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Increased incidence of hypertensive disorders of pregnancy in women with a history of spontaneous preterm birth: A longitudinal linked national cohort study

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ABSTRACT

Objective: Determine the risk of hypertensive disorders of pregnancy (HD) in women with a history of spontaneous preterm birth (SPTB).

Study design: Longitudinal linked national cohort study within the Dutch Perinatal Registry (1999–2009) on linked data among 349,291 women with a first and second singleton pregnancy in the Netherlands.

Main outcome measures: The incidence of HD, small for gestational age (SGA) and placental abruption in the second pregnancy.

Results: Out of 349,291 women with a singleton first pregnancy, 19,991 (5.7%) had a SPTB. The incidence of HD in the second pregnancy was 8.1% in women with a previous SPTB, as compared to 5.6% in women with a previous term birth (aOR 1.49 (CI 1.41–1.57)). Also after excluding HD, SGA and/or placental abruption in the first pregnancy, women with a history of SPTB had a higher risk of HD in their second pregnancy compared to women with a previous term birth (4.6% versus 2.7%, aOR 1.77 (CI 1.64–1.91)). Similarly, the incidence of SGA and placental abruption was higher in the second pregnancy in women with a history of SPTB compared to term birth in the first pregnancy.

Conclusions: Women with a history of SPTB are at elevated risk of HD in the subsequent pregnancy. These results support shared pathophysiology between SPTB and HD.

1. Introduction

Preterm birth is the main cause of neonatal morbidity and mortality [1,2]. One third of preterm births is due to medical intervention, because of fetal and/or maternal conditions such as hypertensive disorders of pregnancy (HD) and fetal growth restriction (FGR), while two thirds of preterm births occur after spontaneous onset of labour or premature rupture of membranes [2]. Spontaneous preterm birth (SPTB) is defined as birth before 37 weeks of gestation after spontaneous rupture of membranes and/or spontaneous onset of contractions. Often an individual cause for SPTB is not identified, but causes include infection, inflammation, uterine over distension and vascular disease of the placenta or a combination of these [3].

The role of vascular disease of the placenta is described extensively in pregnancies complicated by hypertensive disorders of pregnancy (HD), small for gestational age (SGA) or placental abruption, yet it is hypothesized to be of significant importance as well in the onset of SPTB [4]. The hypothesis is that a proportion of SPTB is due to abnormal placentation, similar to hypertensive disorders of pregnancy and fetal growth restriction [5]. Abnormal placentation is observed in one third of women with SPTB [6–8]. An abnormal angiogenic/anti-angiogenic profile in maternal plasma has been described in twice as many women with SPTB compared to women with a term birth [9]. Women with a distorted placentation, determined by an increased resistance at midtrimester Doppler measurement of uterine artery flow, are at increased risk of hypertensive disorders as well as SPTB [10].

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Some epidemiologic studies described that women with history of SPTB have a slightly increased risk of cardiovascular disease in later life, similar but less strongly associated, as do women with a history of pre-eclampsia [11].

HD, SGA and abruption of the placenta share clinical risk factors, have an increased risk of recurrence, and have an increased risk of co-occurrence within a pregnancy. Previous studies have investigated the recurrence in a subsequent pregnancy of preterm delivery, pre-eclampsia, small for gestational age (SGA) offspring, placental abruption and stillbirth [12–22]. However, the association between SPTB and HD or SGA in the subsequent pregnancy is not yet studied extensively. Nonetheless, a common pathophysiological pathway of spontaneous and indicated PTB has been suggested [13]. In the current literature there is a lack of knowledge concerning SPTB and the possible link with HD and SGA, not only in the pregnancy complicated with SPTB but also in the subsequent pregnancy.

2. Study objective

Therefore, we hypothesize that women with a history of SPTB have an increased risk of HD in the subsequent pregnancy, as well as an increased risk of SGA and placental abruption. Abnormal placentation might play a role in shared pathophysiology of SPTB as well as HD, SGA and placental abruption.

