PREVALENCE OF SEXUALLY TRANSMITTED INFECTIONS AMONG PREGNANT WOMEN ATTENDING INSTITUTE OF VENEREOLOGY

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CERTIFICATE

Certified that this dissertation entitled "PREVALENCE OF SEXUALLY TRANSMITTED INFECTIONS AMONG PREGNANT WOMEN ATTENDING INSTITUTE OF VENEREOLOGY" is a bonafide work done by DR.A.KRISHNAVENI, Post Graduate Student in M.D. Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 600 003, during the academic year 2005 – 2008. This work has not been formed previously the basis for the award of any degree.

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INTRODUCTION

Sexually transmitted infections (STIs) are an important cause of morbidity and mortality worldwide, especially in women and children particularly in resource poor settings of developing world.

The World Health Organization (WHO) estimates that around 340 million new cases of STIs occurs world wide.¹ 50% of new cases of STIs occur in South East Asia. STIs most commonly affect people aged 15 to 44 years, the most economically productive age.

Many STIs are asymptomatic or people are reluctant to seek health care due to stigma attached to it. Women of child bearing age are at particular risk of sequelae from STIs due to the impact of many STIs on their reproductive health.² Burden of STIs is not only due to acute episode of the infections, but it has long term and severe sequelae like infertility, ectopic pregnancy and pelvic inflammatory diseases. Both ulcerative and non ulcerative STIs enhance the risk of acquiring and transmitting HIV infection by five to ten folds.²

'Infections in pregnancy are common but few cause fetal infection and damage, particularly urgent is the need to control fetal wastage and congenital abnormalities due to maternal sexually transmitted infections including HIV.³ Pregnancy is vulnerable time for women. Hence STIs in

the pregnant women are more serious than in non pregnant women. Risk factors like multiple sex partners may independently affect pregnancy outcome. ¹

Adolescent sexual activity is increasing globally. With the change in social norms, peer pressure and media influences, teenagers are engaging in premarital sex earlier leading to unintended pregnancy and STIs.³ In general, sexually transmitted infections appears to pose a much greater problem in pregnant adolescents than in older pregnant women due to their monogamous relationship. Infections in young adult have their most serious consequences later in life. Adolescents have miscarriages more often and are partly attributable to STI. Teenage pregnancy remains common in many societies. The incidence of Medical termination of Pregnancy is particularly high among adolescents.

Violence and sexual abuse by men is another aspect where women and girls are the most frequent victims.³ Pregnancy may also be a sign of ongoing sexual abuse. Meticulous examination should be performed looking carefully for STIs in those cases.

Physiological changes, anatomical changes in the genital tract, immunological alteration in a pregnant woman have been postulated to influence the course of STIs which pose special risk of infection for both mother and fetus.⁴

Pregnancy modifies the manifestation of many STIs and presents unique problems for diagnosis and management. Antenatal attendees are a section of population routinely used as a reference point for STI prevalence in the general population of women. Thus routine screening and treatment of certain genital infections in high risk population eventually may lead to reduction in adverse pregnancy outcomes.

REVIEW OF LITERATURE

SEXUALLY TRANSMITTED INFECTIONS IN PREGNANCY

PHYSIOLOGICAL CHANGES DURING PREGNANCY

Immunologically T-Lymphocytes are reduced in number in peripheral blood samples. Decrease in CD-4 T helper subset to nearly half the pregnancy level which is the maximum during the third trimester. No significant change in CD-8 subset. Humoral substances that potentially suppress in vitro lymphocyte function are present in plasma. And this leads to impaired response to microbial antigens and phytohemagglutin. Suppressed maternal immunocompetence leads to higher attack rate of candidiasis. Incidence and severity of viral hepatitis are increased in pregnancy. Susceptibility to renal infection increases during pregnancy due to changes in ureterovesicular muscular tone induced by higher levels of progesterone or partial ureteral compression by gravid uterus.

Anatomical changes include hypertrophic and engorged vaginal walls. Glycogen content of vaginal epithelium increases and intravaginal pH decreases significantly.⁴ These changes influence vaginal micro flora.

The cervix becomes hypertrophic and larger area of columnar epithelium as exocervix is exposed to micro organisms. But cervical

mucous plug limit the access of microorganisms into the uterus. Risk of salphingitis decreases during pregnancy after 12th week and risk of chorioamnionitis increases after 16th week of gestation.⁷

Placental trophoblastic epithelium with langhans layer and syncytiotrophoblast regulate fetal uptake of many substances. Placental macrophages or Houfbauer cells acts as first line of fetal defense to transplacental infection.

SYPHILIS IN PREGNANCY

Syphilis is a systemic, chronic infectious disease transmitted through sexual intercourse, from mother to infant during pregnancy and through transfusion of infected blood. Syphilis in pregnancy is a major cause of fetal and neonatal loss in developing countries.

`Spirochetal transmission still occurs as a result of untreated, inadequately treated or failed treatment of syphilis in the mother.

About 9,00,000 gestations occur annually among 6 million infected woman worldwide.⁹ Increasing number of congenital syphilis have been reported from Asian and African countries.¹⁰ Prevalence of syphilis seroreactivity among pregnant woman varies from 0.02 to 12.1%in different parts of world.¹¹

Syphilis may be acquired in pregnancy or preexist in any of its clinical stages.¹² The clinical presentation of syphilis is not significantly altered by pregnancy; in fact the manifestations of primary and secondary

syphilis may be milder during pregnancy due to immunosuppression. 13,14

Primary syphilis follows incubation period of 10 to 90 days. Primary lesions are smaller size or be so located as to go unnoticed. Cervical chancre is more common due to easily inoculable friable cervix. Increased tissue vascularity contributes toward non formation of primary chancre as it prevents epithelial breakdown. ¹⁵

After 4 to 10 weeks of healing of primary chancre, secondary syphilis appears with variable skin rash. In 25% of patients it may go mild and unnoticed. Consequences of in utero infection depend on factors such as maternal stage, duration of pregnancy at the time of infection and adequacy of treatment.¹⁶

Risk of congenital syphilis is high during first four years after acquisition. Rate of transmission to the fetus in untreated women is approximately 70 to 100% in primary and secondary syphilis, 40% in early latent syphilis and 10% in late latent stage.¹⁶

Kassowit's law states that risk of congenital syphilis is inversely related to the duration of untreated maternal infection. Fetal infection with Treponema pallidum can occur as early as 9 weeks of gestation. ⁹

Pathogenesis of congenital infection depends on immune response of fetus and not due to cytopathic effects of Treponema pallidum.¹⁴ The devastating effects of maternal syphilis on the fetus are many. Untreated maternal syphilis may lead to spontaneous abortion (most common), still

birth, prematurity, perinatal death and congenital syphilis. Overall it is estimated that one third of syphilis seroreactive pregnancies will result in fetal wastage, one third in the birth of infant with congenital syphilis and one third in the delivery of healthy uninfected child.¹²

Recently syphilis is becoming leading cause of non immune hydrops fetalis.¹⁶ Adverse pregnancy outcome due to syphilis is preventable by screening and early treatment of both partners.

VDRL screening should be done at first prenatal visit and repeated at third trimester in high risk patients. As reagin test lack specificity, specific test like Fluorescent Treponemal Antibody absorption test or Treponema Pallidum Haemagglutionation Assay for antibodies to Treponema pallidum is used to confirm positive results.

Women with an epidemiological history of recent exposure to an individual with proven syphilis should be treated regardless of serological results. Prior to syphilotherapy, all women should be offered counselling to undergo testing for antibodies to HIV.

Penicillin therapy during pregnancy is 98% effective in preventing congenital infection.¹⁷ The Jarisch-Herxiheimer reaction commonly occurs during treatment of acquired early syphilis. Patients who do not show four fold drop in titre at 30 months or four fold rise in titre should be retreated.

GONORRHOEA IN PREGNANCY

Prevalence of gonococcal infection during pregnancy ranges from 0.5 to 7%. ¹⁸ In European countries prevalence is below 1%.

Risk factors for acquisition of gonococci include being single, adolescent, poverty, drug abuse, prostitution, lack of prenatal care. Gonococcal infection is also a marker of concomitant chlamydial infection in about 40% of infected pregnant women.

More than half of the pregnant women with gonococcal infection are asymptomatic and clinical signs are associated with this infection is not sufficiently sensitive or specific. ¹²

Several studies have reported an increased risk of pharyngeal gonorrhea to about 15 to 35%. And this pharyngeal gonorrhoea also increases risk of disseminated gonococcal infection in pregnancy particularly in the second and third trimester.¹⁸

Increased gonococcal dissemination could be due to engorged pelvic vasculature or altered immune status of pregnancy.

