

Association Between Placental (Pro)Renin Receptors and Small for Gestational Age in Preeclampsia

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Abstract: Preeclampsia is a pregnancy-specific syndrome of new onset hypertension and proteinuria in 2-8% of pregnancies worldwide and is a major cause of maternal and perinatal morbidity and mortality. Aim to explore the relationship between placental (pro)renin receptor to assess its predictive and concentration and small-for-gestational age rates in women who developed preeclampsia. Cross-sectional observational study at Basrah Maternity Hospital during December 2024-July 2025. A total of eighty pairs of pre-eclamptic pregnant women with their newborns admitted to the labor ward with singleton term pregnancies. Those who participated were divided into two groups based on neonatal birth weight for gestational age: preeclampsia with SGA (n = 40) and preeclampsia with AGA (n = 40). Placental (P)RR concentration was measured using enzyme-linked immunosorbent assay (ELISA) Written informed consent was obtained from all participants before enrollment. Placental (pro)renin receptor levels were significantly increased in the SGA group compared to appropriate-for-gestational age (AGA) group, and there was a statistically significant inverse correlation with neonate birth weight and a direct correlation with maternal systolic blood pressure. The placental (pro) renin receptor was predictive at the cut-off of 81.5ng/ml. The 78% sensitivity and 50% specificity, and an area under the curve of 0.69, were obtained with small-for-gestational age births. In preeclamptic pregnancies, (pro)renin receptor concentration in the placenta is negatively correlated with fetal birth weight and maternal SBP. It has a moderate predictive value for small for gestational age newborns.

Keywords: Preeclampsia, Placental Prorenin Receptor, Impaired Fetal Growth

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Introduction

Preeclampsia (PE) is a multisystem pregnancy disorder defined as new onset hypertension after 20 weeks of gestation, with potential proteinuria, organ dysfunction in the mother and/or impaired uteroplacental perfusion [1]. Its prevalence in the majority of pregnancies is estimated to be between 2-8% and it is an important cause of maternal and perinatal morbidity and mortality worldwide [2]. Its pathogenesis is characterized by a failure of the trophoblast to invade the maternal tissue and remodelling the spiral arteries, leading to placental hypoperfusion, hypoxia, oxidative stress, angiogenic imbalance and generalised endothelial dysfunction in the mother [3].

The placental insufficiency that occurs with PE can impact on fetal growth and is closely correlated with fetal growth restriction and small-for-gestational-age (SGA) infants [4]. The normal definition of SGA is a birth weight below the 10th percentile of the gestation-age curve and can be either constitutional smallness or pathological growth restriction [5]. Infants with growth restriction are more likely to be premature, be born with asphyxia, become hypoglycaemic, be hypothermic, to have sepsis and to die in early childhood [6]. It is very common for early-onset PE to have associated placentation and fetal growth restriction [7].

There is growing evidence that PE and abnormal fetal growth may be related to dysregulation of the placental renin–angiotensin system (RAS). The (pro)renin receptor [(P)RR] is a receptor for renin and prorenin, which increases local angiotensin production and activates intracellular pathways that play a role in oxidative stress and inflammation as well as vascular dysfunction [8]. In preeclamptic pregnancies, it has been reported that placental (P)RR expression and serum soluble (P)RR levels are increased, which may result in increased uteroplacental RAS activity and endothelial dysfunction [9]. Furthermore, an increase in placental RAS activity could affect the fetoplacental circulation and the function of the trophoblast [10]. Increased placental (P)RR level also has been seen in pregnancies that lead to SGA newborns [11]. Thus, understanding the relationship between placental (P)RR levels and SGA in preeclamptic women may be able to yield additional clues to the mechanisms through which placental dysfunction is associated with impaired fetal growth.

Methodology

This cross-sectional observational study was done at Basrah Maternity hospital from December 2024 till July 2025. 80 women with preeclampsia and singleton pregnancies admitted to the labor ward and delivered at term were included in the study. Neonatal birth-weight percentiles for gestational age were used to divide participants into two equal groups according to birth weight percentile. There were 40 women in each group, 40 of whom had delivered small-for-gestational-age (SGA) newborns and 40 others who had delivered appropriate-for-gestational-age (AGA) newborns. The study included women aged between 20 and 40 years and who were at a gestational age of 36–40 weeks. Patients who were multiple pregnancies had significant fetal structural abnormalities or placental structural abnormalities were excluded.

