CONTINUING MEDICAL EDUCATION

EFFECTS OF SEXUALLY TRANSMITTED DISEASES ON THE FOETUS AND NEONATE

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Rapid developments in the fields of immunology, microbiology and other branches of medicine and easy availability of improved diagnostic facilities have made us understand in detail the effects of various sexually transmitted diseases (STD) on the unborn foetus and the neonate. In recent years, there has been a tremendous increase in the incidence of these diseases in different parts of the world including India. Parallel to this increase, the number of foetuses and neonates affected by these STDs are also on the increase. Improved diagnostic facilities in developed countries have demonstrated an apparently widening clinical spectrum of these diseases in neonates and infants.

Congenital syphilis

Despite decades of experience with congenital syphilis, problems still arise in case definition, diagnostic evaluation, treatment and follow up.[1] It still continues to be a major health problem in many under-developed and developing countries.[2-5] The incidence of congenital syphilis mostly correlates with the incidence of primary and secondary syphilis.[6] In most developed countries, in spite of an increase in infectious early syphilis, the incidence of congenital syphilis has been brought down by an efficient antenatal care system. In these countries, the only mothers who give birth to congenital syphilitic babies are those who fail to attend antenatal clinics or those who acquire the disease during the later period of pregnancy.[7] Still, even in some developed countries it continues to occur.[8]

Congenital syphilis, more accurately termed prenatal syphilis, is a disorder in which the foetus becomes infected with *Treponema pallidum* by way of the placenta. Though pregnancy has a benign effect on the course of syphilis, the disease is very hazardous in a pregnant woman since she carries the potential risk of transmitting the disease to the foetus. For decades, it was believed that *Treponema pallidum* does not affect the foetus before the 16th week of gestation. It was attributed to the barrier function of the Langhans layer of chorionic villi which gets atrophied only after the 16th week of pregnancy.[9] But in 1976, Harter and Benirschke demonstrated treponema-like organisms in the foetal products terminated on medical grounds, in untreated syphilitic women before the 12th week of gestation, and suggested that Langhans layer is not a barrier for invasion of the foetus by *Treponema pallidum*.[10] The demonstration of persistence of Langhans layer even at the time of delivery in some women, also disproves the old view of its barrier function. In spite of getting infected early during pregnancy,[11] the foetus develops appreciable tissue damage only in the later part of pregnancy after an adequate immune response has developed in the foetus.[12,13] Attenuated virulence of syphilis during the early weeks of pregnancy is perhaps related to the inability of the foetal

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immune system to marshall a plasma cell response or other unrecognizable biochemical requirements of the treponemes in foetuses.[10] Tissue changes recognized as congenital syphilis do not occur until the foetus is approximately 20-week-old.[14,15] The outcome of pregnancy in a syphilitic woman will depend on the duration of pregnancy and on the time when the foetus was infected with *Treponema pallidum*. A foetus infected at the fourth month of gestation may die in utero and will subsequently be expelled from the uterus resulting in abortion. Women with syphilis abort more than 60% of their pregnancies.[16] If on the other hand, infection occurs in the latter half of pregnancy, it will be delivered as a still-born or as a fatally ill neonate.[17] An analysis in Madras showed that in 274 untreated syphilitic women, 30% pregnancies resulted in still-births and neonatal deaths, 30% in living syphilitics and 40% in living non-syphilitics.[18] Out of 41 pregnancies in a group of 19 married sero-positive women detected in a community survey by Kapoor et al 26.8% resulted in abortion, 14.6% in still-births and 58% in live births which included 3 congenital syphilitics.[19] Foetal wastage in Vaidya's study was 37.3%,[20] whereas in Jayasing's it was 63%.[21] Fifty percent of the sero-positive pregnant women studied by Venkitakrishnan had bad obstetric history.[22] History of still-births and second trimester abortions were recorded in 11 out of 20 cases in a study by Adeobar.[23] Sometimes a woman acquiring syphilis late in pregnancy may have infectious lesions on the genitalia. The child born to her may develop lesions of acquired syphilis due to contact with these lesions in the birth canal of the mother, during delivery. In such cases primary syphilitic chancres develop on the face or neck after an appropriate incubation period.

