

BACTERIAL VAGINOSIS AND PRETERM PREMATURE RUPTURE OF MEMBRANES

Thesis

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INTRODUCTION

Bacterial! vaginosis:

Bacterial vaginosis (B.V) is a polymicrobial disorder characterized by increase in vaginal pH over 4.5 a reduction in or absence of lactobacillus colonization and overgrowth of several facultative and obligatory anaerobic bacteria (*Hay et al, 1992 and Guise et al., 2001*).

Bacterial vaginosis is diagnosed in up 23% of pregnant women (*Gallagher et al., 2004*), Although BV itself is harmless condition it is linked to pregnancy complication including miscarriage. Preterm delivery and pelvic infection after child birth, uterine infection is a common trigger of preterm labor BV may lead to this type of infection or could be a red flag for another problem causing preterm labor. Experts continue research whether BV is a direct or indirect causes (*Gattagher, 2004*).

No single microorganism is detected in all women with B.V. though Gardnerella Vaginalis (G.V) Bacteroides species Mycoplasma hominis (M. hominis) and with B.V. in most studies. The concept the a symbiotic relationship between G.V. and anaerobic bacterial flora changes in the vagina is responsible for the condition has been postulated (*Spiegel et al, 1980, Hiller et al., 2004*).

Bacterial vaginosis is the commonest cause of vaginal discharge occurring in women attending Obstetrics and Gynecological clinics (*Guise et al, 2001 & Hillier et al., 1995*). It is

responsible for approximately one third of all cases of vulvovaginitis in women during reproductive age (*Amsel et al, 1983*). Patients always present with malodorous vaginal discharge, although 30-40% may be asymptomatic (*Hay et al., 1992*).

Preterm premature rupture of membranes:

Preterm premature rupture of membranes is the rupture of membranes during pregnancy before 37 weeks gestation. It occurs in 3 percent of pregnancies and is the cause of approximately one third of preterm deliveries (*Tanya et al 2006*). It can lead to significant perinatal morbidity, including respiratory distress syndrome, neonatal sepsis, umbilical cord prolapse, placental abruption, and fetal death (*Tanya et al 2006*)

The etiology of term PROM remains unclear and likely involves a final common pathway for several related intrinsic and /or extrinsic processes. However, studies comparing the tensile strength of membranes from patients with term PROM to membrane from patients show no differences in tensile strengths except in the membranes near the cervix, suggesting an ascending etiology (*Artal et al 1976*).

AIM OF THE WORK

Study the relationship between Bacterial vaginosis and preterm premature rupture of membranes.

PRETERM PREMATURE RUPTURE OF

MEMBRANE

Introduction:

Allantois:

The allantois arises as a tubular diverticulum of the posterior part of the yolk-sac; when the hind-gut is developed the allantois is carried backward with it and then opens into the cloaca or terminal part of the hind-gut: it grows out into the body-stalk, a mass of mesoderm which lies below and around the tail end of the embryo. The diverticulum is lined by endoderm and covered by mesoderm, and in the latter are carried the allantoic or umbilical vessels (*Gray, 2000*),

It exists merely as a narrow, tubular diverticulum of the hind-gut, and never assumes the form of a vesicle outside the embryo. With the formation of the amnion the embryo is, in most animals, entirely separated from the chorion, and is only again united to it when the allantoic mesoderm spreads over and becomes applied to its inner surface. The human embryo, on the other hand, is never wholly separated from the chorion, its tail end being from the first connected with the chorion by means of a thick band of mesoderm, named the body-stalk (Bauchstiel); into this stalk the tube of the allantois extends (*Gray, 2000*).

The amnion:

The amnion is a membranous sac which surrounds and protects the embryo. In the human embryo the earliest stages of the formation of the amnion have not been observed; in the youngest embryo which has been studied the amnion was already present as a closed sac and appears in the inner cell-mass as a cavity. This cavity is roofed in by a single stratum of flattened, ectodermal cells, the amniotic ectoderm, and its floor consists of the prismatic ectoderm of the embryonic disk—the continuity between the roof and floor being established at the margin of the embryonic disk. Outside the amniotic ectoderm is a thin layer of mesoderm, which is continuous with that of the somatopleure and is connected by the body-stalk with the mesodermal lining of the chorion. (*Gray, 2000*).

When first formed the amnion is in contact with the body of the embryo, but about the fourth or fifth week fluid begins to accumulate within it. This fluid increases in quantity and causes the amnion to expand and ultimately to adhere to the inner surface of the chorion, so that the extra-embryonic part of the coelom is obliterated. The liquor amnii increases in quantity up to the sixth or seventh month of pregnancy, after which it diminishes somewhat; at the end of pregnancy it amounts to about 1 liter. It allows of the free movements of the fetus during the later stages of pregnancy, and also protects it by diminishing the risk of injury from without. It contains less than 2 per cent, of solids, consisting of urea and other extractives, inorganic salts, a small amount of protein, and frequently a trace of sugar. That some of the liquor is swallowed

by the fetus is proved by the fact that epidermal debris and hairs have been found among the contents of the fetal alimentary canal (*Gray, 2000*).

