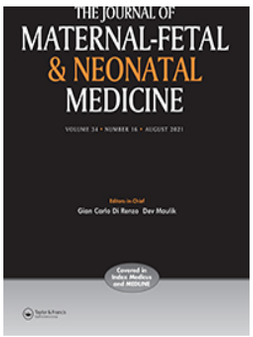

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ORIGINAL ARTICLE

Maternal vascular malperfusion in spontaneous preterm birth placentas related to clinical outcome of subsequent pregnancy

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ABSTRACT

Introduction: Spontaneous preterm birth (SPTB) has several causes and its pathophysiology remains unclear. In a significant proportion of SPTB, placental histology shows signs of maternal vascular malperfusion (MVM); commonly associated with hypertensive disorders of pregnancy (HD), fetal growth restriction (FGR) and placental abruption, together referred to as clinical ischemic placental diseases (IPD). We hypothesized that women with SPTB and placental MVM are at elevated risk for IPD in a subsequent pregnancy.

Methods: We included women with SPTB in our cohort and followed the subsequent ongoing pregnancy ($n = 110$). Histological placental characteristics in the index were reported according to new international guidelines, and related to the clinical outcome of the subsequent pregnancy.

Results: In the SPTB placentas, we observed MVM in 61.8% ($n = 68$). In the subsequent pregnancies in 19.1% ($n = 21$) at least one clinical sign of IPD was present (HD (12.7%), FGR (5.5%) or placental abruption (0.9%)). There was no significant difference in the prevalence of clinical IPD or recurrence of SPTB in the subsequent pregnancy between women with and without placental MVM in the index pregnancy, although our study was not powered to detect small differences.

Discussion: Women with a history of SPTB have an elevated risk of IPD in the subsequent pregnancy. MVM is present in a large proportion of SPTB placentas. The presence of placental MVM in the index pregnancy does not predict clinical IPD or recurrent SPTB in a subsequent pregnancy.

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Ischemic placental disease; maternal vascular malperfusion (MVM); placental insufficiency; preterm birth (PTB); preterm labor; spontaneous preterm birth (SPTB)

Introduction

Preterm birth (<37 weeks of gestational age (GA)) is the leading cause for neonatal morbidity and mortality [1]. Despite the attention to spontaneous preterm birth (SPTB) in medical research, the etiology of SPTB remains largely unexplained [2]. It is considered a syndrome caused by multiple mechanisms such as infection, inflammation, utero-placental ischemia, hemorrhage, stress, uterine over distention and other immunologically mediated processes [3]. In some cases, the cause of SPTB can be identified through histopathological evaluation of the placenta. Histopathologic signs of increased inflammation, excessive response to or stimulation of infection and oxidative stress are known promotors and have been demonstrated in SPTB [4] as well as in fetal growth restriction (FGR) and preeclampsia [5]. A significant proportion of SPTB placentas shows maternal vascular malperfusion (MVM) [6]. MVM is the most common histological placental finding in pregnancies complicated by hypertensive disorders of pregnancy (HD), FGR or placental abruption, together referred to as clinical ischemic placental diseases (IPD).

Shared inflammatory processes and genetic determinants have been suggested as mechanisms of action for both SPTB and cardiovascular disease [7]. Placental vascular lesions and placental bed pathology are common findings in women with a SPTB [8–11]. In a subset of patients with SPTB [12], abnormal angiogenic/antiangiogenic profile in maternal plasma is seen, as well as in women with preeclampsia. This

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suggests an overlap in etiology of preeclampsia and SPTB. Additionally, increased resistance in midtrimester Doppler measurement of uterine artery flow is associated with an increased risk of SPTB [13], as well as with preeclampsia and growth restriction. Also, fetal growth restriction is associated with both SPTB and hypertensive disorders of pregnancy [14]. Moreover, in the long-term, women with a SPTB seem to have an increased risk of developing cardiovascular disease later in life [15,16]; analogous to preeclampsia.

