STUDY OF THE PLACENTA IN PREMATURE LABOURS

by

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Introduction

There have been few reported pathological studies on the prematurely delivered placenta and umbilical cord. The causes of spontaneous premature deliveries are unknown as are the sequence of events causing onset of labour at term in normal pregnancy. The structural changes found in the premature placenta may suggest the existence of an abnormality in the uteroplacental relationship. These placentae usually attain early maturation and it is possible that the morphological maturity is accompanied by changes in the endocrine function.

Material and Method

One hundred and two placentae were collected from the indoor patients of Kamla Nehru Hospital and Swaroop Rani Nehru Hospital, Allahabad. Eighty-six specimens were from cases of premature labour from 28-37 weeks of gestation, and 16 were control specimens from normal labour at term. The premature labour group included cases of anaemia, antepartum haemorrhage, toxaemia, hydramnios, hepatitis and Rh negative cases.

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After gross examination of the placenta, 4 pieces of tissues of 1 cm thickness were taken from different quadrants of the maternal surface, and from those areas which showed gross abnormalities. These were fixed, processed sectioned and stained by standard techniques for histopathological study.

Points noted in the Study

Gross lesions: Calcification, Infarction and retroplacental clots.

Histopathology

I. Villi: (1) Trophoblast, with the presence or absence of syncytial knots and bridges. (2) Villous stroma for signs of degeneration or inflammation. (3) Villous vessels—Thrombosis was looked for. The grading of thrombosis was done by screening the slide under a scanner field. When in a random field, ¼ of the field showed thrombosis it was graded as grade I, when ½ the field showed thrombosis, it was grade II, ¾ of the field showing thrombosis was grade III and involvement of the full field, with the exception of a few villi, was classified as grade IV.

II.Foetal Stem Arteries for the presence or absence of thrombi.

III. Other associated pathological findings: (1) Fibrin deposition, (2) Fibrinoid degeneration, (3) Calcification, (4) Inflammation.

Observations and Discussion

Gross Placental Lesions: These were seen in 38 out of 86 placentae (44.2%). Infarction was seen in 30 out of 38 cases an incidence of 78.9%. Calcification was seen in 12 out of 38 cases (31.6%) and retroplacental clots were seen in 6 out of 38 cases, an incidence of 15.8% (Table I). There was a considerable overlap in the three entities, more than one type of gross lesion being found in the same placenta.

Microscopic Placental Lesions: A marked variation in the villous structure was seen in different areas of the same placenta. The incidence of such a variation was 46.5%, it being seen in 42 out of 86 cases. The villi in most of these cases were crowded and well vascularized as is seen in term placentae. Varying degrees of syncytial knotting was seen in about 75% of cases, and here a mild to moderate amount of fibrin deposition was also seen. The stroma was dense containing very few Hofbauer cells. In some areas sclerosis of the foetal vessels with cellularity of the villi was seen. An intact Langhan's layer was not seen in any case of premature labour.

TABLE I

Gross Placental Lesions in Different Clinical Entities

Sl. No.	Gross abnormality	A.P.H.	Still birth	Toxaemia	Anaemia	Hydramnios
	No. of cases	6	9	5	8	2
1.	Infarction					
	Percentage	20%	30%	16.66%	26.6%	6.66%
	30 / 78.9%					
	No. of cases	2	5	3	1	1
2.	Calcification					
	Percentage	16.16%	41.6%	25%	8.33%	8.33%
	12 / 31.61%					
	No. of cases	6	—	—	—	—
3.	Retroplacental clot.					
	Percentage	100%	—	—	—	—
	6 / 15.8%					

The present findings are in support of Sauramo (1951, 1961) who found a number of infarcts in the placentae of premature labours. Other workers such as Wigglesworth (1962) Wilken (1965) and Fox (1968) found however, a low incidence of placental lesions. Wentworth (1967) found infarcts in the majority of their cases of pre-eclamptic toxaemia. Novak (1974) said that calcification had no significance clinically and that it was found at areas of trophoblastic degeneration.

Holland (1974) said that at least 90% of the placenta would have to show abnormal changes to cause foetal death, and that lesser grades of these lesions were compatible with life.

