

BACTERIOLOGICAL EVALUATION OF PREMATURE RUPTURE OF MEMBRANES (P.R.M.)

by

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Introduction

Although there has been voluminous literature in last four decades on artificial rupture of membranes with a view to induce labour yet there have been relatively few studies on spontaneous premature rupture of membranes. Aetiology of premature rupture of membranes is probably multifactorial. Varied explanations such as congenital defect (Morton and Peabody, 1946), amniotic chorionic leakage (Schuman, 1951), uterine hypermotility (Taylor *et al*, 1961) or ascorbic acid deficiency (Bruchell, 1964) are not acceptable in the aetiology of premature rupture of membranes. However, in majority of cases, maternal infection especially latent urogenital infection has been held responsible (Knox and Horner, 1960; Biskind and Biskind, 1957). Therefore, the present study has been undertaken to evaluate the role of bacterial infections in the causation of spontaneous premature rupture of membranes among the patients admitted in Queen Mary's Hospital of King George's Medical College, Lucknow.

Material and Methods

1. *Selection of cases:* Patients for the present study included 3030 delivery cases admitted in Queen Mary's Hospital, K.G.

Medical College, Lucknow from September 1974 to May 1975. The cases were divided into 2 groups:

(i) Control group: This included 2850 primi and multigravidae with intact membranes. Out of these 200 cases were studied.

(ii) Study Group: This included 180 cases with a history of leaking per vaginam. A detailed clinico-gynaecological examination was done in each patient. The criteria observed for diagnosing a case of premature rupture of membranes were (a) the rupture should have been spontaneous and (b) it should have occurred any time prior to the onset of uterine contractions.

The socio-economic status of each patient of study group was assessed by inquiring the monthly income of the family and accordingly the cases were divided in lower, middle and upper income groups.

(2) *Collection of specimens:* Following specimens were collected from both groups for bacteriological study.

(i) Freshly passed urine samples were collected for complete urine examination including microscopic examination.

(ii) High vaginal swabs were collected on admission, just after delivery and on 3rd postpartum day.

(iii) Naso-pharyngeal swabs of the newborn were collected at birth and after 24 hours.

(iv) In cases, where mothers became febrile, urine samples were cultured to detect any postpartum urinary tract infection.

(3) *Bacteriological Study*: All the specimens collected were transported to the laboratory within one hour of collection and were processed as follows:

(i) *Smear preparation*: Smears were prepared and Gram's staining was done to screen out the type of infection.

(ii) *Culture*: Cultures were done on blood agar plates using 8 to 10% defibrinated sheep blood and lactose bromthymol blue agar plate. The plates were incubated at 37°C for 18 hours and the bacteria were identified by the usual methods of identification.

Results

A total of 3030 cases were delivered in Queen Mary's Hospital and 180 cases had premature rupture of membranes, that formed the study group of this work. Out of 2850 remaining cases, 200 cases were studied as control group.

The overall incidence of premature rupture of membranes in this series was 5.8%. It was highest in the age group of 20-30 years (87.0%) and no cases of premature rupture of membrane was noticed after 40 years of age.

Premature rupture of membranes was common in lower income groups (7.3%) as compared to middle (5.3%) and higher (2.3%) income groups.

The average latent period, that is the interval between premature rupture of membranes and onset of uterine contractions, was 13 hours. The shortest latent period was 40 minutes and was seen in 2 cases only and the longest period observed was 72 hours.

The overall maternal and neonatal morbidity rates in study group were 6% and 9.4% respectively, whereas in control group, they were 1.2% and 6.1% respectively. When the causes of maternal and neonatal morbidity rates were analysed after detailed clinico-bacteriological evaluation of the cases, it was observed that bronchopneumonia and diarrhoea were common in neonates, while puerperal sepsis and urinary tract infection were common in mothers (Tables I, II and VI).