If a woman with untreated syphilis has a series of pregnancies the chance of foetal infection decrease with the number of pregnancies and the length of time elapsed between successive pregnancies. An untreated syphilitic mother may have in successive pregnancies a still-birth followed by an infant with congenital syphilis and then one or more healthy children.[24] Though occasional reports on third generation syphilis are recorded in the literature,[25,26] it does not usually occur because the congenital syphilitic mother becomes non-infectious by the time of her marriage. True third generation syphilis should fulfil all the Fournier-Finger criteria, for its diagnosis.[27] The presence of histopathologic evidence of syphilis including *Treponema pallidum* in the placenta proves the presence of syphilis in the mother but not necessarily in the foetus. The normal placenta at term weighs less than $\frac{1}{4}$ of the weight of the foetus. Syphilitic placenta is usually pale, greasy and bulky and weighs more than $\frac{1}{4}$ weight of the foetus. But these features are seldom used as diagnostic criteria at the present time. Syphilitic still-born foetus may have a macerated appearance with collapse of the skull. Skin is of a livid red colour and is covered with multiple bullae. The florid manifestations of prenatal syphilis in neonates and infants closely resemble the manifestations of secondary syphilis in adults. Uncommon manifestations like ascites, hepato-splenomegaly, protracted diarrhoea and nephrotic syndrome have been recorded.[28-32] Renal lesions are thought to result from the deposition of immune complexes and features of both acute nephritis and nephrotic syndrome may be seen.[29,30,32] Palatal perforation as a manifestation of congenital syphilis in a neonate was reported by Mishra et al.[33] But in the absence of a supportive histopathological evidence of gumma, syphilitic aetiology in this case is uncertain.[34] Even though clinical evidence of CNS involvement is an uncommon finding, CSF abnormalities may be detected in 40-50% of infants with congenital syphilis.[1,35,36]

Congenital syphilis is a completely preventable disease. Its incidence in a community parallels the prevalence of acquired early syphilis and also depends on the efficacy of antenatal care system. Detection of syphilis and its early treatment in a pregnant woman prevents the transmission of the disease to the foetus.[36] There should ideally be 2 serological tests for syphilis during pregnancy, the first one when she presents at the antenatal clinic and the second during the last trimester of pregnancy. In 1973 Bellingham described 2 cases of foetal loss due to transplacental syphilitic infection in the patients in whom routine antenatal screening tests in early pregnancy had been negative.[37] So it is necessary to have serological tests at the beginning of the third trimester and again at the time of admission for labour and delivery. Even a low titre of VDRL reaction should be taken as due to syphilis and appropriate treatment must be instituted in order to forestall foetal wastage, neonatal mortality or infant morbidity. In a recent study by Rattan et al it was observed that 35 of VDRL positive sera out of 1836 antenatal cases had titres below 1 : 8, but 91.4% of these low titre sera were positive by TPHA test.[38] Pregnant women with a reactive serologic test for syphilis in the absence of clinical, historical or epidemiological evidence of syphilis present a difficult diagnostic problem. It is not uncommon to get a biological false positive result to VDRL test with sera from pregnant women.[39,40] Boak et al reported that 73% of the reactive serologic tests for syphilis (STS) in pregnant women were false positive.[41] Wilkinson and Sequeira however, found that 27.5% of 244 sero-positive antenatal patients gave negative TPI reactions.[42] Schofield studied 285 VDRL positive sera from pregnant women, and confirmed syphilis in only 39.[43] In a recent study by Venkitakrishnan, syphilis was detected in only one out of 1000 samples of sera from pregnant women.[22] The large number of biological false positive (BFP) results in the pregnant as compared to the non-pregnant women supports the hypothesis that in some cases, pregnancy or an unknown factor associated with pregnancy results in an alteration of the serum constituents so as to cause reactivity with lipoid antigens employed in STS.

Absolute diagnosis of congenital syphilis depends on the demonstration of *Treponema pallidum* from the skin or mucous membrane lesions of the affected child. A reactive serology in a child born to a syphilitic mother need not always be due to syphilitic infection of the new born. It may be due to passive reaginemia where the reagin is simply transferred from the maternal blood through the placenta to the new born. If there is a true neonatal syphilis, the titre of reagin in the cord blood should show a four-fold increase than the titre in the maternal blood and the titre should rise progressively in the infant in the absence of specific treatment.[44] A negative blood VDRL test in the new born does not always exclude the possibility of congenital syphilis. A pregnant woman getting infected late in pregnancy may give birth to a new born who is initially non-reactive serologically but becomes reactive some months after delivery.[35,45] In such cases, a long follow-up is essential. Active infection of the new born can be detected by FTA-ABS IgM test. But the test has certain fallacies. Some babies may show a delayed onset, the IgM-FTA test being negative at birth but becoming positive after an interval of several weeks.[46,47] So serial tests are required. False positive as well as false negative results to this test occur occasionally.[48] So its practical use is limited. False negative results to FTA-ABS IgM test has been explained by blocking of the IgM receptors on the surface of the antigen by IgG.[49] Mamunes et al proved gamma M FTA test to be an accurate additional way of diagnosing congenital syphilis in the neonatal period.[50]

