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ORIGINAL ARTICLE

Placental thickness-to-estimated foetal weight ratios and small-for-gestational-age infants at delivery

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ABSTRACT

This study aimed to determine the correlation between the placental thickness-to-estimated foetal weight ratio on midterm ultrasonography and small-for-gestational-age (SGA) infants. In this retrospective study, the placental thickness at the umbilical cord insertion site was measured and adjusted for foetal body weight at 18–24 weeks gestation. Investigators compared the data of women who delivered SGA infants (birth weight <10th percentile) with those of women who delivered non-SGA infants. Among the 1281 women in this study, those who delivered SGA infants were younger and less likely to be obese. Women with higher placental thickness-to-estimated foetal weight ratios delivered more SGA infants. In logistic regression analysis, a higher placental thickness-to-estimated foetal weight ratio remained associated with SGA infants. Since the placental thickness-to-estimated foetal weight ratio in midterm pregnancy was associated with infant body weight at delivery, this ratio could be an effective, adjunctive screening marker for predicting SGA status.

KEYWORDS

Placental thickness; foetal body weight; small-for-gestational-age; infant; midterm; ultrasonography

Introduction

Small-for-gestational-age (SGA) status is one of the main causes of perinatal morbidity and mortality, and it is associated with a higher risk of stillbirth, neonatal asphyxia, neurodevelopmental disorders and other neonatal complications (Lee et al. 2013). SGA is detected antenatally in 50% of cases and early identification can improve birth outcomes (American College of Obstetricians and Gynecologists 2013).

Several risk factors are associated with SGA neonates. These include maternal age over 40 years, smoking, cocaine use, daily vigorous exercise, prior history of SGA baby, stillbirth, maternal history of SGA, chronic hypertension, diabetes, renal disease, antiphospholipid antibody syndrome, paternal history of SGA, antepartum haemorrhage, foetal echogenic bowel during the second trimester ultrasound, preeclampsia, low maternal weight gain and pregnancy-associated plasma protein-A < 0.4 MoM (RCOG 2013). However, the pathophysiology of SGA is multifactorial and is still being investigated.

In addition to the maternal and foetal factors, the placenta is an important part of normal foetal growth. Indeed, asymmetric, late onset, foetal growth restriction (FGR) is caused by uteroplacental insufficiency, which commonly occurs after midterm (Nardozza et al. 2012). It is well-known that foetuses with asymmetric FGR have higher morbidity and mortality rates than foetuses with symmetric FGR. To date, researchers have evaluated placental size, volume and blood flow in order to better predict abnormal foetal growth (Hafner et al. 2006; Zhong et al. 2010; Odibo et al. 2011). Generally, a thick and globular-shaped placenta with placental infarction and abruption is associated with SGA (Ness and Sibai 2006; Salafia et al. 2006; Damodaram et al. 2010). However, the exact correlation between placental thickness and SGA remains controversial. Indeed, the findings from two recent studies indicate that placental thickness and diameter are reduced in SGA foetuses (Schwartz et al. 2012, 2014). These inconsistent results may be due to the relatively subjective criteria for measuring placental thickness (e.g. maximal or central placental thickness). Since the measurement of placental thickness is relatively simple, the use of placental thickness for the prediction of SGA would seem clinically valuable. Also, rather than measuring the placental size itself, measuring the relative placental size adjusted for foetal size appears reasonable; for example, Hafner et al. used a placenta quotient (placental volume/crown-rump length) applied in the first trimester (Hafner et al. 2006). Given the late onset of asymmetric-type FGR, the measurement of placenta thickness in the second trimester, as opposed to the first trimester, would better detect the pathologic placenta related to foetal growth problems due to uteroplacental insufficiency.

Therefore, the purpose of this study was to investigate whether the relative placental thickness-to-estimated foetal weight measured objectively on midterm ultrasonography might be used to predict SGA status.

Materials and methods

Trained staff retrospectively collected the data through a review of the medical records of women who presented to the Korea University Medical Center and underwent an

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ultrasonography during their second trimester (18–24 weeks) between 1 January 2011 and 30 April 2014. The hospital's institutional review board approved this study (ED15039) and waived the requirement to document informed consent. We screened 1639 women who underwent ultrasonography. Exclusion criteria included insufficient ultrasonographic data, the absence of delivery or neonatal data, congenital infections and congenitally anomalous infants. The final study population comprised 1281 women.

