HISTOPATHOLOGY OF PLACENTA AND ITS CORRELATION WITH FOETAL OUTCOME

K. AVASTHI, U.MIDHA, B.D. SABHARWAL & KIRNA DEVI

SUMMARY

125 placentae, 25 from normal term pregnant patients and 100 from abnormal term pregnant patients were studied histopathologically and changes found were correlated with foetal outcome. The most important indicator of foetal outcome was villous fibrosis. Increased villous fibrosis has a high incidence of foetal distress & still births. The placentae of still births showed increased syncytial knots, fibrinoid necrosis, villous fibrosis and cytotrophoblastic proliferation. The placentae in foetal distress cases showed increased syncytial knot formation,and thickening of basement membrane and villous fibrosis.

INTRODUCTION

The scientific interest in the placenta derives not only from its enormous diversity of form and function but also from the varied histopathological changes in different disease entities. Much effort is being put into understanding the placental changes and their effects of the foetus. Perinatal period is described as the most delicate period in human life.

Noseman (1937) define the placenta as a fusion of the foetal membranes to the uterine mucosa for the transfer of oxygen and metabolites between the maternal and foetal blood.

Deptt. of Obstet. and Gynec., Dayanand Medical College and Hospital, Ludhiana (Punjab).

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As the placenta grows and ages,histologic changes suggest an increase in the efficiency of transport to meet the metabolic requirements of the growing foetus. Such changes involve the decrease in the thickness of syncytium,partial disappearance of Langhan's cells, decrease in the stroma, increase in the number of capillaries, and their approximation to the syncytial surface.

The pathologic changes in the placenta are not by and large specific to a particular disorder and therefore a variety of disorders may show similar changes. Final picture is often very complicated and no particular complication of pregnancy produces specific morphological changes within the placenta

which allow one to make a specific morphological diagnosis.

MATERIAL AND METHODS

The present study was conducted on 125 patients selected at random from the normal and abnormal pregnant women delivered in the Department of Obst. & Gynaecology at Dayanand Medical College & Hospital, Ludhiana.

The cases were divided in two groups.

Group 1. The control normal term parturient.

Group 2. The abnormal term parturient.

A detailed history & general physical examination, systemic examination were done on every patient. The various investigations like Hb, TLC, DLC,Routine urine,Blood grouping and other tests pertaining to the individual patients were carried out.

For histopathological examination the placenta with membranes and cord was dipped in 4% formaldehyde solution for 48 hours for fixation. The placentae were then washed and examined thoroughly on external surface for any abnormality.

They were then cut at every 2 cms.interval. 5u thin slides were cut from each block, stained with Haemotoxyline and eosin, special staining was done with masson's trichrom for fibrin deposition and periodic acid schiff for basement membrane thickening.

OBSERVATION

Out of 125 placentae,25 were from normal pregnancies, while rest 100 were from pregnancies complicated by various disorders as given in table I.

TABLE I

Distribution of Cases

Group		No. of cases
Normal		25
Abnormal		100
(a)	Toxaemia of pregnancy	40
(b)	Anaemia	20
(c)	IUGR (Intra Uterine Growth Retardation).	10
(d)	IUD (Intra-Uterine Death) and congenital anomalies of foetus.	10
(e)	Post-term	10
(f)	Foeto maternal rhesus incompatibility.	10

The maternal age distribution of various cases is shown in table II.

TABLE II

The maternal age distribution of various cases

Group	Maternal age in years			
	20-24	25-30	31-35	36-40
Normal	14	9	2	–
Abnormal				
Toxaemia	18	21	1	"
Anaemia	8	11	1	"
IUGR	8	2	"	"
Post-Term	2	7	1	"
IUD	4	6	"	
Rh Incompatibility	5	3	1	1
Total	45	50	4	1

TABLE III
Histopathology of the placenta in normal and abnormal pregnancies

	Normal	PIH	Anaemia	IUGR	RH Incompatibility	Post term	IUD
1. Syncytial knot count							
Grade I (0-29%)	25(100%)	14(35%)	20(100%)	3(30%)	2(20%)	3(30%)	4(40%)
Grade II (30-59%)	–	24(60%)	–	7(70%)	7(70%)	6(60%	5(50%)
Grade III(60-89%)	–	2(20%)	–	–	1(10%)	1(10%)	1(10%)
Grade IV Above 90%	–	–	–	–	–	–	–
2. Fibrinoid necrosis							
0-5%	25(100%)	29(72.5%)	16(80%)	10(100%)	10(100%)	8(80%)	7(70%)
5-10%	–	11(27.5%)	4(20%)	–	--2(20%)	2(20%)	
Above 10%	–	–	–	–	–	–	1(10%)
3. Basement membrane thickening							
Absent	25(100%)	16(40%)	20(100%)	3(30%)	3(30%)	3(30%)	5(50%)
0-2%	–	20(50%)	–	7(70%)	–	5(50%)	5(50%)
Above 2%	–	4(10%)	–	–	–	2(20%)	–
4. Villous Fibriosis							
Absent	25(100%)	20(50%)	20(100%)	5(50%)	5(50%)	4(40%)	2(20%)
Grade I	–	18(45%)	–	4(40%)	–	4(40%)	4(40%)
Grade II	–	2(5%)	–	1(10%)	–	2(20%)	4(40%)
5. Cytotrophoblastic proliferation							
Absent	25(100%)	14(35%)	20(100%)	3(30%)	3(30%)	4(40%)	2(20%)
Present	–	26(65%)	–	7(70%)	7(70%)	6(60%)	6(60%)
6. Inter villous Haem							
Absent	–		–	–	–	–	–
Present	25(100%)	40(100%)	20(100%)	10(100%)	10(100%)	10(100%)	10(100%)