Early detection and appropriate treatment of syphilis is essential during pregnancy in order to forestall foetal wastage, neonatal mortality and infant morbidity.[38] To meet the need to be constantly on the alert for this protean disease, every pregnant woman should have repeated blood VDRL tests performed during her visits to the antenatal clinic. Penicillin administered to a syphilitic pregnant woman is most effective in preventing infection of the foetus. Erythromycin is the drug of choice in women allergic to penicillin since foetal teratogenicity to erythromycin does not appear to be a hazard.[51] But recent reports reveal that the ability of erythromycin to cross the placenta is very poor.[52] A few cases of congenital syphilis have been reported in infants born to mothers whose early syphilitic infections were treated with erythromycin.[53-56] These reports emphasize the need for a close follow-up of pregnant women after erythromycin treatment for early syphilis. So it is now recommended that all infants born to such erythromycin treated mothers should be given penicillin even if they are not having any clinical or laboratory evidence of congenital syphilis. Benzyl penicillin is the drug of choice for the treatment of syphilis in the neonates. Since a high percentage of syphilitic neonates have associated asymptomatic involvement of the CNS, benzathine penicillin which causes only a low level of penicillin in CSF,[57] cannot be recommended for the treatment of syphilis in the neonates.[58]

Gonorrhoea

Short incubation period, lack of immunity, asymptomatic nature of infection in some males and females, and emergence of beta-lactamase producing strains of gonococci—all have played their role in causing a tremendous increase in the incidence of gonorrhoea in different parts of the world, including India. In women, it is an important cause for cervicitis and salpingitis. In many countries, neonatal gonococcal infection still continues to be a significant problem. Infants lack bactericidal serum IgM antibody to *Neisseria gonorrhoeae* and may therefore be at an increased risk for developing gonococcal sepsis if exposed to this organism.[59] Although many types of conjunctival infections in neonates are relatively inconsequential, gonococcal, chlamydial and herpetic infections can cause permanent ocular damage and serious systemic complications if not detected early and treated adequately.[60] The affected eyes in gonococcal ophthalmia neonatorum are acutely inflamed within 48 hours of birth. The eyelids are swollen and the pus can be seen oozing from between the eyelids. If treatment is not given, the cornea develops ulcers that lead to opacities and permanent blindness. Diagnosis is easy by examination of the purulent material which on Gram-stained smears show Gram negative, reniform, intracellular diplococci. Signs and symptoms similar to gonococcal ophthalmia may rarely be caused by infection with *Branhamella catarrhalis*.[61]

Emergence of beta-lactamase producing strains of gonococci has been reported from different parts of the world including many states of India.[62-64] Incidence of ophthalmia neonatorum due to these strains of gonococci also are on the increase.[65-68] The treatment regimen for penicillin susceptible gonococcal conjunctivitis includes parenteral penicillin, frequent ocular irrigation and topical ophthalmic gentamicin or tetracycline ointments. If the expected therapeutic response is not achieved after penicillin therapy, it should be viewed with suspicion and the organism should be cultured and subjected to tests for beta-lactamase production and for antibiotic susceptibility. If conjunctivitis is caused by penicillinase producing *Neisseria gonorrhoeae* (PPNG), then the antibiotics required are : spectinomycin or cefoxitin parenterally and topical chloramphenicol.[69] Whereas urethritis is frequently treated

with singular injections of penicillin or spectinomycin, cases of PPNG ocular infections require much longer courses of treatment, often involving 7 days of intramuscular or intravenous antibiotics.[69] Single dose kanamycin therapy also has been tried with variable results in these cases.[70] An eye ointment that is effective against both gonococci and chlamydiae is ideal for prophylactic purposes in neonates. Prophylaxis with 1% silver nitrate eye drops has reduced the incidence of gonococcal ophthalmia neonatorum but does not always prevent it. Rarely, it may cause chemical conjunctivitis in the neonate.[60] Substitution of erythromycin for silver nitrate for the prophylaxis of gonococcal ophthalmia in neonates has caused an increase in the incidence of non-gonococcal ophthalmitis. It is attributed to the ointment vehicle, difficulty in its application and the mechanical introduction of bacteria.[71]

