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Original Research Article

Correlation between changes in placental morphological features with abnormal Doppler flow in pregnancy induced hypertension

Vijay N. Gadhavi, Mansi K. Gadhavi*, Manish Pandya

Department of Obstetrics and Gynecology, C. U. Shah Medical College, Surendranagar, Gujarat, India

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***Correspondence:**
Dr. Mansi K. Gadhavi,
E-mail: drmansigadhavi@gmail.com

ABSTRACT

Background: Placenta is one of the most challenging organs; it is an instrument of transfer of essential elements, i.e. nutrients and oxygen from mother to fetus and waste product of metabolism in reverse manner.
Methods: Cases of PIH between 20-36 weeks of gestation will be studied over a peri-od of 2 years having B.P ≥140/90mm Hg and protienuria ≥1+ in this prospective analytical study having color Doppler scanner with PI, RI of umbilical, uterine artery and middle cerebral artery PI along with placental morphological changes are observed.
Results: In present study where, placental weight was <300g also has LBW babies born were higher 51(100%). In placental gross examination 58% infarction, 42% calcification and 48% retroplacental were found. among 58 samples with infarction 76%, 42 placentas had calcification 48% and among 49 samples retroplacental clots 61% were belonged to uterine artery PI >1 group. While 78% placental infarction, 57% calcification and 69% retroplacental clots be-longed to uterine artery RI > 0.6 group. Infarction were 77.50%, calcification were same as infarction 77.50% while retroplacental clots 80% in group having MCA PI <1.3 that were higher than group of cases having MCA PI >1.3.
Conclusions: In recent years placenta has drawn attention as valuable indicator for maternal and fetal diseases in preeclampsia. Decreased circulation in placenta reflects on its morphological features and these changes causes alterations in Doppler flow velocities of uterine, umbilical and middle cerebral vessels pregnancy induced hyper-tension.

Keywords: Calcification, Infarction, Placenta, Retroplacental clots

INTRODUCTION

Placenta is a leading cause of maternal and perinatal mortality and an important factor in fetal growth retardation as it is commonly associated with placental in-sufficiency.[1,2] Fox and Langley described the placenta as the mirror of the perinatal period. In normal pregnancies the wall of the spiral arteries are invaded by trophoblastic cells and transformed into large, tortuous channels that carry a large amount of blood to the intervillous space and are resistant to the effects of vasomotor agents.[3] Trophoblastic invasion begins from 16-20 weeks of gestation causing destruction of the muscularis layer of spiral arteries land is completed by 24 weeks' time. These physiologic changes are restricted in patients with preeclampsia. The main feature of abnormal placentation is inadequate trophoblastic invasion of the maternal spiral arteries. This results in persistence of muscular and elastic tissues of the media of spiral arteries. As a result, the vessels fail to dilate and remain responsive to vasomotor influences that lead to high resistance low flow choriodecidual circulation.[4]

With progress of pregnancy, the metabolic demand for the feto-placental unit increases but the spiral arteries are unable to dilate to accommodate the required increase in

blood flow, resulting in placental dysfunction that manifest clinically as preeclampsia Although the pathophysiology of preeclampsia is poorly understood, it is characterized by abnormal trophoblast invasion of uterine blood vessels, immunological intolerance between feto-placental and maternal tissues.[5]

Careful examination of placenta can give information that may be important in the immediate and later management of mother and infant.

Pathophysiology of abnormal FVWs in placental insufficiency.[6] In the presence of placental insufficiency, there is greater placental resistance, which is reflected in a decreased end-diastolic component of the UA FVWs.[7-12] An ab-normal UA FVW has a S/D ratio above the normal range.

As their placental in-sufficiency worsens, the end-diastolic velocity decreases then become absent and finally it is reversed Some fetuses have decreased end-diastolic velocity that re-mains constant with advancing gestation and never become absent or reversed, which may be due to a milder form of placental insufficiency Pitfalls can be caused due to e.g. fetal breathing.

Abnormal UA Doppler studies, but not nor-mal results were found to be associated with lower arterial and venous pH values, an increased likelihood of intrapartum fetal distress, more admissions to the neonatal intensive care unit (NICU), and a higher incidence of respiratory distress in IUGR fetuses.