Gonococcal infection is limited to lower genital tract including cervix, urethra, periurethral and vestibular gland in most pregnant women. Endocarditis rarely complicates pregnancy.

Gonococcal salphingitis have been reported between 7 and 12th week of gestation. ¹⁹ Pelvic inflammatory disease is rare event during pregnancy. Local cervical factors and increased serum progesterone

concentration decreases the risk of ascending infection during pregnancy.

Gonorrhoea has adverse effect upon both mother and child. Maternal complications are spontaneous or septic abortions, premature rupture of membranes, postpartum endometritis.¹⁵ Increased risk of prematurity, low birth weight, gonococcal ophthalmia neonatorum are the most important perinatal complication.¹⁹

Neonates can present with bilateral mucopurulent conjunctivitis, corneal ulcer, perforation, anterior staphyloma, anterior capsular cataract, and panophthalmitis. Risk of amniotic fluid infection syndrome and gonococcal ophthalmia appears to be increased after premature rupture of membranes. ¹⁹

Neonates can have gonococcal meningitis, arthritis, septicemia, rarely neonatal vaginitis²⁰, endocarditis, anorectal infection, funisitis and urethritis. ²¹ Gonococcal scalp abscess can occur at scalp electrode site.

The gold standard diagnostic test for gonococcal infection in women is endocervical culture. But this is neither available nor practical in most antenatal care settings. All pregnant women should be screened for infection during first visit and repeat cultures at 36 to 38 weeks in high risk population. ²²

All patients treated during pregnancy should have repeat cultures in the third trimester and for those patients whose partners have not been treated. Nucleic acids amplification assays for the detection of gonorrhoea and Chlamydia trachomatis are more sensitive for screening urine and cervical samples, but not available easily due to its high cost.

CHANCROID IN PREGNANCY

Chancroid is seen less common in women.²¹ It appears that women are asymptomatic carriers.²²

The disease has no influence on pregnancy outcome and the disease is not yet reported in neonates. Hemophilis ducrey causes painful non-indurated genital ulcer or soft sore and at times accompanied by painful inguinal lymphadenopathy. Chancroidal infection is a high risk factor for HIV and syphilis transmission.

Diagnosis by culture is difficult because appropriate media are not widely available. Instead clinical diagnosis is made when typical painful genital ulcers are present with negative dark field examination and herpes viral test.

GRANULOMA INGUINALE IN PREGNANCY

Vagina is often infected with autoinoculation with Calymmatobacterium granulomatis, as the organism has fecal habitat.

Prevalence of granuloma inguinale is very low and about less than 1% in STI clinic attendees.

Lesions of Donovanosis tend to proliferate or recur and show

diminished response to standard antimicrobial therapy.²³ Cervical lesions rapidly enlarge, extend to pelvic structures and disseminate resulting in fatal hemorrhage at the time of delivery. The hypertrophic variant is more commonly seen during pregnancy, But these effects could not be established according to a South African study.²⁴ Miscarriage and abortion due to Granuloma inguinale are rarely reported.

Perinatal transmission has occasionally reported in untreated patients and manifest as suppurative otitis media, cervical lymphadenopathy, lesions on umbilicus, labia and penis.^{25,26,27}

LYMPHOGRANULOMA VENEREUM IN PREGNANCY

Lymphogranuloma venereum caused by L1, L2, and L3 serovars of Chlamydia trachomatis is characterized by transient primary genital infections and followed by inguinal adenitis.

The course and diagnosis of lymphogranuloma venereum are not known to be altered by pregnancy.

There is little evidence of transplacental or perinatal transmission, but no problems have been recognized in babies.²⁷ Infection may be acquired through infected birth canal.²⁸

CHLAMYDIA TRACHOMATIS INFECTION IN PREGNANCY

Chlamydia trachomatis is the most common sexually transmitted disease in women of reproductive age.¹² The rate of Chlamydial infection among pregnant women ranges from 2 to 20%.²⁹ Rate of isolation of the organism is higher during third trimester.

Among pregnant women risk factors for chlamydia infection includes unmarried status, age below 20years. presence of other STIs, partners with non gonococcal uretheritis, presence of mucopurulent cervicitis, sterile pyuria, resident of socially disadvantaged community, late or no prenatal care. ^{10,15}

Up to 70% of women infected with chlamydial infections may be asymptomatic or experience subclinical symptoms.³⁰ Cervical colonization is present in 3 to 25% of pregnant patients.

There is transplacental transfer of maternal IgG antibodies as early as day 38, but not completely protective. Organism causes several clinical syndromes including uretheritis, mucopurulent cervicitis, and acute salphingitis. Upper genital tract infections are rare in pregnancy. Maternal infection present as bartholinitis, perihepatitis, conjunctivitis and reactive arthritis.

Fetal wastage approaches 50% in gestational pelvic inflammatory diseases. ¹² It produces chronic cervical infection. There is high coinfection rate of Chlamydia with gonorrhoea in pregnant population.

Studies have shown association between chlamydial infection in pregnancy and adverse pregnancy outcomes such as spontaneous abortion, premature rupture of membranes, preterm labour, low birth weight infants and delayed post partum endometritis. ^{12,31}

The risk of vertical transmission in untreated genital infection approaches 70% as documented by seroconversion in infant. ¹² Infection occurs during passage of infant through an infected birth canal resulting in ophthalmia neonatorum, pneumonia, serous otitis media. ²⁷

Increased number of WBCs occur in the cervix in normal pregnant women, hence the diagnosis of mucopurulent cervicitis does not correlate well with the presence of Chlamydia in pregnancy and cannot be used as a screening tool.³²

The gold standard method of diagnosis is cell culture which is not universally available. Centre for disease control recommends screening of all pregnant women less than 25 years old or those who have new or multiple sex partners. Repeat cultures in third trimester were recommended in those who are at high risk.

Greater importance should be given for screening unmarried women less than 20 years of age before medical termination of pregnancy, or delivery. DNA probe assay and enzyme immuno assay have greater sensitivities in detection. Newer nucleic acid amplification tests are much more sensitive in screening pregnant women.

VULVOVAGINAL CANDIDIASIS IN PREGNANCY

Candidal vaginal infections are classically considered with other STIs. Candida albicans is present in the vagina of approximately 25% of sexually active women.

Symptomatic vaginal candidiasis affects 15% of pregnant women. About 10% of women in the first trimester and 36 to 50% of patients in the third trimester have symptomatic disease. ³³ Vulvovaginal candidiasis is recurrent in 8% of women in the reproductive age. ³⁴

Clinical signs of infection could be detected in all pregnant women from whom the fungus was isolated, even in those without symptoms.³⁴

Increase in glycogen content of vaginal epithelial cells and vascularity predisposes to candidal infection in pregnancy. Proteinases along with hyphal invasion cause swelling, erythema, desquamation, exfoliation and acute inflammatory response.

Severe vulval pruritus is the most common symptom. Vulvovaginal candidiasis manifest as edematous vulva with fissuring between intra vulval folds and thick curdy white discharge adherent to introitus and vestibular area. Occasionally there may be rash with satellite micro pustules around outer labia and involvement of perianal area. There is increase in the prevalence of vaginal candidiasis in women infected with HIV. ³⁵

There are rare case reports of intrauterine acquired candidiasis

(ascending vaginal infection) resulting in spontaneous abortion.³⁶ Also there are occasional case reports of candidal chorioamnionitis.²²

The most common route of perinatal infection is by direct contact during delivery through an infected vagina and oral thrush of the neonate is the most common problem.²⁸ So the prepartal vaginal colonization should be treated to protect the newborn.

The clinical appearance is confirmed by gram staining, KOH mount and culture. Gram staining shows diagnostic pseudo hyphae in 30 to 50% of cases. Culture in sabouraud's dextrose agar (most commonly employed) or chrom agar medium is even more sensitive.²⁸

TRICHOMONIASIS IN PREGNANCY

Trichomoniasis is the most common protozoal STI and is isolated with many perinatal complication, male and female genitourinary tract infections and an increased incidence of HIV transmission.³⁷

Prevalence of Trichomonas vaginalis infection in pregnancy has ranged from 3 to 10% from several antenatal clinics.³⁸ Factors associated with Trichomonas infection are black race, cigarette smoking, unmarried females, less education, a history of gonorrhoea, multiple sex partners.