Table III (Cont.)

	Normal	PIH	Anaemia	IUGR	RH Incompatibility	Post term	IUD
7. Calcification							
Absent	20(80%)	6(15%)	11(55%)	2(20%)	–	2(20%)	2(20%)
Present	5(20%)	34(85%)	9(45%)	8(80%)	10(100%)	8(80%)	8(80%)
8. Infarction							
Absent	23(92%)	30(75%)	17(85%)	7(70%)	9(90%)	6(60%)	7(70%)
Present	2(8%)	10(25%)	3(15%)	3(30%)	1(10%)	4(40%)	3(30%)
9. Inflammation of membrane							
Absent	25(100%)	30(97.5%)	20(100%)	6(60%)	10(100%)	8(80%)	8(80%)
Present	–	1(2.5%)	–	4(40%)	–	2(20%)	2(20%)

In normal pregnancies (25cases) only syncytial knots (Grade I changes), Fibrinoid necrosis (0.5%) were found in the placentae. Where as placentae in abnormal pregnancies showed more exaggerated response as in table III.

As shown in the table IV, 20 cases (40.8%) showing syncytial knots in 30.59% of villi and 4 (80%) cases with syncytial knot formation in 60.89% of villi had foetal distress. Similarly incidence of still birth was 22.44% (7cases) in group showing syncytial knots in 30.59% of villi and 40% (2cases) in group with syncytial knot in 60.89% of villi. Statistically the incidences of foetal distress ($p< 001$) and still birth ($p<05$) were significant.

Thicknened basement membrane in more than 2% of villi showed foetal distress in 5 cases (62.5%), low apgar score in 22 cases (25.0%),still births in 3 cases (31.5%) and perinatal deaths in one case (1.5%). Statistically the incidences of foetal distress was significantly correlated with thickness basement membrane ($p<001\%$).

The presence of cytotrophoblastic proliferation was associated with foetal distress in 20 cases (37.04%), low apgar score in 8 cases (14.75%), still birth in 16 cases (29.63%) and perinatal deaths in 3 cases (5.55%). Statistically significant correlation was seen with birth cases ($p<001$).

As shown in table V, the incidence of foetal distress was highest in post maturity and Rh-incompatibility group, 6 cases (60%) in each group, followed closely by IUGR in 5 cases (50%) and toxemia in 14 cases (35%). Low apgar score was more frequently observed in toxemia, in 10 cases (25%) and IUGR, 2 cases (20%). Percentage of still births was highest in IUGR and Rh-incom-

TABLE IV

Correlation of foetal outcome with microscopic placental pathology

Pathological changes in the villi	Total No.	Foetal distress (39)		Low apgar score (16)		Still birth (20)		Perinatal death (4)	
		No.	%	No.	%	No.	%	No.	%
Syncytial knot count									
Grade I 0-29%	71	15	21.12	6	8.45	7	9.85	1	1.40
Grade II 30-50%	49	20	40.81	9	18.36	11	22.44	3	6.12
Grade III 60-89%	5	4	80.00	1	20.00	2	40.00	–	–
Grade IV Above 90%	–	–	–	–	–	–	–	–	–
Fibrinoid necrosis of villi									
0.5%	109	33	30.27	14	12.84	13	11.92	4	3.66
5-10%	15	6	40.00	2	13.33	6	40.00	–	–
Above 10%	1	–	–	–	–	1	100.00	–	–
Thickness basement membrane									
Absent	75	13	17.33	7	9.33	8	10.66	1	1.33
0-2%	42	21	50.00	7	16.16	9	21.42	2	4.76
Above 2%	8	5	62.50	2	25.00	3	31.50	1	12.50
Villous fibrosis									
Absent	81	18	22.22	9	11.11	5	6.17	2	2.46
Grade I	33	16	48.48	7	21.21	9	27.27	2	6.06
Grade II	11	5	45.45	–	–	6	54.54	–	–
Cytotrophobastic proliferation									
Absent	71	19	26.77	8	11.25	4	5.63	1	1.41
Present	54	20	37.04	8	14.75	16	29.63	3	5.55

TABLE V
Foetal outcome in various groups

Foetal Status	Normal (25)		Toxaemia (40)		Anaemia (20)		Rh. incompatibility (10)		IUGR (10)		Post Term (10)	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Foetal distress	4	16	14	35	4	20	60	5	50	6	60	
Low Apgar score	1	4	10	25	2	10	1	10	2	20	–	–
Still birth	–	–	5	12.5	–	–	2	20	2	20	1	10
Perinatal deaths	1	4	2	5	–	–	–	–	1	10	–	–

patibility groups (20%), 2cases in each group followed by toxaemia in 5 cases (12.5%).