Previous research focusing on (S)PTB and placenta pathology is extensive, however until recently no international consensus existed on how to report on placental pathology. Therefore, comparison of results proved to be difficult. In 2016, the Amsterdam Placental Workshop Group Consensus Statement was published. This contains a standardized, reproducible, and biologically based classification system, of histological evaluation of the placenta [17].

Systematic and standardized placental histologic evaluation after an SPTB might assist in uncovering possible causes of SPTB for the purpose of diagnosis, prevention and treatment in a subsequent pregnancy [18]. Women with a history of SPTB and histological signs of MVM in the placenta might benefit more from a treatment with focus on attacking the pathological process of development of MVM, by for example low dose aspirin use (which is proven to be effective in reducing recurrent FGR and/or preeclampsia [19]) together with or instead of standardized treatment with progesterone in order to reduce recurrent SPTB. Insight in the contribution of MVM to the genesis of SPTB might therefore lead to more "tailor-made" preventive management in a subsequent pregnancy after SPTB.

We hypothesized that women with a SPTB, and in particular women who also have histological signs of placental MVM, have an increased risk of developing (other) clinical IPD such as HD and FGR in a subsequent pregnancy. The aim of this study was to describe placental histological characteristics in women with SPTB by using an international accepted classification with special attention to MVM, and to relate these findings to the prevalence of clinical IPD in the subsequent pregnancy.

Materials and methods

This is a cohort study of women of with a history of SPTB, which was defined as preterm birth (<37 weeks of gestational age) after spontaneous onset of contractions or after preterm prelabor rupture of membranes (PPROM). The subsequent pregnancy was followed for clinical outcome.

Study subjects

Women were included if a SPTB occurred between 16 and 37 weeks of gestation [20] in the Amsterdam UMC, location VU Medical Center in Amsterdam, the Netherlands from January 2004 to December 2014, and the antenatal care of the subsequent pregnancy was provided in our hospital and placental tissue of the index pregnancy was available for histopathological evaluation. We excluded women with multiple gestations (both in index and subsequent pregnancy), fetal congenital malformations, congenital malformations of the uterus and delivery <16 weeks of gestation in both pregnancies. For the selection of women, an electronic database was used containing data of all women with a previous SPTB under care in our dedicated antenatal care clinic for a subsequent pregnancy.

Outcome

Our primary outcome measure was presence of one or more clinical phenotypes of IPD in the subsequent pregnancy. We defined IPD as presence of at least one of three obstetric complications; HD (preeclampsia, HELLP-syndrome and pregnancy induced hypertension), FGR or placental abruption. HD were defined according the International Society for the Study of Hypertension in Pregnancy (ISSHP)-classification [21]. Fetal growth restriction (FGR) was defined as a birthweight < P10 on the Dutch reference curves for birthweight by gestational age (separate for parity and sex) [22] or birth weight < P10 plotted on the Hadlock 3 fetal growth curve [23,24]. Abruption of the placenta was defined as a clinical diagnosis of (partial) detachment of the placenta from the uterine wall prior to delivery.

Outcomes and covariates

We collected general data concerning characteristics, general and obstetric medical history from an electronic database for both the index and the subsequent pregnancy. Missing or incomplete data were completed by reviewing medical records. We analyzed demographic and obstetric characteristics including maternal age at delivery of the index pregnancy, ethnicity, mean gestational age at delivery, mode of conception, Body Mass Index (BMI), intoxications, educational level based on occupation, interpregnancy

interval. We also recorded comorbidity that could possibly be of influence on the primary outcome including; presence of cardiovascular disease such as (gestational) diabetes and preexisting hypertension.

