PLACENTAL MORPHOLOGY IN HYPERTENSIVE DISORDERS OF PREGNANCY & ITS CO-RELATION WITH FOETAL OUTCOME

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SUMMARY

This study is an analysis of morphological changes of placenta in hypertensive disorders of pregnancy. In total 100 placentae are studied out of which 80 placentae are collected from different categories of hypertensive disorders and 20 placentae from normal cases - as control.

Morphologically placenta of hypertensive disorders of pregnancy differs from that of normal cases. The placenta of hypertensive disorders are lighter in weight, lesser in diameter & thickness, with high incidence of abnormal shape and cord insertion and the foeto-placental ratio is diminished. Infarction, retroplacental - haematoma, subchorionic fibrin are also higher in incidence in hypertensive placenta.

Placental changes are directly proportional to the duration of the disease process and its severity. Morphologically placentae of P.I.H. are more severly affected than the placentae of Essential hypertension.

Foetal outcome is adversely influenced by the pathological changes observed in placenta.

In the recent years placenta has drawn attention as valuable indicator of maternal & foetal deseases. Many of the disorders of pregnaney which are associated with high perinatal morbidity & mortality are accompanied by gross pathological changes in placenta. Hypertensive disorders of pregnancy is also well reflected on placenta with remarkable changes both macroscopic and microscopic. This is an analysis of macroscopic placental changes in hypertensive disorders of pregnancy.

MATERIALS & METHODS

The patients for this study were selected at random from O.P.D. and from Ward admitted as an emergency, during the period from May, 1990 to May, 1991. 100 placentae both from normal and hypertensive patients were studied out of 1380 deliveries. Cases were sistributed as follows :

A. Normal : 20 cases as control.
B. Mild pre-eclampsia : 20 cases.
C. Severe pre-eclampsia : 20 cases.
D. Eclampsia : 20 cases.
E. Essential hypertension : 6 cases.
F. Pre-eclampsia superimposed on Essential hypertension : 14 cases.

Just after delivery all the placentae were collected in clean tray. The membranes and cord at their attachment, near to placenta were cut off. Then the placenta was expressed gently so as to remove its blood content and then washed thoroughly under tap water and following examinations were made.

* Size
* Shape
* Diameter
* Thickness
* Weight
* Mode of attachment of cord and membranes
* Vascular pattern
* Number of cotyledons
* Presence of | Subchorionic fibrin
  | Infarction
  | Calcification
  | Retroplacental haematoma
* Co-relation of the placental changes with duration and severity of hypertension
* C0-relation of placental findings with foetal outcome

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OBSERVATIONS & DISCUSSION

Weight of the placenta : Normally a placenta weighs from 450 - 500 gms. This study observed the reduction of placental weight in hypertensive disorders. Out of 80 hypertensive cases, weight of 25 placentae were below 300 grms. at term, whereas none of the placenta was below 300 gms. in normotensive control group. Bhatia & Sharma (1981); Dutta (1989) & Nabis & Das (1991) also reported the same findings. (Table I)

Placental weight was more reduced in proteinuric patients and also where duration of hypertensive disorder was prolonged. (Table II & V). Although still birth was not significantly increased in this study, but foetal asphuxia, low birth weight were frequent when placenta was less than 300 gms. (Table IV). There was no significant relationship between placental weight with age and parity of the patient.

Placental-foetal ratio : At term the placental-foetal ratio lies between 1 : 6 to 1 : 8 (Morrison, 1963). Fox (1964) reported hypertrophy of placental mass in rewponse to shronic hypoxia in hypertensive patients. This hypertrophy along with low birth weight contributes to low placental-foetal ratio. In this study ratio was found to be decreased with increasing toxaemia. (Table III).

Diameter and thickness : Diameter and thickness both were reduced in hypertensive disorders: there was more reduction in thickness than in diameter. (Table I)

TABLE I
MACROSCOPIC FINDINGS OF PLACENTA IN DIFFERENT GROUPS.