DISCUSSION

In our study, syncytial knot formation was increased in PIH, IUGR, post term and IUD cases. 26 cases of PIH (65%) showed high syncytial knots which is consistent with earlier reports by Sayeed et al (1976) 93%, Mallik et al (1979) 100%, Mirchandani et al (1979) 60% and lately Masodkar et al (1985) 69%. We did not find increased counts in anaemic patients. Sabharwal et al (1987) have also reported the same findings. This in sharp contrast to studies by Sayeed et al (1976) 93% reporting marked increase in counts in anaemic patients.

Sayeed et al (1976) has even reported increased counts in normal patients (77%). But like other workers, we did not observe any increase in normal patients. Mirchandani et al (1979) foetal jeopardy was not increased even if syncytial knot formation was seen in more than 90% of villi. In present study, a statistically o73 significant correlation was seen between high syncytial knot and count of 30-59% and 4 cases (80%) with syncytial knot formation in 60-89% of villi, had foetal distress ($p<0.001$). 11 cases (22.44%) of still birth with syncytial knot count 30-59% of villi and 2 cases of still births with syncytial knot formation in 60-89% were seen ($p<0.5\%$). No significant correlation was seen low apgar score perinatal mortality. Perinatal mortality was seen in 3 cases (6.12%) with syncytial knot formation in 30-59% of villi. In this respect, our observations are more similar to those reported by Masodkar et al (1985) who have observed significant correlation between excessive syncytial knot formation and foetal distress and still birth.

In relation to foetal outcome, 6 cases (40%) of still birth had fibrinoid necrosis in 5-10% of villi and one case (100%) of still birth had fibrinoid necrosis in more than 10% of villi, which was statistically significant (p<.001). This observation is consistent with earlier report by Sen and Langley (1974) who reported 4 cases (66%) of still birth with increased fibrinoid necrosis. In contrast, Fox (1968), Mirchandani et al (1979) and Masodkar et al (1985) have not observed any significant correlation of increased fibrinoid with any of the foetal parameters observed.

Correlation of foetal distress with thickening of basement membrane was quite significant in our study. 21 (50%) cases of foetal distress had basement membrane thickening in 0-2% of villi and 5 (62.5%) cases had in more 2% of villi (p<0.001).

Masodkar et al (1985) also observed 30% cases with basement membrane thickening 0-2% of villi and 50% cases with basement membrane thickening in more than 2% of villi having foetal distress. Incontrast Mirchandani et al (1979) did not report statistically significant correlation of basement membrane thickening with any of the foetal parameters.

As regards foetal outcome, villous fibrosis was the most important factor affecting foetal well being. 16 cases (40.40%) with grade I changes and 5 cases (45.45%) with grade II changes were having foetal distress (p<0.001). Again 9 cases (27.27%) with grade I changes and 6 cases (54.54%) with grade II changes had still birth (p<0.001). This is consistent with earlier reports by Mirchandani et al (1979) and Masodkar et al (1985).

Cytotrophoblastic proliferation was statistically significantly correlated with still birth: 16 cases (29.63%) of still birth and cytotrophoblastic proliferation (p<0.001). Fox (1964) also found high langhans cells counts in 12 out of 13 still birth cases. Masodkar et al (1985) also drew attention to significant correlation with still birth, 21. 1% cases of still birth having langhans cells proliferation.

Inter-villous haemorrhage was found almost all the 125 cases. We do not attach any significance to this finding. Inflammation of the membrane was not found significant in any of the maternal conditions.

BIBLIOGRAPHY

1. *Fox,H. J.Obstet.Gyec.Bri.C'wealth,71: 759,1964.*
2. *Fox,H. Fibrinoid necrosis of placental villi. J. Obstet.Gynaec.Bri.C'wlth.,75:448-451,1968a.*
3. *Mslik,H.B.;Mirchandani,J.J. and Chitra, S. J. Obstet.Gynec.India.,29:805,1979. o73*
4. *Asodkar,A.R.;Kalaskar,L.B.;Patki,P.S. J.Obstet. Gynec.India. 35:294,1985.*
5. *Mirchandani, J.J>;Mallik,G.B. and Chitra,S. J.Obstet.Gynec.INdia. 29:407,1979.*
6. *Noseman,H.W. uterine structures. Cont. Embryo.Carney,INstn.26:129-1937.*
7. *Sabharwal,B.D.;Malhotra,V.;Sofat,R.and Duggal,A. J.Obstet.Gynec.India.37:773-1987.*
8. *Sayeed,M.;Chakrawarti,R.N.;Devi,P.K. J.Obstet. Gynec.India.26:216,1976.*