Preeclampsia was defined as the presence of new onset hypertension at ≥ 24 weeks gestation (systolic ≥ 140 mmHg and/or diastolic ≥ 90 mmHg on at least two occasions) and proteinuria (UPC ≥ 0.3 or proteinuria ≥ 300 mg/24 hours). All participants had a detailed demographic, obstetric, medical and medication history obtained, and general and obstetric examinations undertaken. Complete blood count, liver function tests, renal function tests and coagulation profile were performed as routine laboratory investigations.

Gestational age was confirmed with the first day of the last menstrual period, and/or early ultrasound examination prior to 20 weeks gestation if available. The fetal biometry parameters measured were biparietal diameter, head circumference, abdominal circumference, and femur length. Estimated fetal weight was estimated by validated biometric equations, such as Hadlock and Shepard, and compared to a fetal growth chart according to gestation. A fetal weight < 10 th percentile was defined as suggestive of SGA. Investigations for constitutionally small fetuses and pathological growth restriction were carried out using Doppler and amniotic fluid assessment.

Placental tissue samples were taken within 30 minutes of delivery. About 5 g of tissue was collected from the central placental parenchyma, away from the placental margin and the umbilical cord insertion and

infarctions, as well as from the placental tissue in the center of an infarction and of a calcification. The samples were placed in phosphate buffered saline and stored at 2-8 °C prior to processing. Placental tissue was homogenized with IKA ULTRA-TURRAX T25 digital homogenizer for 20 minutes at 20,500 rpm then centrifuged for 20 minutes at 4,000 rpm. The supernatant was kept at -40 °C for future analysis. The concentration of placental soluble (pro)renin receptor [s(P)RR] was determined by commercially available enzyme-linked immunosorbent assay (ELISA) kit following the manufacturer's instructions.

Venous blood from the mother was drawn into EDTA tubes and spun at 2,000 rpm for 5 minutes. Plasma was separated and stored at -40 °C until the time of analysis, and only once after thawing before testing. Soluble (pro)renin receptors in plasma was measured with validated ELISA method, plasma aldosterone concentrations and plasma renin activity was measured with validated ELISA method. The blood pressure of the mother was taken at least twice, by a calibrated mercury sphygmomanometer.

Newborn's birth weight was measured in a calibrated digital neonatal scale immediately after delivery. Birth weight was defined as appropriate for gestational age based on the use of an internationally accepted growth reference. Infants who had birth weight below the 10th percentile for GA were defined as SGA and those within the expected normal range were defined as AGA.

The study was approved by the IRB of Basrah Maternity Hospital. All participants were informed on the purpose and process of the study and informed consent written forms were obtained prior to the recruitment. All participants were kept anonymous and confidential during the collection, analysis and reporting of data.

SPSS version 26 was used for statistical analysis. Qualitative data were described as frequencies and percentages and quantitative data were described as mean $\pm$ standard deviation for normally distributed quantitative data. The Kolmogorov–Smirnov test was used to assess normality. The Chi-square test was used to compare categorical variables, and Fisher's exact test and Monte Carlo correction were performed when the cells in the contingency tables had values of less than five. Student's independent-samples t-test was used to compare the two independent groups for normally distributed continuous variables. A P value ≤ 0.05 was considered statistically significant.

Results

Table 1. Distribution of demographic data between the studied groups.

Mean $\pm$ SD	Preeclampsia with SGA (N=40)	Preeclampsia with AGA (N=40)	P-value
Maternal age (years)	32.2 $\pm$ 6.1	29.9 $\pm$ 6.3	0.10
Body mass index (kg/m ²)	33.1 $\pm$ 1.5	33.8 $\pm$ 0.99	0.01
Gestational age at delivery (weeks)	37.05 $\pm$ 1.1	37.3 $\pm$ 0.96	0.2
Birth weight(g)	2410.3 $\pm$ 234.1	2795.5 $\pm$ 232.5	<0.001
Placental weight(g)	433 $\pm$ 99	547 $\pm$ 96.8	<0.001
Family history of hypertension N (%)	7(17.5%)	8(20%)	0.77

Maternal age, gestational age at delivery and family history of hypertension were not significantly different between preeclamptic women with SGA and AGA infants (p=0.10, p=0.20 and p=0.77, respectively) (Table (1)). On the other hand, BMI was significantly higher in the AGA group than in the SGA group (33.8 $\pm$ 0.99 vs 33.1 $\pm$ 1.5 kg/m², p=0.01). Furthermore, SGA group had significantly lower levels of both birth weight (2410.3 $\pm$ 234.1 g) and placental weight (433 $\pm$ 99 g) than the AGA group (2795.5 $\pm$ 232.5 g and 547 $\pm$ 96.8 g, respectively; p<0.001 for both).

