BACTERIAL INFECTION OF AMNIOTIC FLUID

by

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Introduction

Amniotic fluid infection, a common event during pregnancy, occurs most frequently in association with rupture of membranes, particularly prolonged rupture and with premature delivery. It has been an important cause of perinatal mortality (Naeye, 1977, 1979). The one encountered in premature infants in the Boston Hospital is usually the infection of the amnion by Escherichia coli, which begins by producing an amnionitis or chorioamionitis before the birth of a premature infant. Such processes sometimes but not always occurring with early rupture of membranes-infect the fetus through the swallowed or inhaled amniotic fluid. Hence the infection gets across the membranes, it may often be the cause of the premature labour itself, for such amnionitis is distinctly less common in full term infants. It tends to occur especially in the 1 lb or 2 lb premature infants rather than in the larger, more mature ones (Smith, 1960). Only a portion of amniotic fluid infections are clinically apparent intrapartum (Russell, 1979). They are usually recognised only on the basis of maternal leukocytosis, maternal or fetal tachycardia, uterine tenderness and irritability or foul-smelling amniotic fluid (Gibbs *et al* 1980). These signs represent late findings of amniotic buid infections, contamination of the fluid by pathogenic bacteria probably occurs several hours before the appearance of clinical signs of infection (Bobitt and Ledger 1978). The resultant intrauterine infections of infants are not usually epidemiologic nursery problems, but they interest us greatly in the possibility of early identification, treatment through the mother before birth and the use of cord sections and amniotic fluid cultures for the pathological and bacteriological diagnosis necessary to useful action. So the present paper deals with the condition of the patient and bacteriological culture of amniotic fluid, culture of cervical swabs to show the possible mode of infection were taken.

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Material and Methods

One hundred patients were taken who delivered in the hospital. These women were having either rupture of leaking

membranes. None of the women had used antibiotics within 30 days before admission. Amniotic fluid was obtained at the time of caesarean section. All patients had undergone L.S.C.S. either as an elective or in emergency. An aliquot amniotic fluid was transported to microbiology laboratory and was inoculated on to blood and MacConkey's agar. Cultures were incubated at 37°C. Facultative isolates were identified by a standard microbiologic technique. Cultures testing for mycoplasma, chlamydiae, viral pathogens were not routinely performed. The cervical swabs were also taken of these patients before or during the caesarean section.

Results

Patients with amniotic-fluid infection did not differ from normal patients in age, race, prenatal care, gravidity, gestational age, or rate of prematurity. However, patients with clinical amniotic-fluid infection had a significantly longer interval from rupture of membranes until delivery and longer labours. Out of 100 patients only 30 had ruptured membranes or leaking membranes and signs and symptoms of amniotic fluid infection. And 20 patients had absent membranes and signs and symptoms of amnionitis. Bacteria were recovered from the amniotic uuid from 33 out of 100 clinically infected patients (Table I). The cervical swabs showed the isolation of *E. coli* and then 2 patients the isolation of E. coli and then 2 patients showed strepto. haemolyticus and 5 patients Klebsiella. None of the cervical swabs showed Pseudomonas and. Mima polymorpha (Table II).

TABLE I

Organism	No. of patients	Incidence
E. coli	20	20%
Ps. aeruginosa	10	10%
Ps. aeruginosa Minor polymorphia	1	1%
E. coli + Ps. aeruginosa	2	2%

TABLE II
Cervical Swabs

Organisms	No. of patients	Incidence
E. coli	25	25%
Strep. to haemolyticus	2	2%
Klebsiella	5	5%

Discussion

The incidence reported by Michael *et al* (1982) was about 88 per cent. 17 per cent of the deaths were due to amniotic fluid infection (Naeye, 1977). Gibbs *et al* (1980) have reported the organism as E. coli, S. pyogenes, S. viridans and F. meningosepticum and 9 infants and perinatal infection. Russell (1979) had observed E. coli the principal organism from amniotic fluid. Previous studies have shown that bacteria are responsible for most if not all of these infections (Naeye, 1977; Lauweryns *et al* 1973). Some amniotic fluid infections appear to be the consequence of bacterial spread from mother's urinary tract (Naeye, 1979). The number of amniotic fluid infection is reduced more in blacks than in white race (Naeye, 1979). Patients with caesarean section had much more complicated courses and its concluded that caesarean section should be reserved for patients with obstetric indications in addition to chorioamnionitis. E. coli was also common in cervical swabs so this is possible that the infection travels from genital tract to amniotic fluid across the membranes. So our results correspond with

the previous workers. So we advise that the amniotic fluid culture must be performed to identify the specific organism involved and to give the proper chemotherapy after sensitivity tests in cases of ruptured or leaking membranes and in cases of prolonged labour.

Summary

100 cases were taken who had leaking membranes more than 24 hours. The amniotic fluid was cultured and the incidence of amniotic fluid infection was 33 per cent. The main organisms were E. coli and Pseudomonas aeruginosa. And E. coli was most common in cervical swabs also so this is possible that infection spreads from genital tract to amniotic fluid.

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