HISTOPATHOLOGICAL STUDY OF FOETAL ADNEXA IN AMNIOTIC INFECTION SYNDROME

by

SAVITA SAWHNEY
VEENA K. SHARMA
V. K. PRATAP
B. DEVDUTT
and
M. L. MEHROTRA

SUMMARY

A total of 120 placenta, cord and membranes (50 study and 70 control cases) were studied. The difference in incidence of inflammation of placenta and membranes was statistically significant in two groups ($p < 0.001$) while it was statistically insignificant in cord inflammation ($p > 0.05$). A significantly higher correlation was found between prolonged rupture of membranes in cases with history of repeated unclean vaginal examination, history of fever 38°C before onset of labour, malodorous intrauterine contents and adnexal inflammation. It is felt that proper antibiotic cover should be given after amniotic fluid culture where history of any of the maternal factors is present.

Introduction

Perinatal infection is an important cause of foetal loss and neonatal death. Clarification of the real relationship between the pathogenesis of perinatal infection and inflammatory reactions in the placenta, membranes and cord referred to as "amniotic infection syndrome" is a problem of considerable importance. About two thirds of the neonates with chorioamnionitis were found to have congenital pneumonia or sepsis presumbly related to the antenatal aspiration of infected amniotic fluid. (Naeye, *et al* 1971). Fewer studies are available regarding maternal events which might have been related to infection. (Maudsely *et al* 1966) and Fox and Langely, 1971). Such maternal information is needed to unravel the pathogenesis of amniotic infection syndrome. The present study links specific maternal events in the late gestation with the amniotic infection syndrome.

From: Deptt. of Pathology, L.L.R.M. Medical College, Meerut-250 102.

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Material and Methods

One hundred and twenty cases were chosen at random from deliveries at S.V.B.P. Hospital attached to L.L.R.M. Medical College, Meerut from May' 83 through May' 84. All the women had a period of gestation of at least 28 weeks.

Of these, 50 were in the study group having one or more of the following features: Prolonged rupture of membranes for more than 24 hours, fever 38°C or more after rupture of membranes, malodorous intra-uterine contents and history of unclean vaginal examination during delivery. Control group included 70 cases in which all the above mentioned features were absent.

Following delivery the placentas along with their adnexa were collected in formal saline. Blocks were prepared from the segments of umbilical cord within 5 cms of its origin at the placenta and from its middle portion; from a full thickness segments of the central part of the placenta including maternal and foetal surfaces and two sections from lateral margin including immediately adjacent membranes and a roll of the membranes from the point of the rupture to the placental margin were taken. Additional sections from any seemingly abnormal tissue were also taken. All the sections were stained by haematoxylin and eosin. The inflammation in cord was graded according to criteria of Wilson and Armstrong (1964), while the inflammation of placenta and membranes was categorized as mild, moderate and severe.

Mild — When sprinkling of neutrophilic leukocytes was seen.
Moderate — In between mild and severe.
Severe — Heavy infiltration by neutrophilic leukocytes.

Observations

The patients were in the age group of 16 to 45 years, the majority of them (115 cases) were below 35 years. Almost all were multipara, and 90.8% of the cases were full term (36 weeks or more) at the time of delivery. Of the 50 cases in study group, majority 40 gave history of prolonged rupture of membranes for more than 24 hours before delivery, 16 had history of fever of more than 38°C from onset of ruptured membranes up to delivery, malodorous intra-uterine contents were present in 6 cases, 6 gave history of more than two unclean vaginal examination, while history of less than two unclean vaginal examination was present in 5 cases.

The cord inflammation was found in 7/50 and 3/70 cases in study and control group respectively. The difference was not statistically significant ($p > 0.05$). The phlebitis was found in all the cases while artery involved in 50% of the cases. Grade I and Grade III inflammation was more common in both groups than grade II (Fig. 1).

The incidence of chorioamnionitis was higher in cases of study group as compared to control group and difference was statistically highly significant ($p < 0.001$) (Table I). Chorionitis was found to be more frequent than amnionitis (Fig. 2).

TABLE I
Frequency of Various Inflammatory Changes in Membranes

Grade of inflammation	Study Group (50 cases)		Control Group (70 cases)	
	Chorionitis	Amnionitis	Chorionitis	Amnionitis
Mild	10 (20%)	7 (14%)	7 (10%)	5 (7%)
Moderate	10 (20%)	10 (20%)	6 (8.6%)	6 (8.6%)
Severe	5 (10%)	5 (10%)	3 (4.3%)	3 (4.3%)
Total	25 (50%)	22 (44%)	16 (22.3%)	14 (20%)

p Value = <0.001.

The inflammation of placenta was found in 22/50 study and 16/70 cases in control groups respectively. The difference was statistically significant ($p < 0.001$). The commonest morphological lesion in both the groups (Table II) was neutrophilic leukocytic infiltration in chorionic plate (Fig. 3) followed by subchorionic intervillositis (Fig. 4), Chorionic vasculitis, and focal areas of necrosis. Abscess formation was seen only in study group.

