HISTOPATHOLOGY OF PLACENTA AND ITS CORRELATION WITH FOETAL OUTCOME

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SUMMARY

Placenta from 100 cases were studied. Foetal outcome in these cases was noted. It has been observed that two-third of placenta may be infarcted without foetal death.

Introduction

Much effort is being put into understanding the placental changes and their effects on the fetus.

In this study, an attempt has been made to correlate various placental changes to the fetal outcome in normal and abnormal pregnancies.

The pathological changes in the placenta are not by and large specific to a particular disorder and therefore a variety of disorders may shows similar changes. The quantification of the placental changes is essential to correlate the outcome in terms of the fetus (Fox 1963).

Material and Methods

In the present study, placentae from 100 cases delivered at Indira Gandhi Medical College and Hospital, Nagpur, were studied. Fetal outcome in these cases was noted.

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On initial examination of the placenta, any gross abnormality of shape, such as accessory lobe or bilobed placenta was noted. Site of cord insertion was recorded. Cut surface of cord was examined and number of vessels recorded. For examination of the membranes, a segment of membranes was cut from the point of rupture to placental margin and then rolled like a "Jam-roll" and subsequently sectioned transversly. Each placenta was then trimmed of membranes, washed and weighed and was then examined for calcificaiton and infarction.

The entire placenta was cut into vertical strips with a sharp knife. Four placental slices from the peripheries were taken and then cut through full thickness block from fetal to maternal surface, from the central area of each slice sections from each of these blocks were cut and stained with H & E stain. Special staining with Masson's trichrome for fibrin deposition and P.A.S. for basement membrane was done.

Cases selected for study were divided

into following groups: Group I Control (Normal) term parturients—25 cases. Group II Toxaemia of pregnancy with blood pressure of 130/90 mm of Hg and above with or without oedema and albuminiuria—42 cases. Group III Diabetes associated with pregnancy—5 cases. Group IV Cases with intrauterine death of fetus—10 cases. Group V Cases with intrauterine growth retardation. Group VI Cases with miscellenous medical disorders.

Anaemia—1, tuberculosis 1, infective hepatitis 1.

Group VII Cases with fetal complications Hydrocephalus 2, anencephalus 1, Conjoined monster 1, Rh incompatibility 1.

The following data was recorded for each fetus:

1. Any sign of fetal distress during labour. 2. Birth weight. 3. Apgar score at 1 minute. 4. Stll birth. 5. Neonatal morbidity and mortality.

One hundred villi in each section from placenta were counted for histological changes and expressed as percentage for

1. Syncytial knot count.
2. Fibrinoid nerosis of villi.
3. Thickened basement membrane.
4. Villous fibrosis.
5. Cytotrophoblast proliferation.
6. Intervillous haemorrhages.

Observations

Table I presents the corresponding percentage counts of the various villous changes in different groups.

TABLE I

Histopathology of the Placenta in Maternal Disorders—Quantitative Analysis

Histopathology	Grade %	Toxaemia %	Diabetes %	IUD %	IUGR %	Normal
Syncytial knot Count	0-29	13 (39.03)	1(20)	6(60)	7(70)	25
	30-59	24 (57.1)	4(80)	2(20)	2(20)	—
	60-89	5 (11.9)	—	2(20)	1(10)	—
	Above 90		—	—	—	—
Fibrinoid necrosis of villi	0-5	16 (38.9)	—	6(60)	7(70)	25
	5-10	25 (89.5)	3(60)	4(40)	3(30)	—
	Above 10	1 (2.3)	2(40)	—	—	—
Thickened basement membrane	Absent	4 (9.5)	—	—	1(10)	25
	0-2	3 (78.5)	3(60)	9(90)	7(70)	—
	Above 2	5 (11.9)	2(40)	1(10)	2(20)	—
Villous fibrosis	Absent	14 (33.3)	—	—	7(70)	25
	I	18 (42.8)	3(30)	2(20)	2(20)	—
	II	10 (23.8)	2(20)	8(80)	1(10)	—
Cytotrophoblast proliferation	Absent	15 (35.7)	2(40)	3(30)	3(30)	25
	Present	27 (64.2)	3(60)	7(70)	7(70)	—
Inter-villous haemorrhages	Absent	—	—	—	—	—
	Present	42 (100)	—	—	—	—

The frequency of syncytial knots was graded according to the percentage of villi showing syncytial knot formation. Less than 30% was grade I, 30-59% as grade II, 60-89%, as grade III and more than 90% as grade IV. The counts of syncytial knots were found to range between 0.39% in normal control group while between 30-59% in all other groups. Villi showing fibrinoid necrosis of more than 5% were found in cases of toxemia (59.5%) and diabetes 60%. The count was between 0-5% in all uncomplicated cases and in most cases of intrauterine death. (60%) and in cases with intrauterine growth retardation (70%).

