Original Article

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Evaluation of Placental Weight Ratio in Preterm Births and Small for Gestation Age Babies in Preeclampsia in Sikkimese Population

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Abstract

Background: Studies have established placental weight ratio (PWR) as a relevant marker of fetal uteroplacental function which reflects whether the fetal and placental growth is proportionate. It decreases across gestation as the placental growth slows and fetal growth accelerates. Preeclampsia and preterm birth are both risk factors of small for gestational age (SGA) off springs with the smaller placenta. The PWR is elevated in both these conditions which share a common pathophysiology.

Aim: The study aims to determine if PWR is a reliable indicator of placental function and fetal growth and to evaluate the PWR by gestational age in preterm births in preeclampsia in Sikkimese population.

Methods: A total of 150 placentae were analyzed, out of which 100 were from normotensive mothers and 50 from pre-eclamptic mothers. Data analysis was done using Statistical Package of Social Sciences 17. Student's *t*-test and Fisher's Chi-square test. $P < 0.05$ was considered to be statistically significant.

Results: Significantly, higher PWR was observed in pre-eclamptic pregnancies (0.001) and in both preterm (0.009) and term (0.048) births. The elevated ratio was observed only in SGA off springs (0.037) of pre-eclamptic mothers and not in normotensive mothers. Decreasing PWR across gestation was documented only in pre-eclamptic patients. The mean birth weight (0.004) and placental weight (0.0001) were significantly reduced in pre-eclamptic pregnancies while the PWR was elevated. The peak placental growth was delayed by several weeks in preeclampsia. No significant difference between preterm and term off springs of cohorts or controls was observed.

Conclusion: Preeclampsia is a confounding variable in SGA leading to raised PWR and is a reliable indicator of pregnancy complications. The influence of gestational age on this ratio and uteroplacental function could not be established. The PWR is not a relevant marker of fetal growth as it is not raised in all SGA off springs.

Key words: Gestation, Preeclampsia, Weight

INTRODUCTION

Placental weight ratio (PWR) is regarded by many as an appropriate marker of uteroplacental function and reflects the balance between fetal and placental growth.[1-4] It is predictive of maternal diseases, obstetric outcomes, perinatal mortality, morbidity, childhood development, and fetal origin of adult onset diseases.[5-9] It is postulated to decrease across gestation as the placental growth slows and fetal growth accelerates.[3,10-12] Although, the placental and birth weights are lower in small for gestational age (SGA) neonates. and preeclampsia the PWR is said to be elevated in them.[4,5,13-16] Insufficient implantation and impaired placental development in preeclampsia cause low placental weight and subsequently intrauterine growth restriction (IUGR).[17] The placenta compensates to minimize fetal growth restriction with lower weight and higher PWR to increase transfer of substrates.[2,10] Elevated PWR indicates an inefficient placenta unable to meet fetal growth requirement while lower ratio indicates increased placental efficiency.[18-20] Neonates with elevated PWR had increased incidence of meconium-stained liquor, low 1 min Apgar

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scores, neonatal jaundice, and respiratory complications.[21] Preeclampsia often necessitates iatrogenic preterm delivery to curtail the placental and fetal adversities.[22] SGA is used as an indicator of fetal growth restriction and defined as birth weight <10th percentile for age and sex. Placental weight has been found to differ between SGA and average-for-gestational age (AGA) infants and in preeclampsia.[2,18,23] PWR could be a crucial indicator of pregnancy complications. The findings would be crucial in understanding the risks of mortality in preterm SGA offspring in preeclampsia and in specifically targeting the disorder for appropriate interventions.

METHODS

A prospective study of cohort design carried out in the Department of Obstetrics and Gynecology at Central Referral Hospital and the department of Anatomy at Sikkim Manipal Institute of Medical Sciences, Gangtok Sikkim from May 2008 to April 2009. A total of 150 placentae were analyzed out of which 100 were from normotensive mothers and 50 from pre-eclamptic mothers. Age matching was done to identify each cohort with two corresponding controls. The study of the gross morphology of placenta was conducted in the Department of Anatomy. After delivery, the placenta were collected from the labor room and operation theatre, examined, washed, dried, and fixed in 10% formalin. The placental weight, volume size, calcification, infarction, and PWR were observed in preterm and term births in preeclampsia and normotensive mothers. The collected data were tabulated and analyzed using the Statistical Package of Social Sciences, version 20.0 for windows. Findings were expressed in terms of proportion and depicted in the form of tables. Student's *t*-test and Fisher's Chi-square test were applied for univariate analysis to study the effect of each variable over the outcome. $P < 0.05$ was considered to be statistically significant all these 150 participants had singleton pregnancies, and their outcome was live births.