Macroscopic Findings	Normal (Total case-20)	Mild pre -eclamp- -sia. (Total case=20)	Severe Pre-ec- lampsia. (Total case=20)	Eclamp sia. (Total case=20)	Essential hyperten- -sion. (Total case=6)	Pre-eclamp -sia super -imposed on essential hypertension (Total Case=14)
WEIGHT						
i.<300 gms.	-	1 (5%)	8 (40%)	11 (55%)	2(33.33%)	3(21.43%)
ii. 301-400gms.	3(15%)	5 (25%)	7 (35%)	6(30%)	3(50%)	6(42.86%)
iii. 401-500gms.	13(65%)	12(60%)	4(20%)	3(15%)	1(16.67%)	4(28.57%)
iv. Above 500 gms	4(20%)	2(10%)	1(5%)	-	-	1(7.14%)
DIAMETER						
i. 15-17 cms.	2(10%)	3(15%)	6(30%)	10(50%)	2(33.33%)	4(28.57%)
ii.18-20 cms.	18(90%)	16(80%	)13(65%)	10(50%)	4(66.67%)	10(71.43%)
iii. Above20cm.	-	1 (5%)	1(5%)	-	-	-
THICKNESS						
I. Below 1.5cms.	-	-	4(20%)	4(20%)	1(16.67%)	-
ii. 1.5-1.9 cms.	6(30%)	5(25%)	9(45%)	10(50%)	3(50 %)	6(42.86%)
iii. 2 -2.5cms.	14(70%)	15(75%)	7(35%)	6(30%)	2(33.33%)	8(57.14%)
SHAPE						
I. Normal.	17(85%)	15(75%)	12(60%)	12(60%)	4(66.67%)	9(64.29%)
ii. Abnormal.	3(15%)	5(25%)	8(40%)	8(40%0)	2(33.33%)	5(35.71%)
INSERTION OF THE CORD						
i. Central.	16(80%)	12(60%)	9(45%)	10(50%)	4(66.67%)	10(71.43%)
ii. Ecentric.	4(20%)	6(30%)	10(50%)	8(40%)	2(33.33%)	4(28.57%)
iii. Marginal.	-	2(10%)	-	2(10%)	-	-
iv. Vellamentous.	-	-	1(5%)	-	-	-
VASCULAR PATTERN						
i. Disperse type.	19(95%)	18(90%)	19(95%)	20(100%)	6(100%)	14(100%)
ii. Magistral type.	1 (5%)	2(10%)	1(5%)	-	-	-
SUBCHORIONIC FIBRIN						
i. Absent.	16(80%)	13(65%)	9(45%)	12(60%)	4(66.67%)	8(57.14%)
ii. Present.	4(20%)	7(35%)	11(55%)	8(40%)	2(33.33%)	6(42.86%)

RETROPLACENTAL HAEMATOMA						
i. Absent.	20(100%)	18(90%)	17(85%)	19(95%)	6(100%)	13(92.86%)
ii. Present. (Above 5% of Parenchyma)	-	2(10%)	3(15%)	1(5%)	-	1(7.14%)
INFARCTION						
i. Absent.	15(75%)	10(50%)	6(30%)	8(40%)	3(50%)	6(42.86%)
ii. 0-5% of parenchyma	5(25%)	2(10%)	1(5%)	1(5%)	1(16.67%)	214.29%)
iii. Above 5% of parenchyma	-	8(40%)	13(65%)	11(55%)	2(33.33%)	6(42.86%)
CALCIFICATION						
i. Absent.	14(70%)	13(65%)	11(55%)	11(55%)	4(66.67%)	8(57.14%)
ii. Present.	6(30%)	7(35%)	9(45%)	9(45%)	2(33.33%)	6(42.86%)

TABLE II
SHOWING CO-RELATION OF THE PLACENTAL WEIGHT WITH DURATION OF THE DISEASE IN TOXAEMIA OF PREGNANCY CASES

Duration of the desease	Number of Toxaemia Cases Total=74	Weight of the placenta 300 grams	300-500 grams	500 grams
Disease starts before 36 weeks of gestation.	55	36(65.45%)	19(34.55%)	-
Disease starts after 36 weeks of gestation	19	8(42.1%)	10(52.63%)	1(5.26%)

Shape & cord insertion : The shape of the placentae were normal in 85% normotensive and 65% hypertensive cases. So, abnormal shape of the placenta was more in hypertensive disorder of pregnancy. The insertion of the cord was central in 80% cases of control group and it was 56.25% in hypertensive group. This study revealed interestingly a higher incidence of ecentric or marginal insertion of the cord ("Battledore Placenta") in hypertensive group (42.2%), which could not be logically co-related with the disease process.

Vascular pattern and cotyledons : Disperse type of vascular pattern was found to be more than magistral type in both groups. Table I). However, the shape of the placenta, its vascular pattern and nature

TABLE III
SHOWING FOETO - PLACENTAL WEIGHT RATIO

GROUPS OF CASES.	NO. OF CASES	MEAN BIRTH WEIGHT (In kgs.)	MEAN PLACEN -TAL WEIGHT (In grams)	F/P RATIO
Normal	20	2.9	442	6.56 : 1
Mild pre-eclampsia	20	2.6	422.5	6.15 : 1
Severe pre-eclampsia	20	2.05	377.5	5.43 :1
Eclampsia	20	1.84	355	5.21 : 1
Essential hypertension	6	2.25	375	6 : 1
Pre-eclampsia superimposed on essential hypertension	14	2.15	378.86	5.67 : 1

TABLE IV
SHOWING CO-RELATION OF THE HYPERTENSIVE PLACENTAL CHANGES WITH PROTEINURIA

Type of cases	Number of cases Total = 80	FINDINGS			
		weight of placenta		Infarction	
		< 300 gms	> 300 gms	Absent	Present
Protein absent	32	3(9.38%)	29(90.63%)	25(78.13%)	7(21.87%)
Protein Present	48	26(54.17%)	22(45.83%)	17(35.42%)	31(64.58%)

of the cord insertion have no effect on baby's weight. Usually the number of cotyledons in normal placenta varies from 10 - 30. But it is found to be less in P I H cases because in case of severe pre-eclampsia and eclampsia it is very difficult to count the number of cotyledons, as the inter-cotyledon septas are obliterated by excess deposition of fibrin. The number of cotyledons has also got no co-relation with foetal outcome.