Absence of thickening of basement membrane was conspicuous in normal group while count of thickened basement membrane was found to range between 0-2% in most of the other groups. Grade-II type of villous fibrosis was found in placentae following intrauterine death. Cytotrophoblastic proliferation was found in toxemia (64.2%), diabetes (60%), intrauterine death (70%), intrauterine growth retardation (70%).

Incidence of fetal distress was found to be more in toxemia (40.4%). Low apgar score was observed more in toxemia (38.09%) and intrauterine growth retardation (30%) cases. Percentage of still births in toxemia was 11.9% and in diabetes 20%. With placenta weighing less than 300 gms fetal distress (43.3%) and low apgar score (43.3%) were observed. Three periratal deaths were also noted (Table II).

Placental pathology was studied microscopically and it was correlated with fetal outcome. Cases with fetal distress (46.8%) and still births (62.5%) were showing syncytial knot count of grade II and grade III respectively (Tables III & IV).

High incidence of fetal distress, low apgar score, still births and perinatal deaths were observed when the thickening of basement membrane was observed in more than 2% of villi.

Highly significant correlation was found between fetal distress, still births and perinatal death with villous fibrosis and cytotrophoblastic proliferation.

Significant intervillous and intravillous haemorrhages were observed in cases of pre-eclampsia and eclampsia (42 cases).

In the present study leukocytic infiltration of umbilical cord was found in 1 case of toxemia (2.3%) and 1 case from normal group (4%). Only 1 case of chorioamnionitis was observed of all the 100 cases studied. No correlation with fetal hypoxia was noted.

TABLE II

Foetal Outcome in Various Groups

Foetal status	Normal (25)	Toxaemia (42)	IUGR (10)	Diabetes (5)	Fever anaemia (1)
Foetal distress	1(4%)	17 (40.4%)	2(20%)	1 (20%)	—
Low apgar score	—	16 (38.09%)	3(30%)	1 (20%)	1(100%)
Still birth	1(4%)	5 (11.9%)	—	1 (20%)	—
Perinatal deaths	—	—	3(30%)	—	—

TABLE III
Correlation of Foetal Outcome With Gross Placental Changes

Grading	Total No.	Foetal Distress %	Low apgar score %	Still births %	Perinatal deaths %
Placental weight					
0-300 gms	23	10(43.3)	11(43.3)	3(13.03)	3(13.04)
300-500 gms	74	15(22)	13(17.5)	8(10.8)	—
Above 500 gms	3	1(33.3)	1(33.3)	2(66.6)	—
Placental infarction					
0-5%	35	7(20)	7(20)	2(5.8)	3(8.5)
5-10%	23	2(8.6)	2(8.6)	2(8.6)	—
10% and above	42	17(40.4)	16(38.09)	9(21.9)	—
Calcification					
Absent	59	20(33.8)	13(22.2)	8(13.5)	3(5.09)
+	35	5(14.2)	10(28.7)	2(5.4)	—
++	6	1(16.6)	2(33.3)	3(50)	—

Discussion

Fox (1975) states that placenta being a fetal organ is small when fetus is small and weight is a poor indicator of placental adequacy. However, low placental weight does decrease functional reserve. Although still births were not significantly increased in this study, the fetal distress, low apgar score and perinatal deaths were significantly more frequent when placenta weighed less than 300 gms at term.

It has been reported that two-third of the placenta may be infarcted without fetal demise and it has been found that placental infarction has no apparent effect upon growth or welfare of the fetus as the placenta has considerable functional reserve capacity. Fox (1967 and 1975) is of the opinion that in an uncomplicated pregnancy, placental infarction plays little role in perinatal mortality and morbidity. But extensive infarction as found in hypertensive complications in pregnancy is associated with high incidence of fetal hypoxia. However, in the present study, we found no corelation between placental infarction with fetal outcome. We found no association between fetal hypoxia, low birth weight or intrauterine death and calcification of placenta. This finding is consistent with the earlier reports made by Tindall and Scott (1965).

Syncytial knot formation is seen with increased frequency in the last weeks of normal pregnancy and in cases of toxamia. (Benirsehke, 1961; Fox, 1965). This seems to be the result of ageing as suggested by electron microscopy. The loss of nuclei as a result of such sequestration is made up by fresh nuclei from cytotrophoblast (Fox, 1965), hence fetal jeopardy is not increased (Mirchandani-Malik-Chitra, 1979). But in present study, a significant correlation was found between excessive syncytial knotting and fetal distress and still births.