TABLE I
Causes of Neonatal Morbidity

Causes	Control group	Study group
Puerperal sepsis	18	6
Urinary tract infection	10	4
Other foci of infection	7	1
Total	35	11

TABLE II
Causes of Maternal Morbidity

Causes	Control group	Study group
Bronchopneumonia	32	4
Cord-sepsis	28	3
Jaundice	31	3
Scleroma	19	1
Diarrhoea	46	5
Haemorrhagic disease of new born	10	1
Septicemia	19	—
Total	176	17

A total of 200 vaginal swabs from control group and 180 vaginal swabs from study group were bacteriologically examined. Positive cultures were found in 79 cases of control group and 93 cases of study group. Among these positive cultures, 17 from control group and 25 from study group showed the growth of pathogens (Table III).

TABLE III
Bacteriological Examination of Vaginal Swabs

Culture	Control group	Study group
Sterile	121	87
Positive	79	93
Cultures		
(i) Pathogens	17	25
— On admission	3	14
— Just after birth	5	6
— 3rd day postpartum	9	5
(ii) Non-pathogens	62	68

Among pathogens, *E. Coli* and *Klebsiellae* were the common isolates (Table IV) and remaining cultures showed the

Bacteriological examination of nasopharyngeal swabs of new borns revealed that 48 out of 200 cases of control group

TABLE IV
Maternal and Neonatal Morbidity Rates in Two Groups of Cases

	Control group		Study group	
	No.	%	No.	%
Maternal morbidity	35	1.2	11	6.0
Neonatal morbidity	176	6.1	17	9.4

TABLE V
Pathogens From Vaginal Swabs

Bacteria	Control group	Study group
<i>E. coli</i>	4	7
<i>Klebsiella</i>	3	5
<i>E. coli</i> + <i>proteus vulgaris</i>	3	2
<i>Klebsilla</i> + <i>proteus mirabilis</i>	2	4
<i>Klebsiella</i> + <i>Pseudomonas aeruginosa</i>	2	3
<i>Staphylococcus aureus</i>	2	2
<i>Streptococcus pyogenes</i>	1	2
Total	17	25

growth of non-pathogens such as diphtheroids, *aero-bacter aerogenes*, *lactobacilli*, etc. It was further observed that in cases of study group, the vaginal swab cultures were positive for pathogens in 14 out of 25 cases at the time of admission as compared to control group where number of positive cultures were more on 3rd day postpartum (9 out of 17 cases).

and 30 out of 180 cases of study group showed positive cultures for different organisms and among positive cultures only 5 out of 48 in control group and 7 out of 30 in study group revealed pathogens (Table V).

Positive cultures for pathogenic organisms were obtained more frequently at the time of birth in study group (6 out

TABLE V
Bacteriological Examination of Naso-pharyngeal Swabs

Nasopharyngeal swabs of new borns	Control group	Study group
Sterile	152	150
Positive	48	30
Non-pathogens	43	23
Pathogens	5	7
— Just after birth	1	6
— After 24 hours	4	1
	200	180

of 7 cases) as against control group where the maximum positive cultures were seen 24 hours after birth (4 out of 5 cases).

The high vaginal swabs of 6 out of 25 patients of study group who suffered from puerperal pyrexia showed a mixed growth of klebsiella and proteus mirabilis in 4 cases and streptococcus pyogenes (Group B) in 2 cases. The babies of the mothers showing streptococcus pyogenes infection developed bronchopneumonia and their naso-pharyngeal swabs revealed the same organisms. Puerperal pyrexia did not occur in the remaining 19 mothers of study group who showed pathogenic organisms, such as *E. coli*, klebsiella, *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Four babies belonging to this group developed bronchopneumonia and naso-pharyngeal swab cultures of the babies revealed the same organism in addition to pneumococci. Urinary tract infection in mothers was observed on 3rd postpartum day in 6 out of 200 cases of control group and 11 out of 180 cases of study group and predominant organisms were *E. coli* and Klebsiella.

Discussion

The term premature rupture of membranes is applied to rupture of membranes any time before the onset of uterine contractions. The most dreaded complication in patients of premature

rupture of membranes is 'infection'. The incidence and severity of infection is directly proportional to the latent period that is the time interval between premature rupture of membranes and onset of uterine contractions. The present study details the bacteriological evaluation of 180 cases of spontaneous premature rupture of membranes and the results have been compared with 200 control cases of normal full term delivery.