The following factors were assessed as basic demographic characteristics or possible confounders: age (years) at the time of delivery, height (cm), weight (kg), body mass index (BMI) (kg/m^2), parity and cigarette smoking history. We recorded blood glucose levels (mg/dL) measured during a 50-g oral glucose tolerance screening test.

Infants' sex, birth weight (kg) and gestational age at delivery (weeks) were evaluated as delivery-related factors. The following other medical or surgical conditions were investigated as confounding factors for foetal growth: hypertension, including pregnancy-associated forms of hypertension; diabetes mellitus, including gestational diabetes; thyroid dysfunction; history of pelvic inflammatory disease; prior caesarean section; and previous conisation of the uterine cervix. Concerning hypertension, gestational hypertension and preeclampsia were not distinguished owing to the partial lack of maternal postpartum data.

We measured the placental thickness in centimetres from the foetal side of the echogenic chorionic plate to the maternal side of the placental–myometrial interface at the umbilical cord insertion site, perpendicular to the chorionic and basal plates, after verification that the umbilical vessels were attached to the placenta using colour Doppler ultrasonography (Figure 1). Primarily, the anterior and posterior positions of the placenta were included to achieve accurate measurement of placental thickness, and the fundal and lateral positions were mainly excluded. To evaluate placental thickness relative to foetal body size, we calculated the placental thickness-to-estimated foetal weight ratio. The gestational age at the time of ultrasonography, estimated foetal body weight auto-calculated via ultrasound (Hadlock et al. 1985), and single deepest pocket estimation of the amniotic fluid volume were recorded. SGA was defined as birth weight less than the 10th percentile for gestational age. Sex-specific charts were not used for the definition or classification of SGA; however, we did include infantile sex in the analysis.

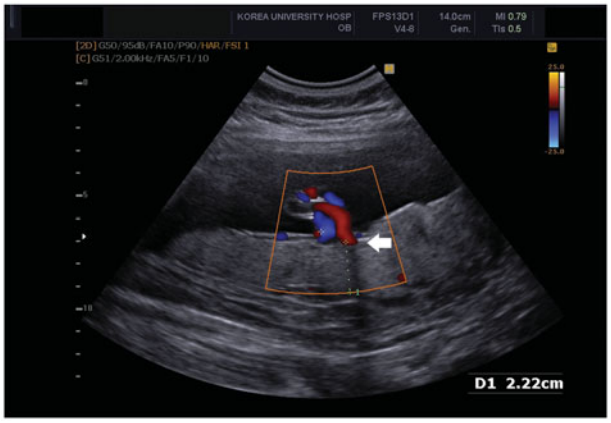

Figure 1. Measurement of placental thickness using ultrasonography during the second trimester. The placental thickness measured at the insertion site of the umbilical cord (white arrow), with a thickness of 2.22 cm.

We expressed the normally distributed continuous variables as means ± standard deviations, using the Student's *t* test for inter-group comparisons of the continuous variables. Differences in the percentages of the groups were analysed using the χ^2 tests or Fisher's exact test. We performed multivariate logistic regression analysis and reported the odds ratios and 95% confidence intervals to determine the risk factors associated with SGA infants. All statistical analysis was performed using the Statistical Package for the Social Sciences version 13.0 (SPSS Inc., Chicago, IL). We used two-sided tests and a p value of $p < .05$ was considered statistically significant.

Results

In the current study, 10.2% (131/1281) of the women delivered SGA infants. The women who delivered SGA infants were younger ($p = .02$) and less likely to be obese ($p = .002$) (Table 1). Lower parity status ($p = .001$) and hypertension in pregnancy ($p = .007$) were associated with a higher delivery rate of SGA infants that were delivered at a younger gestational age ($p = .045$). The body weights of the newborn infants were significantly lower in the SGA group compared to those in the non-SGA group ($p < .001$). Female infants were also more likely to be SGA than were male infants ($p = .008$).

The ultrasonographic findings showed that while gestational age and placental thickness at the time of ultrasonography did not differ between the SGA and non-SGA groups, the placental thickness-to-estimated foetal weight ratio was significantly higher in the SGA group ($p = .003$) (Table 2). Women who delivered SGA infants had a significantly lower estimated foetal body weight at their mid-trimester ultrasonography ($p < .001$). The amniotic fluid volume, which was calculated using the single deepest pocket technique, did not differ between the groups.

Table 1. Demographic and perinatal characteristics of the study participants.

