HISTOPATHOLOGICAL CHANGES IN PLACENTA IN PRETERM LABOUR

by

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Introduction

Preterm parturition is a leading cause of neonatal morbidity and mortality. The precise aetiology of preterm labour or term parturition is still unknown. It has been estimated that premature rupture of membranes occurs before term labour in 10 to 14 per cent of cases. The close association between preterm labour and premature rupture of membranes has been shown by many authors like Danforth *et al* (1953) and Lebherz *et al* (1961). The study of the cause of spontaneous premature rupture of membranes may contribute to the knowledge of aetiology of preterm labour in atleast 30 per cent of the cases. Driscoll (1973) and many others suggested that the inflammation and friability of the membranes may result in premature rupture of membrane.

Material and Methods

This analysis studies the changes in placenta, membranes and cord in cases of preterm labour and also to observe its changes and correlate its relationship with premature rupture of membranes. The cases in the present series were selected from the Antenatal clinic, Out-patients' Department, Labour Room and indoor wards of Hospital for Women, Patna Medical College Hospital, Patna. A total of 100 cases under observation comprised of 75 cases of premature rupture of membranes associated with preterm labour and 25 cases of preterm labour without rupture of membranes.

Study of placenta

All the placentae were studied immediately after the delivery and both gross and histopathological examinations were carried out. The placental inflammation was grouped as:

Grade I—Neutrophilic infiltration is limited to the decidua capsularies and merginalis (Photograph I).

Grade II—Neutrophilic infiltration has extended upto chorion. Sometimes, neutrophilic infiltration includes the placental surface and umbilical cord. If neutrophilic infiltration is only found in the cord, it is considered as grade II (Photograph II).

Grade III—Marked neutrophilic infiltration is present in the chorion. Neutrophilic infiltration frequently extends to the placental surface and umbilical cord.

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TABLE I
Distribution of Age

Age groups in years	No. of cases	Preterm labour with premature rupture membranes		Preterm labour without premature rupture of membranes	
		No.	Percentage	No.	Percentage
15-19	28	16	21.33	12	48
20-24	10	7	9.33	3	12
25-29	11	9	12.00	2	8
30-34	17	13	17.33	4	16
35-39	34	30	40.00	4	16
40 and above	Nil	—	—	—	—
Total	100	75	—	25	—

Preterm labour is frequently associated with premature rupture of membranes in the age group of 35 to 39 years.

The incidence of placental inflammation is more in preterm labour with premature rupture of membranes than without premature rupture of membranes. The frequency of inflammation is inversely proportional to the duration of gestation.

The incidence of Grade I placentitis is more in cases having membrane rupture to delivery intervals of less than 48 hours, Grade II and III placentitis are more often found in association with prolonged

TABLE II
Frequency of Placental Inflammation in Different Weeks of Gestation

Period of gestation in weeks	Total No. of cases	Preterm labour with premature rupture of membranes		Preterm labour without premature rupture of membranes	
		No. of cases	Placental inflammation	No. of cases	Placental inflammation
28 or less	18	15	15 (100%)	3	2 (66.67%)
29-32	46	35	21 (60%)	11	4 (36.36%)
33-36	36	25	10 (40%)	11	—

TABLE III
Relationship Between Membrane Rupture to Delivery Interval and Severity of Placental Inflammation in the Study Group

Membrane rupture to delivery intervals in weeks	No. of cases studied	Cases showing placentitis	Placental inflammation (Placentitis)		
			Grade I	Grade II	Grade III
Less than 48 hours	34 (45.33%)	5 (10.87%)	4 (8%)	1 (20%)	—
More than 48 hours	41 (54.67%)	41 (89.13%)	24 (58.54%)	13 (31.71%)	4 (9.76%)
Total	75	46 (61.33%)	28 (60.87%)	14 (30.43%)	4 (8.70%)

TABLE IV
Grade of Placentitis in the Study and Control Group

Grades of placentitis	Preterm labour with premature rupture of membranes			Preterm labour without premature rupture of membranes		
	Total cases	20-28 weeks	29-36 weeks	Total cases	20-28 weeks	29-36 weeks
Grade I	28 (60.87%)	2 (7.14%)	26 (92.86%)	4 (66.67%)	2 (50%)	2 (50%)
Grade II	14 (30.43%)	9 (64.29%)	5 (35.71%)	2 (33.33%)	—	2 (100%)
Grade III	4 (8.70%)	4 (100%)	—	—	—	—
Total	46 (88.46%)	15 (32.61%)	31 (67.79%)	6 (11.54%)	2 (33.33%)	4 (66.67%)

rupture of membranes (more than 48 hours).

