ASSOCIATION OF BACTERIAL VAGINOSIS IN PRETERM LABOUR -COMPARATIVE STUDY

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CERTIFICATE

This is to certify that the dissertation entitled "**ASSOCIATION OF BACTERIAL VAGINOSIS IN PRETERM LABOUR-COMPARATIVE STUDY**" is the bonafide original work of **Dr. G. SUBATHRA,** under the guidance of **Dr.T. RUKMANI MD., DGO.,** Prof. of Department of Obstetrics and Gynaecology SMC, Chennai in partial fulfillment of the requirements for MD (Obs and Gynae) Branch II Examination of the Tamil Nadu Dr. M.G.R Medical university to be held in March 2010. The period of postgraduate study and training was from May 2007 to Feb 2010.

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INTRODUCTION

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Preterm birth is a condition that occurs in 6-15% of all deliveries and is the most frequent cause of fetal and neonatal death and morbidity. Preterm birth is more common in African - American than in Caucasian women (Paul et al 2008). Preterm birth is defined as birth before 37 weeks of gestation.

The incidence of Preterm births ranges from 10%-15%. The incidence of perinatal mortality rate in India varies from 40 to 150 per 1000 births in contrast to 10-20 in the developed countries.

The importance of preterm labour lies in the fact that 75% of all perinatal deaths occurs in preterm infants, and when lethal anomalies are excluded 85% of all neonatal deaths occur in preterm infants.

The molecular basis of initiation of labour is unclear but a number of theories have been proposed. Of these, progesterone withdrawal, oxytocin stimulation and premature decidual activation are the important ones. Regardless of the stimulus, the final pathway seems to converge towards a central role of inflammatory mediators, the cytokines.

Infection induces an intraamniotic inflammatory response involving the activation of a number of cytokines and chemokines which in turn

trigger preterm contractions, cervical ripening and rupture of membranes. Leucocytes are recruited in response to the infection and undergo activation. Intrauterine bleeding is also thought to be an important trigger for cytokine release. The concentration of cytokines in amniotic fluid and cervicovaginal secretions are significantly higher in patients with preterm labour with evidence of infection.

Evidence suggests that women with documented intra amniotic infection are often refractory to tocolytics. Preterm babies exposed to cytokines in utero are also likely to have lung and brain damage causing bronchopulmonary dysplasia, periventricular leucomalacia and cerebral palsy.

Normal genital tract flora are dominated by lactobacillus which produce lactic acid keeping the vaginal pH below 4.5 and discouraging the growth of other organisms. During pregnancy, the concentration of lactobacillus species increases 10 fold as pregnancy progresses. Increased levels of lactobacilli make the vaginal ecosystem inhibitory to the growth of many pathogens. In Bacterial vaginosis, lactobacilli are altered resulting in 1000 fold increase in anaerobes, mobiluncus species and genital mycoplasmas.

Anaerobes produce the ketoacid succcinate which synergistically promotes the growth of other organisms. In addition, the chemotactic response of polymorphonuclear leucocytes and killing ability are reduced.

Bacterial vaginosis has been suggested as a causative agent in upto 50% cases of idiopathic preterm labour (Subtil et al, 2002). Hay et al., (1994) suggested that odds ratio for preterm labour in presence of bacterial vaginosis detected antenatally was 2.8. If bacterial vaginosis was detected before 16 weeks the risk of preterm birth and late miscarriage was increased with odds ratio 5.5.Bacterial vaginosis is a risk factor for preterm delivery and maternal infectious morbidity (Leitich et al, 2007).

This effect was independent of other recognized risk factors such as previous preterm birth. Gravett et al, 1986 showed that bacterial vaginosis was significantly associated with preterm labour.

The triggers for the change in vaginal flora are currently unidentified. Many factors have been related to changes in vaginal flora including menstrual cycles, socioeconomic factors, concomitant infections, sexual activity, number of sexual partners, genital hygiene, contraceptive methods, immunologic status, use of antibiotics, genital pathology.

However most patients with Bacterial Vaginosis have none of these factors that would explain the change in flora. Hormonal factors and exogenous factors (Sobal, 1989) may play a role because it is a condition that affects mainly in women of reproductive age.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Bacterial vaginosis is a vaginal syndrome associated with alteration of normal vaginal flora rather than an infection specific to any one organism (Preistly et al, 1996). Abnormal vaginal bacterial flora is an important cause of obstetric and gynaecological adverse sequelae (Sheehan et al,1996). In obstetrics, Bacterial vaginosis and its related organisms have been implicated in higher rates of late miscarriage, preterm premature rupture of membranes, chorioamnionitis, spontaneous preterm labour, preterm births, postpartum endometritis (yudin et al,2005).

The prevalence of bacterial vaginosis ranges from 4 - 64% depending on the racial, geographic and clinical characteristics of the study population. In asymptomatic women, the prevalence varies from 12 - 25% and similar percentages are observed in pregnant women. (Gauschino et al,2006).

NOMENCLATURE:

The term ‘bacterial vaginosis’ has evolved over more than a century. The discovery of lactobacillus species in vaginal secretions by Albert Doderlein in 1892 marked the beginning of extensive research into the detailed composition of the vaginal flora.

Following his findings, normal vaginal flora was regarded as homogenous, consisting only of gram positive rods, mainly of the lactobacillus species. Any individual with a more heterogenous pattern was regarded as unhealthy and women with this pattern were described as having an infection known then as non-specific vaginitis.

In 1954, Gardner and Dukes discovered Hemophilus vaginalis and was thought to be the sole organism responsible for non-specific vaginitis.

As identification techniques have improved with time, this organism was then categorized into the genus Corynebacterium and then became known as Corynebacterium vaginalis.

Further identification revealed this to be a new genus and, in honour of the work carried out by gardner in this field, it was renamed Gardnerella vaginalis and the condition became known as Gardnerella vaginitis.

In the early 1980's various anaerobic bacteria were implicated in causing the characteristic fishy malodour produced by volatile amines in vaginal secretions. This led to the term "anaerobic vaginosis" being adopted until, in 1984, the term bacterial vaginosis was adopted to reflect the polymicrobial alteration in vaginal flora causing an increase in vaginal pH,

sometimes associated with a homogenous discharge, but in the absence of a demonstrable inflammatory responses.

EPIDEMIOLOGY AND RISK FACTORS:

Bacterial vaginosis is the commonest cause of abnormal vaginal discharge in young women of reproductive age. The trigger for the change from lactobacilli dominated flora to bacterial vaginosis associated flora has been linked to many possible factors including age at first sexual intercourse, change in sexual partners, and concurrent sexually transmitted diseases. Cigarette smoking and use of intra-uterine contraceptive device are both linked to an increased rate of acquiring bacterial vaginosis.

Vaginal douching has also been implicated as a risk factor for bacterial vaginosis, by aiding the ascent of microorganisms into the upper genital tract. There is also evidence of racial disparity of bacterial vaginosis, which is seen to occur more frequently in women of Afro-Caribbean origin living in the UK compared to Caucasian women (Holzmann et al,2008).

Other research has also shown that black women have a higher prevalence of bacterial vaginosis compared to white women (Paige et al, 1998). Royce et al., found differences in vaginal flora between black women and white women in pregnancy. They demonstrated a 2.6 fold increase in bacterial vaginosis in over 300 black women in their third trimester of

pregnancy compared to a similar number of white women even after adjustment for confounding factors.

Presence of symptomatic bacterial vaginosis amplifies the risk of spontaneous preterm birth in those with a “susceptible” tumour necrosis factor genotypes (Macones et al., 2004)

ETIOLOGY:

Even after many years of research the underlying pathogenesis of bacterial vaginosis is still unknown.

In studies where a relationship between the menstrual and bacterial vaginosis could be demonstrated, change in microbial flora were found to occur more often during follicular phase of cycle at a time when estrogen concentration was relatively high compared to progesterone. Relative estrogen dominance favour candida colonisation of the vagina and infection which has led to the speculation that sex hormones whether by virtue of absolute levels (or) by change of relative concentration, may influence the development of bacterial vaginosis.

The prevalence of bacterial vaginosis decreases as the pregnancy progresses and although concentration of estrogen is elevated throughout,

the relative concentrations of estrogens and progesterone alter as pregnancy progresses.

Evidence against sexual transmission comes from studies that have demonstrated no benefit in treating male partners and the detection of bacterial vaginosis in 12% of girls who are virgo intacta post-menarche.

Other theories propose an enzymatic role in the pathogenesis of bacterial vaginosis. Mucinase and sialidase levels measured in samples of vaginal fluid in women with bacterial vaginosis were found to be significantly elevated compared to women with normal vaginal flora.

More recently, there has been evidence to suggest a role of phage viruses in the etiology of bacterial vaginosis. Phages that affect lactobacilli strains have been found in dairy products & in meat processing factories, where in lactobacilli starter cultures used in meat processing, their action is known to delay acid production and reduce lactobacilli numbers, slowing down ripening. This process, if applied to vaginal lactobacilli could result in inhibition of acid production and decreased numbers of these bacteria and an overgrowth of anaerobes.

In an analysis of products containing lactobacillus strains such as yoghurts, Tao et al., found 43 different types of lactobacilli. Phages were

detected in 11 of these strains, 7 of which were found to inhibit vaginal lactobacilli.

MICROBIOLOGY:

Bacterial vaginosis associated microorganisms are Mobiluncus, Prevotella, Peptostreptococcus species, Porphyromonas asaccharolytica, Fusobacterium nucleatum, Mycoplasma hominis and high numbers of Gardnerella vaginalis (Holst et al., 1994).

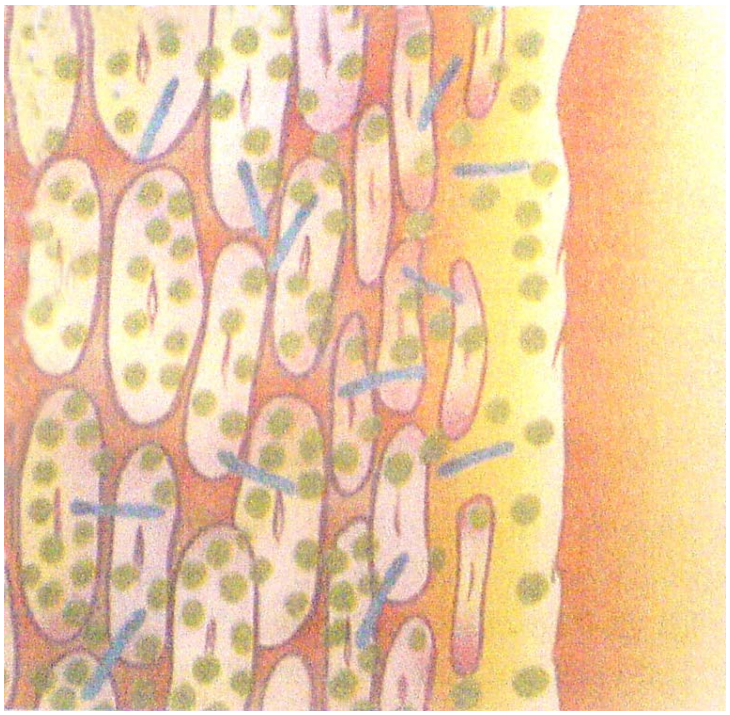
The normal vaginal flora is dominated by lactobacillus species which play a major part in maintaining the dynamic ecosystem in vagina. By metabolizing glycogen in vagina, Lactobacilli produce lactic acid which lowers vaginal pH to below 4.5. This creates a hostile environment which deters the growth of potentially pathogenic bacteria, particularly Gardnerella vaginalis and anaerobes. The low pH generated by the production of lactic acid also reduces the adherence of bacteria to the vaginal epithelium. Other compounds produced by lactobacilli such as lactacin B, acidolin and hydrogen peroxide inhibit the growth of other bacteria. Certain lactobacilli are capable of producing hydrogen peroxide and have been shown to reduce bacterial vaginosis and trichomoniasis and have a bactericidal effect on Gardnerella vaginalis and Prevotella bivia in vitro.