3. Methods

3.1. Dataset

The Netherlands Perinatal (Perined) Registry contains information of mother and child on pregnancies and deliveries up until 28 days after the delivery. The coverage of the Perined registry is approximately 96% of all deliveries in The Netherlands. It contains pregnancies of > 22 weeks' gestation. The Perined database is obtained by a validated linkage of 3 different registries: the midwifery registry, the obstetrics registry, and the neonatology registry of hospital admissions of newborn neonates [23,24]. The data in the registry are recorded by the health care professional responsible for the delivery, this registrations takes place in addition to the general medical documentation, directly after the delivery. Records are entered in the Perined registry at the child's level.

There is no unique maternal identifier in the registry that allows follow up of subsequent pregnancy outcomes in the same mother. A longitudinal probabilistic linkage procedure was performed to create a cohort with complete data on first and second deliveries of the same mother. The details on entry, linkage, aggregation, validation and verification of the data are published elsewhere [25].

The Perined Registry gave their approval for the use of the data for this study (Approval no. 12.56).

3.2. Inclusion and exclusion criteria

From the longitudinal database ($n = 391,026$) we included all 378,958 women who delivered 2 subsequent singleton pregnancies (first and second delivery) in The Netherlands between Jan. 1, 1999 until Dec. 31, 2009. We excluded all women with a gestational age above 43.6 weeks ($n = 125$) as well as pregnancies complicated by congenital anomalies ($n = 19,271$) or antenatal deaths ($n = 3892$). Women with iatrogenic preterm birth < 37 weeks in first pregnancy ($n = 5143$) and women with a preexisting hypertension ($n = 1236$) were also excluded, see Fig. 1.

3.3. Outcome measures

Our primary outcome measure was presence of HD in a subsequent pregnancy. Secondary we analyzed SGA and placental abruption. HD

Fig. 1. Overview of reasons for exclusion from the longitudinal studied cohort.

included pregnancy-induced hypertension, and (pre)eclampsia. We accepted cases identified as preeclampsia or eclampsia in the Perined database. The clinical diagnosis of hypertension in the Perined database was made with a diastolic blood pressure of ≥ 95 mm Hg and pre-eclampsia was diagnosed with hypertension and proteinuria (≥ 300 mg proteinuria in 24 h). SGA was defined as a birthweight smaller than the 10th percentile ($< P10$) on the Dutch reference curves for birthweight by gestational age, separate for parity, sex, and ethnic background [26]. Abruption of the placenta was defined as a clinical diagnosis of (partial) detachment of the placenta from the uterine wall prior to delivery. HD,

Table 1
Baseline demographics and clinical characteristics 1st pregnancy.

Characteristics	SPTB 1st pregnancy (n = 19.991)		Term birth 1st pregnancy (n = 329.300)		p-value
Maternal Age, years mean (± SD)	28.5	(4.1)	28.6	(4.2)	0.07
Non Caucasian race, % (n)	2.053	(10.3)	37.994	(11.5)	< 0.001
Gestational age at delivery, weeks mean (± SD)	2.7	(34.2)	1.3	(40.0)	< 0.001
Assisted conception, % (n)	761	(3.8)	9.826	(3.0)	< 0.001
Low socioeconomic status, % (n)	4.384	(21.9)	72.993	(22.2)	0.43
Inter-pregnancy interval					< 0.001
< 1 Year % (n)	472	(2.4)	3004	(0.9)	
> 5 year(s), % (n)	1.190	(6.0)	17.472	(5.3)	
SGA < p10, % (n)	1.016	(5.1)	30.196	(9.2)	< 0.001

Abbreviations: SD = standard deviation, n = number, SGA = small for gestational age.

SGA and placental abruption were also evaluated as a combined outcome.

