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Original Research Article

Gross Features in Pregnancy Induced Hypertensive and Normotensive Placentae

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ABSTRACT

Introduction: The fetus, placenta and mother form a composite triad of dynamic equilibrium and dysfunction of any one of them can affect the other. It is only recently that attention has been directed to the study of the uterine side of the placenta to look for possible defects that might explain otherwise inexplicable pregnancy complications. Preeclampsia and intrauterine fetal growth retardation are two such disorders in which new information has come to light by the study of placental bed.

Aims: To study gross morphological features of placenta in pregnancy induced hypertensive and normotensive parturients.

Material and Methods: The present study of placenta was conducted in the Department of Anatomy and Pathology, Government Medical College, Patiala. The placentae were collected from labour room and gynaecological operation theatre, Rajindra Hospital, Patiala. The study was carried out on seventy five cases of pregnancy induced hypertension and twenty five cases of normotensive pregnancies. An attempt was made to throw light to any changes in morphological and histological features of placentae of pregnancy induced hypertensive cases and compare it with the normotensive placentae.

Results: Gross examination of placentae in PIH group and control group showed significant decrease in placental weight, volume and diameter, a constant fetoplacental ratio and increased incidence of infarction and calcification in PIH placentae as compared to normotensive placentae (control). The site for umbilical cord insertion showed no statistical significance between the two groups, however, the marginal cord insertion was more related to poor fetal outcome and severe PIH.

Conclusion: Gross changes in placentae associated with PIH are due to occlusion or narrowing of the uteroplacental vasculature, placental ischaemic damage and accelerated placental maturation. The perinatal mortality and morbidity associated with this condition is probably related to alterations in the uteroplacental flow.

Key Words: Calcification, Infarction, Placenta, Umbilical cord.

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INTRODUCTION

The growth and development of fetus in utero reflects a balance between fetus, placenta and the mother. The placenta is the organ providing nutrition and respiratory support to sustain fetal life. The fetus depends both on an adequate maternal fuel supply and ability of the maternal vascular tree to deliver an adequate fuel supply to the fetal-placental unit.[1]

At full term the placenta has the shape of a flattened cake (plakuos = placenta = cake). It has a diameter of 15 – 20 cm. and a thickness of rather over 3 cm. The mature placenta has a volume of about 500ml. Its weight is about 500 grams.

During the second half of pregnancy it increases in weight less rapidly than the fetus, so that at term it is presumably becoming insufficient to meet the nutritional requirements which is compensated by thinning of the “placental barrier” separating the maternal and fetal blood streams, thus increasing its efficiency in transmission.[2]

The umbilical cord is inserted on the fetal surface of the placenta at or near its centre. It is usually about 35 to 50 cm long and 1.5 to 2.0 cm in diameter. It contains two umbilical arteries which are the continuation of hypogastric arteries, and one umbilical vein. The main substance of the cord is composed of a mucoid connective tissue called Wharton’s jelly.[3]

Infarcts are common; they occur in about 25% of otherwise normal term placentas. The finding of a small infarct in an otherwise normal placenta is of no clinical significance. Multiple or large (>3 cm) infarcts, central infarcts, or infarcts in the first or second trimester are indicative of significant, underlying maternal vascular disease, especially preeclampsia.[4]

The placenta provides a 'diary' of the pregnancy. The present study has been undertaken to correlate the various morphological and histological placental changes in normotensive and pregnancy induced hypertensive cases.

MATERIAL AND METHODS

The present study was conducted in the Department of Anatomy and Pathology, Government Medical College, Patiala during a period of January 1, 2008 to December 31, 2009. A total of 100 placentae were collected from labour room and from gynecological operation theatre, Rajindra Hospital, Patiala. Ethical approval was taken prior to study from institutional ethics committee, GMC, Patiala, Punjab, India. Cases were broadly divided into two groups:

- Group I (Study/PIH group) : 75 cases of clinically proved PIH
- Group II (Control group): 25 singleton normotensive pregnancies

Cases with period of gestation more than 35 weeks were taken for study. The placentae were grouped depending on the degree of hypertension as described by Cunningham et al (2005).