Table 2. Distribution of clinical data between the groups studied.

Mean $\pm$ SD	Preeclampsia with SGA (N=40)	Preeclampsia with AGA (N=40)	P-value
Systolic blood Pressure (mmHg)	167.8 $\pm$ 19.1	157.4 $\pm$ 11.6	<0.001
Diastolic blood Pressure (mmHg)	107.4 $\pm$ 11.9	98.9 $\pm$ 15.7	<0.001

Table (2) revealed that, both systolic and diastolic blood pressures were significantly elevated in preeclamptic women with SGA newborns compared to those with AGA newborns, with the mean systolic blood of 167.8 $\pm$ 19.1 mmHg in SGA group versus 157.4 $\pm$ 11.6 mmHg in AGA group, $p < 0.001$. In the same way, mean DBPs of SGA group (107.4 $\pm$ 11.9 mmHg) and AGA group (98.9 $\pm$ 15.7 mmHg) were statistically significantly different ($p < 0.001$).

Table 3. Distribution of laboratory data between study groups.

variable	Preeclampsia with SGA (n0.40)	Preeclampsia with AGA (N=40)	P-value
Placental sPRR (ng/ml)	87.9 $\pm$ 7.12	80.8 $\pm$ 8.81	<0.001
Plasma sPRR (ng/ml)	44 $\pm$ 5.4	43.8 $\pm$ 9.6	0.90
Proteinuria (g/day)	4.9 $\pm$ 1.6	2.4 $\pm$ 1.2	<0.001
eGFR (ml/min/1.73 m ²)	61.3 $\pm$ 13.1	85.2 $\pm$ 14.7	<0.001
GDM	3 (7.5%)	2 (5.1%)	0.2

When comparing the placental PRR levels in women with preeclampsia and those delivering SGA vs AGA newborns, placental levels were significantly higher in preeclampsia women who delivered SGA newborns (87.9 $\pm$ 7.12 ng/mL vs 80.8 $\pm$ 8.81 ng/mL, $p < 0.001$) (Table 3).

Proteinuria was also markedly higher in the SGA group (4.9 $\pm$ 1.6 vs 2.4 $\pm$ 1.2 g/day, $p < 0.001$), while eGFR was significantly lower, indicating worse renal function (61.3 $\pm$ 13.1 vs 85.2 $\pm$ 14.7 ml/min/1.73 m², $p < 0.001$).

However, there were no significant differences in plasma sPRR level and the incidence of gestational diabetes mellitus between the two groups ($p = 0.90$ and $p = 0.20$, respectively).

Table 4. Relationship between placental (P)RR levels and selected maternal and neonatal parameters

Placental PRR		
Birth weight	Pearson Correlation	-.311**
	Sig. (2-tailed)	.005
Systolic BP	Pearson Correlation	.259
	Sig. (2-tailed)	.012
Diastolic BP	Pearson Correlation	.111
	Sig. (2-tailed)	.326
Proteinuria	Pearson Correlation	.158
	Sig. (2-tailed)	.162

Table 4 indicates that birth weight ($r = -0.311$, $p = 0.005$) is significantly and negatively correlated with placental (P)RR, suggesting that higher placental (P)RR levels were associated with lower birth weight. Furthermore, statistically significant correlation was found in ($r = 0.259$, $p = 0.012$). However, correlations with diastolic blood pressure and proteinuria were not statistically significant ($r = 0.111$, $p = 0.326$; and $r = 0.158$, $p = 0.162$, respectively).