Placental inflammation is more frequent in the study group. The percentage of cases showing grade I and II placentitis in both groups is quite similar, while only grade III can be correlated with premature rupture of membranes. Among the cases showing placental inflammation (study group), most of those after 28 weeks gestation had grade I placentitis, while most of those before 28 weeks had grade II and/or III placentitis.

Discussion

One hundred pregnant women were studied. Seventy-five had spontaneous premature rupture of membranes leading to preterm delivery and 25 had preterm delivery without prior rupture of membranes. Placental histology was done in 100 preterm cases, 75 (study group) with premature rupture of membranes and 25 (control group) without premature rupture of membranes. Placental inflammation was detected in 61.33% of the study group and 24% of the control group. The severity of placental inflammation (grade of placentitis) was directly related to the duration of membrane rupture to delivery interval, thus stressing the importance of ascending infection.

Grades I and II placentitis were found in both the groups, but grade III placentitis was exclusively related to premature rupture of membranes. This proves that though infection causes premature onset of labour it is intensified after the rupture of membranes. The earlier in gestation that premature rupture of membranes occurs the higher is the incidence of placental inflammation. The incidence of inflammation of the placenta and its appendanges in the present series, is in accordance with that of Fox and Langley (1969), and Malkani and Bhasin (1970).

The pathogens responsible for the intrauterine infection are mostly amongst the normal inhabitants of female birth canal (Douglas and Stander, 1943; Benirschke and Driscoll, 1967). The common bacteria involved are E.coli, anaerobic streptococci, staphylococcus faecalis lactobacilli, diphtheroids. It has been shown in in-vitro studies that E.coli endotoxins stimulate guinea-pig uterus and significantly increase the tone and

frequency of uterine musculature (Wiederman *et al* 1962).

The membranes (specially amnion) can synthesize prostaglandins as well as serve as a source of arachidonic acid to be used by decidua for the generation of prostaglandins (Creazy and Liggins, 1969). It has also been suggested that phospholipase A_2 activity found in the foetal membranes (especially amnion) is localised, in part, in the lysosomes (Gustavii, 1973). Gustavii (1973) proposed that the lysosomes in the decidua are maintained in a stable form throughout most part of the pregnancy. Uterine infection releases active phospholipase A_2 which frees arachidonic acid from phospholipid stores and makes it available for prostaglandin synthesis. Prostaglandings have the capacity of initiating smooth muscle contraction, thereby causing uterine contractions, and cervical dilatation. The cervical membranes already weakened by infection and devitalized due to stretching become unsupported with cervical dilatation. The "death blow" is dealt by a strong Braxton Hick's contractions or other strain leading to a breach of foetal membranes and ultimately expulsion of the foetus.

It can be safely concluded that endocervical infection from vaginal flora can initiate premature rupture of membranes and subsequently preterm labour either by releasing bacterial phospholipase A_2 or by destabilizing the intracellular lysosomes which house the phospholipase A_2 (Gustavii, 1973). The latter splits the arachidonic acid from the membrane (chiefly amnion phospholipids) and this in turn leads to the synthesis of prostaglandin F_2 and E_2, leading to the onset of labour by stimulating thinning of the cervix an altering calcium metabolism in the myometrium (Gustavii, 1973, and Creazy and Liggins, 1979).

Therefore, infection can bring about premature rupture of membranes and preterm labour. It is suggested that early recognition and adequate treatment of endocervical/vaginal infections in women of child bearing age, specially multigravidae will go a long way in prevention of preterm labour. This gives a new dimension to the scope of good antenatal care leading to a substantial decrease in perinatal mortality and morbidity as well as maternal mortality and morbidity.

Summary

Placentitis was a common finding in preterm cases, specially those associated with premature rupture of membranes. The severity of placental inflammation was directly related to the duration of membrane rupture to delivery interval. This proves that though infection causes premature onset of labour, it is intensified after the rupture of membranes. The earlier in gestation that premature rupture of membranes occurs the higher is the incidence of placental inflammation. Grades I and II placentitis were found in both the groups, but grade III placentitis was exclusively related to premature rupture of membranes before 28 weeks of pregnancy.

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See Figs. on Art Paper I