- Normotensive < 140/90 mmHg
- Mild hypertension ≥ 140/90 - <160/110 mmHg
- Severe hypertension ≥ 160/110 mmHg

The placentae were received in adequate amount of 10% formalin. The placentae were examined grossly and following morphological features were noted.

- Weight of the placenta
- Volume of placenta was recorded by water displacement technique.
- Assuming the placenta to be a perfect circle, the mean diameter was estimated.
- Birth weight of infant was recorded in each case to calculate feto-placental ratio.
- Site of insertion of umbilical cord
- Any gross abnormality in size and shape, gross infarcts, hemorrhages, necrotic areas and calcification was observed.

Besides gross morphological examination of placenta, clinical evaluation of patients and routine hematological and biochemical tests were also considered. Data was compiled in a Performa.

Table 1: Placental weight in Study and Control groups.

Placental weight (gms)	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
≤ 300	12	16.00	0	0
301 – 400	37	49.33	3	12
>400	26	34.67	22	88
Total	75	100	25	100
Statistical Analysis				
Group	**Range**		**Mean ± SD**	
Study	250 – 542		375.95 ± 67.195	
Control	385 – 610		479.8 ± 60.38	

Table 2: Placental volume in study and control groups .

Placental Volume (ml)	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
≤300	11	14.67	0	0
301 – 400	28	37.33	2	8
>400	36	48.00	23	92
Total	75	100	25	100
Statistical Analysis				
Group	**Range**		**Mean ± SD**	
Study	235 – 575		391.2 ± 82.246	
Control	380 – 582		483.96 ± 58.49	

Table 3: Mean Diameter of Placenta in study and control groups .

Mean Diameter (cms)	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
11 – 15	40	53.33	2	8
16 – 20	35	46.67	22	88
> 20	0	0	1	4
Total	75	100	25	100
Statistical Analysis				
Group	**Range**		**Mean ± SD**	
Study	11 – 18		14.55 ± 1.696	
Control	14.5 – 21.4		17.52 ± 1.682	

Fig 1: Measurement of placental weight.

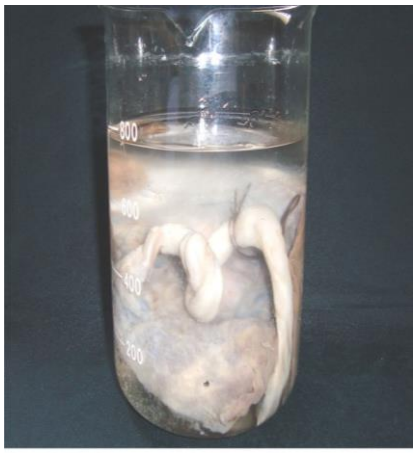

Fig 2: Measurement of placental volume using water displacement method.

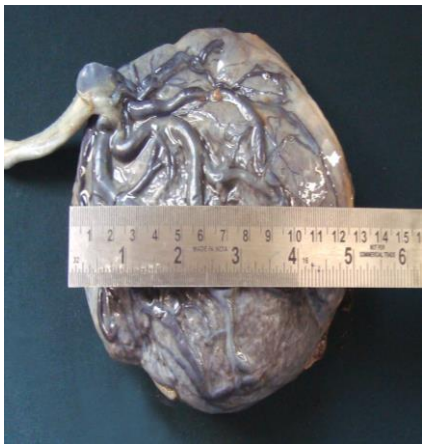

Fig 3: Measurement of placental diameter.

RESULTS

Placental weight (Fig 1)

Table 1 shows that out 75 cases of study group, 12 (16%) cases were of placental weight ≤300 gms, 37 (49.33%) cases had placental weight within the range of 301-400 gms and 26 (34.67%) cases had >400 gms.

In control group, majority of the placentae 22 (88%) weighed >400 gms and only 3 (12%) cases had placental weight within the range 301-400 gms.

The statistical difference between placental weight in study and control group was significant ($p < 0.05$).

Placental Volume (Fig 2)

Table 2 shows that in study group 11 (14.67%) cases had placental volume ≤300 ml, 28 (37.33%) cases had volume within the range of 301-400 ml whereas in control group none of the placentae had volume ≤300 ml and only 2 (8%) cases had placental volume within the range of 301-400 ml. Rest of the cases i.e. 23 (92%) of control group were of placental volume >400 ml as compared to 36 (48%) cases in study group. The statistical difference was significant ($p < 0.05$).