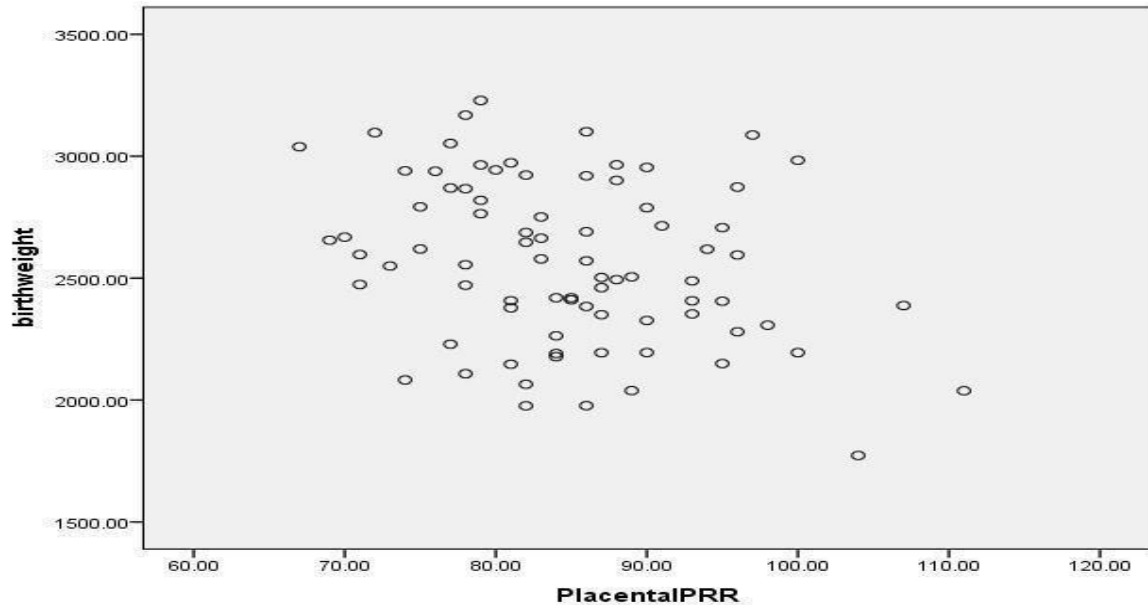


Figure 1. Demonstrates an inverse relationship between placental (P)RR and neonatal birth weight

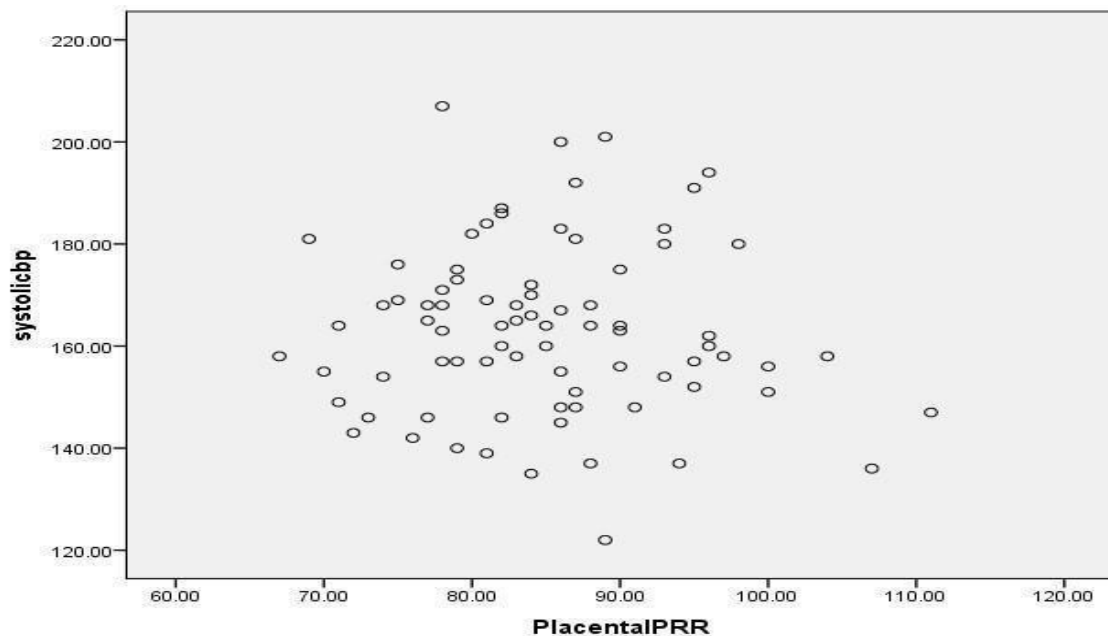
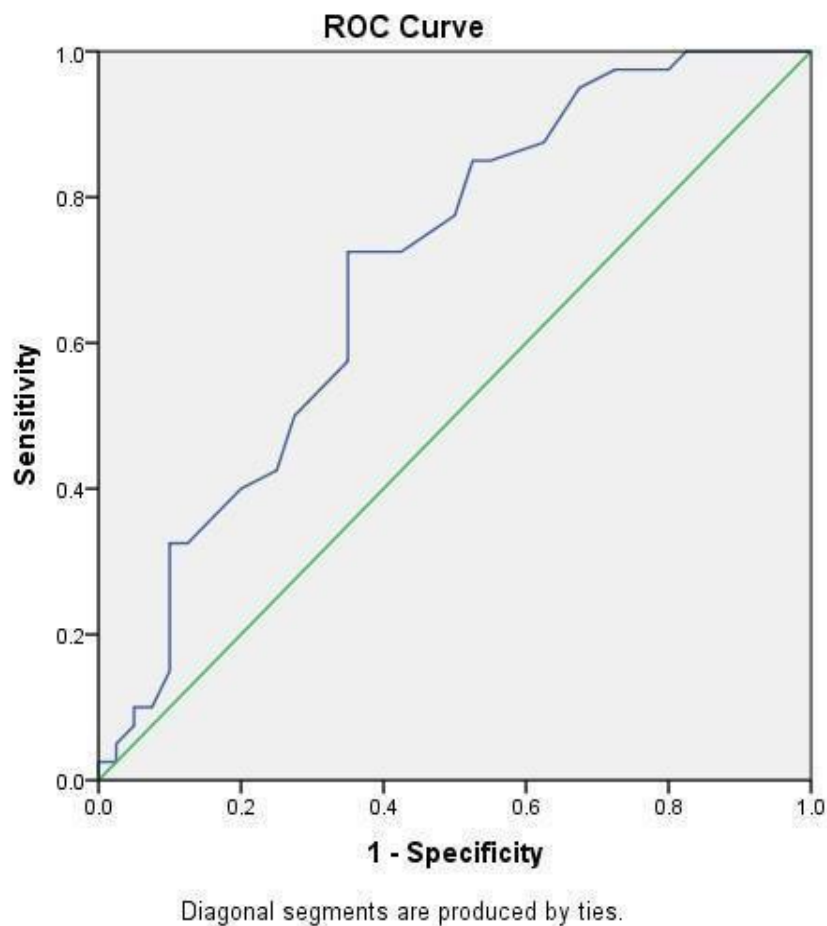