Placentas are examined macroscopically and microscopically for a variety of reasons - diagnostic, either for the mother or for the neonate, prognostic prediction of future pregnancies, investigative and for legal purpose. The frequency of laboratory placental examination varies from institution to institution, from occasional to universal (< 1% to 100%)) from occasional (<1% of deliveries) to universal 9100% of deliveries.[13] Naeye opines that all placentas should be examined grossly, and an immediate microscopic examination indicated for those cases with adverse pregnancy outcome are when placenta is grossly abnormal.[14]

METHODS

The present study is conducted in the department of obstetrics and gynecology, C.U Shah medical college and Hospital, Surendranagar Saurashtra university, Gujarat. The study design is prospective analytical study.

This study was conducted during the period from May 2011 to April 2013.A total 100 Cases of PIH between 20-36 weeks of gestation will be studied over a period of 2 years having B.P ≥140/90 mm Hg and protienuria ≥1+ in this prospective analytical study having col-or Doppler scanner with PI, RI of umbilical ,uterine artery and middle cerebral artery PI along with placental morphological changes are observed.

Inclusion criteria

- All Pregnancy beyond 20 weeks of gestation having Systolic BP >140mm Hg, Diastolic BP >90mm Hg on two occasions four hours apart in previously normo-tensive woman with Proteinuria ≥1+ on urine dip stick test.

Exclusion criteria

- Molar pregnancies
- Renal disease
- Chronic Hypertension
- Hematological Diseases
- Other causes of seizures
- Heart Disease including IHD.

Prior to the commencement of the study ethical clearance was obtained from the Institutional Ethical committee, C.U Shah Medical college, Surendranagar, Gujarat. Pregnant women between 20-36 weeks of gestation attending ante-natal clinic were screened for eligibility by detailed history, antenatal examination and investigations by trained residents in department of Obstetrics and Gynecology.

Women fulfilling selection criteria are explained about the purpose of the study and the need for randomization. A written informed con-sent was obtained from all participants before the enrolment than demo-graphic data, obstetric history and current pregnancy details were obtained, the data was recorded on predesigned and pretested proforma.

RESULTS

There was association with low birth weight<2.5kg in group where placental weight was <300g, no of LBW babies were higher 51(100%) while birth weight of 10(83%) patients were more than 2.5kg in group of placenta weighing >501g (Table 1).

Table 1: Comparison of weight of placenta and birth weight.

Weight of placenta (g)	No	Birth weight <2.5kg (%)	Birth weight >2.5kg (%)
<300	51	51 (100)	0
301-500	37	29 (78)	08 (22)
>501	12	02 (17)	10 (83)
Total	100	82	18

In placental gross examination, total 58(58%) cases of placental infarction seen and 42 (42%) calcification while retroplacental clots were seen in 48 (48%) cases of placenta (Table 2).

Table 2: Different placental gross changes.

Gross findings	Number	Percentage
Infarction	58	58
Calcification	42	42
Retroplacental clots	48	48

In present study highest morphological changes in placenta which were 48% in-farction,43% calcification and 49% retroplacental clots seen with reduced diastolic flow pattern in umbilical artery (Table 3).

Table 3: Umbilical artery diastolic pattern and placental gross features.

Diastolic flow	Infarction (%)	Calcification (%)	Retroplacental clots (%)
Normal	13 (22)	18 (43)	12 (25)
Absent	7 (12)	3 (7)	4 (8)
Reversed	10 (17)	3 (7)	9 (18)
Reduced	28 (48)	18 (43)	24 (49)

The data shows that out of all placentas studied, among 58 samples with infarction 44 (76%), same as among 42 placentas, calcification 20 (48%) and among 49 samples with retroplacental clots 30 (61%) were belonged to uterine artery PI >1 group (Table 4).

Table 4: Uterine artery PI and associate placental gross features.

Uterine artery PI	Infarction	Calcification	Retroplacental clot
>1	44 (76)	20 (48)	30 (61)
<1	14 (24)	22 (52)	19 (39)
Total	58	42	49

Table 5 data suggests that 45 (78%) samples of placenta with infarction out of 58 placental samples, 24(57%) out of 42 samples having calcification and 34(69%) no of samples having retroplacental clots among 49 samples of placenta belonged to uterine artery RI > 0.6 group.

Table 5: Uterine artery RI and gross placental changes.

Uterine artery RI	Infarction	Calcification	Retroplacental clots
>0.6	45 (78)	24 (57)	34 (69)
<0.6	13 (22)	18 (43)	15 (31)
Total	58	42	49

In present study gross histological examination of placenta, no. of infarction were 31(77.50%), no of calcification were same as infarction i.e. 31 (77.50%) while no. of retroplacental clots in placenta were 32 (80%) in group having MCA PI <1.3 that were higher than group of cases having MCA PI >1.3 (Table 6).