Histological examination

Histological examination of preserved placental tissue was performed by a clinical pathologist specialized in perinatal pathology (PvdV) who was blinded to the outcome of the subsequent pregnancy. The classification was performed according to the recently formulated international consensus (by the Amsterdam Placental Workshop Group) for the histopathological evaluation of placentas [17]. This classification distinguishes three main pathological placental processes; vascular, inflammatory-immune and other. MVM describes pathological placental processes leading to clinical phenotypic expressions of IPD such as HD and FGR and is often referred to as (utero-)placental insufficiency in previous publications. Characteristics of MVM are defined as placental hypoplasia (defined as placenta weight below the 10th percentile [25]), (early or late) accelerated villous maturation (AVM), infarction of more than 5% of the placenta, (focal or diffuse) distal villous hypoplasia, retro-placental hematoma and decidual arteriopathy. Based on the histopathological characteristics, the women were divided into women with and without MVM. Positive for MVM was defined as any one of these features present. Potential intra-amniotic infection was screened for by screening for acute chorioamnionitis (defined as neutrophils in the chorionic plate) or funisitis.

Statistics

The combination of the histological characteristics (normal perfusion, mild and severe signs of MVM) and the clinical manifestation of IPD in the index pregnancy, were compared by using either a chi square, Fisher's exact, one-way ANOVA or Kruskal Wallis test.

The prevalence of IPD in the subsequent pregnancy in women with a previous SPTB, with either mild, severe and no signs of MVM during examination of placenta in the index pregnancy, were also compared by using chi square test and Fisher's exact test for discrete variables. A one-way ANOVA test was performed to compare the continuous data, provided the data were normally distributed. When data were not normally distributed, a Kruskal–Wallis test was used. We used a 95% confidence interval.

We compared women with normal perfusion of the placenta to women with signs of MVM in the placenta. For these analyses, we performed a chi square or Fisher's exact test for discrete variables and an independent *t*-test (if normally distributed) or nonparametric test (if not normally distributed) for continuous variables.

Because diabetes and cardiovascular disease are associated with MVM and clinical IPDs [26], the three groups (normal perfusion, mild and severe signs of placental MVM) were compared in prevalence of these diseases. The data were analyzed with the Statistical Package for the Social Science software, version 22.

Results

After application of our in- and exclusion criteria, we have evaluated 110 women. The mean maternal age was 30.3 years (SD 4.7) at birth of the index pregnancy and 70.9% were nulliparous. The mean GA at birth of the index pregnancy was 27.7 weeks (16.80–36.71 weeks); 51% ($n = 56$) had an extreme premature birth (GA $<$ 28 weeks), 36.4% ($n = 40$) an early premature birth (GA 28–34 weeks) and 12.7% ($n = 14$) a late premature birth (34–37 weeks). The clinical characteristics of the index and subsequent pregnancy are presented in Table 1.

In the index pregnancy, besides the SPTB, clinical signs of IPD were seen in 20.9% ($n = 23$) of the women; 0.9% ($n = 1$) developed preeclampsia, 7.3% ($n = 8$) developed pregnancy induced hypertension, 1.8% ($n = 2$) had an abruption of the placenta and 10.9% ($n = 12$) delivered a neonate with a birth weight $<$ p10 on either the growth curves for neonates ($n = 1$) or Hadlock 3 fetal growth curves ($n = 11$).

Evaluation of the preserved placental tissue revealed MVM in 61.8% ($n = 68$) of cases. Accelerated villous maturation was most prevalent with 48.2% ($n = 53$), followed by placental hypoplasia 23.6% ($n = 26$). Classification of the characteristics of the observed MVM can be found in Table 2. In 31.8% ($n = 35$) of cases, MVM was classified as mild, in 30% ($n = 33$) as severe. Severe fetal vascular malperfusion was observed in 12.7% ($n = 14$). Maternal and fetal inflammatory response was observed in 51.8% ($n = 57$) and 46.4% ($n = 51$), respectively.

In the placentas with MVM, maternal inflammatory response was observed as a secondary finding in 42.6% ($n = 29$) and fetal inflammatory response in 36.8% ($n = 25$) of the placentas with MVM. In 19.1% of the subsequent pregnancies, IPD was diagnosed and

Table 1. Clinical characteristics of index and subsequent pregnancy.