	Non-SGA ($n = 1150$)	SGA ($n = 131$)	p Value
Age, years	32.9 ± 4.4	31.9 ± 4.2	.02*
Height, cm	160.5 ± 5.3	159.3 ± 5.0	.02*
Weight, kg	66.0 ± 12.9	61.5 ± 12.2	<.001*
Body mass index, kg/m^2	25.6 ± 4.9	24.2 ± 4.6	.002*
Parity, *n* (%)	0.6 (0.7)	0.5 (0.6)	.001*
Hypertension, any type, *n* (%)	46 (4.0)	12 (9.2)	.007*
Diabetes mellitus, overt or gestational, *n* (%)	88 (7.7)	10 (7.6)	.992
Thyroid dysfunction, *n* (%)			.739
Hyperthyroidism	18 (1.6)	3 (2.3)	
Hypothyroidism	45 (3.9)	4 (3.1)	
Prior caesarean delivery, *n* (%)	282 (24.5)	23 (17.6)	.075
Prior conisation of the uterine cervix, *n* (%)	13 (1.1)	3 (2.3)	.217
History of pelvic inflammatory disease, *n* (%)	2 (0.2)	0 (0)	1.0
Smoking[a], *n* (%)	0 (0)	0 (0)	NA
Gestational age at delivery, weeks	38.2 ± 2.4	37.7 ± 2.8	.045*
Foetal body weight at delivery, g	3211 ± 539.6	2413.6 ± 526.7	<.001*
Female infant, *n* (%)	552 (48.1)	79 (60.3)	.008*

Data were presented as mean ± standard deviation.
SGA: small for gestational age (<10th percentile).
*Statistically significant.
[a]Current or past smoker.

Table 2. Ultrasonographic findings in the study subjects.

	Non-SGA ($n = 1150$)	SGA ($n = 131$)	p Value
Gestational age at ultrasonography, weeks	21.0 ± 1.1	20.9 ± 1.1	.642
Foetal body weight at ultrasonography, kg	0.427 ± 0.092	0.396 ± 0.095	<.001*
Ultrasonographic placental thickness[a], cm	2.6 ± 0.7	2.7 ± 0.7	.394
Placental thickness-to-estimated foetal weight ratio[b]	6.3 ± 2.0	7.2 ± 2.6	.003*
Amniotic fluid, single deepest pocket, cm	4.5 ± 1.0	4.5 ± 1.2	.813

Data were presented as mean ± standard deviation.
SGA: small for gestational age (<10th percentile).
*Statistically significant.
[a]At the insertion site of the umbilical cord.
[b]This parameter was calculated with placental thickness in centimetres and estimated foetal weight in kilograms.

Table 3. Demographic and ultrasonographic risk factors for small-for-gestational-age status (<10th percentile) at delivery.

	OR[a]	95%CI[a]	p Value[a]	OR[b]	95%CI[b]	p Value[b]
Age	0.963	0.91–1.018	.184	0.964	0.911–1.021	.209
Body mass index	0.937	0.891–0.985	.011*	0.938	0.892–0.986	.012*
Parity	0.822	0.576–1.174	.281	0.824	0.577–1.175	.285
Hypertension	3.951	1.746–8.94	.001*	4.055	1.784–9.216	.001*
Female infant	1.805	1.128–2.886	.014*	1.786	1.115–2.86	.016*
Placental thickness-to-estimated foetal weight ratio	5.812	2.242–15.07	<.001*	3.412	1.0985–10.599	.034*
Foetal body weight estimated at ultrasonography				0.997	0.994–1.0	.087

OR: odds ratio; CI: confidence interval.
*Statistically significant.
[a]Logistic regression analysis performed on age, body mass index, hypertension, female sex and placental thickness-to-estimated foetal weight ratio.
[b]Logistic regression analysis performed on age, body mass index, hypertension, female sex, placental thickness-to-estimated foetal weight ratio and foetal body weight estimated at ultrasonography.

In multivariate logistic regression analysis, lower BMI, hypertension, female sex and higher placental thickness-to-estimated foetal weight ratio were significantly associated with SGA status ($p < .001$). The placental thickness-to-estimated foetal weight ratio had a stronger and more significant relationship with SGA ($p = .034$) compared to that of foetal body weight ($p = .087$) (Table 3).

We used a receiver operating characteristic (ROC) curve analysis to determine the predictive accuracy of the placental thickness-to-estimated foetal weight ratio for SGA infants. The placental thickness-to-estimated foetal weight ratio was the only parameter with statistical significance in predictive performance as compared with estimated foetal weight and placental thickness ($p = .003$ vs. .063 and .109, estimated foetal weight and placental thickness, respectively) (Figure 2). The mean placental thickness-to-estimated foetal weight ratio of non-SGA infants (6.3) had a sensitivity and specificity of 55.7% and 54.5%, respectively, whereas the ratio of the SGA group (7.2) had a sensitivity and specificity of 39.8% and 71.9%, respectively (Table 4). The placental thickness-to-estimated foetal weight ratios showed high negative predictive and low positive predictive values for SGA status.