In many countries, neonatal gonococcal infection is still a significant health problem. No data exists on neonatal morbidity caused by this infection which however, has been reported to be significantly associated with prematurity, chorio-amnitis, delayed delivery following rupture of membranes and clinical features of neonatal sepsis.[72,73] Oppenheimer et al reported an unusual case of gonorrhoea in utero which resulted in deep seated foetal tissue inflammation. The 30-week-old foetus died in utero 4 hours prior to delivery. Infection therefore occurred by aspiration of the infected amniotic contents rather than by the more common route of passage through an infected birth canal.[74] Gonococcal salpingitis developing during pregnancy[75,76] may be mistaken for rupture of an ectopic pregnancy and sometimes exploratory laparoscopy may be needed for confirmation of the diagnosis. After adequate treatment of salpingitis, the pregnancy will continue without any adverse effects on the foetus.[75]

All pregnant women should have endocervical cultures for gonococci as an integral part of the prenatal care at the time of her first visit in the antenatal clinic. A second culture, late in the trimester should be obtained from women at high risk of gonococcal infection. This simple step will completely eradicate gonococcal ophthalmia neonatorum and the subsequent disastrous complication of blindness. Adequately treated maternal gonorrhoea is without any adverse effects on the outcome of pregnancy.[77]

Chlamydial infections.

The *Chlamydiae* are obligate intracellular micro-organisms belonging to the order chlamydiales. The serotypes D to K are pathogenic to the mucous membranes especially of the genitalia. In women, it produces cervicitis and salpingitis.[78,79] High prevalence of the genital chlamydial infection has been reported in pregnant women.[80,81] In them, it has been implicated for still birth, neonatal death, ectopic pregnancy, spontaneous abortion, intrauterine death and prematurity.[82-84] The risk of foetal or neonatal death is increased ten-fold if the mother has chlamydial infection during pregnancy.[82] The transmission of *Chlamydia trachomatis* from the infected cervix of a mother to the eyes of an infant with resultant conjunctivitis was documented in humans and in primates 80 years ago by cytologic methods. With modern microbiologic methodology, it is now possible to quantitate this infection.[85] Inclusion conjunctivitis of the new born developing approximately 7-14 days after birth unlike gonococcal ophthalmia, has less severe signs and symptoms and tends to be self-limiting in its course. Mothers of these infants invariably have associated chlamydial muco-purulent cervicitis. Several microbiologic pathogens have been implicated in neonatal conjunctivitis. In one study the organisms isolated included *Haemophilus*, *Staphylococcus aureus*, *Chlamydia trachomatis*, enterococci and *Staphylococcus pneu-*

moniae. Of these, *Chlamydia trachomatis* constituted only 14%.[86] Since these micro-organisms have differing susceptibilities to antimicrobial agents, culture and susceptibility tests are required as a guide to therapy. Topical erythromycin was found to be a more effective prophylactic agent against chlamydial ophthalmia neonatorum when compared to topical silver nitrate.[87]

Even though, chlamydial ophthalmia neonatorum has a benign and self-limited course, the disease is never considered benign because it indicates an active genital infection in the mother. Further, the infection in the neonate if not treated may spread from the eyes to the pharynx,[88] and from there to the lungs and middle ear.[89] Rarely, the disease may result in ocular scarring and pannus.[90] One of the most serious complications of chlamydial infection in infants is development of a distinct pneumonia syndrome.[89,91,92] A retrospective diagnosis of chlamydial ophthalmia neonatorum can be made in most of these infants with pneumonia. The affected babies are afebrile and have a distinctive staccato cough similar to that of pertussis but without the inspiratory whoop.[93] Clinical examination reveals tachypnoea and radiographic studies show diffuse pneumonia. The chlamydia is usually isolated from the naso-pharyngeal and trachco-bronchial aspirates of these babies. Raised chlamydia specific antibody especially of the IgM fraction and recovery of the causative agent from the lung biopsies of some affected infants further support the chlamydial aetiology of this pneumonia.[94] Chlamydia is becoming an important cause of infantile pneumonia. In one study it was associated with 30% of 30 consecutive admissions for pneumonia in infants less than 6 months of age.[95] Myocarditis developing as a complication of chlamydial pneumonitis has been reported in an infant.[96] Since the chance of infection spreading from the eye of an affected infant to the naso-pharynx, lungs and ears are there, many authors strongly recommend systemic treatment for all cases of chlamydial ophthalmia neonatorum.[97,98]