Vaginitis is usually asymptomatic and characterized by purulent greenish yellow frothy vaginal discharge, vulvovaginal erythema and strawberry cervix. Trichomoniasis has been associated with increased risk

of preterm delivery, maternal puerperal infection, premature rupture of membranes. 39,40

Due to combination effect of organism, proteases and host inflammatory response there is decrease in elasticity leading to rupture and bursting of chorioamniotic membranes.⁴¹

Due to the estrogenic effect during pregnancy, vaginal mucosa of female neonates is susceptible to infection. This is reversed spontaneously within 3 to 4 weeks after delivery. Risk of neonatal infection in exposed neonates is 5%. Purulent vaginitis, urinary tract infection and neonatal pneumonia have been described.⁴¹

Wet mount examination for trichomonads is positive only in 50% of infected women. These findings are unaltered by pregnancy, except for an increase in the number of WBCs in the vaginal fluid.¹⁰

Vaginal culture is more sensitive than direct microscopy of vaginal secretions. Screening of all pregnant women who are at increased risk of preterm birth should be done with wet mount and culture. Trichomonal infection suspected on papanicolaou smear of the cervix should be confirmed by wet mount and culture before treatment during pregnancy.

BACTERIAL VAGINOSIS IN PREGNANCY

Bacterial vaginosis is a polymicrobial imbalance of vaginal flora.⁴² Bacterial vaginosis is detected in 12 to 23% of pregnant women,

increasing to more than 30% in women who deliver prematurely.⁴³ Pregnant black women and unmarried are especially at higher risk.

Half the populations of women with bacterial vaginosis are asymptomatic and current standard antenatal procedures do not provide regular screening for bacterial vaginosis.⁴⁴

Bacterial vaginosis is characterized by nonpurulent, homogenous, malodorous vaginal discharge and by an increase in vaginal pH and by the presence of characteristic amines and organic acids in the vaginal fluid.

Bacterial vaginosis increases the risk of post operative infections, and acquisition of HIV and other STIs because of lack of peroxidase producing lactobacilli which allow local cytokine production.

Bacterial vaginosis increases the risk of maternal and fetal morbidity. Bacterial vaginosis is associated with adverse sequelae such as spontaneous abortion, premature rupture of membranes, preterm labour, intrauterine infections (choroiamnionitis), postpartum endometritis, low birth weight babies, neonatal sepsis and cutaneous abscess, congenital pneumonia, jaundice of the newborn ^{43, 45, 46}

Early detection by gram staining and clinical criteria is easy and inexpensive. For the interpretation of Bacterial vaginosis, Nugent's criteria was used. Bacterial groups proposed are 1.large gram positive rods(lactobacilli) 2. small gram negative or variable rods (Gardenella

vaginalis or Prevotella spp) 3. curved gram negative or variable rods (Mobilincus spp). Each group is quantitatively weighted on a scale of 0 to 4.

Score 0 = no morphotype per oil field

Score 1 = less than one morphotype per field

Score 2 = one to four morphotype per field

Score 3 = five to thiry morphotype per field

Score 4 = more than thirty morphotype per field.

A total score of 7 to 10 is considered to be indicative of Bacterial vaginosis.

Systemic antimicrobial therapy for bacterial vaginosis is necessary to prevent these complications. ^{43,46}

GENITAL HERPES IN PREGNANCY

The incidence of genital herpes is increasing worldwide and at present, Herpes simplex virus type 2 is the most common cause of genital ulceration all over the world.⁷ Antenatal cases infected with herpes simplex virus differs considerably according to the incidence of STI in the area, but it is roughly 1%. According to various reports from developed countries the attack rate per 100 pregnancies for herpes simplex virus is 12%.²¹

Primary herpes simplex virus is symptomatic in only one third of

cases, mostly mild and asymptomatic.⁴⁷ Systemic dissemination of herpes virus leading to hepatitis, encephalitis, pneumonia is mostly associated with primary herpetic infection.¹⁰ Maternal mortality with systemic herpetic infection in pregnancy is high.

Up to 80% of pregnant women with genital herpes simplex virus infection have recurrences during pregnancy, on an average 2 to 4 times more frequently in the third trimester. ^{10, 12} Cervical involvement is less frequent with recurrent infection.

Recurrences in pregnancy will be asymptomatic in 10% and frequently involve perineum, buttock, back and thighs and this allows consideration for vaginal delivery.

Acquisition of genital herpes during pregnancy has been associated with spontaneous abortion, premature labour, intrauterine growth retardation, fetal death, congenital and neonatal herpes.¹⁰

Frequency of these complications is higher with first episode of herpetic infection. Primary infection in the first trimester may be teratogenic but the risk is probably minor and is not an indication for therapeutic abortion. ²²

Third trimester primary genital herpes infection carries a higher risk of fetal and neonatal involvement due to maternal viremia. ¹⁰

The major perinatal problem is neonatal herpes infection. Herpes infection acquired during labour is about 90% by direct contact with

infected maternal genital secretions. In 5% of cases in utero (ascending infection or transplacental) and in another 5% of cases herpes infection is acquired post partum.⁴⁹

If herpes infection is present clinically the risk of neonatal infection is higher than from asymptomatic maternal viral shedding. Newborn infection presents as three forms namely asymptomatic, localised, and disseminated.

The laboratory techniques used to establish an accurate diagnosis of herpes infection include virologic, serologic and cytological methods.²¹ The gold standard diagnostic test to detect the presence of herpes infection has been the viral culture. But the diagnosis of herpes infection is still made largely on clinical grounds.³⁶

Under ideal circumstances routine prenatal screening for detection of genital herpes could be advocated in all pregnant women. Caesarean delivery is indicated in women with an active genital lesion at the time of labour.

HEPATITIS B VIRUS INFECTION IN PREGNANCY

The prevalence of chronic hepatitis B viral infection in the general population worldwide is 360 million out of which 78% of infected individuals are seen in Asia.⁵⁰ HBV carrier rate in India varies from 2 to 5%.⁵¹

Overall prevalence of hepatitis B surface antigen in pregnant women in several Indian studies varies from 2.6 to 12%. It is estimated that 1.17 to 1.64% of infants out of 24 million births occurring annually in this country would be infected with hepatitis B virus perinatally.

The chances of acquiring hepatitis infection are directly proportional to the number of sexual contacts and maximum positivity of surface antigen in the second and third trimester.

There is no evidence that the clinical presentation or the course of the disease in healthy pregnant women differs from that in non pregnant women ⁵³

Acute viral hepatitis during pregnancy may cause spontaneous abortion, premature labour, and intrauterine death. ^{22,36} However the major concern in pregnancy is vertical transmission to the neonate. ³⁶

Infection of fetus is thought to occur either by transplacental route or during birth process. The risk of perinatal infection is 80 to 90% when maternal disease occurs in the third trimester. By contrast only up to 10% become infected by maternal infection occurring earlier in gestation.²² The frequency of transmission is relatively low when the mother is an asymptomatic carrier of hepatitis B surface antigen.³⁶

The risk of perinatal transmission is 10 to 20% among women who are HBs Ag positive and HBe Ag negative, but rises to 50 to 90% among

HBe antigen positive women. 22,54

Routine screening of pregnant women for HBs antigen is suitable in interrupting vertical transmission of hepatitis B viral infection. For high risk mothers who are antigen negative, vaccine can be provided during pregnancy.

MOLLUSCUM CONTAGIOSUM IN PREGNANCY

This is a common asymptomatic viral disease of vulval skin. A usual crop includes up to 20 lesions but hundreds may exist in an immunocompromised host. The diagnosis is often clinical but confirmation may include histological evidence of molluscum bodies. ²¹

SCABIES/PEDICULOSIS IN PREGNANCY

No reported effect on pregnancy. 12

HUMAN PAPILLOMA VIRUS INFECTION IN PREGNANCY

Genital papilloma virus infection affects young women of 16 to 25 years, which is also the age group with highest rate of pregnancy occurs. Genital human papilloma viral infection has the prevalence of 20 to 40% in the sexually active women under the age of 30 years.

True incidence of genital warts in pregnancy is uncertain. 28% of

individuals were seropositive for HPV 16 viral capsid antibodies. 12

Human papilloma virus subtype 6 & 11 are most commonly associated with genital warts. But it may also be caused by 16 & 18 oncogenic subtypes.