Lactobacillus crispatus and Lactobacillus jensennii both produce hydrogen peroxide and presence of increased number of these species is linked to a lower prevalence of bacterial vaginosis. Mobiluncus species and Bacteroides species produce the ketoacid succinate, as a major biochemical metabolite and this is found in elevated concentrations in women with bacterial vaginosis. The absence of lactic acid and production of succinate is found in women with bacterial vaginosis. A high vaginal interleukin-8 concentration, abnormal vaginal gram stain, absent hydrogen peroxide-producing lactobacillus, and anaerobic vaginal flora were strongly associated with preterm labour (Eschenbach et al., 2001).

NORMAL VAGINAL SMEAR WITH LACTO BACILLI

It raises the pH and blunt the chemotactic response of polymorphonuclear leucocytes thereby reducing their killing ability. This may explain why bacterial vaginosis produces no cellular inflammatory response despite the presence of high number of potentially pathogenic micro-organisms.

CLUE CELL PREDOMINATES



Gram negative anaerobic bacteria such as Bacteroides species, Prevotella species and Porphyromonas species can be found in over 50% of healthy women. However, these obligate anaerobes are also important pathogens and are strongly linked to bacterial vaginosis.

Common yeasts such as Candida albicans may also be seen in healthy women. Genital mycoplasmas such as Mycoplasma hominis and Ureaplasma urealyticum can be a part of the flora in healthy women, but may also be involved in genital tract infections.

Potential pathogens such as klebseilla species, staphylococcus aureus and Escherichia coli can also be present. The presence of mobiluncus species, an anaerobic fastidious curved rods, has been shown to be highly specific for bacterial vaginosis.

While 5-15 species of bacteria may be cultured from normal vaginal secretions, the total bacterial count of normal flora is $<10^6$ organisms/ml, whereas women with bacterial vaginosis have upto 10^9 organism /ml. The normal microbial balance is upset when there is a dramatic rise in anaerobes and other organisms and a decrease in lactobacilli resulting in the polymicrobial pattern, characteristic of bacterial vaginosis.

Anaerobic bacterial number increases 1000 fold and bacterial vaginosis related organisms such as Gardnerella vaginalis, Bacteroides species, Mobiluncus species, and Mycoplasma hominis dominate the flora with a reduction in the quantity and quality of lactobacilli. Gardnerella vaginalis and other anaerobes are capable of producing volatile amines and

organic acids other than lactic acid, which may be responsible for the fishy odour of bacterial vaginosis.

DIAGNOSIS:

Composite clinical criteria (Amsel, 1983)

1) Homogenous vaginal discharge.
2) Elevated vaginal pH > 4.5
3) Positive "whiff" Test on addition of 10% potassium hydroxide to sample of vaginal secretions.
4) Presence of "clue cells" on microscopic examination of a wet preparation of vaginal secretions.

Atleast three of the four criteria is regarded as diagnostic of bacterial vaginosis.

pH:

Vaginal pH is measured using narrow range pH paper and assessing the colour change produced by a sample of vaginal secretion taken from posterior fornix. A low pH virtually excludes bacterial vaginosis. An elevated pH is the most sensitive, but least specific of the criteria used for the diagnosis of bacterial vaginosis, as an increase can also be associated with menstruation, recent sexual intercourse (or) infection with Trichomonas vaginalis.

ODOUR:

Whiff test involves addition of a drop of 10% potassium hydroxide to a sample of vaginal secretions. It produces a characteristic fishy odour. Pheifer et al., were the first to report the presence of fishy odour in bacterial vaginosis. It has been demonstrated that some bacterial vaginosis microorganisms such as mobiluncus species produce trimethylamine, a substance linked to the smell of rotten fish which explains the malodour. Whiff test has a positive predictive value of 90% and specificity of 70%.

CLUE CELLS:

Clue cells are desquamated vaginal epithelial cells that are densely coated in adherent bacteria such that their borders are indistinct. The detection of clue cells on direct microscopy is the single most sensitive and specific criterion for bacterial vaginosis but is operator dependent.

Debris and degenerated cells may be mistaken for clue cells and lactobacilli may adhere to epithelial cells in low number. Clue cells can be identified on gram stain or wet preparation (small sample of vaginal secretions to which a drop of saline has been added) and are regarded as pathognomonic of bacterial vaginosis.

The most objective way of identifying the clue cell is to observe the cell borders. If the vaginal cell border has a serrated appearance and cannot

be identified clearly because of the attachment of large number of bacteria, a clue cell is present. The appearance of a dirty, hazy or cloudy interior of the epithelial cell is more subjective in identifying a clue cell than utilizing the cell border.

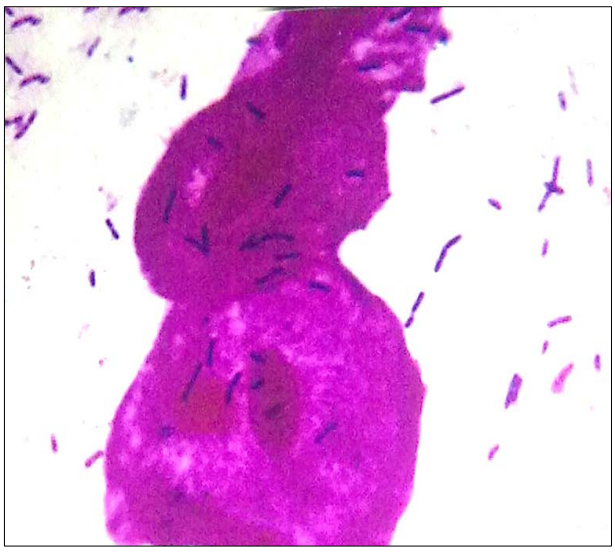
Atleast 20% of epithelial cells should be having the appearance of clue cells in a wet mount of vaginal fluid (Eschenbach et al., 1998) to diagnose bacterial vaginosis.

GRAM STAIN:

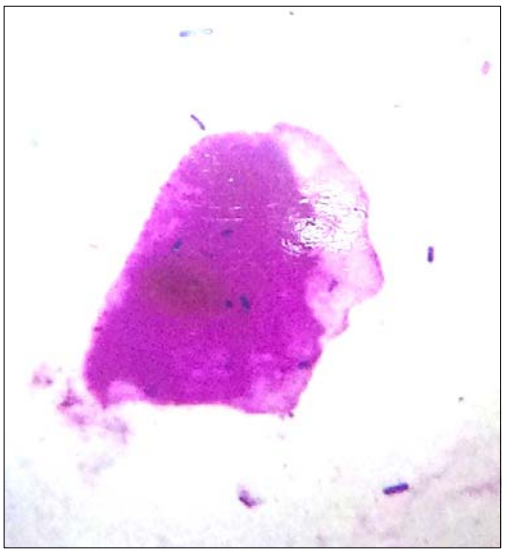
Gram stain of the vaginal fluid has been used for confirmation of bacterial vaginosis since 1965. Dunkelberg reported that all 132 women having clinical signs of bacterial vaginosis and clue cells on wet mount examination had gram stain consistent with bacterial vaginosis. (Kurki et al., 1992).

The advantages of the gram stain are that it provides a more objective method of diagnosis. The slide can also be stored for future reference and patient collected ‘blind’ vaginal swabs have been demonstrated to be as separate as swabs taken using a speculum.

NORMAL VAGINAL SMEAR WITH PREDOMINANT LACTOBACILLI



VAGINAL SMEAR IN BACTERIAL VAGINOSIS WITH FEW LACTOBACILLI



SPIEGEL ET AL., 1983 CRITERIA:

Less than one organism per field	1+
1-5 organisms per field	2+
5-30 organisms per field	3+
>30 organisms per field	4+

Presence of large number of Gram positive lactobacilli morphology alone (or) greatly exceeding other morphological types is labelled as negative Gram stain for bacterial vaginosis. When lactobacilli morphologial types are present at <2+ levels and there are 3+ to 4+ levels of mixed flora including Gardnerella cocci, rods, fusiform bacteria (or) curved rods, slides are interpreted as positive for bacterial vaginosis.

HAY / ISON CRITERIA (2002):

Grade 1 (Normal): Lactobacillus morphotypes predominate

Grade 2 (intermediate): Mixed flora with some lactobacilli present, but Gardnerella (or) Mobiluncus morphotypes also present.

Grade 3 (Bacterial vaginosis): Predominantly gardenerella and / or mobiluncus morphotypes. Few (or) absent Lactobacilli (Hay et al., 1994)

NUGENT CRITERIA (1991):

In this scale, a score of 0-10 is generated from combining three other scores. It is time consuming and requires trained staff but it has high inter observer reliability.

0-3 - negative for bacterial vaginosis

4-6 - intermediate

7+ - indicative of Bacterial vaginosis.

Atleast 10-20 high power (1000 x oil immersion) fields are counted and an average determined.

Lactobacillus morphotypes : Average per high powered (1000xoil immersion) field.View multiple fields	Gardnerella / Bacteroides morphotypes : Average per high powered (1000xoil immersion) field. View multiple fields	Curved gram variable morphotypes : Average per high powered (1000xoil immersion) field view multiple fields (note that this factor is less important – scores of only 0-2 are possible)
Score 0 for >30 Score 1 for 15-30 Score 2 for 1-14 Score 3 for <1 (this is an average, so results can be >0, yet <1) Score 4 for 0	Score 0 for 0 Score 1 for <1 (this is an average, so results can be 0, yet <1) Score 2 for 1-4 Score 3 for 5-30 Score 4 for >30	Score 0 for 0 Score 1 for <5 Score 2 for 5+

CRITERIA OF THOMSON ET AL., (1990)

1) Thin homogenous vaginal discharge
2) Vaginal pH > 4.5
3) Presence of clue cells
4) Presence of fishy odour with 10% potassium hydroxide.
5) Non- Lactobacilli morphological types greater than lactobacilli morphological types in wet mount examination.

Bacterial vaginosis present if four of the five criteria were met. Special diagnostic method for bacterial vaginosis.

1) Papanicolaou smear:

Clue cells and changes in bacterial flora can be found in the papanicolaou smear (Kurki et al., 1992), which normally would be incidental finding and has limited diagnostic potential in comparison with other methods. Studies have been shown to have a sensitivity of 90% and a specificity of 97%. Schnading et al., reported excellent correlation between pap smear and gram stain smear for diagnosis of bacterial vaginosis.

2) Culture:

Culture generally plays no role in the diagnosis because the isolation of Gardnerella vaginalis and / or anaerobic bacteria from the vagina does not

define the clinical entity and can be observed in women without bacterial vaginosis.

3) Affirm VPIII Test :

A recent study compared the gram stain using the Nugent criteria and the DNA hybridization test Affirm VPIII in diagnosing bacterial vaginosis. The Affirm VPIII test detected Gardnerella in 107 (93.0%) of 115 vaginal specimens positive for bacterial vaginosis diagnosed by gram stain. The Affirm VPIII test has a sensitivity of 87.7% and specificity of 96% and may be used for the rapid diagnosis of bacterial vaginosis in symptomatic women.

Early screening for bacterial vaginosis in pregnant women who experienced a preterm delivery may help in predicting the risk of adverse outcome (Guerra et al., 2006).

OBSTETRIC COMPLICATIONS ASSOCIATED WITH BACTERIAL VAGINOSIS:

1) Spontaneous preterm labour and preterm births:-

In the industrialized world, preterm labour accounts for 8-10% of all births and is the major cause of perinatal morbidity, mortality and subsequent neuro developmental problems such as cerebal palsy.Bacterial

vaginosis has a significant association with preterm labour and adverse pregnancy outcome (Goval et al., 2004).

The mechanism by which bacterial vaginosis can induce preterm labour is linked to the ascending genital tract infection, with an immune response resulting in the production of pro-inflammatory cytokines such as interleukin-1, interleukin 1β and tumour necrosis factor alpha. Cytokines are the products secreted during inflammatory processes with an immunological basis and play a role in intercellular signaling. They are present during the process of normal labour, but higher concentrations have been found in the amniotic fluid of women in spontaneous preterm labour due to infection, which sets off a cascade resulting in the recruitment of inflammatory mediators such as prostaglandins. This eventually leads to cervical ripening and uterine contractions that may result in preterm labour. Phospholipase A2 and phospholipase C are enzymes responsible for cleaving arachidonic acid, the obligate precursor for prostaglandin synthesis, from glycerophospholipids in the cell membrane and have been found to be elevated in the lower genital tract of women with bacterial vaginosis.