Mean Diameter of Placenta (Fig 3)

Table 3 shows that in study group 40 (53.33%) cases had mean placental diameter in the range of 11-15 cms and 35 (46.67%) cases between 16-20 cms. Mean placental diameter was not more than 20 cms in any of the cases of the study group. Mean placental diameter in maximum number of the control group cases i.e. 20(88%), was found to be in the range between 16-20 cms. In 2(8%) cases of control group, mean placental diameter was between 11-15 cms and it was more than 20cms in onlsy 1 (4%) case of control group. The statistical difference between two groups for mean placental diameter was highly significant ($p < 0.001$).

Table 4: Umblical cord insertion in study and control groups.

Umblical Cord Insertion	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
Central	25	33.33	12	48
Eccentric/medial	23	30.67	7	28
Lateral	11	14.67	4	16
Marginal	16	21.33	2	8
Total	75	100	25	100
Statistical Analysis				
χ^2	P value		Significance	
3.01	>0.05		NS	

Table 5: Infarction of placenta in study and control groups.

Infarction	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
Absent	24	32.00	19	76
Mild (< 10%)	35	46.67	6	24
Extensive (>10%)	16	21.33	0	0
Total	75	100	25	100
Statistical Analysis				
χ^2	P value		Significance	
16.1	<0.001		S	

Table 6: Calcification of placenta in study and control groups

Calcification		Group I (Study)		Group II (Control)	
		No.	%age	No.	%age
Absent		28	37.33	15	60
Present	Mild	27	36	10	40
	Extensive	20	26.67	0	0
Total		75	100	25	100

Statistical Analysis

χ^2	P value	Significance
8.99	<0.05	S

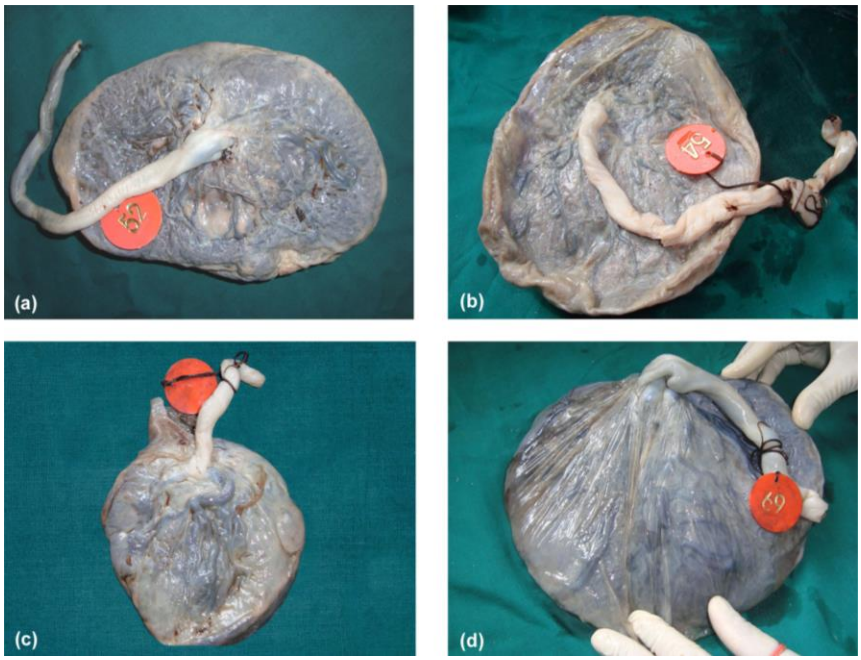

Fig 4: Gross photograph of placenta showing: 4a) Central insertion of umbilical cord. 4b) Eccentric insertion. 4c)) Lateral insertion. 4d) Marginal insertion.