Infarction : There was definite increase in incidence of placental infarction involving > 5 % of parenchyma in hypertensive

TABLE V
SHOWING CO-RELATION OF THE MACROSCOPIC PLACENTAL FINDINGS WITH FOETAL OUTCOME IN HYPERTENSIVE AND CONTROL CASES

Macroscopic placental findings	No. of hypertensive cases (Total=80)	State of the newborn Birth <2.5 kgs	weight >2.5 kgs	Low apgar - score	Still -born	No. of normal cases (Total-20)	Birth <2.5 kgs.	State of the newborn weight >2.5 kgs	Low Apgar - score	Still - born
Weight of the placenta.										
i. <300gms.	25	14(56%)	11(44%)	16(64%)	8(32%)	-	-	-	-	-
ii. 301-500"	51	19(37.25%)	32(62.75%)	17(33.33%)	4(7.84%)	16	2(12.5%)	14(87.5%)	1(6.25%)	1(6.25%)
iii. > 500 gms	4	-	4(100%)	1(25%)	-	4	-	4(100%)	-	-
Infarction										
i. Absent.	33	9(27.27%)	24(72.73%)	2(6.06%)	1(3.03%)	15	-	15(100%)	-	-
ii. 0-5 %	7	2(28.57%)	5(71.43%)	2(28.57%)	1(14.29%)	5	1(20%)	4(80%)	-	-
iii. > 5%	40	34(85%)	6(15%)	26(65%)	8(20%)	-	-	-	-	-
Retro-placent-tal haematoma.										
i. Absent.	73	18(24.66%)	55(75.34%)	36(49.32%)	-	20	1(5%)	19(95%)	1(5%)	-
ii. Present.	7	3(42.86%)	4(57.14%)	4(57.14%)	2(28.57%)	-	-	-	-	-
Calcification										
i. Absent.	47	9(19.15%)	38(80.88%)	-	3(6.38%)	14	1(7.14%)	13(92.86%)	-	-
ii. Present.	33	9(27.27%)	24(72.73%)	7(21.21%)	3(9.09%)	6	-	6(100%)	1(7.14%)	-
Subchorionic fibrin.										
i. Absent.	46	11(23.91%)	35(76.09%)	9(19.57%)	3(6.62%)	16	1(6.25%)	15(93.75%)	-	-
ii. Present.	34	14(41.18%)	20(58.82%)	11(32.35%)	5(14.71%)	4	-	4(100%)	-	-

disorders, specially in pre-eclampsia and eclampsia cases. In this study it was present in 10 (50%) mild pre-eclampsia, 14 (70%) severe pre-eclampsia cases and 12 (60%) eclampsia cases. It was found to be present in 3 (50%) out of 6 essential hypertension cases and 8 (57.14%) out of 14 pre-eclampsia superimposed on essential hypertension cases. Infarction was absent in 15 (75%) normal cases and when present it involved less than 5 % of the total surface area (Table I &IV)

Proteinuric patients were having higher incidence of infarction than non-proteinuric hypertensive cases (Table V). Fox (1975) was of the opinion that extensive infarction was found in hypertensive disorders is associated with high incidence of foetal hypoxia, growth retarded babies and foetal death. So also in our observation, as infarcts reduce the amount of placental tissue available for nutrition of the foetus.

Retro-placental Haemotoma : Fox (1978) & Mohan et al (1989) reported higher incidence of retro-placental haemotoma in pre-eclampsia. Our study revealed 8.75 % cases of pre-eclampsia & eclampsia were associated with retro-placental haemotoma involving more than 5% of placental parechyma; where as it was absent in control group. These cases were associated with low apgar score babies. Larger haematoma was also associated with intra uterine foetal death; be cause a considerable portion of the villi were acutely separated from the maternal utero-placental circulation. (Table I & VI)

Calcification & Subchorionic fibrin : Mohan et al (1989) found frequency of calcification was same in control as well as in hypertensive group. This study revealed a little higher incidence of calcification in diseases placentae but it does not alter the foetal outcome. Most of the studies (Fox, 1967 & Mallik et al, 1979) did not record increase of subchorionic fibrin in hypertensive disorders. But in the present study, the incidence of subchorionic fibrin was more than double in hypertensive cases (42.5%) than in control group (20%); however it didnot affect the foetal outcome (Table I & VI).

CONCLUSION

From the above findings it can be concluded that hypertensive disorders of pregnancy have some definite adverse influences on the morphology of the placenta and growth of the foetus. The placental changes are more prominent with severity and duration of the disease process.

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