3.4. Population characteristic and clinical characteristics

Women were classified into two groups: women with a SPTB < 37 weeks of GA in their first pregnancy and women with a term birth in their first pregnancy ($\geq$ 37 weeks). We analyzed demographic and obstetric characteristics including maternal age, ethnicity, mean gestational age at delivery, mode of conception, socioeconomic status, inter-pregnancy interval and SGA < P10 (Table 1) in the first pregnancy. The mode of conception is described as percentage of conception after assisted reproductive technology (ART). The socioeconomic status score was based on the 2010 national data from the Netherlands institute of Social Research (mean income level, the percentage of households with a low income, the percentage of inhabitants without a paid job, and the percentage of households with, on average, low education level) in a 4-digit postal code area and is expressed as percentage of women with a low economic socioeconomic status score (P25). Inter-pregnancy interval is categorized as < 12 months, 12–60 months or $\geq$ 60 months. The severity of the first preterm birth was subdivided in three groups: extreme premature birth as < 28 weeks of gestation, early premature birth as 28–32 weeks of gestation and late preterm birth as 32–37 weeks of gestation. We evaluated if HD, SGA or placental abruption was present in the first pregnancy both for women with a SPTB in the first pregnancy as for women with a prior term birth, and we evaluated these conditions as a combined outcome as well. Finally, we evaluated the difference in incidence of HD, SGA or placental abruption in women after SPTB with preterm and term delivery in the second pregnancy.

3.5. Statistics

We performed univariate analyses with the Student *t* test for the continuous variables and the χ^2 test for the categorical variables to compare baseline characteristics. If the continuous variables were normally distributed the equal variance test was used and for skewed distributions the unequal variance test was used.

We tested separately for SPTB and term birth with or without HD, SGA and/or placental abruption in the first pregnancy. Analyses were

also performed stratified for severity of preterm birth, based on gestational age at delivery in the first pregnancy (< 28 weeks, 28–32 weeks and $\geq$ 32 weeks).

We determined the crude effect of risk factors on HD and also SGA and/or placental abruption in the second pregnancy using logistic regression modeling, expressed as odds ratios with 95% confidence interval (CI). We adjusted the combined outcome (the combined presence of HD, SGA and/or placental abruption) for maternal age (< 25, $\geq$ 35 years old), Non Western ethnicity, inter-pregnancy interval (< 1 year, $\geq$ 5 years interval) and low socioeconomic status in a multivariable analysis.

Analyses were repeated for women without HD, SGA and/or placental abruption in the 1st pregnancy and with HD, SGA and/or placental abruption in the 1st pregnancy. All statistical tests were 2-sided; we chose a probability value of 0.05 as threshold to indicate statistical significance. The data were analyzed with the SAS statistical software package, version 9.3.

4. Results

After application of our in- and exclusion criteria we have identified a total of 349,291 women with two consecutive singleton pregnancies in the Perined database in the period from January 1, 1999 until December 31, 2009. Flow diagram 1 shows the selection process. In the first pregnancies 19,991 children were born prematurely after spontaneous onset of the delivery, 329,300 children were born at term.

Table 1 shows the baseline characteristics for both groups. Gestational age at delivery was lower in women with a pregnancy complicated by preterm delivery, non-Caucasian ethnicity was also lower, as was the percentage of pregnancies after assisted conception, pregnancy interval and percentage of SGA. No differences were found in maternal age and low socioeconomic status between women with a history of SPTB and women with a first term birth.

In Table 2 all the women in the cohort are presented. Of all women studied in the cohort 8.1% (1615 women) of those with a history of SPTB had HD in the subsequent pregnancy versus 5.6% (18,332 women) of women with a term birth in the first pregnancy (OR 1.49 (CI 1.41–1.57)). Also when we analyzed SGA and placental abruption we found significantly increased incidences in the group of women with a history of SPTB (Table 2). When we analyzed the combined outcome of

Table 2
Incidence of HD, SGA, placental abruption and combined outcome in the 2nd pregnancy in all pregnancies studied.

Outcome 2nd pregnancy	SPTB 1st pregnancy (n = 19.991)		Term birth 1st pregnancy (n = 329.300)		Odd ratio with 95% CI*
HD, n(%)	1.615	(8.1)	18.332	(5.6)	1.49 (1.41–1.57)
SGA < P10, n(%)	1.853	(9.3)	27.811	(8.4)	1.11 (1.05–1.16)
Placental abruption, n(%)	11	(0.06)	87	(0.03)	2.10 (1.11–3.90)
Combined outcome*	3.253	(16.3)	43.954	(13.3)	1.26 (1.21–1.31)

Abbreviations: HD: hypertensive disorders of pregnancy, SGA: small for gestational age < P10.

* Combined outcome = presence of HD, SGA and/or placental abruption.