Characteristics	*n* (%)	
Non-Caucasian maternal ethnicity	36 (32.6)	
Low educational level	25 (22.7)	
Interpregnancy interval		
0–12 months	38 (34.5)	
12–60 months	70 (63.6)	
>60 months	2 (1.8)	
Pregnancy specific characteristics	**Index pregnancy**	**Subsequent pregnancy**
Parity, *n* (%)		
0	78 (70.9)	
1	24 (21.8)	78 (70.9)
2	6 (5.5)	24 (21.8)
≥3	2 (1.8)	8 (7.3)
Weeks of GA, median (min–max)	27.7 (16.8–36.7)	38.8 (16.3–41.4)
<28 weeks, *n* (%)	56 (50.9)	6 (5.5)
28–34 weeks, *n* (%)	40 (36.4)	8 (7.3)
34–37 weeks, *n* (%)	14 (12.7)	18 (16.4)
>37 weeks, *n* (%)		76 (69.1)
Age in years, mean (SD)	30.3 (4.7)	32.7 (4.9)
BMI, *n* (%)		
<18.5 kg/m^2	2 (1.8)	1 (0.9)
18.5–25 kg/m^2	56 (50.9)	48 (43.6)
25–30 kg/m^2	20 (18.2)	29 (26.40)
>30 kg/m^2	6 (5.5)	7 (6.4)
Smoking, *n* (%)	7 (6.4)	7 (6.4)
Diabetes, *n* (%)	3 (2.7)	6 (5.5)
Pre-existent	2 (1.8)	3 (2.7)
GDM	1 (0.9)	3 (2.7)
Fetal male gender, *n* (%)	68 (61.8)	51 (46.4)

GA: gestational age; SD: standard deviation; BMI: Body Mass Index; GDM: Gestational Diabetes Mellitus.

Table 2. Placental histological characteristics of the index pregnancy.

Characteristic	*n* (%) of cohort
Any sign of maternal vascular malperfusion[a]	68 (61.8)
Placenta hypoplasia (weight <10th percentile)	26 (23.6)
Infarction >5%	
Early	12 (10.9)
Late	4 (3.6)
Distal villous hypoplasia	
Focal	3 (2.7)
Diffuse	8 (7.3)
Accelerated villous maturation	53 (48.2)
Retroplacental hematoma	10 (9.1)
Decidual arteriopathy	0 (0)
Severe fetal vascular malperfusion	14 (12.7)
Maternal inflammatory response (≥stage 2)	57 (51.8)
Fetal inflammatory response	51 (46.4)

[a]Because of overlap, the total number of "sign of malperfusion" is lower than the total count of the individual components.

classified as 2.7% ($n = 3$) preeclampsia, 10% ($n = 11$) pregnancy induced hypertension, 5.5% ($n = 6$) birth weight < P10 and 0.9% ($n = 1$) abruption of the placenta.

Hypertensive disorders of pregnancy occurred in 16.2% of women with MVM versus 7.1% in women without MVM, this was not statistically significant. Also, difference in the prevalence of other IPD in the subsequent pregnancy between women with or without MVM were not significantly different. Neither a difference in recurrent SPTB was seen in women with MVM in the first pregnancy (31.8%) compared to women without MVM (26.2%), see Table 3. Three women used low dose aspirin in the subsequent pregnancy because of a history of eclampsia, hypertension and antiphospholipid syndrome. Prevalence of comorbidity did not differ between the group with and without MVM. Subgroup analyses of mild and severe MVM also showed no significant difference.

Discussion

In the present cohort study, we observed MVM in 61.8% ($n = 68$) of SPTB placentas. This rate is much increased when compared to unselected (near)term pregnancies. A large prospective cohort study in an unselected at or near-term population observed MVM in 8% ($n = 90$) of 1.122 pregnancies [27].