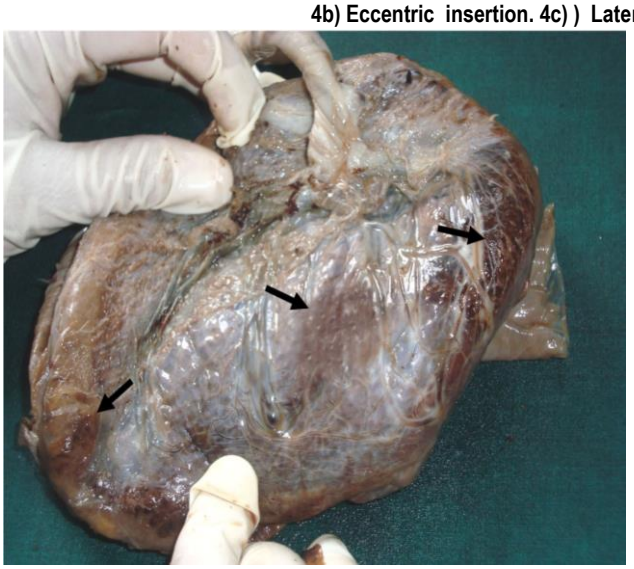

Fig 5: Gross photograph of placenta showing areas of red infarction

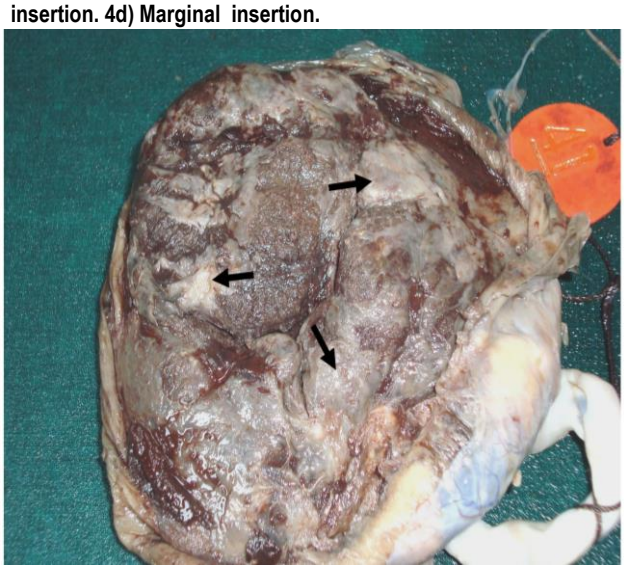

Fig 6: Gross photograph of placenta showing areas of calcification.

Umblical Cord Insertion

Central and eccentric insertion of umblical cord is considered normal (Fig 4a, 4b, 4c and 4d). Table 4 shows that out of 75 cases of study group 25 (33.33%) cases had central and 23 (30.67%) cases had eccentric insertion of umblical cord. Marginal insertion of umblical cord was found in 16 (21.33%) cases of study group as compared to only 2 (8%) cases of control group. In control group central insertion of umblical cord was found in 12 (48%) cases, eccentric in 7 (28%) cases and lateral in 4 (16%) cases. The statistical difference between two groups was not significant.

Infarction

Table 5 shows that in the present study, infarction was absent in 24 (32%) cases and mild in 35 (46.67%) cases of the study group. 16 (21.33%) cases of the study group showed extensive infarction

(Fig 5). No significant infarction was noted in the control group. 6(24%) cases of the control group had only mild infarction. The statistical difference between two groups for infarction was significant.

Calcification

The presence of calcification is considered as a normal physiological phenomenon (Fig 6). Table 6 shows that out of 75 cases of the study group, 47 (62.67%) cases showed presence of calcification on gross examination and calcification was extensive in 20 (26.67%) cases. In the control group, out of 25 cases only 10 (40%) cases had mild calcification in their placentae. None of the cases of the control group had extensive calcification. In majority of the cases of control group, calcification was absent. The statistical difference between two groups for calcification was significant.

DISCUSSION

Pregnancy complicated by hypertension not only affects maternal health but also jeopardizing fetal normalcy. In this study, the emphasis has primarily been made on finding gross changes in placenta in PIH and normal cases and its relation with fetal outcome.

Placental weight

In the present study, mean placental weight for study group (PIH) was 375.95 gm and 16% placentae were of weight ≤ 300 gm. In the control group, mean placental weight was 479.28 gm and no placenta had weight ≤ 300 gm. Majority of the placentae (88%) were of weight > 400 gm in control group [Table 7(a)].