Women appear to manifest HPV infection as symptomatic warts during pregnancy. Human papilloma viral infection is usually multifocal and most women with vulvar lesions also have cervical infection and vice versa. 15,22

Genital warts frequently increase in size and number during pregnancy and mechanically obstruct labour. Increased vaginal mucus secretion throughout pregnancy offers ideal moist conditions for viral growth and accelerated viral replication with advancing pregnancy and progression to cervical neoplasm. HIV infection also encourages the growth of warts in pregnancy. The warts may regress spontaneously in the postpartum period either due to loss of vascularity, excessive moisture or due to immunosuppression of pregnancy.²²

Transmission by ascending or transplacental route may be possible in the antepartum period. In the intrapartum period transmission rate is very low. Risk of transmission of the virus in the neonatal oropharynx is 30%. ^{12,55} But the virus was cleared by 5 weeks.

Occasionally baby develops genital, perigenital or perianal warts or papillomas of respiratory mucosa. Perinatal transmission during vaginal delivery is the likely mode of spread, since the child borne of caesarean section seems to have lower risk of acquiring recurrent respiratory papillomatosis. ^{12,56} But caesarean delivery is not entirely found to be protective against juvenile onset recurrent respiratory papillomatosis [JORRP]. ⁵⁷

The clinical appearance of genital warts is characteristic but histopathology is confirmatory.²²

HUMAN IMMUNODEFICIENCY VIRUS INFECTION IN PREGNANCY

HIV and AIDS epidemic continues to advance at an alarming rate in India. Over 5 million individuals are already infected with HIV & AIDS and women constitute 1.5 million and 84% of these women are in the child bearing age group of 15 to 45 years.⁵⁸

Screening women for antibody to HIV in various geographic areas throughout the world has shown the seroprevalence to be from 0.5 to 20%. ⁵⁹

In western countries HIV-1 seroprevalence among women of child bearing age is generally under 1%, wheras in developing countries the seroprevalence among pregnant women can be as high as 32% and about 1600 infected infants are born daily. Every year approximately 20,000 deliveries are likely to be conducted in seropositive Indian mothers.

Surveillance data of HIV infection among antenatal mothers by NACO in Tamil Nadu was 0.64% and the seroprevalence of HIV infection was in the range of 0.5 to 3.3%.

Prevalence is said to be on rise and no longer confined to high risk population group. Prevalence of HIV infection among STD clinic attendees in Tamil Nadu is 8.4%. 60 About 89% of pediatric HIV infection occurs by mother to child transmission.

It is estimated that in India almost 30% of HIV infection occurs in those women who have sexual intercourse with a single male partner. Many women who delivered an infant who subsequently develops AIDS have been asymptomatic during pregnancy.⁶¹ But it is noted that seropositive pregnant women have increased incidence of infections such as gastroenteritis, pyelonephritis, pneumonia, and cellulites as complication.⁶²

Ulcerative STIs increase the risk of acquiring HIV infection by 10 fold and genital discharges increases the risk by 5 fold.

Male to female transmission is 17 times higher than female to male transmission. Younger women's immature cervix, relatively low vaginal mucous production and larger surface area of mucosa exposed during intercourse and higher viral concentration favours male to female transmission. ⁶³

For HIV infected women it is unknown whether pregnancy by

itself or possibly along with certain cofactors such as genitourinary infection increases the rate of progression to AIDS. Immuno suppression including fall in CD-4 lymphocyte count allow extended viral replication and disease progression. In addition antigenic stimulation of fetal tissues during pregnancy could lead to T cell activation and viral replication.

According to certain studies in seropositive mothers there has been high incidence of premature rupture of membranes, preterm labour, intrauterine fetal death, low birth weight and higher neonatal death rate which increase further with co-existing STIs.

Rupture of membranes, chorioamnionitis and other maternal STIs favours transmission of HIV infection to the fetus. Adverse pregnancy outcomes are associated with CD-4 count proportion less than 14%.

The rate of vertical transmission of HIV from mother to the fetus varies between 15 to 48%. Transplacental transmission can occur as early as 8 weeks of gestation. The risk factors associated with transmission are low CD-4 count, decreased CD-4: CD-8 ratio, plasma viremia, p24 antigen levels, high viral copy number, AIDS defining illness/symptomatic disease in the mother, placental membrane inflammation, prematurity, prolonged breast feeding. 62,65

Few studies show increased risk with female gender of the infant, higher maternal age, maternal vitamin A deficiency, premature rupture of

membranes for more than 4 hours, smoking and drug abuse. The presence and amount of virus in the genital tract may affect transmission risk.⁶⁶

Transmission of virus from the mother to infant may occur during gestation by crossing the placenta, during delivery by contact with maternal blood and body fluids and post partum via breast feeding.⁶⁶

Studies show inconsistent results that whether women with monocytotrophic HIV strains or T cell line tropism viruses are more likely to transmit the disease to the offspring.⁶⁷

Intra partum transmission is thought to account for more than half of perinatal infections. The risk of transmission is most likely in the third trimester and at the time of labour. Absolute risk of in utero transmission is 5 to 6% and intra partum is 13 to 48% and via breast feeding is 16%. ⁶⁸

The newborns immature gastro intestinal system with low gastric acidity, thin mucosa and microvilli facilitate HIV transmission.

So the best way to prevent maternal to child transmission is by offering integrated counselling and testing services to all antenatal women. ⁵⁸

AIMS AND OBJECTIVES

- 1. To determine the prevalence of sexually transmitted infections including HIV among the pregnant women.
- 2. To study about the age distribution, socio economic status and Educational level of the pregnant women.
- 3. To study the prevalence of concomitant sexually transmitted infections among the pregnant women.

MATERIALS AND METHODS

NATURE OF THE STUDY

It is a prospective observational study to find out the prevalence of STIs among the pregnant women attending the STD outpatient department at the Institute of Venereology, Government General Hospital, Chennai from January 2006 to June 2007.

MATERIALS

Out of 140 pregnant women attending STD outpatient department at Institute of Venereology, Government General Hospital, Chennai, 90 pregnant women was found to have sexually transmitted infections.

Majority of the pregnant women were referred from Institute of Obstetrics and Gynecology, Egmore, Chennai and Kasthuriba Gandhi Hospital for Women and Children, Triplicane, Chennai, Corporation Hospitals and other Hospitals. Pregnant women were referred for VDRL reactivity. Also many attended on their own for various genitourinary symptoms.

Unmarried pregnant women were brought from vigilance home to screen for sexually transmitted infections including HIV infection.

METHODS

A well structured proforma was prepared and used for the study. It consists of various informations including their age, socio economic status, marital status, sexual history and obstetric history apart from detailed clinical examination. Pregnant women were screened for sexually transmitted infections and a provisional diagnosis was made. Routine and special investigations were done accordingly to confirm the diagnosis.

SPECIMEN COLLECTION

Vaginal discharge was examined after adding one drop of normal saline for wet mount and 10% potassium hydroxide one drop is added for KOH mount and for Whiffs test. pH of the discharge was noted. Discharge was also smeared on glass slide for Gram stain.

In case of genital ulcer, after thorough cleaning of ulcer, serous exudate was used for dark field microscopy. Smear was taken from the base of the ulcer for Tzanck smear and Gram staining was also done. Tissue smear was taken and stained with leishman's stain for Klebsiella granulomatis.

MICROSCOPY

Wet mount Examination

This is a simple diagnostic procedure used to visualize normal epithelial cells, motile trichomonads, clue cells, pus cells and also candidal hyphae in some cases.

KOH Mount

This test is used for the diagnosis of genital candidiasis. Addition of pottasium hydroxide to vaginal discharge gives fishy amine odour in bacterial vaginosis.

Gram stain

A thin smear was prepared on a clean glass slide by rolling the swab on the slide. After air drying and heat fixing the smear, it was stained with gram stain. Smear was examined under microscope to look for the presence of epithelial cells, polymorphonuclear leucocytes and the intracellular and extracellular gram negative diplococci, clue cells of bacterial vaginosis, candidal hyphae and spores and streptobacilli in school of fish appearance.

Dark field Examination

Treponema pallidum is demonstrated from moist lesions of primary, secondary syphilis using dark field examination. Clear serous exudate from the lesion is obtained on a glass slide after cleaning the lesion using physiological saline and abrading the lesion with dry gauze.

This slide is examined immediately under dark field microscope.