The incidence of premature rupture of membranes in the present study was 5.8% which is in conformity with many observers (Biskind and Biskind, 1957; Taylor *et al*, 1958; Danier *et al*, 1965; Sachs and Baker, 1967). However, many other workers have noticed higher incidence (Embrey, 1953; Lebherz *et al*, 1963).

The bacteriological evaluation of P.R.M. cases revealed that the high vaginal swab cultures were positive for pathogens in 25 out of 180 cases in study group as compared to 17 out of 200 cases in control group. Out of these 25 positive cultures in study group, maximum number (14) were positive on admission as against only 3 out of 17 cases being positive in control group. Further, in control group maximum number of cultures were positive on the 3rd postpartum day indicating that the infection in study group was preexisting even before the delivery, while in control group the infection was

acquired after delivery. Therefore, the high incidence of infection in study group may be attributed to be one of the causes of premature rupture of membranes. Out of 6 cases of puerperal pyrexia seen in study group only 1 was under latent period of less than 24 hours and 5 were above latent period of 24 hours.

Seven out of 180 cultures were positive for pathogenic organisms in nasopharyngeal swabs taken from the babies born of mothers in study group and 6 were positive at the time of birth and cultures showed almost same organisms as found in vaginal swabs of mothers indicating that babies must have acquired infection from mothers. Six of these babies developed bronchopneumonia 24 hours after delivery. Mothers of two of these babies suffered from puerperal pyrexia and the vaginal swabs of mothers and the nasopharyngeal swabs of the babies showed positive cultures for *Streptococcus pyogenes*. Only 5 out of 200 babies of control group showed positive cultures for pathogens, mostly 24 hours after birth and the organisms isolated were *staphylococcus aureus*, *streptococcus pyogenes* and *Pneumococci* but none of the mothers in this group developed any infection.

The observations in the present study simulate to fair extent those in the studies carried out earlier. Pryles (1963) observed that preexisting infection in the mother as revealed by bacteriological culture of high vaginal swabs at the time of admission greatly contributes to the causation of premature rupture of membranes. The author has also shown that the bacteria isolated from vaginal swabs of mothers at the time of admission were

same as those isolated from nasopharyngeal swabs of neonates. Brelji and Kaltreider (1966) isolated both pathogenic and non-pathogenic organisms similar to the present study.

Therefore, it can be concluded that pre-existing bacterial infection carried by a mother at the time of admission, as revealed by high vaginal swab cultures greatly contributes to the causation of premature rupture of membranes. This observation is further supported by the fact that the bacteria isolated from the nasopharyngeal swabs of newborns were almost the same as were isolated from their mothers at the time of admission.

References

1. Biskind, J. I. and Biskind, L. A.: *Am. J. Obst. & Gynec.* 73: 750, 1957.
2. Brelje, M. C. and Kaltreider, D. F.: *Am. J. Obst. & Gynec.* 94: 889, 1966.
3. Burchell, R. C.: *Am. J. Obst. & Gynec.* 88: 251, 1964.
4. Danier, L. R., Scarbrogue, R. W., Fillingim, D. W. and Baker, R. E.: *Am. J. Obst. & Gynec.* 93: 398, 1965.
5. Kmbrey, M. P.: *J. Obst. & Gynec. Brit. Emp.* 60: 37, 1953.
6. Knox, J. C. and Horner, J. K.: *Am. J. Obst. & Gynec.* 59: 190, 1950.
7. Leberherz, T. B., Helman, L. M., Wadding, R., Actil, A. and Arje, S. L.: *Am. J. Obst. & Gynec.* 87: 218, 1963.
8. Morton, J. H. and Peabody, C. S.: *Am. J. Obst. & Gynec.* 43: 422, 1946.
9. Pryles, C. V., Steg, N. L., Nair, S., Gellis, S. S. and Tonney, B.: *Paediatrics.* 31: 608, 1963.
10. Sachs, M. and Baker, T. H.: *Am. J. Obst. & Gynec.* 97: 888, 1967.
11. Schuman, W.: *Am. J. Obst. & Gynec.* 62: 633, 1951.
12. Taylor, E. S., Mortan, R. L., Bruns, P. D. and Drose, V. E.: *Am. J. Obst. & Gynec.* 82: 1341, 1958.