Figure 2. Correlation between placental (P)RR and (BP) systolic blood pressure changes

Table 5. Diagnostic performance of placental (P)RR levels for identifying small for-gestational-age newborns using ROC curve analysis.

Result Variable(s)	Cutoff value	AUC	sensitivity	specificity	Std. Error	Asymptotic Sig.	Asymptotic 95% Confidence Interval	
							Lower Bound	Upper Bound
placental (P)RR	81.5	0.69	78%	50%	0.053	0.002	.584	.814

Placental (P)RR showed a sensitivity of 78% and a specificity of 50% for prediction of small-for-gestational-age newborns at a cutoff value of 81.5ng/ml. AUC=0.69 (95% CI : p=0.002).

**Figure 3.** ROC curve demonstrating the diagnostic performance of placental (P)RR levels in identifying small-for-gestational-age newborns

Discussion

Preeclampsia is a pregnancy-specific condition which is a new onset of hypertension and proteinuria and occurs in 5-8% of all pregnancies. Pre-eclampsia is considered severe if there is severe hypertension (arterial pressure $\geq 160/110$ mmHg on two occasions 6 hours apart); proteinuria (> 5 g/24 hours or $> 3+$ in

two urine samples); or any of the following: cerebral/visual disturbances, abdominal pain, abnormal liver function, oliguria, pulmonary edema, thrombocytopenia, or fetal growth restriction [12]. Pre-eclampsia was found to be a risk factor for birth complication, but the effect was quite varied between studies. Some of this difference may be attributed to the different clinical types of pre-eclampsia and inability to distinguish the different types in most studies [13].

Fetal complication with pre-eclampsia is directly related to the degree of maternal disease, gestational age and is associated with higher rate of SGA, preterm delivery, Intra-uterine Growth Restriction (IUGR), placental abruption and perinatal death [14].

The Pro renin receptore (P)RR was first identified in mesengial cells in 2002, which is a single transmembrane protein (350 amino acid) that binds renin and its inactive proenzyme form prorenin. The (P)RR expression has been demonstrated with the highest amount of mRNA found in brain, heart and placenta while lower amount was expressed in liver, pancreas and kidney and other tissue. The current study revealed that, there was no significant difference among the studied groups with respect to maternal age, Gestational age at delivery and Family history of hypertension while, there was statistically significant difference among the studied groups with respect to BMI, placental weigh and birth weight.