Tzanck smear

This smear can be used for the diagnosis of genital herpes. Scrapings from the vesicle, base of the ulcer was stained with Leishman's and examined for the presence of multi nucleated giant epithelial cells.

Culture of Candida species

Sabouraud's Medium is the commonest fungal culture media used. It consists of ingredients like glucose, cycloheximide, agar etc. Sterile vaginal swabs were used for taking specimens, cultures were incubated at room temperature for 2 weeks.

Identification is based on the colony appearance (creamy, white and mucoid) and the morphology of the fungus by microscopy. The method of identifying Candida albicans was based on the ability to form germ tubes within two hours when incubated in human serum at 37°C.

SEROLOGY

Blood samples were collected for VDRL test, Trepenonema pallidum haemagglutination test, for detection of Hepatitis B surface antigen by Enzyme Linked ImmunoSorbent Assay and for detection of HIV specific antibodies by ELISA. HIV Positive patients were registered

in well health clinic and CD-4count along with investigations for opportunistic infections were done.

RESULTS

In this study, 90 pregnant women were examined and following

results were obtained.

1. AGE DISTRIBUTION

Table No: 1 – Age distribution of pregnant women (n=90)

Age group in years	Total No.	Percentage
16 -20	27	30
21-25	42	46.7
26-30	14	15.6
31-35	4	4.4
Above 35	3	3.3

Majority of the pregnant women were in the age group of 21-25years (46.6%). The youngest and oldest pregnant women encountered in the study were aged 17 and 39 respectively.

2. EDUCATIONAL STATUS

Table No: 2 – Educational status of pregnant women (n=90)

Education	Total No.	Percentage (%)
Uneducated	13	14.4%
Upto 5th	21	23.3%
Upto 10th	39	43.3%
+1 &+2	14	15.6%
College	3	3.3%

Majority of the pregnant women studied upto 10th standard (43.3%). Only 3 women obtained degree (3.3%).

3. SOCIO-ECONOMIC STATUS

Table No: 3 - Socio-economic status of pregnant women (n=90)

Monthly Income	Total No.	Percentage (%)
Up to 1000	51	56.6%
1000-2000	29	32.2%
2000-3000	6	6.7%
Above 3000	4	4.4%

Majority of the pregnant women belonged to lower socio-economic strata (56.7%), i.e. income less than Rs.1000/month.

4. MARITAL STATUS

Table No:4 – Marital status of the pregnant women (n-90)

Marital status	Total No.	Percentage (%)
Married	85	94.4%
Single (unmarried)	5	5.6%

Majority of the pregnant women were married (n=85, 94.4%).

5. REFERENCE

Table No: 5 – Referral status of the pregnant women (n=90)

Reference	Total No.	Percentage (%)
IOG	32	35.6

KGH	7	7.8
Other Hospitals	28	31.1
Self	8	8.9
Contact sake	7	7.8
Vigilance Home	6	6.7
NGO	2	2.2

Majority of the pregnant women were referred from Institute of Obstetrics and Gynecology (35.6%), and also from other hospitals like Kasthuriba Gandhi hospital (7.8%) and from corporation hospitals. Few attended STD outpatient department on their own for genital complaints (8.9%) and for contact sake (7.8%).

6. DURATION OF PREGNANCY

Table No: 6 – Duration of pregnancy among women (n=90)

Trimester	Total No.	Percentage (%)
First	16	17.8
Second	41	45.5
Third	33	36.7

Majority of the pregnant women were in the second trimester (45.5%).

7. PRESENTING COMPLAINTS

Table No: 7 – Presenting complaints of pregnant women (n=90)

Complaints	Total No.	Percentage (%)
Check up	32	35.6
Genital discharge	22	24.4
Soreness of genitalia	15	16.7
Itching in Genitalia	14	15.6
Growth in genitalia	12	13.3
Ulcer in genitalia	9	10
Swelling in genitalia	8	8.9
Burning micturition	4	4.4
Lower abdominal pain	1	1.1

Majority of the pregnant women had visited the STD outpatient department for check up (35.5%). Some pregnant women attended the outpatient department with multiple complaints. Genital discharge (24.4%) and soreness of genitalia (16.7%) were the most common presenting complaints, followed by itching in the genitalia (15.6%), ulcer in the genitalia (10%) and growth in the genitalia (13.3%). Some pregnant women also had other symptoms like burning micturition (4.4%), and lower abdominal pain (1.1%).

8. SEXUAL HISTORY

Table No: 8 – Premarital / extramarital contacts among married

Pregnant women (n=85)

Contacts	Total No.	Percentage (%)
Only MC	75	88.4
MC + EMC	6	7
PMC + MC	3	3.5
PMC + MC + EMC	1	1.1

When married pregnant women were interwieved for this study, around 7% gave the history of extramarital contact (n=6) and 3.5% gave the history of premarital contact (n=3). One woman gave history of both premarital and extramarital contact.

9. STATUS OF UNMARRIED PREGNANT WOMEN

Table No: 9 – Status of the unmarried pregnant women (n=5)

Group	Total No.	Percentage (%)
C.S.W.	3	60
Kept	1	20
Deserted	1	20

In the women who were unmarried, majority were commercial sex workers with history of multiple partners (n=3, 60%). One woman was kept mistress (20%).

10.PAST HISTORY OF SEXUALLY TRANSMITTED INFECTION

Table No: 10 – Past history of STIs (n=90)

Past H/O STIs	Total No.	Percentage (%)
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Present	8	8.8
Absent	82	91.1

Table No: 11 – Distribution of previous STIs (n=8)

Disease	Total No.	Percentage (%)
Discharge	4	50
Wart	2	25
Ulcer	2	25

Among the 8 pregnant women who gave the past history of STI, 4 had genital ulcer (50%), 2 had genital discharge (25%), and 2 gave history of warts (25%).

11. BAD OBSTETRIC HISTORY (BOH)

Table No: 12 – BOH among the pregnant women (n=17)

ВОН	Total No.	Percentage (%)
Spontaneous abortion	5	29.4
Neonatal death	3	17.6
Preterm labour	2	11.8
Abortion +IUD	3	17.6
Abortion + Preterm	2	11.8
IUD	1	5.8
Low Birth Weight	1	5.8

Bad obstetric history was found in 17 patients (18.8%) out of 90 pregnant women screened in the study. Majority of them had spontaneous abortion (29.4%), followed by neonatal death in 3 patients (17.6%),

preterm labour (11.8%). Three women (17.6%) had both spontaneous abortion and intrauterine death (IUD) and only one women had low birth weight (5.8%).

12. CLINICAL SIGNS

Table No: 13 – Clinical signs in the pregnant women (n=90)

Clinical sign	Total No.	Percentage (%)
Soddening of vulva	20	22.2
Genital wart	18	20
Genital ulcer	13	14.4
Odema of vulva	4	4.4
Bilateral inguinal lymphadenopathy	3	3.3
Bartholin's cyst	3	3.3
Excoriations of vulva	1	1.1
Molluscum contagiousum	1	1.1
Condylomata lata	1	1.1
Furunculosis of Vulva	1	1.1
Intertrigo groin	1	1.1
Tinea cruris	1	1.1

Soddening of Vulva was the most common clinical sign seen in 21 women (22.2%). Genital wart was the next common sign seen in 18 women (20%), followed by genital ulcer (14.4%). Out of 14 women with genital ulcer, one had single painless ulcer suggestive of chancre, eight women had multiple painful superficial ulcers of herpes genitalis and others had linear superficial fissures suggestive vulvovaginal candidiasis. Bartholin's cyst was seen in 3 women (3.3%), molluscum contagiosum

lesion was seen in one women(1.1%). Genital warts were distributed over labia majora, labia minora, fourchette.

Table No: 14 – Nature of genital discharge (n=90)

Nature of discharge	Total No.	Percentage (%)
Mucoid	42	46.7
Mucopurulent	26	28.9
Curdy white	21	23.3
Frothy	1	1.1

Majority of pregnant women had mucoid discharge (46.7%).

13. RESULTS OF INVESTIGATION IN THE STUDY GROUP

Table No: 15 - Results of investigation (n=90)

Investigations	Positive results	Percentage (%)
Culture for candida spp.	27	30
Gram stain for candida	23	25.6
KOH mount for candida	21	23.3
Gram stain for clue cells	18	20
Tzanck smear for GEC	8	8.9
Wetmount	4	4.4
DF for Treponema	2	2.2

Smear for gonococcus and culture for gonococcus were negative in all pregnant women in the study.