† Adjustment of the combined outcome for maternal age, ethnicity, inter-pregnancy interval and socioeconomic status showed no effect on the results

Table 3

Incidence of HD, SGA or placental abruption in the second pregnancy after a 1st pregnancy without one of these conditions.

Outcome 2nd pregnancy	SPTB 1st pregnancy (n = 16.963)		Term birth 1st pregnancy (n = 267.520)		Odd ratio with 95% CI*
HD, n(%)	781	(4.6)	7.086	(2.7)	1.77 (1.64–1.91)
SGA < P10, n(%)	1.382	(8.1)	16.528	(6.2)	1.35 (1.27–1.43)
Placental abruption, n(%)	8	(0.05)	52	(0.02)	2.43 (1.15–5.11)
Combined outcome*	2.074	(12.2)	22.879	(8.6)	1.49 (1.42–1.56)

Abbreviations: HD: hypertensive disorders of pregnancy, SGA: small for gestational age < P10.

#Combined outcome = presence of HD, SGA and/or placental abruption.

*Adjustment of the combined outcome for maternal age, ethnicity, inter-pregnancy interval and socioeconomic status showed no effect on the results

HD, SGA and/or placental abruption we observed an incidence of 16.3% (3253 women) in the women after SPTB versus 13.3% (43,954 women) after term birth (OR 1.26 (CI 1.24–1.31)) which was similar after adjustment for maternal age, ethnicity, inter-pregnancy interval and socioeconomic status.

We performed sub-group analyses for women with or without HD, SGA and/or placental abruption in their first pregnancy. Table 3 shows the subgroup of women without HD, SGA and/or placental abruption in their first pregnancy, these women developed HD, SGA and placental abruption in the subsequent pregnancy more often than women with a prior term birth. The strongest increase was found in the incidence of HD 4.6% (781 women) versus 2.7% (7086 women) with OR 1.77 (CI 1.64–1.91). When women with a history of SPTB without HD, SGA and/or placental abruption in their first pregnancy had a recurrent SPTB they had less HD, SGA or placental abruption, OR 0.78 (CI 0.62–0.83). However, when women reached full term in the second pregnancy they had a higher occurrence of HD, SGA and placental abruption (OR 1.6 (CI 1.52–1.68)).

Of all women with a history of SPTB 15.1% also suffered HD, SGA and/or placental abruption in the same pregnancy. These women had a higher risk of recurrence of one of these conditions in the second pregnancy compared to women in the term group. Consequently, a history of SPTB together with HD, SGA and/or placental abruption, results in an increased risk of developing one or more of these conditions in the subsequent pregnancy. More overlap between the different conditions is seen in this group. For women with a history of SPTB complicated by HD, SGA and/or placental abruption who have a recurrent SPTB we did not find an increased risk for the development of these conditions OR 1.00 (CI 0.82–1.21) contrary to women who reach full term OR 1.25 (1.15–1.36).

5. Discussion

5.1. Main findings

We found that women with a history of spontaneous preterm birth had a 1.5-fold increased risk of HD in the subsequent singleton pregnancy compared to women with a term birth in their first pregnancy. In 16.3% of women with a history of SPTB in the first pregnancy at least one of the three diseases (HD, SGA, placental abruption) was observed in the second pregnancy. Hypertensive disease of pregnancy was most common. SPTB was associated with an elevated risk of HD, SGA and/or placental abruption in the subsequent pregnancy both in women with and without HD, SGA and/or placental abruption in the first pregnancy, especially if women reached the term period. Women who did have a recurrent SPTB have less risk of developing HD, SGA and/or placental abruption presumably because the recurrent preterm birth occurs before the hypertensive disease becomes clinically manifest. These findings support the theory that SPTB, at least partially, is caused by placental insufficiency. In women with a SPTB due to abnormal placentation the underlying process might be similar to hypertensive disorders of pregnancy, small for gestational age and placental abruption. However, the effect of confounding or the influence of shared risk factors on the outcomes could not be completely evaluated due to

missing data such as maternal morbidities, BMI and smoking.

5.2. Strengths and limitations

In general in this type of research based on data extracted from a national registry one has to be critical concerning the accuracy and validity of the used data. In the used registry the data are recorded by the responsible healthcare professional and a high level of accuracy is to be expected. However an effect of exposure and outcome misclassification cannot be excluded.

