

**MICROBIOLOGY OF AMNIOTIC FLUID**

**OR FETAL MEMBRANES**

**AT CAESAREAN SECTION**

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ  
 اللَّهُ لَا إِلَهَ إِلَّا هُوَ الْحَيُّ الْقَيُّومُ  
 لَا تَأْخُذُهُ سِنَةٌ وَلَا نَوْمٌ  
 لَّهُ مَا فِي السَّمَاوَاتِ وَمَا فِي الْأَرْضِ  
 مَن ذَا الَّذِي يَشْفَعُ عِنْدَهُ إِلَّا بِإِذْنِهِ  
 يَعْلَمُ مَا بَيْنَ أَيْدِيهِمْ وَمَا خَلْفَهُمْ  
 وَلَا يُحِيطُونَ بِشَيْءٍ مِّنْ عِلْمِهِ  
 إِلَّا بِمَا شَاءَ وَسِعَ كُرْسِيُّهُ  
 السَّمَاوَاتِ وَالْأَرْضَ  
 وَهُمَا فِي كَفِّ يَدَيْهِ  
 ذَا كَرَمٍ



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# **INTRODUCTION**

## **AND**

# **AIM OF WORK**

patients (33) delivered vaginally and accounted for 3.8 % of all vaginal deliveries. In 80 % (26) of these 33 patients, the fever resolved spontaneously. The other 32 patients underwent caesarean section and these cases account for 22.5% of all abdominal deliveries. In this group, the fever spontaneously resolved in only 30 %.

Endometritis (based upon clinical criteria) is more frequent and more severe after caesarean section. After abdominal delivery, endometritis has ranged very widely from 12 % to 95 % whereas endometritis rates rarely exceed 3 % after vaginal delivery. Overall the risk is 5 - 10 times greater after caesarean section. Both antibiotic failure and serious complications occur more often in women who deliver by caesarean section. Recent reports, from the State of Rhada Island and from Los Angeles County Hospital found the risk of lethal, maternal sepsis to be 81 times greater after caesarean birth than after vaginal birth. The death rate from sepsis was 1 in 1600 Caesarean sections. In other series, sepsis has been less a threat, as at Boston Hospital for women, where 10,000 consecutive Caesarean section were reported without a single maternal death from any case (*Frigaletto et al., 1980*)

The increased incidence of infection in Caesarean section is probably explained by increased intrauterine manipulation, foreign body (suture) reactions, tissue necrosis at the suture



line, haematomas/seroma formation, wound infections and prolonged and difficult labour (*Gibbs, 1985*).

For many years, it was accepted that there is greater increase in maternal infection and death with classic than lower - segment Caesarean sections. In a retrospective, case control study of 89 patient pairs, however, *Blanco and Gibbs (1980)* found no higher incidence of infection morbidity in patients having a classic Caesarean section than in those having low cervical transverse Caesareans.

Several studies have been made of bacterial colonization in the amniotic cavity as a determinant of infection *Gilstrap and Cunningham (1979)* who collected amniotic fluid at Caesarean section from obstetric patients with rupture of the membranes isolated bacteria in all samples and found endometritis in 95 % of the patients.

Several studies have attempted to relate intrapartum bacterial flora of the amniotic fluid to the development of postpartum infection because of the large variety of organisms recovered in these studies (*D'Angelo and Sokol, 1980*).

Clearly, not all patients undergoing caesarean section are at equal risk. Those patients with electively scheduled operations (with no labour and no rupture of membranes) have

lower infection rate than do those with non elective procedures (with labour, ROM, or both). These observations has been made nearly universally in a large number of studies (*Gibbs, 1985*).

*Gibbs (1985)* suggested many risk factors for postpartum infection include :

Operative and obstetric factors :

- Labour.
- Rupture of the membranes.
- Failure to progress in labour.
- Number of vaginal examinations.
- Number of rectal examinations.
- Internal fetal monitoring.
- Low parity.
- Anesthesia.
- Skill of the operator.
- Duration of the operation(60 minutes).
- Estimated blood loss (800 ml).
- Postoperative anemia.
- Obesity.

Laboratory factors :

- Positive amniotic fluid culture.
- Vaginal colonization group B Streptococci.
- Low phosphate / zink ratio in amniotic fluid.
- S. aureus in patients.

Because bacterial colonization of the amniotic cavity often develops after rupture of membranes, it is reasonable to expect that ROM may play a role in postcaesarean infection (*Gibbs, 1985*).

Although vagina is colonized with numerous organisms, infection of the pregnant uterus seldom occurs. Resistance to infection stems from mechanical barriers (the endocervical mucus plug and intact fetal membranes), the antimicrobial properties of amniotic fluid and cervical mucous.

In addition to serving as a barrier, cervical mucous contains lysozyme, an important bacterial inhibitor, which may be present 100 times the concentration of human serum. Antibody has also been found in cervical mucus. The utility of these systems in the pregnant cervix is uncertain.

Rupture of the membranes, whether initiated by local infection or not, removes these barriers. Vaginal flora has free access to the amniotic cavity and infection of the fetus, placenta, membranes or uterus may occur.

Prior to labour and ROM, the amniotic cavity is nearly always sterile. The physical and chemical barriers formed by the intact placental membranes and cervical mucus are usually effective in preventing the entry of bacteria (Sweet and Gibbs, 1985).

## AIM OF THE WORK

The aim of the present work was to describe the quantitative bacteriologic characteristics of amniotic fluid or the fetal membranes at caesarean section in cases:

- 1) Not complicated with rupture of the membranes.
- 2) Cases complicated with prior rupture of membranes and was tried for vaginal delivery for a variable time

Then to correlate such findings with postpartum morbidity

**REVIEW**

**OF**

**LITERATURE**

It is well known that microorganisms that characteristically colonize a particular tissue may, under certain circumstances be the cause of infectious morbidity in the individual (Galask et al., 1976).

The normal flora may be described as a characteristic but varied group of organisms that colonize a given area of a host without causing disease. This may in some cases include organisms that are commonly considered to be opportunistic pathogens, which strike a balance with the host such that they do not cause disease if the host remains healthy.

It should be noted that age, nutritional factor, hormonal status and general health - each is involved in determining the composition of the flora of a particular tissue in a specific individual. It is suggested, therefore, that the term indigenous flora would be an appropriate description for the autochthonous microbes of a given tissue since it avoids the value judgment inherent in the term normal flora and does not arbitrarily exclude certain species, which although considered to be provisional pathogens are nevertheless characteristic of a limited number of hosts and are not exogenous organisms transiently established.

The mere presence of a particular bacterial species as a member of the indigenous flora may be

# MICROBIOLOGY OF THE FEMALE GENITAL TRACT