CHORIOAMNIONITIS

by

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The term chorioamnionitis was first introduced by Corner *et al.* in 1951 to indicate inflammation of the foetal membranes. It is mainly caused by an ascending infection after rupture of the bag of membranes and may indicate the degree of intranatal and neonatal infection of the baby and probably intrapartum infection of the mother.

Material and Methods

In 54 patients delivered by caesarean section, 2 bits of placenta and membrane, about 3 cm. sq. from each, were snipped off with sterile scissors and forceps during the operation. One piece of each was put up for culture and another was placed in N/10 formaline solution, processed, sectioned and stained with haematoxylin and eosin. Liquor amnii and lower segment swabs were also cultured as described elsewhere (Rao *et al.* 1965). The time of rupture of the membranes, clinical evidence of sepsis, if any, the condition of the child at birth and in the neonatal period, and the nature of the puerperium were also noted. Amongst 54 caesareans 6 were elective sections. Twenty one parturients showed clinical evidence of sepsis and 27 appeared free from infection.

TABLE I

Type of patients	No. of cases	Positive histology	Positive culture	Positive histology and culture
Elective ..	6	1	...	...
Not infected ..	27	12 (44.4)	9 (33.3)	6
Infected ..	21	15 (71.4)	12 (55.6)	9
Total ..	54	28 (51.8)	21 (38.8)	15

(Figures in brackets indicate percentage).

Results

Chorioamnionitis was seen in 51.8 per cent of patients studied (Table

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I). In 6 elective sections, only one showed mild lesion though the cultures were sterile. In those who were apparently free from infection 44 per cent showed chorioamnionitis but in one-third of this group the culture of the membrane was positive. In the clinically infected, group, however, three-fourths of them showed microscopic evidence of inflammation of membranes and in half of them the cultures were positive. Of 21 cases in which the cultures were positive supportive histologic evidence was obtained in 15 (71.4 per cent) but in 28 patients where chorioamnionitis was present histologically, positive culture was obtained in 53.5 per cent.

In patients in whom the interval between rupture of membranes and delivery was less than 6 hours, mild inflammatory reaction was seen in 40 per cent; but when labour was prolonged to over 24 hours following rupture of membranes severe reaction was noted in two-thirds of the cases (Table II).

TABLE II

Duration of labour after rupture of membranes	Incidence of chorio-amnionitis	Remarks
0 — 6 hours	40%	all mild
6 — 24	60%	(40% severe)
over 24 hours	68%	(66.6% severe type)

Amongst 54 caesarean sections, perinatal mortality was 16.6 per cent (6 dead born and 3 neonatal deaths) and 4 were treated for neonatal infections and survived. Amongst those showing histologic evidence of chorioamnionitis, perinatal mortality was 21.4 per cent and the risk of perinatal infection and death was 35.7 per cent. In the histologically negative group this risk was 11.5 per cent.

Discussion

Chorioamnionitis was reported in 12.7 per cent of 1300 deliveries by Benrischke *et al.* (1959). In a series of 709 placentas examined by Emig *et al.* (1961) the incidence was 13.3 per cent, though it was higher when labour was delayed after rupture of membranes or in those with cervicitis or vaginitis. The inflammatory reaction appears to be more severe and massive in the chorionic and amniotic layers than in the uterine decidua. It is mainly produced by ascending bacterial infection from the vagina, and the cervix and is rarely haematogenous in origin (Bourne, 1962). In 6 elective sections, no pathologic lesion was found in the membranes in 5 and in one it was mild. The placental and membranal cultures were negative in these cases showing that normally the uterine cavity is sterile. The longer the labour is delayed after rupture of membranes the greater the chances of infection of the amniotic cavity from the vagina. Blanc (1961) noted amnionitis in only 2-3 per cent of cases where the membranes were absent for 0-6 hours but when this interval increased to 24 hours or more it was 58 per cent. Similar observations have been made by Sidall (1928) and Mcllwaine (1963). In our series, mild infection was seen in 40 per cent of those in whom labour lasted for 6 hours following rupture of membranes, but when it exceeded 24 hours, two-thirds of them showed severe chorioamnionitis. It was particularly marked in those who were handled outside and brought late in labour. Chorio-amnionitis could also be produced occasionally by irritants like meconium, hypertonic glucose etc., in the amniotic cavity (Morison, 1963).