There are some limitations of this specific study. Firstly, not all variables with potential effect of the primary outcome were available in the national perinatal registry such as Body Mass Index (BMI) and smoking. Due to the increased risk of SPTB caused by antenatal smoking and the inverse association between antenatal smoking and development of preeclampsia we did not expect a significant effect of smoking on our primary outcome though [27,28]. In addition, due to low data quality, we were not able to analyze the effect of presence of (gestational) diabetes which might have an effect on the risk of developing HD, SGA and/or placental abruption. The number of women with pre-existing hypertension could be underestimated and not excluded from the study but included in the HD group. The percentage of pregnancies conceived after ART should not be considered representative due to incomplete registration by obstetric caregivers.

Secondly, of particular importance is the exact calculation of the gestational age. The way the expected date of delivery (EDD) of the studied pregnancies used in the Perined database was calculated is not reported on individual level and could either be based on the first day of the last menstrual period and/or early ultrasound, in case of difference of one week dating by ultrasound prevailed.

Finally, we evaluated the proportion of SGA neonates, defined as birthweight below the 10th percentile. However, this cut-off point does not necessarily distinguish the constitutionally small neonate from the SGA neonate whose birth weight is lower than the expected potential. Growth restricted neonates with a birth weight above the 10th percentile are also missed [29,30]. It is difficult to state that a preterm neonate should to be considered SGA because its birthweight is less than the 10th percentile, because birthweight norms do not align well with fetal weight norms prior to term. However, neonates SGA < P10 do have an elevated risk of mortality and morbidity [31].

5.3. Interpretation

A total of 5.7% of women in the studied cohort had a first pregnancy with a SPTB. Since all indicated preterm births, multiple gestations and fetal structural abnormalities were excluded, this percentage was to be expected and is consistent with percentages described in epidemiologic literature [2]. The incidence of the combination of HD, SGA and/or placental abruption in the first pregnancies with SPTB (15.1%) was higher than previously expected because we anticipated that most of the women with one of these conditions would have been excluded by excluding indicated preterm births in the first pregnancy. The incidence of SGA < P10 only in 5.1% of the women with a first SPTB is noticeable. However, neonates born after indicated preterm birth were

already excluded from this analysis. A large proportion of preterm births are indicated due to HD and/or a fetal growth restriction. High recurrence rates of HD, SGA and/or placental abruption were expected and confirmed [14]. An elevated risk of HD and also SGA and/or placental abruption in a pregnancy after a SPTB pregnancy without HD, SGA and/or placental abruption was found, as hypothesized. The association between SPTB and placental insufficiency has been established through studies on histopathologic findings and on increased risk on SPTB with abnormal Doppler measurements among others [2,6–10,18,32–34].

This is one of the first studies to evaluate the risk on clinical phenotypes of placental malperfusion after a SPTB. Our findings support the hypothesis that SPTB is a heterogeneous condition and that abnormal placentation plays an important role [32]. The treatment and follow up of women with different causes leading up to SPTB should be individualized [3]. It has been hypothesized before that SPTB may occur as a protection for mother and/or fetus, and functions as an ‘ejector seat’ before the onset of maternal or fetal problems due to placental ischemia [5]. Previous research showed that the perinatal outcomes of neonates born after SPTB with histologic findings consistent with placental insufficiency were poorer [32]. The question arises whether or not it is wise to delay preterm delivery in women with imminent labor due to placental malperfusion.

6. Conclusion

SPTB is associated with an elevated risk of HD in a subsequent pregnancy. Therefore caregivers of women with a history of SPTB should also be alert to hypertensive disorders of pregnancy and SGA, specifically when these women reach term. A shared role of abnormal placentation is suggested to play an important role.

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.

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Contribution to authorship

LV, MdB and CdG were involved in conception and design of the study. Analysis were conducted by AR and LV. LV drafted the manuscript. MdB, CdG, AR, BM and HG contributed to the interpretation of the analysis and writing of the manuscript. All authors approved the final manuscript.

Details of ethics approval

The data in the perinatal registry are anonymous; therefore, ethical approval was not needed. The Dutch Perinatal Registry gave their approval for the use of their data for this study (approval no. 12.56).

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