If there is a cytokine mediated / immune response related neuro developmental disturbance, there is high incidence of cerebral palsy in babies delivered by women with perinatal infection.

Approximately 15-20% of all pregnant women will have bacterial vaginosis and these women are upto 4 times more likey to have a preterm labour than without bacterial vaginosis. In a longitudinal study, Hillier et al., 1995 demonstrated that women with Bacterial vaginosis are 40% more likely to deliver a preterm, low birth weight infant than women without bacterial vaginosis. The presence of bacterial vaginosis at an early gestational age is associated with preterm delivery (DeSeta et al., 2005).

In a longitudinal study by Hay et al., the gram stained vaginal smears of 718 pregnant women were examined for bacterial vaginosis until 36 weeks gestation. The results showed that of those women who initially had normal flora at their first antenatal visit, only 2.4% had developed bacterial vaginosis by 36 weeks gestation. Of 32 women who had bacterial vaginosis initially, half had abnormal vaginal flora by 36 wks.

Women who are diagnosed with bacterial vaginosis before 20 weeks of gestation were at higher risk of delivering preterm than those who developed bacterial vaginosis after 20 weeks (Schoeman et al., 2005).

2) Late Miscarriage:

The incidence of late miscarriage (13-23 weeks gestation) has been demonstrated to be significantly higher in women who have bacterial vaginosis than those who do not have bacterial vaginosis (Hay et al., 1994).

3) Postpartum Endometritis:

Bacterial vaginosis is also found to be associated with endometritis (Romanik et al., 2005).

Postpartum endometritis is a relatively common obstetric complication and, although the incidence is higher in women undergoing caesarean section, it may also occurs following a vaginal delivery (Zana et al., 1993).Risk factors include prolonged rupture of membranes, prolonged labour and increased number of vaginal examinations. Postpartum endometritis following a caesarean section tends to develop within 2 days and is described as early endometritis. This is most likely to be due to the introduction of bacteria into the endometrial cavity at delivery.

Women who have a vaginal delivery usually develop late endometritis, which can occur upto 6 weeks postnatal. This delayed infection tends to result from ascending infection over a course of time. Facultative anaerobes linked to bacterial vaginosis are commonly isolated in cases of endometritis.

One study demonstrated that, in women diagnosed with early endometritis, the majority were found to have bacteria such as Peptostreptococcus species and Gardnerella vaginalis as predominant isolates. In a study which examined the rate of postpartum endometritis in

women delivered by caesarean section, those women with bacterial vaginosis were nearly 6 times more likely to develop the condition than women without bacterial vaginosis despite antibiotic prophylaxis (John studd 15).

GYNAECOLOGICAL COMPLICATIONS ASSOCIATED WITH BACTERIAL VAGINOSIS:

1) BACTERIAL VAGINOSIS AND CERVICAL INTRAEPITHELIAL NEOPLASIA:

A possible association between bacterial vaginosis and cervical intraepithelial neoplasia has been explored by various studies over many years. Platz-Christensen et al., in a large retrospective study, found a relative risk of 5.0 (95% CI 2.2-11.6)of having CIN-3/Carcinoma in situ in women with bacterial vaginosis when compared to women without bacterial vaginosis.The main criticism of previous studies investigating the possible role of bacterial vaginosis in the etiology of CIN is failure to control for sexually transmitted infections, particularly oncogenic human papilloma virus infections - a known risk factor for cervical neoplasia.

It has been suggested that some vaginal flora, such as the anaerobes associated with bacterial vaginosis, are capable of producing carcinogenic substances called nitrosamines. The mechanism of action by which nitrosamines may act has also been ill defined although thought to be by

exerting an influence via enhanced replication of oncogenic human papilloma virus.

In the only study to date which examined the relationship between bacterial vaginosis and CIN whilst adequately controlling for HPV and other sexually transmitted diseases, bacterial vaginosis has not been shown to be associated with higher rates of CIN and nor has it been demonstrated to produce higher levels of nitrosamines in the vagina of women with the condition than in women who do not have bacterial vaginosis (Boyle DCM et al., in press).

2) PELVIC INFLAMMATORY DISEASES(PID):

Acute PID remains a serious cause of morbidity in young women and is usually polymicrobial in nature. The sequelae of PID such as tubal damage leading to infertility and ectopic pregnancy create further problems for both patient and clinician. In the past, PID was commonly caused by Chlamydia trachomatis or Nisseria gonorrhoea, but current attention is focused on the effects of bacterial vaginosis related microorganisms (Eschenbach et al., 1993).

Cervico vaginal fluids can be sucked through the cervix into the uterus and beyond during spontaneous hormone mediated uterine

contractions at midcycle. This may explain the finding of bacterial vaginosis related microrganisms in the endometrium of women with PID. These organisms tend to be found less frequently in the fallopian tubes and, occasionally, within the abscesses associated with PID. Due to the polymicrobial nature of bacterial vaginosis, it is difficult to attribute bacterial vaginosis associated PID to any one organism.

3) INFERTILITY AND FIRST TRIMESTER LOSS:

Several studies have examined the possible relationship between bacterial vaginosis and infertility. A recent UK study found that there was a high prevalence of bacterial vaginosis in women undergoing invitro fertilization and that women with bacterial vaginosis had a higher rates of first trimester miscarriage than those with normal vaginal flora.

4) POSTHYSTERECTOMY VAGINAL CUFF INFECTION:

In two separate studies, post abdominal hysterectomy vaginal cuff infection occured 3 - 4 times more commonly in women with bacterial vaginosis than in those without. Neither study group received antibiotics prior to the hysterectomy, but screening for bacterial vaginosis took place beforehand.

In one of the studies by Larsson et al., the presence of clue cells on air-dried smears of vaginal secretions was used as a diagnostic basis for

bacterial vaginosis. Of women who had clue cells, 35% developed postoperative infection. Only 8% of women without clue cells developed an infection.

The second study involved the analysis of vaginal secretions prior to surgery in order to detect bacterial vaginosis and trichomoniasis. The patients who had these two conditions diagnosed were 3 times more likely to develop postoperative vaginal cuff cellulitis compared to the control group. This suggest that there is a place for the preoperative assessment of vaginal flora and administration of appropriate treatment is necessary (Koumann et al., 2001).

5) POSTABORTAL SEPSIS:

Postoperative infections such as endometritis occur at rates between 4-12%. Pelvic infection following termination of pregnancy may be due to vaginal infections particularly with Nisseria gonorrhoea, Chlamydia trachomatis and bacterial vaginosis related organisms.

In one study, a 2.4 fold increased risk of postabortal infection was reported if women had clue cells in their vaginal secretions compared to women with normal vaginal flora.

In a later study, the same authors reported a 3.8 fold reduction in the risk of postabortal infection in women with bacterial vaginosis who were

randomized to receive treatment with metronidazole compared to the group who received no treatment.

One double blind trial recently was carried in over 1000 women undergoing termination of pregnancy randomized to receive 2% clindamycin cream or placebo prior to procedure. Their results showed that preoperative treatment with clindamycin cream significantly reduced the risk of postabortal infections (RR 4.2, 95%CI 1.2 - 15.9) among women with bacterial vaginosis but less so in women who had normal preoperative flora.The use of antibiotic prophylaxis before surgical termination of pregnancy demonstrates a protective effect (Koumann et al., 2001).

6) URETHRAL SYNDROME:

Urethral syndrome can be defined as dysuria in women that cannot be explained by the bacteria that normally cause urinary tract infection. Chlamydia trachomatis has been implicated in some cases. The role of lactobacilli in maintaining urinary tract health is disputed, but speculation has arisen that if lactobacilli are important in the prevention of urethral syndrome, and bacterial vaginosis is associated with a reduction or absence of such bacteria, then bacterial vaginosis may be implicated in the etiology of urethral syndrome. This theory needs further investigation.

7) BACTERIAL VAGINOSIS AND SEXUAL ACQUISITION OF HUMAN IMMUNODEFICIENCY VIRUS:

Klebanoff et al., showed that the presence of hydrogen peroxide producing lactobacilli in the vagina results in a more acidic environment which is not only toxic to bacterial vaginosis associated flora but also to HIV. They postulated that a lower vaginal pH may, therefore, block the production of CD4 lymphocytes whereas a higher more alkaline pH associated with bacterial vaginosis, may enhance HIV survival.

Cohen et al., demonstrated that bacterial vaginosis is linked to the increased detection of the anti-inflammatory cytokine, IL-10 in the endocervical secretions, which in turn increases macrophage susceptibility to HIV-1 infection. It has been shown that bacterial vaginosis microorganisms, especially Mycoplasma hominis are able to increase the activity of a soluble HIV inducing factor (HIF) and therefore increase HIV -1 expression. Genital tract infection with Gardnerella vaginalis, which is commonly isolated in bacterial vaginosis, has been shown to stimulate HIV -1 production and hence increase the likelihood of sexual transmission.

TREATMENT:

The treatment of bacterial vaginosis significantly reduced the rates of prematurity and other perinatal complications (Camargo et al., 2005).

1. Metronidazole - orally 400-500 mg bd for 5 - 7 days (cure rate 79-100%) and vaginal gel (0.75%) od for 5 days. Occasionally suppository may cause metallic taste in mouth, disulfiram reaction with alcohol, rarely teratogenicity.

   Intravaginal metronidazole gel application was found to be an effective therapeutic option (Mathew et al., 2001).

2. Clindamyin - orally 300 mg bd for 7 days (cure rate- 90%) and vaginal cream (2%) 5g once daily for 7 days. It may cause pseudomembranous colitis.

   Both drugs are secreted in breast milk.

Only one trial using oral clindamycin (300 mg twice daily for 7 days) compared to oral metronidazole (500 mg twice daily for 7 days) has been performed and both drugs showed high efficacy rates of 94% and 96% respectively. Further research into the efficacy of oral clindamycin is needed.

Klebanoff et al., 2008 showed no benefit was found in treating women with low or average risk pregnancies for asymptomatic bacterial vaginosis.

TREATMENT FAILURE:

In treatment failure cases, the vaginal swab to be taken from the lateral walls and should be sent for culture in an anaerobic transport medium. Then treatment of the dominant organism should be aimed. A broad spectrum agent to be chosen whose main strength is directed against the dominant organism. The patients should be reevaluated within two weeks of completing therapy.

RECURRENT BACTERIAL VAGINOSIS:

Some women have frequent episodes of bacterial vaginosis, which are probably new episodes rather than treatment failures.The reason why some women have their relapses is not fully understood because the underlying factors involved in the pathogenesis of bacterial vaginosis are not clear.

One small study involving 30 women with symptomatic recurrent bacterial vaginosis demonstrated the effectiveness of hydrogen peroxide 3% vaginally as an agent in treatment. In 23 women who completed the study, all had a negative amine test at reassessment after 3 weeks, as well as the absence of the mixed anaerobes which were present prior to treatment.

In practice, some approaches to management of recurrent bacterial vaginosis involve repeated antibiotic treatment, occasionally with antifungal agents. The use of lactobacillus strains cultured under laboratory conditions to replace the vaginal flora is still being investigated (Hay et al., 1998).

AIM OF THE STUDY

AIM OF THE STUDY

1. To find out the incidence of bacterial vaginosis in preterm labour.

2. To study the association of bacterial vaginosis with preterm labour.

3. To evaluate and to find out the occurrence of bacterial vaginosis under the influence of the following factors like age, parity, socioeconomic class, weight, previous preterm labour.

4. To evaluate bacterial vaginosis as a risk for preterm labour.

MATERIALS AND METHODS

MATERIALS AND METHODS

This prospective comparative study was carried out in the Department of obstetrics and gynaecology at RSRM Lying in Hospital, Chennai during the period from Dec 2008 to Sep 2009.

INCLUSION CRITERIA:

1) Study group comprises 200 pregnant women with 100 women in preterm labour (ie) gestational age between 28-37 weeks with painful uterine contractions 2 (or) more in 10 minutes lasting for 45 seconds, cervical dilatation less than (or) equal to 3cm and cervical effacement of 75% (or) more but with intact membranes and 100 women of term gestation in labour.
2) Both primigravida and multigravida were included.