Discussion

In the present study, a thick placenta relative to body weight in the second trimester was associated with small neonates. This finding is consistent with that of a magnetic resonance imaging study that used maximal placental thickness as a parameter (Damodaram et al. 2010); the placental thickness was considered greatest at the point where the umbilical cord entered the placenta. In the case of eccentric cord insertion, the thickest part of the placenta was selected for measurement. We do not consider the method used in that study to assess 'the thickest' placenta site to be an objective measure.

However, we agree that the thickest area of the placenta may not correspond to the cord insertion site. The umbilical cord insertion site during the second trimester should be the same as the implantation site during early pregnancy. In most cases, the umbilical cord enters the centre of the placenta; however, as pregnancy progresses, the trophoblast, chorion and placenta migrate to a more highly vascularised area for superior blood supply (Robinson et al. 1983). From a methodological perspective, our study seems more objective since we measured the placental thickness at the umbilical cord insertion site. It is interesting that the relative placental thickness measured during the second trimester remained significantly associated with foetal body weight at delivery. This finding is consistent with a previous study in which a thick and globular placenta was shown to be associated with restricted intrauterine growth (Damodaram et al. 2010).

Most pathological studies on FGR have reported low placental weight and volume with increased apoptosis within the intramural and endovascular trophoblast, as well as a thicker and globular placenta (Kaufmann et al. 2003; Biswas and Ghosh 2008). Although it seems contradictory, this phenomenon can be explained by the fact that uterine contractions thicken the placenta. Since a tense and hypertonic uterine state is associated with potentially reduced blood flow to the foetus and FGR (Li et al. 2006), it follows that a thickened placenta during the second trimester would be associated with SGA status. Other hypotheses have attempted to reveal the association between a thickened placenta and FGR. Irrespective of hypertonic uterine musculature, placental growth could increase in the presence of problematic placental blood circulation. The hypoxic state of the placenta, due to reduced trophoblast invasion, induces trophoblast

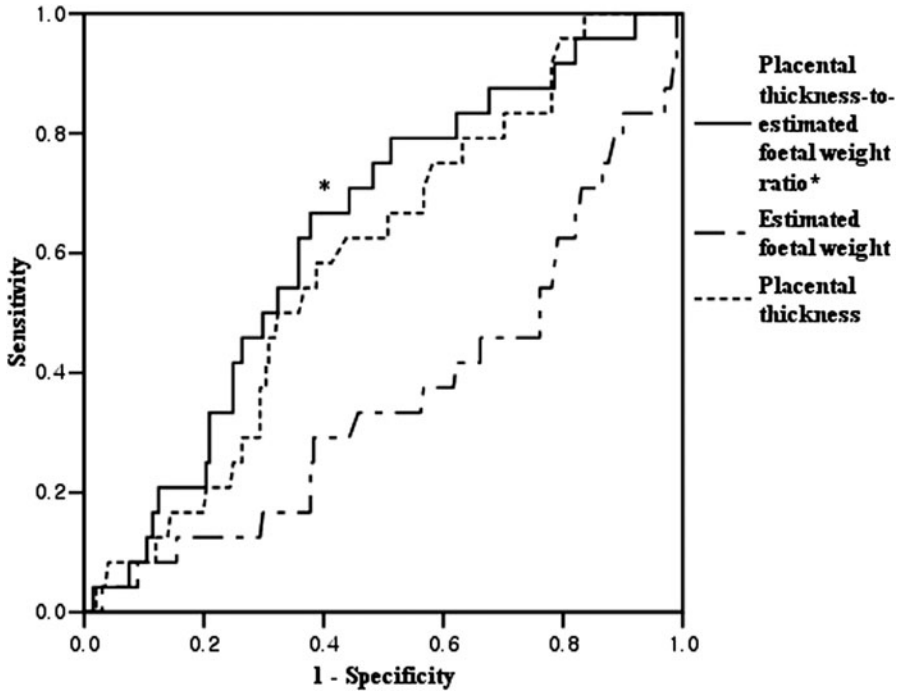

Figure 2. Comparison of predictive accuracy among three parameters using ROC curve analysis. Area under the curve (AUC) were 0.648, 0.426 and 0.597 for the placental thickness-to-estimated foetal weight ratio, estimated foetal weight and placental thickness, respectively. *Statistically significant.

