% CI 1.07, 1.24) and 1.12 (95% CI 1.07, 1.18), respectively.

Table 2
Cox regression showing the hazard ratio of vaginal birth among women with hypertensive disorders of pregnancy compared with Normotensive according to onset of labor.

	Onset of labor					
	All N = 101,531		Spontaneous N = 84,272		Induced N = 17,259	
	Crude HR (95%CI)	Adjusted HR (95%CI)	Crude HR (95%CI)	Adjusted HR (95%CI)	Crude HR (95%CI)	Adjusted HR (95%CI)
Normotensive (N = 95,983)	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)	1.0(ref.)
Hypertensive disorders of pregnancy (N = 5548)	1.06 (1.04, 1.08)	1.07 (1.05, 1.09)	0.96 (0.93, 0.99)	0.98 (0.95, 1.01)	1.07 (1.04, 1.09)	1.07 (1.04, 1.09)
Gestational Hypertension (N = 2370)	1.05 (1.01, 1.10)	1.07 (1.03, 1.12)	0.97 (0.92, 1.02)	0.99 (0.94, 1.04)	1.13 (1.05, 1.21)	1.14 (1.07, 1.24)
Pre-eclampsia (N = 3178)	1.12 (1.08,1.17)	1.15 (1.11,1.20)	0.92 (0.86,0.98)	0.94 (0.88, 1.01)	1.13 (1.07, 1.19)	1.12 (1.07, 1.18)

Hazard ratios with 95% confidence intervals for vaginal birth were calculated via Cox regression. Adjusted analyses include maternal age, maternal height, maternal BMI, country of birth, pre-gestational diabetes and pre-gestational hypertension as covariates. N = 97,433 included in adjusted analyses of all births. N = 80,184 included in adjusted analyses of spontaneous labor onset. N = 16,619 included in adjusted analyses of induced onset of labor.

Table 3
Cox regression showing the hazard ratio of vaginal birth among women with hypertensive disorders of pregnancy compared with normotensive stratified by gestational age.

	Preterm (34–36 gestational weeks) N = 3338		Term (37–41 gestational weeks) N = 91,140		Post-term (≥42 gestational weeks) N = 7053	
	Crude HR (95%CI)	Adjusted HR (95%CI)	Crude HR (95%CI)	Adjusted HR (95%CI)	Crude HR (95%CI)	Adjusted HR (95%CI)
Normotensive	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)	1.0 (ref.)
Hypertensive disorders of pregnancy	1.02 (0.95, 1.09)	1.04 (0.96, 1.12)	1.05 (1.03, 1.07)	1.07 (1.05, 1.09)	0.89 (0.81, 1.01)	0.92 (0.82, 1.02)
Gestational Hypertension	1.10 (0.88, 1.39)	1.14 (0.90, 1.45)	1.05 (1.00, 1.10)	1.07 (1.03, 1.12)	1.00 (0.02, 1.24)	1.00 (0.81, 1.24)
Pre-eclampsia	1.02 (0.88, 1.18)	1.06 (0.91, 1.23)	1.11 (1.07, 1.16)	1.14 (1.09, 1.19)	0.74 (0.57, 0.97)	0.78 (0.60, 1.02)

Hazard ratios with 95% confidence intervals for vaginal birth were calculated via Cox regression. Adjusted analyses include maternal age, maternal height, maternal BMI, country of birth, pre-gestational diabetes and pre-gestational hypertension as covariates. N = 3221 included in adjusted analyses among preterm births. N = 87,444 included in adjusted analyses among term births. N = 6778 included in adjusted analyses of post-term births.

Given that we did not find any effect on duration of labor by hypertensive disorders among spontaneously laboring women, it is unlikely that the presence of increased inflammation in women with hypertensive disorders would contribute to a shorter duration of labor and increased probability to achieve vaginal delivery.

Although we found that among induced women, hypertensive disorders were associated with a shorter labor duration, this difference could be attributed to a number of clinical factors and reflect altered clinical management, including a possible greater urgency to deliver women with hypertensive disorders. Indeed, there was an increased rate of caesarean section among those with hypertensive disorders and in our sub-analysis where we accounted for mode of birth the differences in duration of labor for induced labors were no longer significant. Additionally, among women who were induced gestational age at delivery and birthweight were significantly lower among those who developed a hypertensive disorder of pregnancy. Gestational age and birth-weight are inherently associated with duration of labor [16]. In accordance with prior studies [16,17] we found that maternal age and BMI were associated with labor duration, and hence adjusted for these variables. However, given birthweight and gestational age may mediate the association between hypertensive disorders and labor duration, we did not regard them as confounders and chose not to correct for these variables.

Although there has been little investigation of duration of labor among women with hypertensive disorder of pregnancy, our findings are supported by a previous case-control study published in 1997. This report found no difference in duration of labor between 727 women with preeclampsia and 1454 normotensive women. The authors attributed the clinical perception of shorter labor to more aggressive management [2]. A small case-control study from 2002 compared time from induction to delivery between women with and without preeclampsia and found a longer duration of labor among women with preeclampsia [18]. In contrast, Bregand-White et al. analyzed patterns of labor of 55,572 induced women and found that women with chronic hypertensive disorders had a slower progression of cervical dilatation from 4 cm to 10 cm compared to normotensive women, whereas women with gestational hypertension or preeclampsia progressed more rapidly [4].

Strengths of our study include a population-based setting with a large sample size and detailed information on pregnancy and labor characteristics, which permitted inclusion of potential confounders in the statistical models. Additionally, women who labored but were delivered via caesarean section were censored in the Cox regression analysis and this allowed them to contribute with time and provided an estimate of the hazard ratio of a successful vaginal delivery. By restricting the analysis to nullipa-rous women, the important assumption of independent observations in Cox models could be fulfilled and each woman contributed only once. To evaluate any clustering effect on reporting labor onset between county units, ICC was calculated to 2.4%, indicating a low heterogeneity between birth units.

However, there are some limitations. Information of cervical dilatation at induction and different time points during labor was not available, and estimation of labor duration was partly based on self-reported onset of established contractions. Women's reports of onset of labor have been reported to be in agreement with

midwife assessment in less than one third of cases [19], which could introduce ascertainment bias and result in skew distribution of labor duration between women that labored spontaneously and those that were induced. Stratification between spontaneous and induced onset may have reduced this bias. Additionally, the time of established contractions was missing in 18% of the eligible population and were thus excluded, which may have resulted in selection bias. Finally, it is plausible that an association between hypertensive disorders of pregnancy and labor duration may be mediated by the onset of labor and gestational age at birth. However, mediation analyses were not performed and analysis of induction methods was not performed, and thus a benefit of an inflammatory state of pregnancy at time of induction could not be ruled out.

Conclusion

Hypertensive disorders of pregnancy do not alter the duration of labor among women with spontaneous onset of labor. However, among induced women, hypertensive disorders were associated with a shorter labor, which is likely to reflect clinical management but a benefit of an inflammatory state of pregnancy at time of induction cannot completely be ruled out.

Author contributions

Susanne Hesselman, Anna-Karin Wikström, Tansim Akhter and Lina Bergman conceived the study. Roxanne Hastie and Susanne Hesselman managed the data set and performed the analyses with intellectual input of Anna Sandström and wrote the first draft. All authors provided technical and intellectual input and contributed to the final manuscript.

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Details of ethics approval

The Regional Ethical Board at Uppsala approved the study the 15th of August 2018 (Dnr 2018/287).

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.ejogrb.2020.05.024.

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