Vaginal discharge for culture of candida species in sabouraud's dextrose agar medium revealed growth in 27 cases (30%). Clue cells were

demonstrated by wet mount and gram stain in 18 women (20%), and Tzanck smear shows giant epithelial cells in 8 women (8.9%), wet mount shows trichomonas vaginalis in 4 patients (4.4%) and treponema pallidum was demonstrated by dark field microscopy in 2 patients (2.2%).

14. SEROLOGY RESULTS AMONG THE PREGNANT WOMEN

Table No: 16 - - Prevalance of VDRL Reactivity (n=90)

Serological test	Patients with reactive serology	Percentage(%)
VDRL	16	17.7

Blood VDRL was reactive in 16 (17.7%) patients and non reactive in 74 (82.2%) patients.

Table No: 17 – Distribution of VDRL titer (n=16)

VDRL Titer	Total No.	Percentage (%)
1 dilution	3	18.8
2 dilution	3	18.8
4 dilution	6	37.5
16 dilution	1	6.2
32 dilution	2	12.5
64 dilution	1	6.2

One Woman with secondary syphilis have VDRL reactive in 64 dilutions (6.2%). VDRL was reactive in 4 dilutions in 6 (37.5%) patients.

Table No: 18 - Prevalance of TPHA Reactivity (n=90)

Serological test	Patients with reactive serology	Percentage(%)
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TPHA	16	17.7
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Blood TPHA was reactive in 16 (17.7%) patients and non reactive in 74 (82.2%) patients.

Table No: 19- Prevalence of HBs Ag (n=90)

Serological test	Patients positive for HBs Ag	Percentage (%)
HBs Ag	2	2.2

ELISA test for Hepatitis B surface antigen was positive in only 2 patients (2.2%). It was negative in 88 Patients (97.8%).

Table No : 20– Prevalence of HIV infection (n=90)

Serological test	Patients positive for HIV	Percentage (%)
HIV	6	6.7

Out of 90 pregnant women, 6 women (6.7%) were found seropositive and 84 women (93.3%) were negative for HIV anti bodies.

15. INFECTIONS DIAGNOSED BY CLINICAL EXAMINATION AND INVESTIGATIONS IN THE STUDY GROUP

Table No : 21 – Distribution of infections among pregnant women(n=90)

Infections	Total no.	Percentage (%)
Vulvovaginal candidiasis	27	30
Genital wart	18	20
Bacterial vaginosis	18	20
Syphilis	16	17.8
Genital herpes	8	8.9
HIV	6	6.7
Trichomonas vaginalis	4	4.4
Bartholins cyst	3	3.3
Non-gonococcal urethritis	4	4.4
Hepatitis-B	2	2.2
Molluscum contagiosum	1	1.1

Out of 16 patients with syphilis, one had primary syphilis (6.2%), one had secondary syphilis (6.2%), 12 had early latent syphilis (75%) and two had late latent syphilis (12.5%).

Table No: 22 – Infection among unmarried pregnant women (n=5)

Infections	Total No.	Percentage (%)
Trichomonas vaginalis	2	40
Syphilis	1	20
Bacterial vaginosis	1	20
Vulvovaginal Candidasis	1	20

Among the unmarried pregnant women (n=5) 2 had trichomonas vaginalis infection (40%), and one woman had primary syphilis (20%).

Table No: 23 – Infection among HIV positive pregnant women (n=6)

Infections	Total No.	Percentage (%)
Genital wart	2	33.3
Bacterial vaginosis	1	16.7
Vulvovaginal candidiasis	1	16.7
Molluscum contagiosum	1	16.7

Among the 6 HIV positive pregnant women examined and investigated 5 had single or multiple infections. Vulvovaginal candidiasis was found in one woman (16.7%), two women had genital wart, (33.3%) and molluscum contagiosum in one woman (16.7%).

16. DISTRIBUTION OF INFECTION AMONG HUSBANDS OF MARREID PREGNANT WOMEN

Table No: 24 – Infections among husbands of married pregnant women (n=37)

Infections	Total No.	Percentage (%)
Genital wart	13	35.1
Syphilis	9	24.3
HIV	6	16.2
Genital herpes	5	13.5
Candidiasis	2	5.4

Among the husbands (n=35) examined and investigated, thirteen had genital wart (35.1%), nine had early latent syphilis (24.3%) and five had genital herpes (13.5%).

17. CONCOMITANT INFECTIONS IN THE STUDY GROUP OF PREGNANT WOMEN

Table No: 25 Concomitant infections in the study group (n=90)

Infections	Total No.	Percentage(%)
Candidiasis + Genital herpes	2	22.2
Candidiasis + Bartholins cyst	2	22.2
Candidiasis + Bacterial vaginosis	2	22.2
Candidiasis + Bacterial vaginosis	1	11.1
Candidiasis + Bacterial vaginosis +TV	1	11.1
Candidiasis + HIV	1	11.1
Bacterial vaginosis + Genital wart	1	11.1
Bacterial vaginosis +Syphilis	1	11.1
Bacterial vaginosis + Hepatitis-B	1	11.1
Bacterial vaginosis + HIV	1	11.1
Trichomonas vaginalis + Genital	1	11.1
Trichomonas vaginalis + Syphilis	1	11.1
HIV + Genital wart	1	11.1
HIV + Molluscum contagiosum	1	11.1

DISCUSSION

In this study, majority of the pregnant women were in the age

group of 21-25(46.7%), followed by 16-20 years of age. A significant number of pregnant women were below 20 years. This study has confirmed that adolescent sexual activity has increased resulting in rise in unintended pregnancy rate. Due to early initiation of sexual activity, they are more prone for STIs including HIV. As they often resort to Medical Termination of Pregnancy, services offered to them should include check up for sexually transmitted diseases and knowledge about contraception.

Majority of the pregnant women belonged to lower socio-economic strata. Lower socio-economic status often co-exists with poor nutritional status of pregnant women which in turn affect the course of the disease and their obstetric outcomes.

Majority of the pregnant women in this study were married (94.4%). A considerable number of pregnant women were unmarried. They form an important risk group for acquiring STIs including HIV infection.

Majority of the pregnant women were reffered to the STD outpatient department for VDRL reactivity and genital lesions. A good number pregnant woman presented with various genitourinary symptoms on their own along with their partners. This increase in number of self referral by these women could be as a result of awareness about sexually transmitted infections including HIV.

Nearly one third of the pregnant women reported only for check

up. However on clinical examination and completion of investigations, it was found that they were suffering from one or other infections. This shows that majority of the pregnant women were asymptomatic. Among the asymptomatic pregnant women (n=28), 11 were found to be affected with early latent syphilis, 8 with bacterial vaginosis, 2 with Hepatitis-B, and 2 with HIV infection.

The significance of this is that pregnant women may not be aware of the silent clinical status of syphilis, Hepatitis-B and HIV. This may lead to delay in detection, progression of the disease to complications and vertical transmission. Early diagnosis by routine antenatal screening for STIs will greatly help in the detection in the initial stages so that effective interventions can be done.

Genital discharge, genital soreness and itching genitalia were the most common symptoms noted among the study group.

Majority of the married pregnant women denied history of premarital or extramarital contacts (88.4%). This stresses the fact that innocent housewives were infected by their promiscuous husbands and were exposed to high risk of acquiring STIs including HIV infection resulting in morbidity and mortality. Their unborn and newborn children were also at risk.

Genital discharge (50%) was the commonest among previous venereal diseases noted. There is an increased rate of recurrence of genital

herpes during pregnancy. 11, 12

Bad obstetric history was noted in a significant proportion of pregnant women (18.8%). Majority of them had spontaneous abortion in the past followed by neonatal death. Among these women (n=17), majority were found to have syphilis (n=5, 29.5%). The other STIs noted were Bacterial vaginosis (n=4, 23.5%), HIV (n=2, 11.8%), Genital herpes and trichomonas infection.

In this study group, soddening of vulva, genital warts and genital ulcer were the most important clinical signs noted.

Generalised lymphadenopathy is an important sign of secondary syphilis and HIV disease. Among the pregnant women, one had generalized lymphadenopathy (1.1%). She was found to have HIV infection.

Moist grayish white, flat topped papules which are highly infective in nature were seen in one pregnant woman. Untreated maternal secondary syphilis carries high risk of perinatal complications.

In this study group 6 cases were found to HIV seropositive. These women were screened to detect signs of AIDS defining illnesses early.