There was no significant correlation between the age and parity and the inflammation of placenta, cord and membranes. A

TABLE II

Frequency of Various Inflammatory Lesions in Placenta

Inflammatory lesions	Study Group (50 cases)		Control Group (70 cases)	
	No. of casse	Percentage	No. of casse	Percentage
Polymorphonuclear leukocytic infiltration in chorionic plate	24	48.0	16	22.9
Subchorionic intervillositis	12	24.0	4	5.7
Chorionic vasculitis	4	8.0	2	2.9
Abscess formation in chorionic plate	4	8.0	—	—
No. of Hofbauer cells	5	10.0	1	1.4
Focal areas of necrosis	4	8.0	1	1.4
Placental Villitis	—	—	—	—

TABLE III

Correlation Between Prolonged Rupture of Membrane, Repeated Vaginal Examination, History of Fever (38°C) Before Delivery, History of Malodorous Intrauterine Contents and Inflammation of Placenta, Membrane and Cord

	Total No. of cases	Placental inflammation	Membrane inflammation	Cord inflammation
Duration of rupture of membranes (hours)				
24-48	25	7 (28%)	12 (46%)	2 (4%)
48-72	12	6 (50%)	10 (83.3%)	2 (16.6%)
More than 72	3	2 (66.7%)	3 (100%)	2 (66%)
History of unclean vaginal examination				
Less than 2 times	5	1 (20.0%)	1 (20.0%)	1 (20.0%)
More than 2 times	6	2 (33.3%)	3 (50.0%)	2 (33.3%)
History of fever 38°C (before delivery	16	8 (50%)	9 (56.2%)	2 (12.5%)
Malodorous intrauterine contents	6	4 (66.7%)	5 (83.3%)	2 (33.3%)

rising trend of adnexal inflammation was noted with history of fever (38°C) before onset of labour, repeated unclean vaginal examination, malodorous intrauterine contents and with increasing duration of rupture of membranes (Table III).

Discussion

In the present study frequency of inflammation of cord, membranes and placenta worked out to be 14%, 50%, and 44% for study group, and 4.3%, 22.8%, and 22.9% for control group respectively. The results of this study are in general agreement with those of Fujikara and Benson (1963) for cord; Piskari *et al* (1963) for membranes; and Woods *et al* (1979) for placenta.

It was found that the incidence of inflammation of placenta cord and membrane in full term deliveries was 37.5% in comparison to 54.5% in 11 preterm deliveries. According to Siddall (1927 and 1928) and Blanc (1959) placental inflammation was three times as common in premature births as in term deliveries. The cord lesions tend to be associated with the more advanced types of placental lesions. This finding coupled with the lower incidence of cord lesions suggest that the umbilical cord is only involved at a fairly late stage of the infective process, thus increasing the clinical value of cord inflammation, and a real risk of clinical foetal infection is also observed in relatively advanced stage.

In this study chorionitis was found to be more frequent than amnionitis. This finding is in accord to a number of workers (Benirschke, 1960; Bhaskar Rao *et al*, 1966 and Driscoll *et al*, 1965). The amnion is an avascular structure and therefore the presence of inflammatory exudate within its substance indicates an extension of the inflammatory process from underlying tissue. Thus amnionitis represent a severe form of inflammation of the membranes (Maudsley *et al*, 1966). It is mainly produced by ascending bacterial infection from the vagina and cervix and is rarely haematogenous. Santosh *et al* (1984) have found that although bacteria could be present in amniotic fluid without evidence of placental lesions nevertheless placental leukocytic infiltration was invariably associated with the presence of organisms.

No significant association was found between inflammation of placenta, cord and membranes and the clinical factors such as maternal age and parity. Similar observations were reported by Fujikara and Benson (1964) and Fox and Langley (1971). A highly significant association was found between leukocytic infiltration of placenta, cord and membranes and prolonged rupture of membranes as has also been reported by Blanc, 1959; Bhaskar Rao *et al*, 1966; Overbach *et al*, 1970. The prolonged rupture of membranes predisposes to the intrauterine spread of bacteria and therefore higher incidence of placenta, cord and membrane inflammation.

Similar to the observation of Maudsley *et al* (1966) increasing incidence of adnexal inflammation was also noted in cases with history of repeated unclean vaginal examination and in cases having fever exceeding 38°C prior to delivery. Thus a predelivery fever is an important clinical observation and indicates intrapartum infection. It is also indicative of a general maternal response to tissue injury.

As the differences in the inflammatory reaction exhibited by the foetal adnexa in the two groups was significant, it is imperative that amniotic fluid should be cultured in all the cases having history of any of maternal factors and proper antibiotic cover should be given after performing sensitivity.

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See Figs. on Art Paper I