EXCLUSION CRITERIA:

Women were excluded from analysis if they had

Fever

Urinary tract infections

Anemia

Diabetes

Multiple pregnancy

Pregnancy induced hypertension

Antepartum hemorrhage

Hydramnios

Cervical incompetence treated with cervical encirclage.

Antibiotic therapy within last 30 days.

Absent Membranes.

CLINICAL STUDY:

A complete history was taken in the menstrual history and obstetric history. The gestational age was confirmed from last menstrual period and was correlated with clinical examination and ultrasonographic gestational age. Any previous history of preterm labour was carefully analysed. Pervaginal and perspeculum examination was done and vaginal swabs were taken for bacteriological study.

BACTERIOLOGICAL STUDY:

Under all aseptic precautions, the posterior vaginal wall was retracted with sims speculcum. The vaginal swabs were taken from posterior fornix by 3 sterile cotton swabs.

1) pH of vaginal discharge was measured by Nitrazine paper. Care was taken to avoid cervical mucus. pH >4.5 is considered alkaline and is suggestive of bacterial vaginosis.

2) Wet mount preparation - vaginal swab is stirred in 0.2ml of physiological saline, the drop of it was put on clean glass slide and examined for the presence of clue cells.
3) Amine Test : A drop of 10% potassium hydroxide was added to wet mount specimen and fishy odour is noted.
4) Gram staining : A direct smear was done on clean glass slide and gram staining was done and smear was examined for the presence of clue cells and gram negative coccobacilli.

Specimen were adequate if atleast 10 epithelial cells per high power field (x400) were seen. The presence of even as few as one clue cell per field in 20 field (X400) was considered positive.

RESULTS AND ANALYSIS

OBSERVATION

Table: 1

Distribution of Study subject according to their age

Age group (in years)	Preterm Group		Term Group	
	No.	Percentage	No.	Percentage
<20 yrs	17	17.00	14	14.0
20-24	60	60.00	63	63.0
25-29	20	20.00	18	18.0
30 and above	3	3.00	5	5.0
Total	100	100	100	100
Mean age	22.6 $\pm$ 3.1 years		22.9 $\pm$ 3.1 years	

Table: 2

Age of preterm		Age of term		Mean difference	t	Degrees of freedom	Significance
Mean	S.D	Mean	S.D				
22.6	3.1	22.9	3.1	0.3	0.810	198	P > 0.05

The above tables compare the study subjects in terms of their age. The mean age of preterm mothers was 22.6 ± 3.1 years and the term mothers was 22.9 ± 3.1 years. The difference between the mean ages of two groups (0.3 years) was not statistically significant (p > 0.05).The percentage distribution of the ages of the two groups were not statistically significant (P>0.05).

FIGURE – 1

DISTRIBUTION OF STUDY SUBJECT ACCORDING TO THEIR AGE

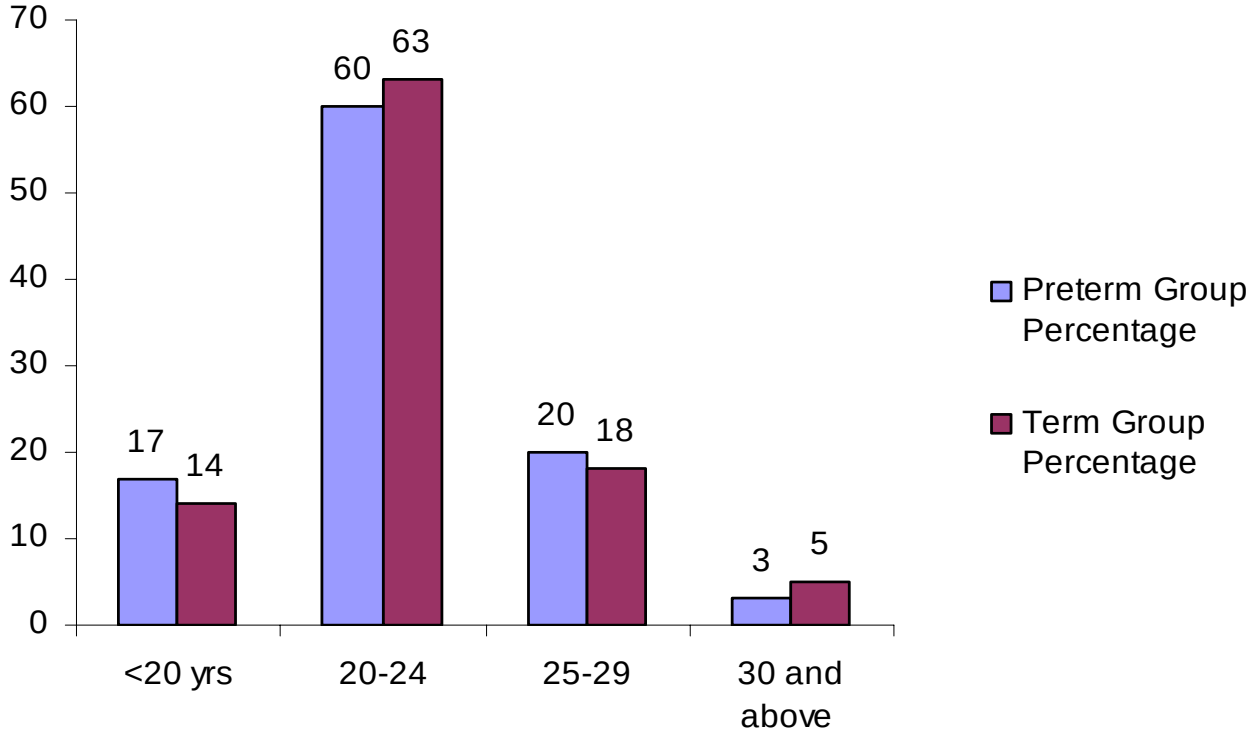


Table: 3

Distribution of Study Subject According to Obstetric History

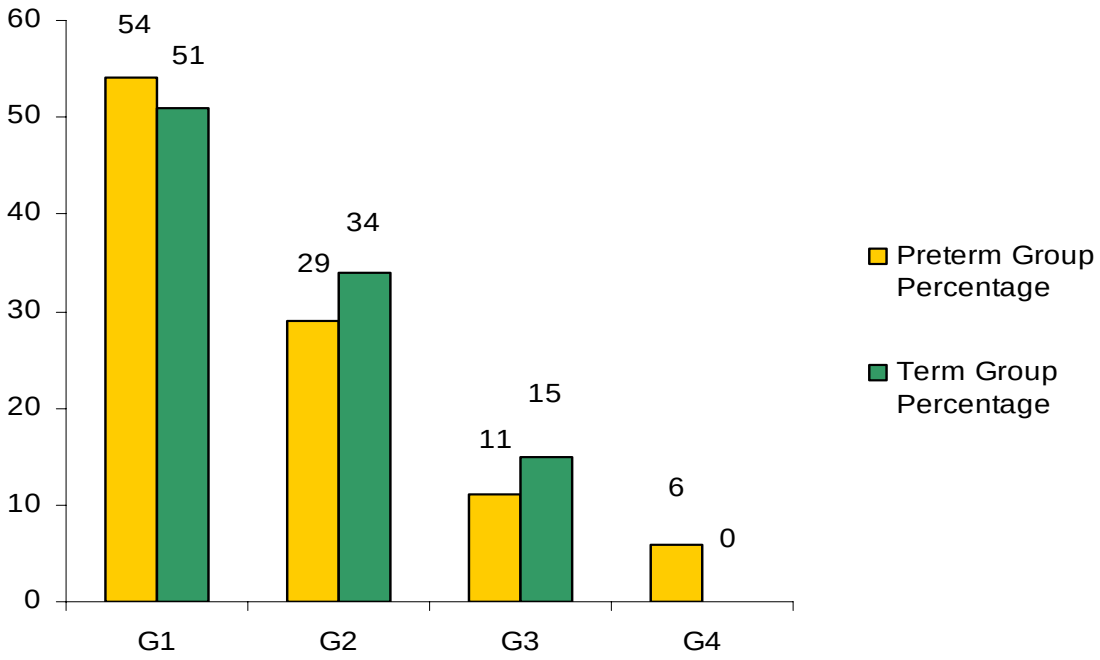
Gravida	Preterm Group		Term Group		Significance
	No.	Percentage	No.	Percentage	
G1	54	54.00	51	51.00	p> 0.05
G2	29	29.00	34	34.00	p> 0.05
G3	11	11.00	15	15.00	p> 0.05
G4	6	6.00	0	0.00	P< 0.05
Total	100	100	100	100	-

The table-3 explains the obstetric history of the study subjects. Primigravidas of both groups were 54% and 51% respectively. The difference was not statistically significant. Similarly, G2 and G3 mothers were also not statistically significant. The G4 mothers of preterm labour was 6%, whereas no mothers were with above category among term mothers.

In respect of age and obstetric history, the two categories of mothers had not been differed much. Hence the two groups of mothers were comparable for studying the prevalence of bacterial vaginosis and its impact among the preterm and term mothers.

FIGURE - 2

DISTRIBUTION OF STUDY SUBJECT ACCORDING TO OBSTETRIC HISTORY



Prevalence of Bacterial Vaginosis:

The prevalence of bacterial vaginosis was analysed and assessed by different methods like pH value of vaginal discharge, amine test, clue cells and gram stain among the preterm and term mothers.

Table: 4

Prevalence of bacterial vaginosis among preterm and Term mothers by different investigations

S.No.	Tests	Preterm Group		Term Group		Significance of preterm with term group
		% in the sample	Estimated proportion in the population at 95% CI	% in the sample	Estimated proportion in the population at 95% CI	
1.	pH value	79.0	71.0 to 87.0	37.0	27.5 to 46.5	P < 0.001
2.	Amine Test	30.0	21.0 to 39.0	9.0	3.4 to 14.6	P < 0.001
3.	Clue cells	31.0	22.0 to 40.0	11.0	5.0 to 17.0	P < 0.001
4	Gram stain	33.0	24.0 to 42.0	13.0	6.4 to 18.6	P < 0.001

From the above table-4, it was inferred that the prevalence of bacterial vaginosis among preterm mothers was statistically greater than the term mothers by all investigations (P<0.001). The estimated proportion in the population at 95% confidence intervals were also statistically & significantly differed in all investigations.

Impact of Bacterial Vaginosis on Preterm Deliveries:

The impact of Bacterial vaginosis was assessed and compared between the preterm and term mothers by different investigations. The association of preterm mothers with bacterial vaginosis was analysed and tabulated as follows.

Table : 5

Comparison of association with bacterial vaginosis among the preterm and term mothers by pH value.

Bacterial Vaginosis By pH value	Preterm Group		Term Group		Total	
	No	%	No	%	No	%
Positive	79	79.00	37	37.00	116	58.00
Negative	21	21.00	63	63.00	84	42.00
Total	100	100.00	100	100.00	200	100.00

Chi square Test γ^2 - 36.2

P value - < 0.001

The table-5 explains the association between preterm mothers with bacterial vaginosis. The association was highly significant (P<0.001).

FIGURE - 3

COMPARISON OF ASSOCIATION WITH BACTERIAL VAGINOSIS AMONG THE PRETERM AND TERM MOTHERS BY PH VALUE.