The variation in villous structure was also reported by Boyd and Hamilton (1967) and Fox (1964). They found a striking difference in the villous pattern from different areas of the same placenta. The villi were crowded and well vascularized. They inferred that there was a maturation well beyond the period of gestation for some obscure purpose, probably for increasing the ease of transfer in cases of anoxia or placental insufficiency. Becker (1960) found a fully

mature villous morphology in 10% of his cases, and he called this "Maturation Praecox Placentae".

Fox (1969) also noted aberrations of villous morphology in 10% of his cases. Syncytial degeneration is found when there is placental ischaemia and early placental maturation. The supporters of this view are Wigglesworth (1962) and Fox (1964).

In the present series, no correlation was found between the gestation age of the foetus and increasing placental maturity, the foetal age being normal for that period of gestation. Fox (1969) also did not find any evidence of the acceleration of foetal maturity in his series of abnormal placental maturation.

Other Associated Pathological Findings

Fibrinoid Degeneration: In prematurely delivered placentae, fibrinoid degeneration was seen in 67 out of 86 cases (77.9%), in and around the villous stroma and the blood vessels.

An arbitrary grading was done, and it was seen that mild grade of changes were most commonly seen between 28-32 weeks of gestation. Moderate to severe grade of changes were seen chiefly between 33-37 weeks of gestation (Table II).

The fibrinoid degeneration of moderate grade which was seen between 33-37 weeks of gestation suggests that there is premature aging of the placenta. Novak (1974) said that the fibrinoid degeneration is a consequence of syncytial knotting and thinning of the syncytium. In the control series, a mild grade of fibrinoid degeneration was present in all the 16 cases. In his series Fox (1969) said that only 3% of term placentae showed fibrinoid degeneration and that it was present in 29.7% of cases of prematurely delivered placentae.

Thrombosis of the Villous Vessels: The commonest lesion associated with fibrinoid degeneration was thrombosis seen in 46 out of 67 cases (68.6%). The maximum number of cases, 20 (43.48%) were seen with grade II variety of thrombosis—Table III.

TABLE II

Different Degrees of Fibrinoid Degeneration in Different Periods of Gestation

S. No.	Degree of fibrinoid degeneration	Period of gestation			
		28-32 wks		33-37 wks	
		No	%	No.	%
1.	Mild	20	29.85	13	19.37
2.	Moderate	4	5.97	30	44.70
3.	Severe	2	2.98	8	11.92

TABLE III

Grading of Cases of Fibrinoid Degeneration Associated With Thrombosis

Sl. No.	Grades	Thrombosis	
		No. of cases	% of cases
1.	Grade I	12	26.09
2.	Grade II	20	43.48
3.	Grade III	8	17.38
4.	Grade IV	6	13.05
	Total:	46	100

TABLE IV

Different Clinical Entities Seen in Different Sets of Gradings

Sl. No.	Grades	A.P.H.		Toxaemia		Hydramnios		Anaemia		Still births	
		No. of cases	% of cases	No. of cases	% of cases	No. of cases	% of cases	No. of cases	% of cases	No. of cases	% of cases
1.	Grade I	—	—	1	8.33	1	8.33	7	58.31	2	16
2.	Grade II	4	20	4	20	2	10	2	10	4	20
3.	Grade III	2	25	3	37	—	—	2	26	3	37
4.	Grade IV	1	16.66	—	—	—	—	1	16.66	4	66

In the control series thrombosis was seen in 5 out of 16 cases the incidence being 31.25%. It is probable that thrombosis is an end result of syncytial degeneration, which leads to coagulation of maternal blood which comes in contact with exposed subepithelial villous tissue. This in turn leads to ischaemic necrosis and infarction, signifying an early maturation of placentae, Sauramo (1951, 1961).

Calcification: The second finding associated with fibrinoid degeneration was calcification. It was seen in 22 out of 67 cases (32.8%). These were seen around the villi or around the areas of fibrinoid degeneration.

TABLE V

Association of Fibrinoid Degeneration With Calcification in Different Clinical Conditions

Sl. No.	Clinical conditions	No. of cases	% of cases
1.	Stillbirths	11	50
2.	Antepartum haemorrhage	3	16.16
3.	Toxaemia	2	0.90
4.	Anaemia	5	22.74
5.	Hydramnios	1	0.45
	Total	22	100

Inflammation: Inflammation alongwith fibrinoid degeneration was seen in 12 out of 67 cases, 13.8%. In 3% of these cases there was formation of lymphoid follicles. Inflammation was found in those cases of premature labour which were associated with premature rupture of membranes, fever and stillbirths.