RESULTS

The PWR was significantly higher ($P = 0.001$) in pre-eclamptic pregnancies compared to normotensive controls. Decreasing PWR with increasing gestational age was observed only in pre-eclamptic mothers with the maximum at 31 (0.2) weeks and minimum (0.16) at 38 weeks demonstrating that the placental growth peak was delayed in pre-eclamptic mothers. It decreased up to the 34th week after which it was stable at 0.18 up to the 38th week nevertheless in normotensive pregnancies the PWR was constant (0.17) throughout suggesting that placental growth achieved its peak growth much earlier (34 weeks). The PWR in the term (0.048), as well as preterm births (0.009) of pre-eclamptic mothers was significantly higher. No significant difference in PWR was observed between preterm and term births in pre-eclamptic or normotensive mothers. Also, no difference between the PWR of SGA and AGA offsprings in normotensive pregnancies was observed. The mean birth weight (0.004) and placental weight (0.0001) were significantly reduced in pre-eclamptic pregnancies while the PWR was elevated (Tables 1-4).

DISCUSSION

In this retrospective study of the PWR, we observed that though the placental and birth weights were closely correlated and lower in preeclampsia and SGA offsprings the PWR was raised. The impaired endothelial invasion of cytotrophoblasts leads to reduced capillary size of terminal villi and reduced no of arteries in tertiary term villi and reduced perfusion which reduces the efficiency of the placenta as reflected by a raised PWR.[18,24-27] The increased PWR in SGA and preeclampsia as suggested by many authors appears to be an adaptive mechanism to tide over the unfavorable environment.[2,19,28] In the present study elevated PWR in SGA was observed only in preeclampsia suggesting that predetermining risk factors causing reduced placental perfusion must coexist with IUGR to exert their effect on uteroplacental function.[1] However, no significant difference in PWR was observed between SGA and AGA offsprings in normotensive pregnancies contrary to study of Macdonald *et al.* who documented elevated PWR in SGA offsprings, nonetheless he studied the PWR of SGA births in uncomplicated pregnancies.[2] We observed a PWR of 0.21 in premature deliveries of pre-eclamptic mothers and 0.16 in normotensives. Raghunathan *et al.* observed a PWR of 0.22 in premature deliveries which was similar.[4] Our PWR distribution differs from that observed by Macdonald *et al.* who observed that majority of the placental growth occurs before 33 weeks of gestation whereas in our study placenta reached its peak growth at 37-38 weeks in preeclampsia when the PWR was the least which indicates that placental efficiency was delayed in both cases.[2,23] Our observation of the highest PWR (0.2) at 31 weeks in pre-eclamptic mothers indicates that placental efficiency was the least in earlier weeks while in the normotensive mothers the PWR was lower and constant throughout gestation suggesting that placenta achieved its peak growth much earlier in normotensive pregnancies. Our study does not support the hypothesis that the placenta and fetus follow a different growth pattern with the placenta reaching its peak growth between 28 and 30 weeks while the fetus reaches its peak growth at term.[23] The mean birth and placental weight increased from SGA to large for gestational age offsprings yet the PWR significantly decreases in AGA.[2,29] Similarly,

Table 1: Comparison of placental morphometry between preeclamptic and normotensive pregnancies

Morphology of placenta	Mean (SD)		*P*-value from *t*-test
	Preeclampsia	Normotensive	
Placental weight (g)	415.44 (91.6)	484.69 (66.3)	0.0001
Placental volume (cc)	430.0 (81.3)	491.6 (65.3)	0.0001
Placental thickness	2.6 (0.33)	2.7 (0.33)	0.10
Placental diameter	19.9 (1.6)	20.3 (1.92)	0.20
Foetoplacental weight ratio	5.5 (0.40)	5.8 (0.33)	0.001
Placental coefficient/ PWR	0.18 (0.03)	0.17 (0.010)	0.001
Birth weight	2.2 (0.47)	2.8 (0.43)	0.004

P<0.05 was statistically significant, SD: Standard deviation, PWR: Placental weight ratio

Table 2: Comparison of morphometry of the placenta and birth weight between pre-term and term small for gestation age babies in preeclampsia