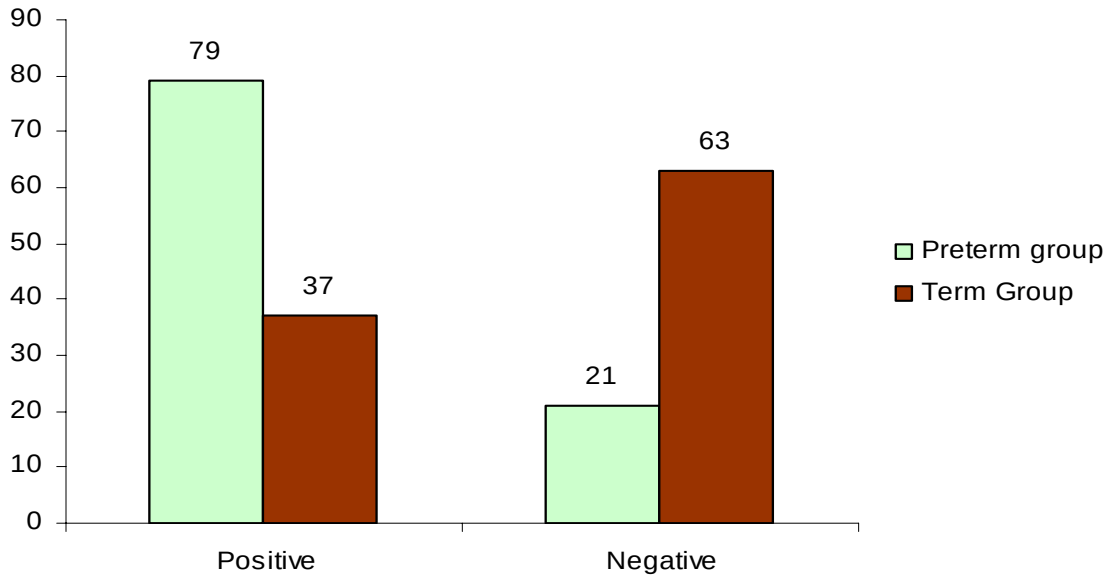


Table : 6

Comparison of Association with Bacterial Vaginosis Among the Preterm and Term Mothers by Amine Test

Bacterial Vaginosis by Amine Test	Preterm Group		Term Group		Total	
	No	%	No	%	No	%
Positive	30	30.00	9	9.00	39	19.50
Negative	70	70.00	91	91.00	161	80.50
Total	100	100.00	100	100.00	200	100.00

Chi square Test γ^2 - 14.047

P value - < 0.001

The table-6 shows that there was a highly significant association between the preterm mothers with bacterial vaginosis (P <0.001).

FIGURE - 4

COMPARISON OF ASSOCIATION WITH BACTERIAL VAGINOSIS AMONG THE PRETERM AND TERM MOTHERS BY AMINE TEST

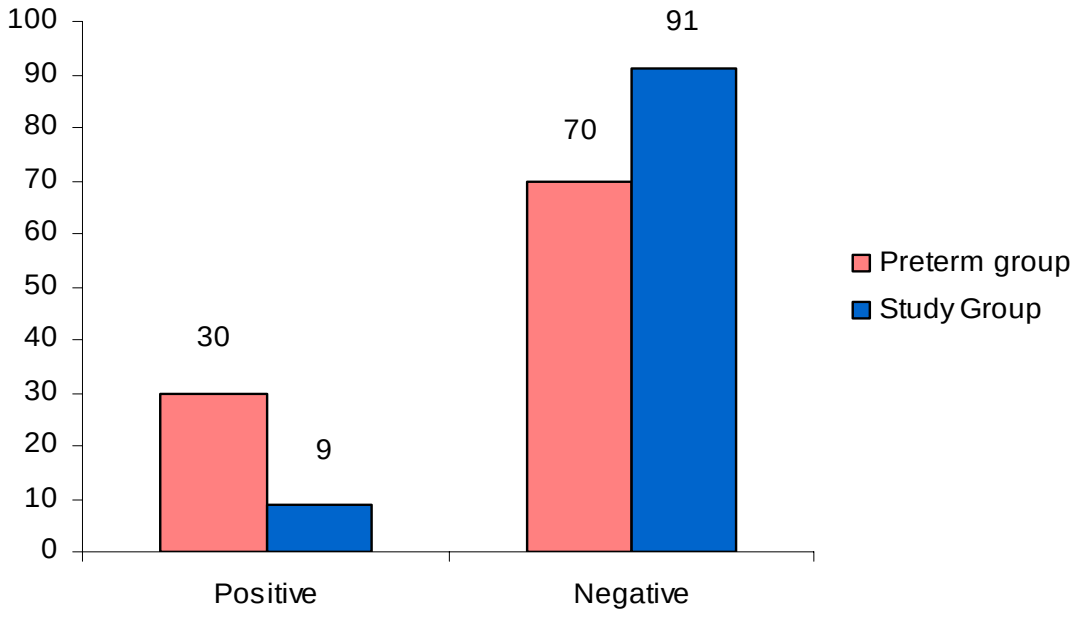


Table : 7

Comparison of Association with Bacterial Vaginosis Among the Preterm and Term Mothers by Clue Cells

Bacterial Vaginosis by Clue cells	Preterm Group		Term Group		Total	
	No	%	No	%	No	%
Positive	31	31.00	11	11.00	42	21.00
Negative	69	69.00	89	89.00	158	79.00
Total	100	100.00	100	100.00	200	100.00

Chi square Test γ^2 - 12.055

P value - < 0.01

The table-7 evaluates the association between the bacterial vaginosis with preterm mothers. It was found to be highly significant (P<0.001).

FIGURE - 5

COMPARISON OF ASSOCIATION WITH BACTERIAL VAGINOSIS AMONG THE PRETERM AND TERM MOTHERS BY CLUE CELLS

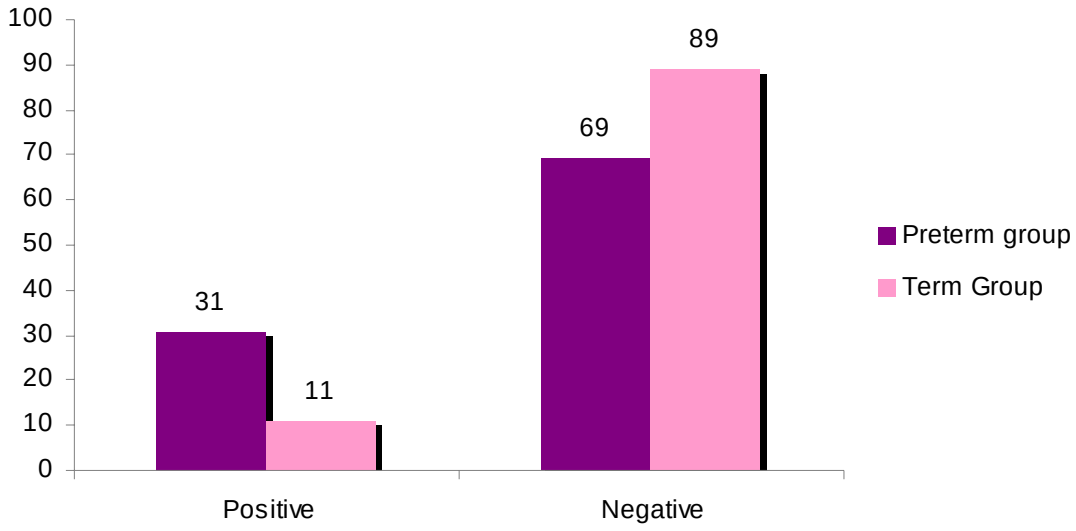


Table : 8

Comparison of Association with Bacterial Vaginosis Among the Preterm and Term Mothers by Gram Stain

Bacterial Vaginosis by Gram Stain	Preterm Group		Term Group		Total	
	No	%	No	%	No	%
Positive	33	33.00	13	13.00	46	23.00
Negative	67	67.00	87	87.00	154	77.00
Total	100	100.00	100	100.00	200	100.00

Chi square Test γ^2 - 11.293

P value - < 0.01

The association illustrated in the above table-8 clearly shows that there was significant positive association between bacterial vaginosis with preterm mothers (P <0.01).

FIGURE - 6

COMPARISON OF ASSOCIATION WITH BACTERIAL VAGINOSIS AMONG THE PRETERM AND TERM MOTHERS BY GRAM STAIN

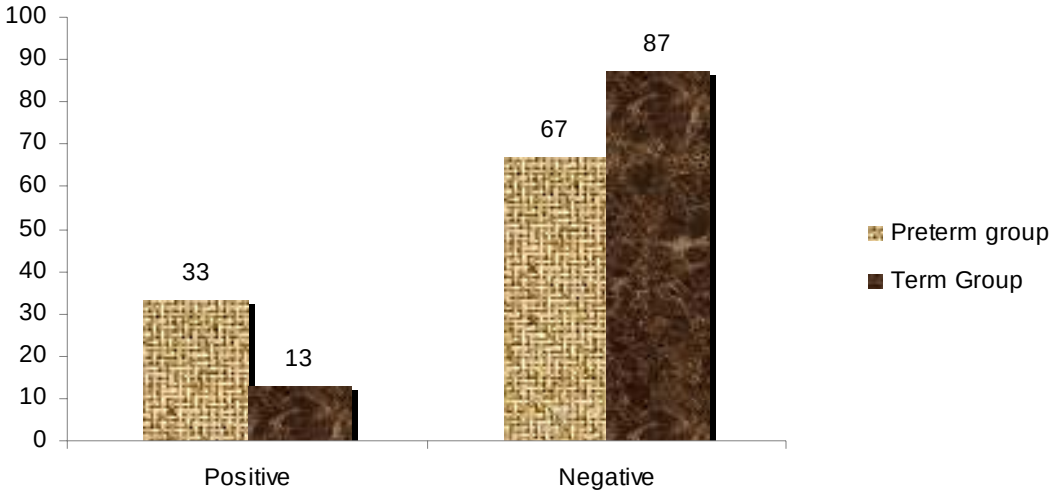


Table : 9

Distribution of Subject Groups According to Past Obstetric History

S.No.	Past obstetric history	Preterm Group	Term Group
1.	Previous Abortion		
	Spontaneous	10	2
	Induced	1	1
2.	Previous still Birth	1	-
3.	Previous preterm delivery	15	1
4.	Neonatal death	2	1

The above table-9 shows there was highly significant association of bacterial vaginosis with previous preterm deliveries ($p<0.001$).

FIGURE - 7

DISTRIBUTION OF SUBJECT GROUPS ACCORDING TO PAST OBSTETRIC HISTORY

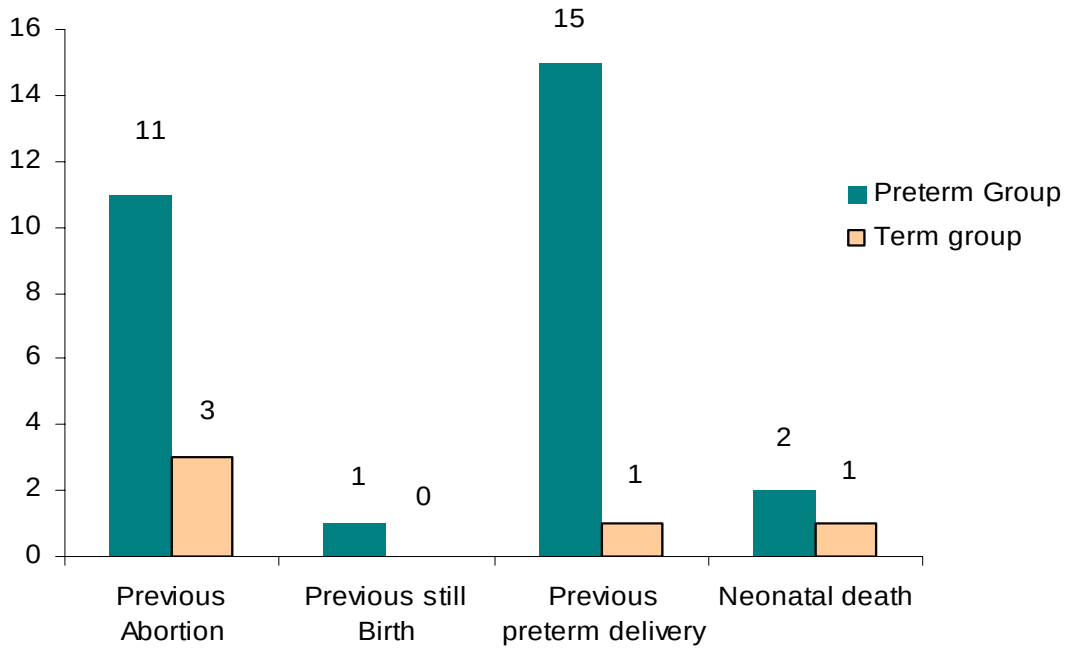


Table: 10

Distribution of Subject Groups According to their Socio Economic Status

Socio economic Status	Preterm Group		Term Group		Significance	Total
	No.	Percentage	No.	Percentage		
Class I	-	-	-	-	-	-
Class II	-	-	-	-	-	-
Class III	4	4.00	11	11.00	P=0.05	15
Class IV	5	5.00	19	19.00	P<0.01	24
Class V	91	91.00	70	70.00	P<0.001	161
Total	100	100.00	100	100.00	P<0.001	200

From the table - 10, among the 15 class III mothers, the preterm and term groups were 4% and 11% respectively which was statistically just significant. In respect of class IV mothers, the percentage of preterm and term groups were 5% and 19% respectively. The difference was statistically highly significant ($p < 0.01$).