Bajaj et al (1970) found 20% placentae having weight ≤300 gm in PIH group.[5] Udainia and Jain (2001),[6] Majumdar et al (2005)[7] and Artico et al (2009) also found significant reduction of PIH placental weight as compared to control.[8]

Placental weight is important predictor of birth weight. In the present study, the low weight placentae were associated with low weight babies and also with IUDs. However, placental weight may vary due to areas of calcification, retroplacental, haematoma or how the postpartum placenta gets prepared. As PIH is associated with decrease utero placental flow and placental villous lesions. This will hamper the growth of the developing placenta and fetus.

Placental Volume

In the study group, mean placental volume 391.20 ml. 11 (14.67%) cases of study group had volume ≤300 ml. Majority of the cases (88%) in the control group had volume more than 400 ml. Placental volume ≤300 ml was not reported in control group and the mean placental volume for control group was 464.96 ml [Table 7(b)].

Aherne and Dunhill (1966),[9] Boyd and Scott (1985),[10] Majumdar et al (2005)[7] and Artico et al (2009)[8] observed lower mean placental volume in PIH than control with significant statistical value.

Present study reveals that placental volume is significantly lower in pregnancy induced hypertension as compared to normotensive and these findings collaborate with the studies of other workers.

Lower volume was due to placental insufficiency and decreased placental growth in PIH.

Mean diameter of placenta

In the present study, the mean diameter of placenta in study and control group was 14.55 cms and 17.52 cms respectively. (Table 3).

Table 7 (a): Gross changes in placentae of pregnancy induced hypertensive subjects; a) Placental Weight.

Author (Year)	Group	≤300 gm	Mean	SD	Significance
Bajaj et al (1979)	PIH	20%	-	-	S
	Control	0%	-	-	
Udainia & Jain (2001)	PIH	-	405	114.11	S
	Control	-	495	101.64	
Majumdar et al (2005)	PIH	-	399.10	90.31	S
	Control	-	485.85	47.31	
Artico et al (2009)	PIH	-	461.1	-	S
	Control	-	572.1	-	
Present study (2009)	PIH	16%	375.95	67.195	S
	Control	0%	479.28	60.38	

Table 7 (b): Gross changes in placentae of pregnancy induced hypertensive subjects; b) Placental Volume.

Aubthor (Year)	Group	Mean	SD	P value
Aherne and Dunhill (1966)	PIH	363	-	S
	Control	488	-	
Boyd and Scott (1985)	PIH	357	-	S
	Control	459	-	
Majumdar et al (2005)	PIH	375.99	173.61	S
	Control	612.98	213.26	
Artico et al (2009)	PIH	437.4	-	S
	Control	542.0	-	
Present Study (2009)	PIH	391.2	82.246	S
	Control	483.96	58.49	

Rath et al (2000)[11] and Majumdar et al (2005)[7] observed that mean surface area of the placentae of hypertensive group was shorter than that of normotensive mothers.

In preeclampsia, placenta is smaller on an average than placenta from uncomplicated pregnancies (Gersell and Kraus, 2004).[12] Rahman et al (2007)[13] reported significantly smaller values of placental diameter in eclampsia group ($p < 0.001$).

In present study, the statistical difference for placental diameter between PIH (study group) and control group was highly significant ($p < 0.001$).

Umbilical Cord Insertion

The umbilical cord typically insert into the placenta near its center. About 90% of cord insertions are central or eccentric and about 7% of umbilical cord insertions occur at placental margin (Joseph, 1998).[14] In the present study, majority of the placentae had eccentric or central insertion of umbilical cord. Marginal cord insertion in PIH (study group) was 21.33% whereas 8% in normotensive placentae. Statistical difference for all types of umbilical cord insertion was not significant between the study and control groups [Table7(c)].

Woods and Malan (1978) found no correlation between the birth weight in sight of cord insertion in normal infants.[15] Rath et al (2002) found no significant differences for umbilical cord insertion between study and control groups and observed that marginal cord insertion results in low birth weight both in normotensive and hypertensive cases, most commonly in severe hypertensive subgroup.[11]

Some of the authors have related the site of insertion of umbilical cord with fetal outcome and PIH and some do not. In the present study, marginal cord insertion was related to poor fetal outcome and with severity of PIH, though the result was insignificant.

