PROPHYLACTIC ANTIBIOTICS IN PREMATURE RUPTURE OF MEMBRANES

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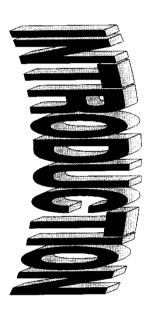
This work is dedicated

To

- * My Parents
- * My Husband
- * My Daughter "Yara"

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INTRODUCTION

Premature rupture of fetal membranes is defined as spontaneous rupture of the membranes prior to the onset of labor regardless of gestational age. It complicates approximately 10% of all pregnancies and is associated with several maternal and fetal problems (Rydstrom et al., 1986).

From the clinician's point of view, preterm premature rupture of membranes is a clinical dilemma because the dangers of preterm delivery must be weighed against the risk of maternal, fetal or neonatal sepsis. Also because complication of prematurity rather than sepsis are the main source of perinatal morbidity and mortality, as well as the general agreement that promote delivery is indicated if these patients have clinical amnionitis, conservation and screening for early detection of infection is best management to improve the perinatal outcome (Vintzileos et al., 1991).

In diagnosing PROM, the history and physical examination done are often inadequate to confirm the status of the membranes, fluid may not present in the vagina for evaluation. Furthermore, at times, fluid may be contaminated with urine, cervical mucus, bath water, vaginal discharge, blood, or meconium. Because of these difficulties, multiple cytological, biochemical colorimetric and sonographic methods have been developed for the the detection of ruptured membranes. Despite significant advances in technology, no one test has been found to be completely accurate and diagnosis still require an integration of historic factors, physical examination and laboratory testing.

Introduction (1)

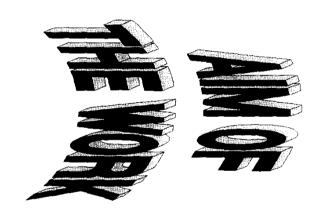
Several investigation used amniocentesis in detection of microbial invasion of amniotic fluid in PROM for example, the use of amniotic fluid cultures and gram staining, amniotic fluid leukocytic count and glucose level, detection of bacterial endotoxins, leuko-attractants as well as leukocytic esterase activity (Garite et al., 1979).

Although cultures method are very sensitiveespecially when compare maternal and neonatal infectious complication in PROM in patient with positive and those with negative culture results, it is tedious, time consuming and the patient may even pass into premature labor or suffer clinical chorioamnionitis while waiting for the culture result. This is in comparison to other rapid and in expensive tests e.g. assessment of leukocyte esterase activity (Gauthier and Meyer, 1992).

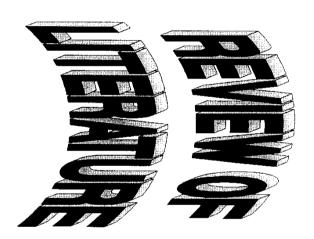
The traditional view is that clinical chorioamnionitis should be managed by immediate delivery. This idea has been extended to the management of microbial invasion of amniotic cavity now the use of antibiotics seems to be the most promising because of its efficacy and minimal side effects (Lackwood et al., 1993 and Owen et al., 1993).

A combination of watchful expectancy and prophylactic antibiotics coverage for cases of PROM has yielded good results in our high risk indigent population. A low perinatal mortality and morbidity from prematurity and infection, and minimal maternal mortality from post partum endometritis (puerperal endometritis).

Introduction (2)



AIM OF THE WORK Evaluate the value of antibiotic to prevent the infection in cases of premature rupture of membrane in both mothers and neonates.



DEVELOPEMENT, ANATOMY, AND HISTOLOGY OF FETAL MEMBRANES

The amnion, derived from embryonic ectoderm, forms as a single layer over the neural ridge of the embryo at the seventh post-conception day. Some investigators have also reported a significant contribution to the amnion originating from trophoblastic cells in addition to the mbryonic ectoderm. Regardless of origin, the amniotic epithelium consists of a single avascular layer of cuboidal or columnar cells, that become more columnar over the placental surface. There are also frequently areas of squamous metaplasia in the amnion.

Although the amnion is only 0.08, 0.12mm in thickness, it has a complex structure. The surface of the amniotic cells is covered with irregular, branched and sometimes confluent microvilli. This unique cellular architecture contributes to the strength and integrity of the membranes. The microvilli extend down the lateral borders of the cells to form intercellular canaliculi in a labyrinthine pattern. Desmosomes, dense discontinuous thickenings of opposing plasma membranes can also be found. The presence of both microvillia and desmosomes allows for greater intercellular communication, an asset necessary for controlling the intraamniotic environment (Van Herendael et al., 1978).

The connections or interconnections made by the microvilli and desmosomes between the amniotic cells provide a mechanical barrier to pathogenic organisms and inflammatory cells. Thus the desmonsomal connections and plethora of microvillar intercellular interdigitations provide

Review of literature (4)

the mechanical strength and resistance to penetration from internal strain forces and external contaminants respectively. The amnionic barrier that protects the fetus becomes a liability when bacterial infections occur or enzymatic systemic go away because it prevents access of therapeutic modalities into the intraamniotic environment. Consequently, the pregnancy is terminated precociously in such instances.

The cytoplasm of amniotic cells is dense and granular with few mitochondria and no demonstrable Golgi apparatuses. The nuclei are located centrally in the cuboidal cells and apically in the columnar cells. Lying beneath the epithelium is a basement membrane of types IV and V collagen firmly anchored to the amnionic epithelium above by podocytes. Such histologic arrangements are usually observed in cells involved in active transport metabolism. Extending down from the basement membrane are collagen projections, composed of type V collagen, that project and intertwine with the type I collagen of the extracellular matrix (Bourne and Lacy, 1960; Verbeck et al., 1967; Modesti et al., 1984; Azzarelli et al., 1987).

This extracellular matrix is an avascular, nerveless stroma composed of banded types I and III collagen bundles, reticular fibrils, and fibroblasts. It is densely laden with macrophages that change their shape from spindle to round when stimulated by foreign particles. There are also scattered mesenchymal cells which, prior to the approximation of the amnion and chorion, compose a contiguous cell layer. With progressive maturation, this cell layer is scattered by the thickening amnionic epithelium (Hoyes, 1970).

Review of literature (5)