FIGURE - 8

DISTRIBUTION OF SUBJECT GROUPS ACCORDING TO THEIR SOCIO ECONOMIC STATUS

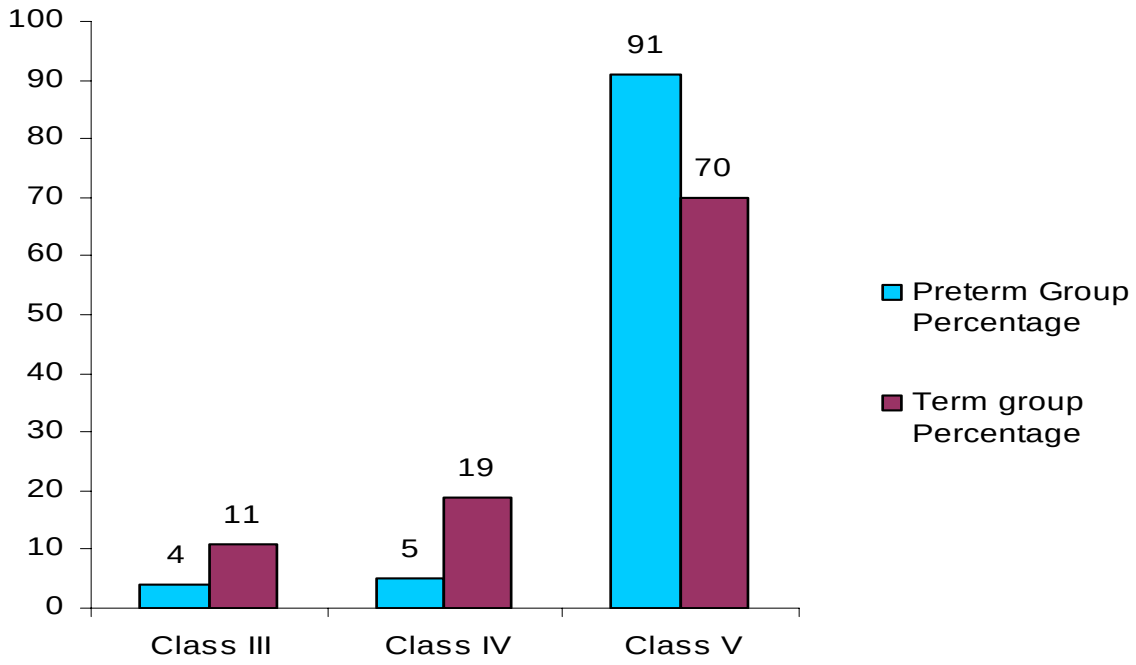


Table: 11

Distribution of Subject Groups According to their Socio Economic Status (Class - V)

Socioeconomic Status	Preterm Group	Term Group	Total
Class V	91	70	161
Other class	9	30	39
Total	100	100	200

Chi square Test γ^2 - 14.047

P value - <0.001

From the table -11, the low socioeconomic class V was predominantly more among the two categories as 91% among the preterm and 70% among the term group. The difference was statistically very highly significant ($p<0.001$).

Table: 12

Association Between weight of the Mother with Preterm Deliveries.

Weight of the mother	Preterm Group		Term Group		Total	
	No	**%**	**No**	**%**	**No**	**%**
< 45 kg	32	32.00	4	4.00	36	18.00
45 kg & above	68	68.00	96	96.00	164	82.00
Total	100	100.00	100	100.00	200	100.00

Chi square test γ^2 - 24.695

P value - < 0.001

The above table-12 shows that there was a significant association between the mother of weight <45 kg with the preterm group.

FIGURE - 10

ASSOCIATION BETWEEN WEIGHT OF THE MOTHER WITH PRETERM DELIVERIES.

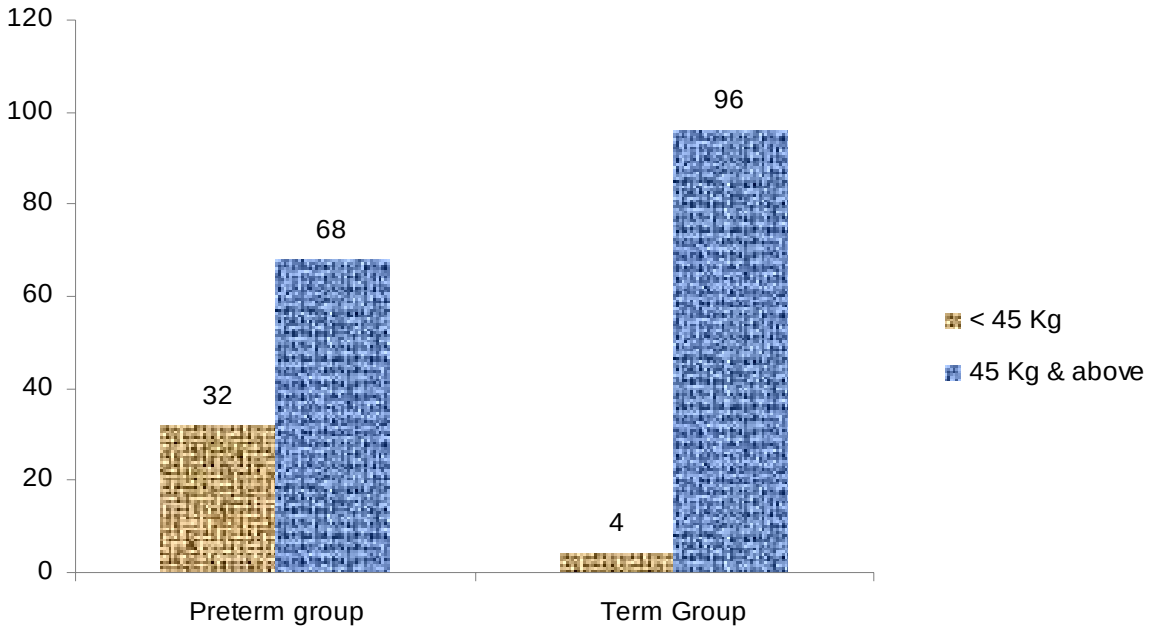


Table : 13

Association of Bacterial Vaginosis among the Preterm Group

According to Gestational Age

Bacterial vaginosis	Preterm Group		
	<34 wks	**34-36 wks**	**Total**
Positive	16	17	33
Negative	14	53	67
Total	30	70	100

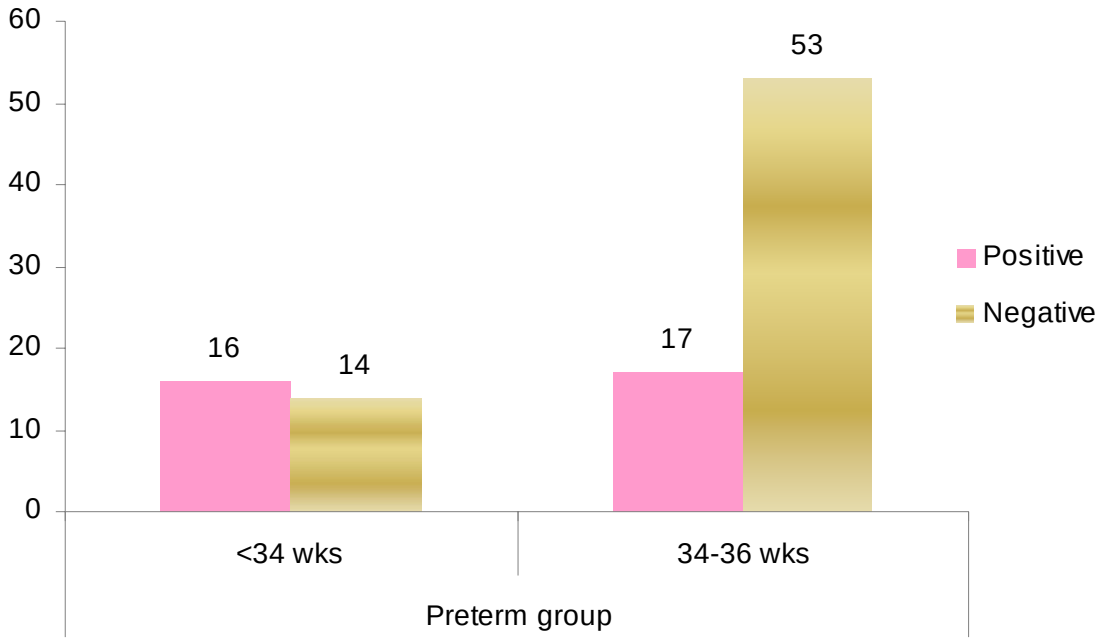
Chi square test γ^2 - 8.014

P value - <0.01

From the above table-13, there is positive association between the bacterial vaginosis with the < 34 wks of gestation. The significant association clearly shows that the bacterial vaginosis induced the preterm labour earliest than that of 34 – 36 wks of gestation ($p < 0.01$).

FIGURE - 11

ASSOCIATION OF BACTERIAL VAGINOSIS AMONG THE PRETERM GROUP ACCORDING TO GESTATIONAL AGE



DISCUSSION

DISCUSSION

By Amsel's criteria, bacterial vaginosis is diagnosed when three of the following four are present:

- Homogenous vaginal discharge.
- pH value >4.5
- Fishy odour on alkalinization.
- Presence of clue cells.

Since during labour, there is increased vaginal discharge and since patients with history of draining per vaginum were excluded from the study subjects, homogenous vaginal discharge as one of the criteria for diagnosis of bacterial vaginosis was not included in this study.

Hence gram stain was also done in addition to pH value of vaginal discharge, amine test and smear for clue cells on wet mount. The presence of Gardnerella outnumbering lactobacilli was studied in gram stained smear and diagnosis made by spiegel's criteria also, as it has high sensitivity of 97% and negative predictive value of 98%.

Evaluation of bacterial vaginosis by culture for Gardnerella vaginalis was not done since it has repeatedly been shown to be of little diagnostic value.

Eschenbach's group found that more than 55% of normal patients had Gardnerella vaginalis positive. Culture generally plays no role in the diagnosis because the isolation of Gardnerella vaginalis and / or anaerobic bacteria from the vagina does not define the clinical entity and can be observed in women without bacterial vaginosis.

- The preterm and term groups were not statistically significant in terms of their age. The mean ages of the above study subjects were 22.6 $\pm$ 3.1 and 22.9 $\pm$ 3.1 years respectively. The difference of mean was not statistically significant ($t=0.4347$, $d.f=198$ and $p>0.05$) (Table 1 and Table 2).

- The preterm and term groups were also not statistically significant in terms of their parity (Table 3).

- The prevalence of bacterial vaginosis among the preterm groups and term groups were identified by different methods namely pH value, Amine test, clue cells and Gram Stain. The prevalence among the preterm groups by all methods were statistically higher than the prevalence among the term mothers. The differences were statistically very highly significant ($p<0.001$) (Table 4).

- The impact of bacterial vaginosis was reflected among the preterm groups by means of positive significant association in respect of the investigations. The pH value had shown the association significantly as $\gamma^2 = 36.2$ and $p<0.001$ (Table 5). The amine test had evaluated the association as $\gamma^2 = 14.047$ and $p<0.001$ (Table 6). The clue cells had interpreted the association as $\gamma^2 = 12.055$ and $p<0.01$ (Table 7). The gram stain had identified the association as $\gamma^2 = 11.293$ and $p<0.01$ (Table 8).

- About 29% of the preterm group had bad obstetric history compared to 4% of term group. ($\gamma^2 = 20.411$ and $p<0.001$). There was also highly significant association of bacterial vaginosis with previous preterm deliveries ($p<0.001$) (Table 9).