No statistical significant difference was found in the prevalence of clinical IPD in the subsequent pregnancy between women with or without MVM in the placenta of the index pregnancy. In the subsequent pregnancies, we observed a hypertensive disorder of pregnancy in 12.7% ($n = 14$). The incidence of hypertensive disorders of pregnancy was increased when compared to national epidemiological data from the Dutch Perinatal Registry, where hypertensive disorders of pregnancy were recorded in 5.6% ($n = 18.332$) of 329.300 multiparous women without a history of SPTB [28]. Our study shows an increased risk of

Table 3. Outcome subsequent pregnancy related to histological placental evaluation in the index pregnancy.

Outcome subsequent, *n* (%) Total *n* = 110		Histological placental evaluation index: Maternal vascular malperfusion, *n* (%). Total *n* = 68	Histological placental evaluation index: No maternal vascular malperfusion, *n* (%). Total *n* = 42	*p*-Value
Hypertensive disorders of pregnancy	14 (12.7)	11 (16.2)	3 (7.1)	.15
Preeclampsia	3 (2.7)	3 (4.4)	0 (0)	.28
PIH	11 (10.0)	8 (11.8)	3 (7.1)	.35
Fetal growth restriction, <P10	6 (5.5)	4 (5.9)	2 (4.8)	>.99
Placental abruption	1 (0.9)	0 (0)	1 (2.4)	.39
Fetal loss	3 (2.7)	1 (1.5)	2 (4.8)	.56
Recurrent SPTB	32 (29.6)	21 (30.8)	11 (26.2)	.14

PIH: pregnancy induced hypertension; SPTB: spontaneous preterm birth.

hypertensive disorders of pregnancy in women with a history of SPTB. Clinicians should therefore be aware of an increased risk of hypertensive disorders in a subsequent pregnancy in women with a history of SPTB.

More than half of the studied women had a history of extreme premature birth. In 20.9% ($n = 23$) an (other) clinical form of IPD was observed in the SPTB pregnancy, more than half of these conditions was FGR.

MVM seems to play a role, also in extreme premature births, even though this group is known to have a higher rate of infection than late and early preterm births [29]. The studied population of a tertiary obstetric care center is possibly not representative for the total SPTB population. As the prevalence of extreme premature birth is over 50% in our cohort, in comparison with 5% in epidemiologic data [1]. Previous studies showed that MVM is most frequently observed in moderate to late preterm births [30], therefore our finding might be an underestimation. However, we still observed MVM in 61.8% of the placentas. This indicates an important role of MVM in the pathogenesis of SPTB, as one of the several factors leading to (extreme) SPTB [31]. We did not find a difference in recurrence of preterm birth depending on placenta pathology of the first pregnancy. The fact that MVM was found in such a large proportion of spontaneous preterm birth placentas adds to the little evidence that exists on the possible effect of low dose aspirin in reducing the number of SPTB [32,33].

Percentile value for birth weight was only available from 24 weeks onward. Therefore, due to the large portion of extremely premature neonates in our cohort, in 28.2% of cases the percentile for birth weight and in 22.7% of growth on Hadlock 3 could not be determined.

Another limitation of this study is the relatively small number of cases. The studied cohort is not comprehensive enough to establish a statistically significant association between MVM and clinical IPD in the subsequent pregnancy. We conclude that the risk of developing hypertensive disorders in subsequent pregnancy after SPTB is elevated. In a large proportion of SPTB placentas signs of MVM are seen, yet these signs are not predictive of recurrent SPTB.

Author contributions

MdB, PvdV, and LV were involved in the conception and design of the study. PvdV performed the histological examination with assistance of HvB. LH and LV analyzed the data. LV and MdB drafted the manuscript. All authors edited the manuscript and read and approved the final version of the manuscript.

Disclosure statement

No potential conflict of interest was reported by the authors.

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