In line with (Watanabe et al.,) studied to determine the relationship between the concentration of soluble (P)RR [s(P)RR] in human cord blood (neonatal blood at birth) and small for gestational age (SGA) birth and they found no significant difference between the studied groups with respect to maternal age, Gestational age at delivery and hypertension. While women with SGA had significantly lower placental weight, and lower birth weight when compared with women AGA, ($p < 0.001$, for both) [15].

Agreed with Ali et al., who compared the weight of the placenta at the time of delivery between women with SGA and women without SGA and found that women with SGA had significantly lower weight of placenta at delivery ($p = 0.003$) and lower weight of the fetus at delivery ($p < 0.001$) [16].

Moreover (Hebbar et al.,) compared between women with small gestational age and women with AGA, reported that there was no statistically significant difference between the studied groups regarding maternal age, Gestational age at delivery. However, there was a significant difference in the birth weight of women with SGA compared to AGA ($p = 0.003$) [17].

Severe and early onset Preeclampsia were found to be more strongly associated with fetal growth restriction and SGA infants, and these had more severe blood pressure abnormalities, and worse fetal growth outcomes [18].

In consistent with (Al-Eraky et al.,) aimed to study the association between placental (Pro) renin receptor with the severity of pre-eclampsia and incidence of small for gestational age birth weight, reported that there was highly statistically significant difference between pre-eclampsia with SGA and preeclampsia with AGA groups regarding systolic blood pressure and diastolic blood pressure, ($P = 0.007$ and 0.01) [19].

The current study indicated that, there was no statistically significant difference between the studied groups regarding plasma sPRR and GDM while, there was highly statistically significant difference between the studied groups regarding placental PRR, proteinuria and eGFR.

This agrees with (Kurlak et al.,) who suggested that the placental RAS responsible for changes in tissue oxygenation and thus affect placental development and function which necessary for fetal growth [48]. In line with (Al-Eraky et al.,) reported that there was no statistically significant difference between pre-eclampsia with SGA and pre-eclampsia with AGA groups regarding plasma sPRR while, there was highly statistically significant difference between the studied groups regarding placental PRR, ($P = 0.002$) [19]. Agreed with (Watanabe et al.,) who found there was no statistically significant difference between

pre-eclampsia with SGA with AGA group with regard to GDM but the difference was statistically significant with regard to sPRR ($p=0.049$) [15].

The current study found that, there was statistically significant negative correlation between placental PRR and birthweight while there was statistically significant positive correlation between placental PRR and Systolic BP. The results were consistent with (Al-Eraky et al.,) which found statistically significant negative correlation between placental PRR and birth weight ($r= -0.368$, $p=0.004$), and statistically significant positive correlation between placental PRR and Systolic BP ($r= 0.627$, $p=0.0001$) [19]. Furthermore, (Nartita et al.,) set out to elucidate the relationship between placental (P)RR expression and plasma soluble (P)RR [s(P)RR] levels and blood pressure rise and renal function during pregnancy found that in preeclamptic women, there was positive correlation of SBP with placental (P)RR levels ($P= 0.004$) [20].

The current study demonstrated that, placental (P)RR had sensitivity of 78% and specificity of 50% with significance for prediction of small for gestational age and at cutoff value 81.5ng/ml.

These results are consistent with those of previous studies, Al-Eraky et al., indicated that placental (P)RR has a significant diagnostic accuracy for detection of small for gestational age newborns with the highest (AUC) 0.736, best cut off value was 81.45ng/ml, specificity was 56.7%, and sensitivity was 80% [19].

Conclusion

Preeclamptic women with SGA newborns had significantly higher placental (P)RR than those with AGA newborns, which also was accompanied by significantly higher levels of proteinuria. Placental (P)RR had a significant negative correlation with neonatal birth weight and a significant positive correlation with maternal systolic blood pressure.

The moderate predictive value of placental (P)RR in preeclamptic pregnancies was demonstrated by ROC analysis (AUC 0.69). For the prediction of SGA, the placental (P)RR had a sensitivity of 78% and a specificity of 50% at a cutoff value of 81.5.

This indicates that elevated placental (P) RR is related to raise the severe of disease also reduced fetal growth in pregnancies complicated by preeclampsia. But larger studies are needed to validate and build upon these findings.

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