Placenta	Mean (SD)		*P* value from *t*-test
	Preterm SGA	Term SGA	
Birth weight	1.7 (0.34)	2.2 (0.15)	0.0006
Placental weight (g)	333 (50.4)	433.7 (27.6)	0.0002
Placental volume (cc)	330.5 (46.8)	455.7 (50.6)	0.0001
Foetoplacental weight ratio	5.3 (0.3)	5.0 (0.38)	0.34
PWR/placental coefficient	0.2 (0.03)	0.18 (0.01)	0.41

P<0.05 was considered to be statistically significant, SD: Standard deviation, SGA: Small for gestational age, PWR: Placental weight ratio

lower weighing placentas in preeclampsia have been reported by several authors.[4,5,30,31] Gestational age greatly influences placental growth with a significantly higher prevalence of smaller placenta in preterm preeclampsia compared to term preeclampsia.[32] Several studies have documented an increasing PWR with advancing gestational age.[3,10-12] Disparate to observations of most of these authors the influence of gestational age on the PWR could not be well established in our study as a decreasing PWR with increasing gestational age was again observed only in pre-eclamptic pregnancies.[1-3] This hypothesis is further strengthened by the fact that though lower weighing placenta were observed in preterm offsprings in both cohorts and controls no significant difference in the PWR was observed between preterm and term births. The slightly lower PWR across gestation observed in uncomplicated pregnancies in our study contrasts with those of other authors however our result is similar to Williams *et al.*[1-4] The mean PWR (0.16) in normotensives as well as pre-eclamptic mothers in our study at 40 weeks was lower (0.17) than that of Raghunathan *et al.*[4] and Macdonald *et al.*[2] (0.19). Lower mean PWR across all groups in our study could be attributed to pre-placental hypoxia and lower birth weight at higher altitudes as observed by various studies. Though, similar reduced placental weights and smaller placentae were revealed in

Table 3: Comparison of morphometry of the placenta and birth weight of preterm and term small for gestation age babies in normotensive pregnancies

Placenta	Mean (SD)		*P* value from *t*-test
	Preterm SGA	Term SGA	
Birth weight	2.0 (0.54)	2.4 (0.26)	0.02
Placental weight (g)	390.8 (76.9)	448.0 (40.3)	0.03
Placental volume (cc)	406.6 (55.7)	457.0 (48.5)	0.02
Foetoplacental weight ratio	5.6 (0.34)	5.4 (0.44)	0.27
PWR/placental coefficient	0.17 (0.008)	0.17 (0.001)	0.05

P<0.05 was considered statistically significant, SGA: Small for gestational age, SD: Standard deviation, PWR: Placental weight ratio

Table 4: Comparison of PWR of different studies in uncomplicated pregnancies

Authors	Year of publication	PWR
Williams *et al.*[3]	1997	0.17 (0.27)
Raghunathan *et al.*[3]	2011	0.19
Almog *et al.*[6]	2011	0.19
Macdonald *et al.*[5]	2014	0.17 (0.044)
Present study	2015	0.17 (0.011)

PWR: Placental weight ratio

our study no significant difference in PWR was elicited between term and preterm births in pre-eclamptic as well as in normotensive mothers.[32] As reported by several authors the placental weight to birth weight were highly correlated and an increased placental to birth weight ratio could be predicted by the birth weight and vice versa.[1,5,32] Considering that the PWR were observed after delivery in our sample similar ultrasound based investigations of placental morphology and PWR in the antenatal period would give more appropriate results. Also, the small size of the sample could be a drawback and studies of larger population based cross-sectional studies need to be undertaken in the near future. The findings would be crucial in understanding the risks of mortality in preterm SGA offspring in preeclampsia and in specifically targeting the disorder for appropriate interventions.

CONCLUSION

The PWR is not correlated with fetal growth as no significant difference between SGA and AGA offsprings was recognized. The PWR is not a relevant marker of fetal growth as it is not raised in all SGA off springs. Nevertheless in complications like preeclampsia, the uteroplacental function is greatly compromised resulting in elevated PWR. Preeclampsia is a confounding variable in SGA leading to raised PWR and is a reliable indicator of pregnancy complications. The influence of gestational age on the PWR and uteroplacental function in any of the groups could not be established.

The mean placental weight and birth weight were lower in preterm pre-eclamptic pregnancies while the PWR is higher with no significant difference between preterm and term births. In normotensive pregnancies, no difference across gestational age in SGA and AGA (appropriate for gestational age off-springs) was observed. The findings would be crucial in understanding the risks of mortality in preterm SGA offspring in preeclampsia and in specifically targeting the disorder for appropriate interventions.

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