- The preterm and term mothers were significantly differed in respect of their Socioeconomic status. There was no mother reported either in the class I and II. In respect of class III (4%) and class IV (5%), preterm groups were significantly lesser than the respective term groups of class III (11%) and class IV (19%). Conversely, the class V category of preterm group (91%) was significantly greater than term group (70%). This interpretation was supported by the significant association

between the class V category with preterm groups as (γ^2 =14.047and p<0.001) (Table 10 and Table 11).

- The low weight of the preterm groups was significantly associated with preterm deliveries. The mothers of less than 45kg of weight were more vulnerable to preterm deliveries, since the low weight was significantly associated with preterm deliveries (γ^2 =24.695 and p<0.001). The mean weight of preterm group was 49.8 $\pm$ 7.4 was significantly less than the mean weight of the term groups 54.1 $\pm$ 5.6 kg (Table 12).

- Among the preterm group, 33% of the patients were positive for bacterial vaginosis. Of which, 16% of the patients were in gestational age of less than 34 weeks and 17% in the gestational age of 34-36 weeks. There is positive association between the bacterial vaginosis with less than 34 weeks of gestation.(γ^2= 8.014 and p < 0.01) (Table 13).

Study	Preterm Group	Term Group
Eschenbach et al., (1984)	49%	24%
Gravett et al., (1986)	43%	14%
Martius et al., (1988)	34%	12%
Mc Gregor et al., (1990)	35%	14.5%
Mc Donald etal., (1991)	15%	6%
Holst et al., Dept. of Microbiology Lund University, (1994)	41%	11%
Goval et al., (2004)	31%	15%
In the present study	33%	13%

From the present study, we have confirmed significant association between the bacterial vaginosis and preterm labour.

In our study, the incidence of Bacterial vaginosis was 33% in the preterm group and 13% in the term group.

Our study corresponds to that of Gravett et al., Martius et al., Mc Gregor et al., and Goval et al.,

Approximately 15-20% of pregnant women will have bacterial vaginosis (Hay et al., 1994). These women are upto 4 times more likely to have preterm labour than women without bacterial vaginosis.

Hillier et al., demonstrated that women with bacterial vaginosis are 10% more likely to deliver a preterm low birth weight infant than women with out bacterial vaginosis.

Hillier et al., showed that pregnant women have predominantly one of the two primary vaginal floral patterns-either normal (or) bacterial vaginosis and a few women will have intermediate pattern. Women with bacterial vaginosis in the second trimester tend to remain bacterial vaginosis positive in the third trimester and those women with intermediate flora had a significant chance of progressing to bacterial vaginosis.

Incidence rates of Bacterial Vaginosis

Year of study	Population studied	Incidence of bacterial vaginosis
Embree et al., (1984)	Women attending an STD clinic, USA	64%
Eschenbach et al., (1988)	Asymptomatic college students, USA	4%
Bump et al., (1988)	Group of virginal post-menarcheal girls, USA	12%
Hay et al., (1992)	Low risk gynaecology clinic, Harrow, UK	11%
Blackweel et al., (1993)	Group of women undergoing termination of pregnancy, USA	28%
Hay et al., (1994)	A routine Antenatal Clinic, Harrow, UK	15%
Lamont et al., (2000)	Group of asymptomatic women attending general practitioner for cervical cytology, UK	9%

Relationship of abnormal colonization to preterm delivery or preterm labour according to gestational age of screening.

Study	Maximum gestational age at screening	Relative risk	Confidence interval
Gravett et al., (1986)	32	2.0	1.1 – 3.5
McDonald et al., (1992)	28	1.8	1.0 – 3.2
Hillier et al., (1995)	26	1.4	1.2 – 1.8
Krolin et al., (1995)	26	1.5	1.1 – 2.2
Hillier et al., (1995)	26	1.5	0.8 – 3.0
Mc Gregor et al., (1990)	24	2.0	1.1 – 6.5
Riduan et al., (1993)	20	2.0	1.0 – 3.9
Hay et al., (1994)	20	5.5	2.3 – 13.30
Kurki et al., (1992)	17	6.9	2.5 – 19

SUMMARY

SUMMARY

The incidence of bacterial vaginosis in the preterm labour was 33%.

The incidence of bacterial vaginosis in the term labour was 13%.

There was significant association between preterm labour and bacterial vaginosis.

No significant association was found statistically between bacterial vaginosis and age groups.

No significant association was found statistically between bacterial vaginosis and obstetric history.

It was found that there was significant association of bacterial vaginosis with low socioeconomic group (Class V).

There was highly significant association of bacterial vaginosis with previous preterm deliveries.

The impact of bacterial vaginosis was reflected among the preterm group by means of positive significant association in respect of the investigations.

There was highly significant association that bacterial vaginosis induced the preterm labour earliest than that of 34 -36 weeks of gestation.

CONCLUSION

CONCLUSION

Bacterial vaginosis is a condition in which the normal hydrogen peroxide producing lactobacillus predominant vaginal flora is replaced with anaerobic bacteria, Gardnerella, Mobiluncus species and mycoplasma hominis.

There is significant association between bacterial vaginosis and preterm labour.

There is also significant association of various factors like women belonging to very low socioeconomic class, women with weight less than 45 kg and history of bad obstetric history, the previous spontaneous preterm birth to the study group.

Bacterial vaginosis being asymptomatic in more than 50% of patients, given these associations, antenatal women belonging to very low socioeconomic class, women with weight less than 45 kg and women with history of previous spontaneous preterm labour can be screened for bacterial vaginosis and can be given therapeutic intervention, which might result in the reduction of preterm labour. This would reduce the hospital admission for preterm labour and premature rupture of membranes and low birth weight babies.

PROFORMA

PROFORMA

Name : Age : IP No :

Socio Economic Class :

Date of Admission :

Date of Delivery :

Obstetric Code :

LMP : EDD :

Menstrual History :

Obstetric History :

Past History :

O/E:

Height :

Weight :

Vital Signs:

G/E :

INVESTIGATIONS

Hb :

Urine : Albumin

Sugar

RH typing :

Blood Sugar :

USG :

VAGINAL DISCHARGE

pH :

Amine Test :

Clue Cells :

Gram Stain :

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BIBLIOGRAPHY

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MASTER CHART

PRETERM GROUP

S. No	Name	Age	Ip no	Socioec status	Weight (kg)	Obstetric code	LMP	EDD	GA (weeks)	Date of delivery	pH	Amine test	Clue cells	Gram Stain	Baby wt
1	Mahalaxmi	19	9475	5	45	G2P1L1A0	14/12/08	21/09/09	32	24/07/09	5.5	+	+	+	1.75
2	Banu priya	20	9623	5	58	Primi	2/12/2008	9/9/2009	34	25/07/09	4	-	-	-	2
3	Kalavathy	21	9617	5	46	G3P2L2A0	3/1/2009	10/10/2009	30	26/07/09	6	+	+	+	2
4	Chitra	25	9738	5	59	G4P3L3A0 (P.pret)	18/11/08	25/08/09	36	28/07/09	5	-	-	-	2.4
5	Vennila	21	16555	3	47	Primi	26/04/08	3/2/2009	32	1/12/2008	6	-	-	-	1.9
6	Meenatchi	22	16610	5	60	G2P1L1A0	1/4/2008	7/1/2009	36	2/12/2008	4	-	-	-	2.3
7	Ramya	28	16627	5	48	G5P4L4A0	7/4/2008	14/01/09	34	2/12/2008	5	-	-	-	2
8	Rehana	22	16643	5	62	Primi	1/4/2008	8/1/2009	36	2/12/2008	4.5	-	-	-	2.45
9	Valli	23	16623	5	39	Primi	25/04/08	2/2/2009	32	2/12/2008	6.5	+	+	+	2
10	Saraswathy	26	16586	5	49	G4P3L2A0	26/04/08	3/2/2009	32	2/12/2008	4	-	-	-	1.75
11	Nageshwari	20	16109	5	59	Primi	8/4/2008	15/01/09	34	3/12/2008	4.5	-	-	-	2
12	Samshath	18	16696	4	49	G2P1L1A0 (P.pret)	1/4/2008	8/1/2009	36	3/12/2008	4	-	-	-	2.4
13	Vanmathy	23	16548	5	68	Primi	2/4/2008	9/1/2009	36	3/12/2008	4.5	-	-	-	2.36
S. No	**Name**	**Age**	**Ip no**	**Socioec status**	**Weight (kg)**	**Obstetric code**	**LMP**	**EDD**	**GA (weeks)**	**Date of delivery**	**pH**	**Amine test**	**Clue cells**	**Gram Stain**	**Baby wt**
14	Chitra	21	16842	5	48	Primi	8/4/2008	15/01/09	34	5/12/2008	5	+	-	+	2
15	Hemavathy	27	16865	5	59	Primi	8/4/2008	15/01/09	34	6/12/2008	5	-	-	-	1.95
16	Priya	24	16889	5	47	G2A1	4/4/2008	11/1/2009	36	7/12/2008	6	+	+	+	2.3
17	Vanitha	24	16928	5	59	Primi	14/05/08	21/02/09	30	8/12/2008	4	-	-	-	1.8

97	Bhavani	28	9407	5	54	Primi	3/12/2008	10/9/2009	34	29/07/09	4	-	-	-	2
S. No	**Name**	**Age**	**Ip no**	**Socioec status**	**Weight (kg)**	**Obstetric code**	**LMP**	**EDD**	**GA (weeks)**	**Date of delivery**	**pH**	**Amine test**	**Clue cells**	**Gram Stain**	**Baby wt**
98	Anu	20	10415	5	41	G3P1L1A1	22/12/08	29/09/09	34	11/8/2009	5	+	+	+	2
99	Yamuna	18	12341	5	55	Primi	10/2/2009	17/11/09	32	19/09/09	5	+	+	+	1.8
100	Kokila	22	12634	5	56	G2P1L1A0	28/02/09	7/11/2009	34	24/09/09	5	-	-	-	1.9

TERM GROUP

S .No	Name	Age	Ip no	Socioec status	Weight (kg)	Obstetric code	LMP	EDD	GA (weeks)	Date of delivery	pH	Amine test	Clue cells	Gram Stain	Baby wt
1	Girija	25	16458	5	47	G2P1L1A0	23/02/2008	23/02/2008	Term	1/12/2008	4.5	-	-	-	2.75
2	Parisha	18	16541	3	54	Primi	23/02/2008	23/02/2008	Term	1/12/2008	5.5	-	-	-	2.6
3	Mehrunisha	20	16582	5	59	Primi	24/02/2008	24/02/2008	Term	2/12/2008	4	-	-	-	3.4
4	Boopathy	21	16632	5	58	G2A1	26/02/2008	26/02/2008	Term	3/12/2008	5	-	-	-	3.05
5	Kowsalya	26	16720	5	61	Primi	27/02/2008	27/02/2008	Term	4/12/2008	4	-	-	-	3.1
6	Shakira	19	16705	5	59	Primi	27/02/2008	27/02/2008	Term	4/12/2008	4	-	-	-	3.2
7	Indumathi	22	16680	5	46	G2P1L1A0	28/02/2008	28/02/2008	Term	5/12/2008	6.5	+	+	+	2.6
8	Shamsath	29	16696	5	57	Primi	26/02/2008	26/02/2008	Term	3/12/2008	4	-	-	-	2.8
9	Vijaya	23	16833	4	56	Primi	28/02/2008	28/02/2008	Term	6/12/2008	5	-	-	-	2.75
10	Lakshmi	27	16838	5	55	G2P1L1A0	28/02/2008	28/02/2008	Term	6/12/2008	5	-	-	-	2.6
11	Varalaxmi	18	16910	5	51	Primi	1/3/2008	1/3/2008	Term	8/12/2008	4.5	-	-	-	3.75
12	Elavarasi	24	16979	5	55	Primi	1/3/2008	1/3/2008	Term	9/12/2008	4	-	-	-	2.5
13	Muniammal	24	17012	5	58	Primi	1/3/2008	1/3/2008	Term	9/12/2008	4	-	-	-	2.614
14	Kuppammal	30	16728	5	59	G2P1L1A0	2/3/2008	2/3/2008	Term	10/12/2008	6	+	+	+	3.2
15	Mahalaxmi	23	17035	4	54	Primi	2/3/2008	2/3/2008	Term	10/12/2008	4	-	-	-	3.38
16	Parimala	28	17020	5	58	G3P2L2A0	4/3/2008	4/3/2008	Term	10/12/2008	5	-	-	-	3.1
17	Marlina	19	17062	5	52	Primi	4/3/2008	4/3/2008	Term	10/12/2008	4	-	-	-	2.75
18	Kavitha	22	17099	5	54	G2P1L1A0	3/3/2008	3/3/2008	Term	11/12/2008	4	-	-	-	3.3
19	Bharathy	21	17053	5	59	Primi	3/3/2008	3/3/2008	Term	11/12/2008	4	-	-	-	2.8