Table 4. Sensitivities, specificities and predictive values of placental thickness-to-estimated foetal weight ratio for predicting small-for-gestational-age infant status (<10th percentile) at delivery[a].

Placental thickness-to-estimated foetal weight ratio[b]	Sensitivity	Specificity	Positive predictive value	Negative predictive value
3.0	1.0	0.017	0.103	1.0
4.0	0.943	0.097	0.105	0.934
5.0	0.841	0.267	0.11	0.934
6.0	0.591	0.494	0.109	0.912
6.3	0.557	0.545	0.11	0.913
7.0	0.443	0.677	0.113	0.912
7.2	0.398	0.719	0.113	0.91
8.0	0.33	0.832	0.12	0.913
9.0	0.193	0.916	0.115	0.91
10.0	0.136	0.956	0.113	0.904

[a]ROC curve analysis was performed.
[b]Ultrasonography performed at 18–24 weeks gestation, with placental thickness-to-estimated foetal weight ratio in centimetres and foetal body weight in kilograms.

apoptosis and interrupts placental growth, resulting in FGR (Ishihara et al. 2002; Levy et al. 2002). To compensate for placental growth interruption, released hypoxia-inducible factor-1 increases the expression of vascular endothelial growth factor and produces a low-oxygen state before mid-gestation stimulates branching angiogenesis (Khaliq et al. 1999; Caniggia and Winter 2002). These effects may partly explain the compensatory placental growth. However, it remains unclear why the compensatory growth tends to occur perpendicularly rather than along the uterine wall. Further studies are needed to elucidate the mechanisms underlying the direction of placental growth in these hypoxic circumstances.

The results from our data indicate that a relatively thick placenta is a better predictor of SGA compared to foetal weight. The placental thickness-to-estimated foetal weight presented high negative and low positive predictive values due to the low prevalence of SGA. Based on the predictive values, the placental thickness-to-estimated foetal weight could be considered an adjunctive rather than an independent screening tool for SGA during the second trimester of pregnancy. Our data suggest that if the placental thickness-to-estimated foetal weight ratio is greater than 6.3 (the mean value of the non-SGA group in this study), then the sensitivity and specificity are both greater than 50%. Although a predictive accuracy of 6.3 is not very high, this result may still be clinically meaningful for physicians when counselling pregnant women with multiple risk factors for foetal growth problems leading to the possibility of delivering an SGA infant. If the association between placental thickness-to-estimated foetal weight ratio and SGA is somewhat weak due to the wide confidence interval (10-fold) and the lower edge of the confidence interval (1.1), a higher placental thickness-to-estimated foetal weight ratio provides evidence for the need to carefully monitor foetal growth during subsequent antenatal care.

Despite the potential clinical importance, our study has some limitations. Since we did not measure the placental volume or uterine wall thickness, a definitive conclusion could

not be reached regarding the correlation between a thick placenta associated with a thickened uterine wall, placental volume and SGA status. Calculations of the Doppler indices within the umbilical artery and vein, middle cerebral artery, uterine artery and ductus venosus would have provided information about the correlation between functional vascular flow changes in the thickened placenta and SGA. Therefore, in our study, we did not differentiate pathologic SGA from normally small infants. We realise that the small size of many of the infants in our results may have been due to constitutional factors. However, we did exclude individuals with congenital anomalies from our study. Moreover, the time of the placental thickness measurement was at midterm and not in the early pregnancy period. A thickened placenta may be associated with pathologic SGA due to uteroplacental insufficiency related to late-onset FGR. Therefore, a certain proportion of the SGA infants in our study would have been pathologic. Further studies on serially measured placental thicknesses and volumes, uterine wall thicknesses at the sites of the placental thickness measurements, placental vascular Doppler examinations during each pregnancy trimester, pathologic information and neonatal outcomes are needed to more clearly differentiate pathologically small foetuses. A second limitation of our study is the lack of measurement regarding the inter- and intra-observer variability of the placental parameters. Information pertaining to measurement reproducibility would make these results more reliable.

In conclusion, the placental thickness-to-estimated foetal weight ratio is associated with infant body weight at delivery. Since this ratio is an easily obtainable, useful ultrasonographic marker for predicting the delivery of SGA infants, our data might be helpful for the obstetric counselling of women with high-risk pregnancies associated with SGA.

Disclosure statement

The authors report no declarations of interest.

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