The Decidua:

Before the fertilized ovum reaches the uterus, the mucous membrane of the body of the uterus undergoes important changes and is then known as the decidua. The thickness and vascularity of the mucous membrane are greatly increased; its glands are elongated and open on its free surface by funnel-shaped orifices, while their deeper portions are tortuous and dilated into irregular spaces.

These changes are well advanced by the second month of pregnancy, when the mucous membrane consists of the following strata (1) stratum compactum, next the free surface; in this the uterine glands are only slightly expanded, and are lined by columnar cells; (2) stratum spongiosum, in which the gland tubes are greatly dilated and very tortuous, and are ultimately separated from one another by only a small amount of interglandular tissue, while their lining cells are flattened or cubical; (3) a thin unaltered or boundary layer, next the uterine muscular fibers, containing the deepest parts of the uterine glands, which are not dilated, and are lined with columnar epithelium; it is from this epithelium that the epithelial lining of the uterus is regenerated after pregnancy.

Distinctive names are applied to different portions of the decidua. The part which covers the ovum is named the decidua capsularis; the portion which intervenes between the ovum and the uterine wall is named the decidua basalis or decidua placentalis; it is

here that the placenta, is subsequently developed. The part of the decidua which lines the remainder of the body of the uterus is known as the decidua vera or decidua parietalis. (*Gray, 2000*),

Coincidentally with the growth of the embryo, the decidua capsularis is thinned and extended and the space between it and the decidua vera is gradually obliterated, so that by the third month of pregnancy the two are in contact. By the fifth month of pregnancy the decidua capsularis has practically disappeared, while during the succeeding months the decidua vera also undergoes atrophy, owing to the increased pressure. The glands of the stratum compactum are obliterated, and their epithelium is lost. In the stratum spongiosum the glands are compressed and appear as slit-like fissures, while their epithelium undergoes degeneration. In the unaltered or boundary layer, however, the glandular epithelium retains a columnar or cubical form. (*Gray, 2000*).

The Chorion:

The chorion consists of two layers: an outer formed by the primitive ectoderm or trophoblast, and an inner by the somatic mesoderm; with this latter the amnion is in contact. The trophoblast is made up of an internal layer of cubical or prismatic cells, the cytotrophoblast or layer of Langhans, and an external layer of richly nucleated protoplasm devoid of cell boundaries, the syncytiotrophoblast. It undergoes rapid proliferation and forms numerous processes, the chorionic villi, which invade and destroy the uterine decidua and at the same time absorb from it nutritive materials for the growth of the embryo (*Gray, 2000*).

The chorionic villi are at first small and non-vascular, and consist of trophoblast only, but they increase in size and ramify, while the mesoderm, carrying branches of the umbilical vessels, grows into them, and in this way they are vascularized. Blood is carried to the villi by the branches of the umbilical arteries, and after circulating through the capillaries of the villi, is returned to the embryo by the umbilical veins. Until about the end of the second month of pregnancy the villi cover the entire chorion, and are almost uniform in size but after this they develop unequally. The greater part of the chorion is in contact with the decidua capsularis and over this portion the villi, with their contained vessels, undergo atrophy, so that by the fourth month scarcely a trace of them is left, and hence this part of the chorion becomes smooth, and is named the chorion laeve; as it takes no share in the formation of the placenta, it is also named the non-placental part of the chorion. On the other hand, the villi on that part of the chorion which is in contact with the decidua placentalis increase greatly in size and complexity, and hence this part is named the chorion frondosum (*Gray, 2000*).

Definition of PPRM:

Preterm premature rupture of membranes is the rupture of membranes during pregnancy before 37 weeks' gestation. It occurs in 3 percent of pregnancies and is the cause of approximately one third of preterm deliveries. It can lead to significant perinatal morbidity, including respiratory distress syndrome, neonatal sepsis, umbilical cord prolapse, placental abruption, and fetal death (*Tanaya et al., 2006*)

Premature rupture of membranes (PROM) is the rupture of the fetal membranes before the onset of labor. In most cases, this occurs near term, but when membrane rupture occurs before 37 weeks' gestation, it is known as preterm PROM. Preterm PROM complicates approximately 3 percent of pregnancies and leads to one third of preterm births. It increases the risk of prematurity and leads to a number of other perinatal and neonatal complications, including a 1 to 2 percent risk of fetal death (*Tanaya et al., 2006*).

Preterm prelabour membrane rupture remains a management problem, particularly at very early gestations, yet obstetric and neonatal care can make a difference to outcome. While at early gestations the prognosis is poor, it is not hopeless (*Lanwnt et al., 2003*).

Incidence:

Low birth weight and IUGR are surrogate measures of fetal growth that are determined at delivery. Low birth weight is defined as <2,500 grams, and occurs in about 7% of US births. Intrauterine growth retardation is commonly defined as birth weight less than the 10th percentile for gestational week, using a standard population. Preterm delivery is birth at <37 weeks gestational age, and occurs in approximately 12% of US births (*Windham et al., 2008*).

Preterm premature rupture of membranes (PROM) at 16 through 26 weeks of gestation complicated approximately 1% of pregnancies in the United States and was associated with significant risk of neonatal morbidity and mortality (*Nourxe et al., 1997*).