In mild cases, amnion retains its normal smooth and shining appearance. In gross infections this sheen is lost and membranes appear dirty grey and oedematous with exudate covering it. In most of the sections studied there was an exudation of polymorphonuclear cells in the connective tissue of the chorion and the subamniotic space which extend into the amnion (Figs. 1 and 2). Perivascular infiltrates were seen around the small vessels of the chorion. The placenta itself showed a minimal or absence of inflammatory cells except in a few cases. (Fig. 3).

Chorioamnionitis does not always indicate evidence of intrapartum infection of the mother or risk of intra or neonatal infection of the baby. The nonpathogenic organisms in the vagina or cervix may give rise to histologic evidence of inflammation of the membranes though clinically there may not be any evidence of puerperal sepsis or foetal infection. This may explain why amongst our 15 controls (in whom no pelvic examinations were done and who delivered spontaneously in less than 6 hours after rupture of membranes) mild chorioamnionitis was seen in 6 and severe in one case, though all babies were born alive and healthy and the puerperium was normal in this group. In 52 deliveries the cervical swab was cultured and placental membranes were examined histologically. In 31 of them organisms were grown from the cervix but chorioamnionitis was noted in 16 or 51.6 per cent. Similarly, in 26 cases where chorioamnionitis was seen, the cervical swab culture was positive in 61.5 per cent. But when the organisms invade from the cervix membranes, amnionitis is more likely to occur. In 18 cases where culture of the membranes was positive, amnionitis was present in 11; and in 10 cases where cultures of placenta and membranes were positive, histological appearances were corroborative in 7 of them. When bacteria are present in the amniotic cavity amnionitis is most likely to occur. In 13 cases where culture of liquor amnii was positive, amnionitis was noticed in 11. In these 13, there were 4 perinatal deaths and 2 neonatal infections. As shown in Table I, in the clinically infected group the risk of chorioamnionitis, often of severe type, is over 70 per cent. In some cases of gross or even fatal infections, however, there may not be any local lesion in the placenta or membrane the patient having died due to severe toxaemia before inflammatory response in the placenta could be evoked. In 4 patients, who died due to severe sepsis, placental histology and culture were positive in 3; but in the fourth case the membranes were healthy and only the lower segment swab showed streptococcus pyogenes on culture. Likewise occasionally there may be gross inflammation of the foetal membranes but no clinical evidence of foetal or maternal infection as noted in one of our control cases.

Should prophylactic antibiotics be

given in labour to protect the foetus and the mother? It is not necessary, unless labour is delayed for over 6-12 hours after rupture of membranes. However, in all cases handled outside or showing clinical evidence of sepsis, antibiotics are essential. It is in this group that three-fourths of cases showed chorioamnionitis and the foetal risk was high. Blanc (1961) advocates histologic study of the membranes and if there is amnionitis advises antibiotic cover for the newborn. However, we feel that amnionitis is not a certain index of intrapartum infection of the mother or intranatal foetal infection, though the risk of perinatal infection and death is three times greater in cases showing chorioamnionitis than in those where the membranes are healthy. The clinical condition of the parturient (for any evidence of sepsis) is a more reliable guide. In caesareans, the culture of amniotic fluid, membranes or swabs from the lower segment gives us valuable information as to the causative organisms and their antibiotic sensitivity. This information is useful when therapy is started in clinically suspect cases.

Summary

1. In 54 caesarean deliveries, bits of placenta and membranes removed aseptically were subjected to bacteriologic culture and histologic study.

2. In 6 elective sections, cultures were sterile and only in one case was mild amnionitis seen. The lesion was noted in over 70 per cent of clinically infected cases.

3. Chorioamnionitis is mainly caused by ascending infection after rupture of membranes. In those delivering within 6 hours after rupture of membranes mild inflammation was seen in 40 per cent; but when this interval exceeded 24 hours two thirds of the cases showed severe lesions.

4. The risk of perinatal infection and death is 3 times greater in cases showing chorioamnionitis than in those where the membranes were healthy.

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Figs. on art paper VII