In this study, mucopurulent vaginal discharge was most commonly seen. Patients with Bacterial vaginosis (n=18) and Trichomonas vaginalis vaginitis had predominantly mucopurulent discharge and typical frothy vaginal discharge of Trichomoniasis was seen in only one woman.

The usual description of discharge in vulvovaginal candidiasis is curdy white and adherent. In this study group 77.7% of the pregnant women had above said clinical sign and remaining had only itching in genitalia. Culture for gonococcus was negative in the study group.

About 30% of the pregnant women had genital candidiasis. This study confirmed the aspect that vulvovaginal candidiasis is more common in pregnancy. Vulvovaginal candidiasis co-existed with many other STIs in this study.

The prevalence of VDRL/TPHA reactivity was 17.7%. This is comparable with prevalence rate of syphilis in STD clinic attendees is 17-30% and the prevalence is between 3-19% in developing countries.

The prevalence of Genital wart in the group of pregnant women in the study was 20%. This high prevalence data could be due to high risk population group screened. ⁵⁶

The prevalence of Bacterial vaginosis in this study population is 20%. This is comparable with prevalence rate of 12 to 23 % in pregnant women in India. ⁴⁵

The prevalence of Genital herpes in the study population is 8.9%. This high prevalence data could be due to high risk population group screened.

In this study group, prevalence of HIV was 6.5% which was relatively higher than some Indian studies.⁶¹ This could be due to

screening of high risk population in the study.

The prevalence of HBs Ag in the study group was 2.2%. This was comparable with Risbund, et al study from Chandigarh. ²⁷

Prevalence of Trichomoniasis in the study population was 4.4%. This was comparable with 6% prevalence of Divekar AA, et al study from Mumbai. 12

The most important sexually transmitted infections found in unmarried pregnant women (n=5) were Trichomoniasis (40%), Bacterial vaginosis (20%), Syphilis (20%), candidiasis (20%). This prevalence data is higher than married pregnant women.

Among the HIV positive pregnant women in this study group, viral STIs were found to be more common than bacterial STIs, which is consistent with other studies. ³⁵

Health education regarding transmission of sexually transmitted infection, safer sex and counselling should be made available to them. Prompt treatment of infected person and their partners by early detection will prevent further transmission. It will also minimize the severity of long term sequelae.

The larger number of pregnant women with an STI provides an important opportunity to reduce the reservoir of infection in the broader community. Hence systematic screening of sexually transmitted infection in pregnancy combined with adequate treatment and follow up will

reduce the risk of maternal, fetal and neonatal adverse consequences.

Appropriate antenatal, intrapartum and postnatal care should be provided.

Unmarried pregnant women have an increased risk of acquiring STIs, hence counselling services, knowledge regarding use of contraceptives and provision of STI care should be made available to them.

Sexually transmitted infections often coexist and a search for them should be instituted in every patient.

CONCLUSION

- Vaginal discharge was more common than genital ulcer diseases. Vulvovaginal candidiasis (30%) was the commonest STI among the pregnant women, followed by Genital wart (20%), Bacterial vaginosis (20%) and syphilis(17.8%).
- HIV infection was prevalent in 6 pregnant women (6.7%).
- Hepatitis B was prevalent in 2 pregnant women (2.2%).

- Around 88.8% of the pregnant women belong to the lower socio economic status i.e., less than Rs.2000/- per month. Nearly 76.7% of pregnant women belong to the age group of 16 25years.
- Nearly 83.4% of the HIV infected pregnant women were affected with other sexually transmitted infections. Genital wart was the commonest viral infection among HIV infected pregnant women (33.3%).
- Vulvovaginal candidiasis was coexisting with multiple STIs among pregnant women.

Routine screening for sexually transmitted infections in all pregnant women should be included in the antenatal care. Effective treatment services should be made widely available for those who are found to be infected.

BIBLOGRAPHY

- 1. WHO "Global prevalence and incidence of selected curable sexually transmitted infections –overview and estimate"- WHO Geneva 2001
- 2. Wasserhurt J N. Epidemiological synergy "Interrelationship between HIV and other sexually transmitted diseases" Sexually Transmitted Diseases 1992.19;61-77
- 3. Greenberg J et al. "Age at first coitus, A marker of risky sexual behavoiur in women" Sexually transmitted diseases 1992.19;331-334
- 4. Singer A "The uterine cervix from adolescence to menopause"
 British Journal of Obstetrics and Gynecology 1975.82;81
- 5. Sridama V et al. "Decreased levels of helper T cells, A possible cause of immunodeficiency in pregnancy"- New England Journal Medicine 1982.37:352
- 6. Paul VK et al. "Chlamydia trachomatis infection among pregnant women, Prevalence and prenatal importance" Natl. Med. J. India 1999.12;11-14
- 7. Gina Dallabetta et al. "Control of Sexually Transmitted Diseases" AIDSCAP\Family Health International. U.S.A. p169-186
- 8. Ambrose King, Claude Nicol and P Rodin. "Venereal Diseases" 4th edition. April 1980
- 9. Lumbiganon P et al. "The epidemiology of Syphilis in pregnancy" International Journal of STD and AIDS 2002.13;486-494
- 10. Katherine A Martens. "Sexually Transmitted and genital tract infections during pregnancy". Pregnant patient Emergency Medicine Clinics of North America. Vol. 12, No.1

- 11. Bartin, Gogath, Karande. "Reproductive Tract Infections, Gynecological morbidity & HIV Seroprevalence among women in Mumbai, India"—Bulletin of WHO 1998.76(3);277-87
- 12.Gray Cunningham, Kenneth J Leveno, Steven L Bloom, John C Haeith, Larry Gilstrap III, Katherine D Wenstrom. "Williams Obstetrics" 22nd edition. 2005 Sexually Transmitted diseases. p1302-1320
- 13.Singh AE et al. "Syphilis: Review with emphasion clinical, epidemiological & some biological features" Clinical Microbiological Review 1999.12;187-209
- 14.Harter CA Benirschkek. "Fetal syphilis in first trimester" American Journal of Obstetrics & Gynecology 1976.70;124
- 15.Ronald S, Gibbs, Richard L Sweet & W Partrick Duff. "Maternal and Fetal Medicine" 5th edition.2004 Maternal and Fetal Infectious Disorders. p773-787
- 16.King K Holmes. "Sexually Transmitted Diseases" 4th edition, 2004
- 17.Centre for disease control and prevention. "Sexually Transmitted Diseases Treatment Guidelines" MMWR 2002:51
- 18.Elliott B et al. Maternal Gonococcal infection as preventable risk factor for low birth weight" Journal of Infectious Diseases 1990.161;531-536
- 19. Amstrong J H et al. "Ophthalmia Neonatorum, A chart review" International Journal of Pediatrics 1976.57;884
- 20.Barton LL, Shija M. "Neonatal Gonococcal vaginitis" Journal of Pediatrics 1981.98;171
- 21.Bhaskar Rao K, SS Ratnam, S Arulkumaran. "Obstetrics and Gynecology for post graudates" Vol. 2, 1st edition. 1996. p370-384
- 22.Margret J Godley. "Sexually Transmitted Diseases in pregnancy" Medicine group (Journal) Ltd 1996

- 23.O Farrel N. "Clinico epidemiological study of Donovanosis in Durban South Africa" Genitourinary Medicine 1993.53;108-111
- 24.Hoozen AA et al. "Granuloma Inguinale in association with pregnancy & HIV infection" Indian Journal of Obstetrics and Gynecology 1996.53;133-138
- 25.Richens J. "Sexually Transmitted Diseases in children developing countries" Genitourinary medicine 1994.70;278-283
- 26.Bowden FJ et al. "Donovanosis causing cervical lymphadenopathy in five month old baby" Pediatric Infectious Diseases Journal 2000.19;167-169
- 27.Bushan Kumar, Somesh Gupta. "Sexually Transmitted Infections" first edition. 2005 Sexually transmitted infections and pregnancy p897-908
- 28.Mc Gregor JA et al. "Prevention of premature birth by screening and treatment of common genital infections, Results of a prospective controlled evaluation" American journal of Obstetrics and Gynecology 1995.173;157-167
- 29.Mc Gregor JA et al. "Chlamydia trachomatis infection during pregnancy" American Journal of Obstetrics and Gynecology 1991.164;1782-1789
- 30. Tibor Nyari, Judith Deak, Elizabeth. "Epistudy of Chlamydia trachomatis infection in pregnant women in Hungary" Sexually Transmitted Diseases 1998.74;213-215
- 31.Andrews WW et al. "The preterm prediction study of association of second trimester genitourinary Chlamydia infection with subsequent spontaneous preterm birth" American Journal of Obstetrics and Gynecology 2000.183;662-668
- 32. Neugen RP, Hiller SL. "Mucopurulent cervicitis as a predictor of Chlamydial infection and adverse pregnancy outcome" Sexually Transmitted Diseases 1992.19;198-202

