

A HISTOLOGICAL STUDY OF PLACENTA DELIVERED BEFORE TERM

by

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Prematurity is often blamed as a cause of foetal wastage. Altered placental morphology has been suspected along with maternal and foetal defects for the causation of prematurity. Many workers have attempted to identify the pathological lesions in the placenta expelled in premature labour.

For this study 212 placentae expelled in premature labour have been selected. Care has been taken to select placentae from pregnancies where prematurity was the only notable abnormality. Placenta from pregnancies with toxaeemias, diabetes, Rh incompatibility and other such disorders have been excluded.

Material and Methods

Placenta were obtained from deliveries conducted in the labour wards of the Obstetric department of the medical college. Pregnancies where either the mothers knew the correct last menstrual period or were attending the antenatal clinic from the first missed period only were taken into account. A gross study was made soon after the placenta were delivered and the placenta was fixed in a solution of 10 per cent formalin for a week. They were then cut into half cm. blocks and processed for histopathological study. All the slides were stained with haematoxylin and eosin. Villus maturity, chorioamnionitis, langerhans cells, fibrosis and fibrinoid necrosis were studied.

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Accepted for publication on 1-6-1978.

Observations

1. Majority of the placenta were apparently normal when seen macroscopically. Ten placentae showed small infarcts at the periphery. Retroplacental clots were noticed in 4 of the placentae.

2. *Villus Maturity*: Villus maturity was studied in all placentae—gross morphological variation in the development could be noted only in 26 of the placentae. Seven placentae showed a pattern of full differentiation which could be seen usually in a mature placenta. It has been observed that variable villous morphology may be a result of continuous placental development extending upto its expulsion (Fox, 1968).

3. *Langerhan's Cells and Syncytial Knots*: These features which suggest an early placenta were seen in 9 placentae.

4. Stromal fibrosis has been noted in 5 cases.

5. *Chorio-amnionitis*: This feature was noted in 59 of the placentae. This constitutes the major lesion in this series. This feature was also noticed earlier by Blanc 1961, Benirschke and Driscoll, 1967, and Clark and Anderson, 1968.

6. *Fibrinoid necrosis*: This was noted in 11 of the placentae.

Discussion

This study comprising of 212 placentae from pregnancies where prematurity was the only apparent abnormality, was done to evaluate as to how far the placenta contributed to the occurrence of premature delivery.

Placental maturity depends on maternal, foetal and environmental factors. In any case, if the mothers were otherwise healthy and the babies normal, then the fault must be with the placenta. Probably, the morphological maturity of the villi is brought about by the multiple endocrinal alterations that are noticeable at term.

Previous work (Fox, 1969) has shown a significant incidence of fibrinoid necrosis in the prematurely delivered placenta suggesting an immunological response. This series shows a low incidence of this feature.

A high incidence of chorio-amnionitis has been seen in this series. This probably is the precipitating cause for the premature onset of labour. It is stated that early rupture of membranes could predispose to bacterial infection (Langley and Smith, 1959). This is probable in causing chorioamnionitis in this series.

Though a majority of placentae were noted to be normal, in those of the placentae where chorio-amnionitis occurred, it is felt that this condition could be responsible for the premature expulsion of the placenta.

Summary

212 Placentae from premature labour, where prematurity was the only apparent abnormality were studied. Though a majority were found to be normal, 59 of them showed chorio-amnionitis while a few others showed minor pathological defects.

Acknowledgement

I express my gratitude to the Professor and Head of the Department of Obstetrics and Gynaecology and staff for the placental collection, documentation and transportation to the department, my Professor Dr. M. S. Parthasarathy for all encouragement and the Professor and Head of the Department of Pathology and staff for their wholehearted co-operation.

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