Table 6: Middle cerebral artery PI and gross placental changes.

MCA PI	Infarction	Calcification	Retroplacental clots
<1.3	31/40 (78%)	31/40 (78%)	32/40 (80%)
>1.3	29/60 (48%)	13/60 (22%)	21/60 (35%)
Total	100	100	100

DISCUSSION

In present study incidence of birth weight less than 2.5kg seen in placental weight less than 500g comparable to Nayereh Ghomian et al high numbers of LBW babies in group of placenta weighing 500g. Normally a placenta weighs from 400 to 800g. This study observed the Reduction of placental weight in the hypertensive disorders.[14]

Udain and Jain et al found linear correlation exits between weight of new-born and weight of placenta in PIH cases.[15] Bandana Das et al, Nobi's, Das et al, Sharma et al, Dutta et al and also reported the same findings.[16-19] The incidence of abnormal Doppler waveforms in umbilical diastolic flow was when the placenta weighed 300gm this was statistically significant.

Fetal morbidity was high in terms of low Apgar and NICU admission and neo-natal outcome is directly related to the low placental weight. Similar result was noted by Harsh Mohan et al in their study.[20]

In present study placental morphological changes shows that percentage of infarction, calcification, retroplacental clots 58%, 42% and 48% respectively which is comparable to Navbir P et al where Percentage infarction, calcification, retroplacental clots are 67%,73%and 30% respectively.[21]

Wide variations in the incidence of placental infarcts have been reported by Fox and Langley ranging from 34% in women with mild pre-eclampsia to 60% in women with the severe form of the disease. The incidence of retroplacental hematoma in toxaemic subjects as ranging from 12-15%, but a correlation with the severity of disease has not been well established. Salvatore et al reported an incidence of retro placental haematoma in mild and severe pre-eclampsia as 3.1% and 25.8% respectively.[22,23]

On examine gross pathological finding of placenta suggested that 58% incidence of infarction in present study and 67% in Navbir P et al study, while 30% in study conducted by Salgado SS et al which is on lesser side than both of above study.[21,24]

In present study infarction, calcification, retroplacental clots were 12%, 7% and 8% with absent umbilical diastolic flow and 17%, 7%, 18% with reversed umbilical diastolic flow along with 48%, 43%and 49 % in reduced flow pattern suggesting effect of hypoxia on placenta and ultimately on fetus. Fox et al postulated that extensive placental infarction is associated with a high incidence of foetal hypoxia, intra-uterine growth retardation and death.[22] Olga genbacev et al observed similar effect of hypoxia causing placental damage and risk factor for PIH and preeclampsia.[25]

Abnormal uterine artery PI >1 group data shows that out of 58 cases of placentas , 76% infarction , 48% calcification , 61% retroplacental clots were present in patients belonging to uterine artery PI >1 group while uterine RI > 0.6 group had 78% placenta , 57 % calcification and 69% retroplacental clots .In abnormal MCA PI calcification 57 %, syntial knots 65% were found which were on higher range than normal MCA PI group where 35 % calcification and 60 % of syntial knots were seen.

CONCLUSION

Many of disorders of pregnancy which are associated with high perinatal morbidity and mortality are accompanied by gross pathological changes in placenta. these changes in placenta leads to abnormal Doppler flow in multiple vessels like uterine, umbilical and middle cerebral vessels, ultimately ends into impending perfusion and undesirable perinatal and maternal mortality.

Placentas from pre eclamptic women tend, on average to be smaller than those from uncomplicated pregnancies, but the decrease is only slight, and a proportion of such placentas are unusually large.

The feto-placental ratio is generally decreased. The incidence of placental infarction ranges from about 33% in cases of mild preeclampsia to approximately 60% in patients with severe form of the disease. Extensive infarction (involving more than 10% of the parenchyma) is found in about 30% of placentas from cases of severe pre-eclampsia, but not a feature of the milder forms of this disease. Placental calcification often regarded as evidence of either placental senescence or degeneration is of no pathological or clinical importance and not associated with any fetal complication. Retroplacental hemotomas are found unduly frequently, occurring in about 12-15% all cases. The retro placental haemorrhage is due to rupture of maternal decadal arteriole, the wall of which is weakened because of the changes that occur in pre-eclampsia. They are found in approximately 5% of all placentas. Large lesions, in which 40% or more of the villous population is acutely deprived of the blood supply, are associated with high incidence of fetal hypoxia, death.

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