An undue thickening of basement membrane has been noted in placenta from cases of pre-eclamptic toxemia (Hall, 1949; Sayeed et al, 1976; Mirchandani, 1979) we found thickened basement

TABLE IV

Correlation of Foetal Outcome With Microscopic Placental Pathology

Pathological changes in the villi		Total No.	Foetal distress (26%)	Low apgar score (25%)	Still birth (13)	Perinatal death (3)
Syncytial Knot Count						
Grade						
I	0-29%	60	9(15.0%)	19(31.6%)	2(3.3%)	—
II	30-59%	32	15(46.8%)	4(12.5%)	6(18.7%)	3(9.3%)
III	60-89%	8	2(25.0%)	2(25.0%)	5(62.5%)	—
Above IV	Above 90%	Nil	Nil	Nil	Nil	Nil
Fibrinoid Necrosis of Villi						
	0-5%	62	19(29.03%)	19(30.6%)	7(11.2%)	1(1.6%)
	5-10%	35	7(20.0%)	4(11.4%)	6(17.1%)	2(5.8%)
	Above 10%	3	1(33.3%)	2(66.6%)		
Thickened Basement Membrane						
	Absent	30	3(10%)	4(13.3%)	1(3.3%)	—
	0-2%	60	18(30%)	15(25%)	5(8.2%)	2(3.3%)
	Above 2%	10	5(50%)	6(60%)	7(70%)	1(10%)
Villous Fibrous						
	Absent	54	11(20.3%)	10(8.5%)	—	—
	Grade-I	25	12(48%)	12(48%)	5(20%)	—
	Grade-II	21	3(14.2%)	3(14.2%)	8(38.09%)	3(14.7%)
Cytotrophoblast Proliferaton						
	Absent	48	2(4%)	4(8.3%)	2(4.2%)	—
	Present	52	24(46.1%)	21(40.3%)	11(21.1%)	3(5.7%)

membrane in 0-2% of villi in 78.5% cases of toxemia, 90% of intrauterine deaths and 60% of diabetic cases. We found a high incidence of fetal distress in 70% cases when low apgar score in 60% cases and still births in 70% cases when more than 2% villi showed basement membrane thickening. These findings are consistent with the reports made by Mirchandani et al, 1979.

Stromal fibrosis increased in pre-edampsia and diabetes (Fox, 1968) and also in placenta following intrauterine death. Fox (1978) states that there is no association between villous fibrosis and any fetal complications such as hypoxia or low birth weight. The present study shows that fetal jeopardy is increased with stromal fibrosis. Our findings are similar with the observations made by Mirchandani et al, 1979.

Mathews (1973) found stromal fibrosis in 0.3% of villi in terms placenta. The two main factors thought to be responsible for the formation of stromal fibrosis are normal ageing process and a reduced uteroplacental blood flow.

A high incidence of fibrinoid necrosis is found in diabetes, toxemia and Rh incompatibility cases (Fox, 1968). Severe degrees of fibrin deposition increases fetal jeopardy (Wigglesworth, 1964). We found 5-10% of villi showing fibrinoid necrosis in toxemia (59.5%) and diabetes (60%), but no association between fetal hypoxia, low apgar score and still birth with a high fibrinoid necrosis count, was noted. This is also reported by Fox (1963 and 1975).

According to Fox (1964), a high proportion of live born fetuses who have suffered intrauterine hypoxia have placentae in which villous cytotrophoblastic cells are unduly increased in number. Similar findings are also seen in placentae of infants of low birth weight and still births: If the syncytiotrophoblast suffers ischaemic damage, the cytotrophoblast will proliferate in an attempt to replace the damaged tissue. It is clear that the degree of cytotrophoblastic hyperplasia is related to the extent of snyncytial damage and thus it is a rough quantitative index of the severity of ischaemia to which the villi have been subjected. A high proportion of live born fetuses who have suffered intrauterine hypoxia have placentae in which villous cytotrophoblastic cells are numerous. In the present study, we found highly significant correlation between fetal distress, low apgar score, still births, perinatal deaths and cytotrophoblastic proliferation.

Furthermore, it was observed that all the placentae from toxemic pregnancies showed intervillous and infravillous haemorrhages.

Conclusions

1. Quantitative estimation of placental changes is essential as none of the individual changes are diagnostic either of maternal associated condition or of fetal hypoxia.
2. Fetal outcome was observed to be worse when placenta weighs less than 300 gms at term.
3. Placenta from cases with high incidence of fetal distress and still births showed increased syncytial knotting.
4. In placentae following intrauterine death, thickening of basement membrane count was increased.
5. Fetal jeopardy was indicated by cytotrophoblastic proliferation.
6. Placentae from cases with still births and perinatal deaths showed marked villous fibrosis.

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