- 33. Sobel JD et al. "Vulvovaginal candidiasis: Epidemiological, diagnostic and therapeutic consideration" American Journal of Obstetrics and Gynecology 1998.178;203-211
- 34.Foxman B et al. "Frequency and response to vaginal symptoms among white and black women: Results of a random digital dialing survey" Journal of woman health 1998.7;1167-1174
- 35.Ruth M et al. "Lower genital tract infections among HIV infected and high risk uninfected women" Sexually transmitted Diseases March 1999.26;(3)
- 36.Sheldon H Cherry, Irwin R Merkat. "Infections in Pregnancy" Complications of pregnancy- medical, surgical, gynecological, psychosocial & prenatal- Williams/Wilkins 4th edition.1991 p302-383
- 37.Petrin D et al. "Clinical and microbiological aspects of Trichomonas vaginalis" –Clinical Microbiological review April 1998.12;300-317
- 38.M Passey et al. "Screening for Sexual Transmitted Diseases in rural women in Papua New Guinea" Bulletin of WHO 1998.76(4);401-411
- 39.Cotch M F et al. "Trichomonas vaginalis associated with low birth weight and preterm delivery" Sexually Transmitted Diseases 1997.24;1-8
- 40.Draper D et al. "Trichomonas vaginalis weakens Human amnion in an invitro model of Premature rupture of membranes" Infectious co infection with vaginal trichomoniasis" British Journal of Venereal Diseases 1976.52;58
- 41.Bramely M. "Study of female babies born of women entering co infection with vaginal trichomonas" British Journal of Venereal Diseases 1976.52;58
- 42.Klein LL et al. "Use of microbial cultures and antibiotics in prevention of infection associated preterm birth" American Journal of Obstetrics and Gynecology 2004.194;1493-1504

- 43.Hiller SL et al. "The role of Bacterial vaginosis & vaginal bacteria in amniotic fluid infection in women in preterm labour with intact fetal membranes" Clinical Infectious Diseases 1995.20(2);276-278
- 44.Ronald F Lamout et al. "A comparison of the use of papanicolaou stained cervical cytological smears with Gram stained vaginal smears for the diagnosis of Bacterial vaginosis in early pregnancy" International Journal of STD & AIDS 1999.10;93-97
- 45. Nicole Woodrow, Ronald F Lamout. "Bacterial Vaginosis: Its importance in Obstetrics" Hospital Medicine June 1998.59
- 46.Obstetrics and Gynecology Today May 1999. Vol.4(5);p296-303
- 47.Langenberg AGM et al. :A prospective study of new infection with HSV type 1 & 2" New England Journal of Medicine 1999.341;1432
- 48.Brown ZA et al. "Risk factors associated with recurrences and asymptomatic shedding of HSV" American Journal of Obstetrics and Gynecology 1985.153;24
- 49.Brikic S, Jovanic J. "Genital herpes with special emphasis in perinatal HSV transmission" Medicinski Pregled 1998.51(1-2);49-9
- 50.Harold S Margolis. "Hepatitis B viral Infection" Bulletin of WHO 76(suppl-2);152-153
- 51. Thakur SK et al. "Hepatitis B carrier a study of possible routes of acquiring the infection in Army hospital Delhi cantt and AFMC, Pune" Indian Journal of Gastroenterology march 1999. 18(suppl-1)
- 52.Sharma R et al. "Hepatitis B virus infection in pregnant women & its transmission to infants" Journal of Tropical Pediatrics Dec1996.42(6);352-367
- 53.Sweet RL. "Hepatitis B infection in pregnancy" Obstetrics and Gynecology Report 1990.2;128-139

- 54.American College of Obstetrics and Gynecologist. "Viral Hepatitis in pregnancy" Educational Bulletin July 1998.No.248
- 55.Kemp EA et al. "HPV Prevalence in pregnancy" International Journal of Obstetrics and Gynecology 1992.79;649-656
- 56.Tenti P et al. "Perinatal transmission of HPV from gravidas with latent infection"-International Journal of Obstetrics and Gynecology199.93;475
- 57. Silverber MJ et al. "Condyloma in pregnancy is strong predictor of Juvenile onset recurrent respiratory papillomatosis"-International Journal of Obstetrics and Gynecology 2003.101;645-652
- 58.National AIDS control Programme, India, Country scenario an update 2004. NACO, Ministry of Health and Family Welfare Government of India p30
- 59.Lokeshwar et al. "AIDS in Children-Perinatal transmission and its prevention" Asian Journal of Pediatric practice 1998.2(2);34-38
- 60.Continuum of care for HIV/AIDS Hand book for health care providers Tamilnadu state AIDS Control Society.p35-38
- 61.Royce RA et al. "Bacterial vaginosis associated with HIV infection in pregnant women from North California" Journal of AIDS Human Retrovirol 1999.20;382-386
- 62.Joshi PL et al. "Changing epidemic of HIV/AIDS in India" AIDS Res. Rev.1999.2;
- 63.STI/ HIV/ AIDS prevention Education Doctors training manual CAPACS.p15-18.
- 64.Burns D N et al. "Cigarette smoking, premature rupture of membranes, vertical transmission of HIV among women with low CD4 counts levels"- Journal of AIDS 1994.7;718-726

- 65.Hague RA et al. :Maternal factors in HIV transmission" International Journal of STD & AIDS 1993.14;142-146
- 66.Stratton P et al. "Obstetric and new born outcomes in a cohort of HIV infected pregnant women-A report of women and infants transmission study" Journal of AIDS Human Retrovirol.1999.20:179
- 67.Ometo L et al. "Viral phenotype and host cell susceptibility to HIV-1 infection as a risk factor for mother to child HIV-1 transmission" Journal of AIDS 1995.9;427-434
- 68.Kuhn L et al. "Distinct risk factors for Intrauterine and intra partum HIV transmission and consequences for disease progression in infected children" Journal of Infectious Diseases 1999.179;52-58

PROFORMA

2. STD OP No.

1. Serial No.

3. Name	4.Age			
5. Educational qualification: Uneducated/<5 th /<10 th /+1&+2/college				
6. Income: <1000/1000-2000/2000-3000/>3000				
7. Marital status &Duration of marriage				
8. Occupation	9.Address			
10. Referred by: IOG/ KGH/Self/or	thers			
11. Presenting complaints:				
Duration of amenorrhoea				
Genital ulcer with duration –Single/Multiple Painful/Painless				
Genital discharge with duration – foul smelling/not				
Itching genitalia				
Swelling in the inguinal region -Painful/not				
Skin rash				
Growth in the genitalia				
Burning micturition				
Lower abdominal pain				
Jaundice/ oral lesion/ fever/ joint pain/ CNS disturbances				
12. Obstetric History:				
Months of amenorrhoea	LMP:	EDD:		

Gravida/ Para

Previous obstetric H/O – Sex /Age/Mode of delivery

Menstrual H/O – Regular/ Irregular

13. Treatment History:

Treatment taken for present complaints

History of previous venereal disease and treatment

14.Exposure History:

Recent exposure with dates MC: PMC: EMC:

15. Contact History:

Partner name and card No.

History and investigations report

16. Examination:

General and systemic examination

Examination of genitalia

Inguinal nodes

External urethral meatus

Speculum examination

 $Genital\ lesion-Soddening\ of\ vulva/\ wart/\ ulcer$

Discharge – Scanty/ moderate/ profuse

Mucoid/ mucopurulent/ purulent

Homogenous/ flocullar/ curdy

Foul smelling/ not

Other systems – Skin and mucous membrane

Bones and joints

17. Investigations:

Wet mount - Saline

KOH

Smear - Urethral/Cervical/Vaginal

Culture - Gonococci/Candida/General microbial culture

Ulcer - Dark field for treponema

Gram stain for Hemophilis ducrey

Leishman stain for gaint epithelial cells

Tissue smear for Calymmatobacterium -

granulomatis

VDRL & TPHA

USG Abdomen

VCTC

Hep B

HCV

Urine Routine and Culture sensitivity

Complete hemogram

Random blood sugar