The association between the site of umbilical cord insertion and low birth weight can be explained by altered distribution of fetal blood in the placenta as a result of different modes of arrangement of intracotyledonary vessels of placentae. This study should be pursued in large number of cases; the mother should undergo regular antenatal checkup and ultrasonography (USG). If USG reveals marginal cord insertion then counseling can be done to get it confirmed by colour doppler imaging so that fetal outcome can be improved by other measures.

Table 7 (c): Gross changes in placentae of pregnancy induced hypertensive subjects; c) Umblical Cord Insertion.

Author (Year)	Group	Central	Medial	Lateral	Marginal
Rath et al (2000)	PIH	20.83%	20.83%	26.38%	31.94%
	Control	24%	22%	27%	27%
Present study (2009)	PIH	33.33%	30.67%	14.67%	21.33%
	Control	48%	28%	16%	8%

Table 7 (d): Gross changes in placentae of pregnancy induced hypertensive subjects; d) Infarction

Authors (Year)	Group	% of cases	Significance
Boyd and Scott (1985)	PIH	64.29	S
	Control	9.68	
Udainia et al (2001)	PIH	96	S
	Control	29	
Majumdar et al (2005)	PIH	-	S
	Control	-	
Artico et al (2009)	PIH	51.2	S
	Control	1.15	
Present Study (2009)	PIH	68	S
	Control	24	

Table 7 (e): Gross changes in placentae of pregnancy induced hypertensive subjects; e) Calcification.

Authors (Year)	Group	% of cases	Significance
Wentworth (1964)	PIH	41.56	-
	Control	-	
Avasthi et al (1991)	PIH	85	S
	Control	20	
Majumdar et al (2005)	PIH	-	S
	Control	-	
Present Study (2009)	PIH	62.67	S
	Control	40	

Infarction

True infarcts were most frequent in placentae from cases of preeclampsia, and when extensive they were associated with low birth weights and stillbirths (Wigglesworth, 1964).[16] In the present study, infarction was found in 60% of PIH and 24% cases of control group [Table 7(d)].

Significant infarction in pregnancy induced hypertension as compared to uncomplicated pregnancies was also observed by

Boyd and Scott (1985),[10] Udainia et al (2001),[6] Majumdar et al (2005)[7] and Artico et al (2009).[8]

In present study, extensive infarction was also found to be more in cases having severe PIH. No control group placenta was associated with extensive infarction.

Infarcts are common; they occur in about 25% of otherwise normal term placentae. The finding of a small infarct in an otherwise normal placenta is of no clinical significance. Multiple or large (>3 cm) infarcts, central infarcts, or infarcts in the first or second trimester are indicative of significant, underlying maternal vascular disease, especially preeclampsia as stated by Gersell and Kraus (2002).[4]

In PIH, there occurs complete occlusion of blood supply to a group of villi due to intense vasospasm, thus leading to ischaemic necrosis or infarcts.

Calcification

In the present study, 62.67% of cases of study group (PIH) placentae had shown calcification as compared to 40% cases of control group. However, in control group all the 40% cases had mild calcification and none of the cases were associated with extensive calcification. In study group, calcification was extensive in 26.67% cases. The statistical difference between two groups was significant [Table 7 (e)].

Wentworth (1964) found calcification in 41.56% cases of PIH[17]. Avasthi et al (1991)[18] and Majumdar et al (2005)[7] also found significant calcification in PIH placentae as compared to control. Increased incidence of calcification in PIH placentae indicates accelerated maturation.

SUMMARY AND CONCLUSION

Gross examination of placentae in PIH group and control group showed significant decrease in placental weight, volume and diameter, a constant fetoplacental ratio and increased incidence of infarction and calcification in PIH placentae as compared to normotensive placentae (control). The site for umbilical cord insertion showed no statistical significance between the two groups, however, the marginal cord insertion was more related to poor fetal outcome and severe PIH.

Gross changes in placentae associated with PIH are due to occlusion or narrowing of the uteroplacental vasculature, placental ischaemic damage and accelerated placental maturation. The perinatal mortality and morbidity associated with this condition is probably related to alterations in the uteroplacental flow.

The fetus, placenta and mother form a composite triad of dynamic equilibrium, and dysfunction of any one of them can affect the other. This information provides new insight into link between fetus, placenta and PIH thus can be a useful adjunct in planning and management of future pregnancies.

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