Fox (1969) found many cases in his series associated with inflammation, but these were cases of premature rupture of membranes. In the control series there was no case of calcification or inflammation.

TABLE VI

Association of Inflammation With Fibrinoid Degeneration in Various Conditions

Sl. No.	Clinical entity	No. of cases	% of cases
1.	Premature rupture of membrane	5	41.5
2.	Stillbirth	5	41.5
3.	Fever before and during delivery	2	17
	Total	12	100

Fibrin Deposition: It was seen in 13 cases of premature labour in 28-32 weeks gestation period, the incidence being 15%.

Villous Degeneration: This was in 16 out of 86 cases of premature labour, the incidence being 12.04%.

(C) Fibrinoid degeneration was seen in 77.9% of cases. The maximum number of cases, 30 out of 67 were seen with a moderate grade of fibrinoid deposition in the 33-37 weeks gestation period.

(D) Fibrin deposition was seen in 15% of cases which were between 28-32 weeks of gestation.

(E) Inflammation was found in 13.8% cases which were associated with premature rupture of membranes, fever and stillbirths.

(F) Thrombosis of the villous vessels was seen in 46 out of 67 cases (68.6%). Thrombosis classed as grade II variety was seen in the majority of cases 20 (43.48%).

In the control series, fibrinoid degeneration was seen in 100% of cases, fibrin deposition in 18.75% of cases and thrombosis in 31.25% of cases.

TABLE VII

Pathological Findings in the Control Series

Sl. No.	Various pathological findings	No. of cases	% of cases
1.	Fibrin deposition	3	18.75
2.	Fibrinoid degeneration	16	100
3.	Thrombosis associated with fibrinoid degeneration	5	31.25
4.	Calcification	—	—
5.	Inflammation	—	—

Conclusion

The histological examination of the placenta from 86 cases of premature labour showed—

1. *Macroscopic lesions*: in the form of infarction, calcification and retroplacental clots in 44.2% of cases.

2. *Microscopic lesions*: (A) Marked variation in the villous structure was seen in different areas of the same placenta in 46.5% cases.

(B) Thinning of the syncytium with the presence of syncytial knots and bridges was seen in 75% of cases.

The intrinsic placental aging mechanism is fascinating, and, as yet, largely unknown. The balance between aging of the placenta and the period of gestation is a delicate one, and what upsets this balance is still a speculation.

In the present series, studies have shown that placentae from premature deliveries do show premature age changes. Fibrinoid degeneration present as 100% in the control series is a normal aging phenomenon. This is present in the

premature placenta in 77.9% of cases. Thrombosis present in 31.25% of cases in term placentae is present in 68.6% in premature placentae, and gross placental lesions were not found at all in the control series studied.

Thus, if the age changes normally present in term placentae show up in premature placentae, it signifies premature aging of the placenta. Therefore, the factors which are responsible for onset of labour at term, are triggered off by premature aging of the placenta. This may upset the hormonal balance responsible for maintenance of pregnancy, or it may be responsible for an exaggerated immunological response, a sort of rejection phenomenon, between maternal and placental tissues, thus precipitating premature labour.

Summary

One hundred and two placentae were examined from 86 cases of premature labour and 16 control cases of labour at term. In the premature labour group:

Macroscopic: lesions were found in 44.2% of cases, in the form of infarction, calcification and retroplacental clot.

Microscopic lesions: were found as variation in villous structure in 46.5% of cases; thinning of syncytium with the presence of syncytial knots and bridges in 75% of cases and fibrinoid degeneration was seen in 77.9% of cases. In addition fibrin deposition was found in 45% cases, inflammation in 15% of cases and thrombosis in 68.6% of cases. In the *In Control series*, fibrinoid degeneration was seen in 100% of cases, fibrin deposition in 18.75% of cases and thrombosis in 31.25% of cases.

Thus in cases of prematurity, the placenta may reach a stage where its function stops, and this may set up hormonal and immunological disturbances leading to the onset of labour.

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