S .No	Name	Age	Ip no	Socioec status	Weight (kg)	Obstetric code	LMP	EDD	GA (weeks)	Date of delivery	pH	Amine test	Clue cells	Gram Stain	Baby wt
20	Beula	20	17149	5	47	Primi	5/3/2008	5/3/2008	Term	12/12/2008	4	-	-	-	2.5
21	Sundari	21	17072	5	61	Primi	5/3/2008	5/3/2008	Term	13/12/08	4	-	-	-	3.05
22	Chitra	25	17214	4	62	G3P2L2A0	5/3/2008	5/3/2008	Term	13/12/08	5	+	+	+	3.25
23	Annie	19	17232	5	59	Primi	4/3/2008	4/3/2008	Term	13/12/08	4	-	-	-	3.05
24	Prema	22	17250	5	58	Primi	6/3/2008	6/3/2008	Term	14/12/08	4.5	-	-	-	2.9
25	Hajitha	23	17246	5	54	Primi	7/3/2008	7/3/2008	Term	14/12/08	4	-	-	-	3.1
26	Sangeetha	26	17407	4	52	G2P1L1A0	9/3/2008	9/3/2008	Term	17/12/08	5	-	-	-	2.6
27	Durga	24	17418	5	52	G2P1L1A0	9/3/2008	9/3/2008	Term	17/12/08	4	-	-	-	3.3
28	Jayanthi	21	17448	5	54	Primi	9/3/2008	9/3/2008	Term	17/12/08	4	-	-	-	2.8
29	Chitra	25	17558	3	45	G3P2L2A0	14/03/08	14/03/08	Term	20/12/08	4	-	-	-	2.5
30	Thilaga	20	17576	5	55	Primi	14/03/08	14/03/08	Term	20/12/08	4	-	-	-	2.5
31	Shanthi	21	17667	5	58	Primi	14/03/2008	14/03/2008	Term	22/12/08	4	-	-	-	3.07
32	Shanthi	19	17669	4	59	Primi	15/03/2008	15/03/2008	Term	23/12/08	5.5	-	-	+	2.76
33	Malathy	22	17681	5	46	Primi	15/03/2008	15/03/2008	Term	23/12/08	5	-	-	-	2.8
34	Laxmi	23	17644	5	64	G2P1L1A0	15/03/2008	15/03/2008	Term	24/12/08	4	-	-	-	2.76
35	Poorni	24	17802	4	68	G2P1L1A0	15/03/2008	15/03/2008	Term	24/12/08	4.5	-	-	-	2.96
36	Kalpana	26	17861	5	47	G3P2L2A0	18/03/2008	18/03/2008	Term	26/12/08	4	-	-	-	2.25
37	Thulasi	27	17865	3	59	G3P2L2A0	19/03/2008	19/03/2008	Term	27/12/08	4	-	-	-	2.8
38	Lakshmi	21	17901	5	56	Primi	19/03/2008	19/03/2008	Term	27/12/08	4	-	-	-	3.1
39	Mahalakshmi	18	17949	5	57	Primi	20/03/2008	20/03/2008	Term	28/12/08	5	-	-	-	3
S .No	**Name**	**Age**	**Ip no**	**Socioec status**	**Weight (kg)**	**Obstetric code**	**LMP**	**EDD**	**GA (weeks)**	**Date of delivery**	**pH**	**Amine test**	**Clue cells**	**Gram Stain**	**Baby wt**
40	Usha	20	17978	5	64	Primi	21/03/2008	21/03/2008	Term	29/12/08	4	-	-	-	3.26
41	Bhavani	24	17918	4	68	G2P1L1A0 (P. Pret)	21/03/08	21/03/08	Term	29/12/08	6	+	+	+	2.3
42	Latha	31	18041	5	42	G2P1L1A0	23/03/08	23/03/08	Term	31/12/08	4	-	-	-	2.5
43	Indhu	25	18070	3	55	G3P2L2A0	23/03/08	23/03/08	Term	31/12/08	5	-	-	-	2.95

44	Shanthi	22	277	5	48	G3P2L1A0	2/4/2008	2/4/2008	Term	7/1/2009	4	-	-	-	3.2
45	Saritha	24	110	5	54	G2P1L1A0	1/4/2008	1/4/2008	Term	4/1/2009	4	-	-	-	2.6
46	Papli	21	326	5	53	Primi	1/4/2008	1/4/2008	Term	9/1/2009	5	+	+	+	3
47	Mari	23	364	4	52	G2P1L1A0	2/4/2008	2/4/2008	Term	10/1/2009	4	-	-	-	2.8
48	Janaki	19	327	5	49	Primi	2/4/2008	2/4/2008	Term	10/1/2009	4	-	-	-	2.69
49	Kalpana	20	321	3	43	Primi	3/4/2008	3/4/2008	Term	11/1/2009	4.5	-	-	-	2.46
50	Amala	28	412	5	46	G2P1L0	3/4/2008	3/4/2008	Term	11/1/2009	4	-	-	-	2.6
51	Devi	21	439	5	58	G2P1L1A0	4/4/2008	4/4/2008	Term	12/1/2009	4	-	-	-	3.2
52	Pushpa	32	299	4	57	G3P2L2A0	5/4/2008	5/4/2008	Term	13/01/09	4	-	-	-	3
53	Gowri	22	506	5	59	G2P1L1A0	5/4/2008	5/4/2008	Term	14/01/09	4	-	-	-	3.1
54	Vijaya	25	589	4	54	G2P1L1A0	9/4/2008	9/4/2008	Term	17/01/09	5	-	-	-	2.6
55	Indhra	23	751	5	53	Primi	10/4/2008	10/4/2008	Term	20/01/09	4	-	-	-	2.8
56	Aruljothi	24	666	5	46	Primi	12/4/2008	12/4/2008	Term	20/01/09	4	-	-	-	2.75
57	Dhanalaxmi	26	792	5	58	G2P1L1A0	13/04/2008	13/04/2008	Term	21/01/09	5	-	-	-	2.75
58	Revathy	19	1098	4	47	Primi	22/04/2008	22/04/2008	Term	29/01/09	4	-	-	-	3
59	Mari	21	1120	5	57	Primi	21/04/2008	21/04/2008	Term	29/01/09	5.5	+	+	+	3.63
S .No	**Name**	**Age**	**Ip no**	**Socioec status**	**Weight (kg)**	**Obstetric code**	**LMP**	**EDD**	**GA (weeks)**	**Date of delivery**	**pH**	**Amine test**	**Clue cells**	**Gram Stain**	**Baby wt**
60	Kala	22	1182	5	56	Primi	25/04/2008	25/04/2008	Term	3/2/2009	4	-	-	-	2.46
61	Kalaivani	23	2278	5	48	G2P1L1A0	21/05/2008	21/05/2008	Trm	27/02/09	4	-	-	-	3.01
62	Renuka	27	2321	3	54	G3P1L1A1	21/05/2008	21/05/2008	Term	28/02/09	5	-	+	+	3.1
63	Parveen	18	2419	5	43	Primi	25/05/2008	25/05/2008	Term	2/3/2009	4.5	-	-	-	2.6
64	Rani	21	2017	4	55	Primi	25/05/2008	25/05/2008	Term	2/3/2009	4	-	-	-	3.1
65	Govindamal	24	2502	5	49	G2P1L1A0	27/05/2008	27/05/2008	Term	4/3/2009	4	-	-	-	2.5
66	Vanitha	22	2450	5	57	Primi	27/05/2008	27/05/2008	Term	4/3/2009	5	-	-	-	2.6
67	Dhanalaxmi	18	2514	5	59	Primi	27/05/2008	27/05/2008	Term	4/3/2009	4	-	-	-	2.5
68	Queenmary	23	2712	3	60	G2P1L1A0	28/05/2008	28/05/2008	Term	4/3/2009	6.5	+	+	+	2.76

70	Prema	24	3091	5	61	G2P1L1A0	11/6/2008	11/6/2008	Term	18/03/09	4	-	-	-	2.6
71	Shenbagam	27	3294	5	58	G2P1L1A0	15/06/2008	15/06/2008	Term	21/03/09	4	-	-	-	2.9
72	Selvi	24	3874	5	47	Primi	24/06/2008	24/06/2008	Term	31/03/09	5	-	-	-	2.7
73	Shenbagam	23	4123	5	57	G2P1L1A0	29/06/2008	29/06/2008	Term	6/4/2009	4	-	-	-	2.9
74	Sudha	21	4198	5	54	Primi	29/06/08	29/06/2008	Term	7/4/2009	4	-	-	-	3.3
75	Komala	22	4168	5	45	Primi	29/06/08	29/06/2008	Term	7/4/2009	4	-	-	-	3.2
76	Kokila	24	4112	3	59	G2P1L1A0	4/7/2008	4/7/2008	Term	11/4/2009	4.5	-	-	-	3.3
77	Masudha	18	4747	5	46	Primi	11/7/2008	11/7/2008	Term	19/04/09	4	-	-	-	2.5
78	Devi	20	5064	4	58	Primi	18/07/2008	18/07/2008	Term	24/04/09	4	-	-	-	2.5
79	Durga	24	6398	5	57	G2P1L1A0	25/07/2008	25/07/2008	Term	1/5/2009	5	-	-	+	3
S .No	**Name**	**Age**	**Ip no**	**Socioec status**	**Weight (kg)**	**Obstetric code**	**LMP**	**EDD**	**GA (weeks)**	**Date of delivery**	**pH**	**Amine test**	**Clue cells**	**Gram Stain**	**Baby wt**
80	Eswari	21	6501	3	55	Primi	1/8/2008	1/8/2008	Term	8/5/2009	4	-	-	-	3.1
81	Rani	24	7002	4	59	G2P1L1A0	14/08/2008	14/08/2008	Term	20/05/09	4	-	-	-	2.75
82	Gowri	22	7208	5	46	Primi	21/08/2008	21/08/2008	Term	28/05/09	4	-	-	-	2.9
83	Rajeshwari	21	7369	5	58	G3P2L2A0	29/08/2008	29/08/2008	Term	4/6/2009	4	-	-	-	2.6
84	Nithya	30	7603	5	45	G2P1L1A0	9/9/2008	9/9/2008	Term	15/06/09	4	-	-	-	2.75
85	Seema	20	8301	5	57	Primi	9/10/2008	9/10/2008	Term	17/07/09	4	-	-	-	2.8
86	Ratna	21	9697	4	47	Primi	28/11/2008	28/11/2008	Term	3/8/2009	4	-	-	-	3
87	Salsa	28	10989	5	56	G3P2L2A0	12/11/2008	12/11/2008	Term	18/08/09	5	+	+	+	3.3
88	Malathy	22	11984	5	55	Primi	15/11/2008	15/11/2008	Term	23/08/09	4	-	-	-	2.95
89	Parimala	23	12299	4	55	G2P1L1A0	13/12/2008	13/12/2008	Term	18/09/09	4	-	-	-	3
90	Parvathy	27	12331	3	54	G2P1L1A0	13/12/2008	13/12/2008	Term	20/09/09	4.5	-	-	-	3.1
91	Manjula	24	12437	5	44	G2P1L1A0	13/12/2008	13/12/2008	Term	21/09/09	4	-	-	-	2.75
92	Anitha	24	12415	5	49	G3P2L2A0	15/12/2008	15/12/2008	Term	21/09/09	6	+	+	+	2.8
93	Parvathy	20	12312	4	52	Primi	15/12/2008	15/12/2008	Term	22/09/09	4	-	-	-	3
94	Thilagavathy	21	12533	5	54	Primi	15/12/2008	15/12/2008	Term	23/09/09	4	-	-	-	2.9
95	Salma	19	12593	5	55	Primi	16/12/2008	16/12/2008	Term	24/09/09	4	-	-	-	3.25