Mycoplasmas

Mycoplasmas are the smallest free living micro-organisms found in man. *Mycoplasma hominis* and T. mycoplasma, also known as *Ureaplasma urealyticum* are associated with genital infections. These are recovered more frequently from pregnant than from non-pregnant women.[99] The rate of colonization is increased between the first and third trimesters of pregnancy.[100] In women it has been associated with post-abortal and post-partum fever, spontaneous abortion, still birth, preterm labor and early neonatal death.[101-104] Whether genital mycoplasmas invade the foetus and cause its death or whether the invasion of the foetal tissue is merely secondary to death of the foetus is not known. Infection of the new born with *Ureaplasma urealyticum* is associated with low birth weight,[105] though some studies did not confirm it.[106,107] Acute respiratory distress, fever and pneumonia in neonates have been associated with isolation of mycoplasmas from the naso-pharynx of affected infants.[108] However, they have been recovered from a significant number of babies without any respiratory symptoms as well.[102]

Herpes genitalis

The effects of herpes simplex virus (HSV) type II on the foetus and neonate have been studied extensively in recent years. The increased prevalence of genital HSV infection among the adult population seems to be associated with a concomitant increase in neonatal HSV infections.[109] Primary herpetic genital infection is usually associated with features like fever, pseudo-chills, myalgia and arthralgia indicating that viremia had occurred. If this occurs in a pregnant woman, the viremia raises the possibility of congenital herpetic infection of the foetus.[110] But congenital infection is very

rare and when this does occur,[111] most HSV infections in utero result in foetal death rather than in birth defects.[112] Herpetic viral inclusion has been demonstrated in the endothelial cells and suggests that foetal infection by transplacental route is possible. It is noted that the chance of abortion is three times more than in general population if herpetic infection occurs before the 20th week of pregnancy. Premature birth rate also is more in these patients.[113] If a pregnant woman has herpetic lesions on the vagina or cervix at the time of delivery, the neonate may acquire the disease while passing through the birth canal, resulting in neonatal herpetic infection. Even in the absence of visible lesions on the genitalia of the mother, asymptomatic shedding of the virus may occur.[113-115] The risk of transmission of HSV from a mother with genital HSV infection to her infant during vaginal delivery is about 50% which may be reduced to less than 10% if a caesarean section is performed before rupture of the membranes or within 4 hours of its rupture.[116]

The clinical picture of neonatal HSV infection ranges from a fulminant disseminated infection to localised CNS, eye, skin or oral infection. Meningo-encephalitis is a disastrous complication of the neonatal herpetic infection. Allen et al described 2 neonates with HSV-2 encephalitis born to mothers in the same commune.[117] Encephalitis in infants usually ends fatally. Infants who survive the acute infection have a bad prognosis, more than half of them being left with microcephaly, hydrocephalus or psychomotor retardation.[116] The virus has been demonstrated in the brain tissue and CSF of such infants. In one case that died of neonatal meningo-encephalitis, autopsy revealed cystic infarction of the brain.[117] Bilateral necrotising retinitis complicating fatal herpetic encephalitis in a neonate was reported by Greer.[118] Respiratory distress, pneumonia and transient bullous lesions may rarely develop in these affected neonates.[119]

Development of neonatal herpetic infection to some extent can be prevented. Genital HSV infection during pregnancy has caused considerable concern among lay and professional personnel in the past few years. Knowledge concerning the potential hazards of HSV to the neonate has increased the use of caesarean section for women who have or are suspected to have genital HSV infection near or at the time of labor.[120] All pregnant women with a past history of genital herpetic infection should be followed closely during the last trimester of pregnancy for evidence of cervical shedding of the virus. Cervical scrapings for cytological, immunofluorescent or immunoperoxidase staining should be obtained at the onset of labor. The route of delivery vaginal or by caesarean section, is determined by the result of cervical culture for virus. An elective caesarean section in a mother with evidence of cervical shedding of virus is probably the best way of preventing neonatal HSV infection and its horrible consequences. Isolation and barrier nursing of all babies born to infected mothers are being stressed now-a-days. Contrary to all these, two recent reports reveal that herpetic infection during pregnancy is not so disastrous to the foetus and neonates, as once thought.[121,122]

Cytomegalo virus infection

Frequent demonstration of cytomegalo virus in the female and male genital tracts suggests that these are sexually transmitted.[123] High prevalence of infection with this virus has been reported in pregnant women.[124-128] Human CMV infection, the most common virus infection of the foetus and neonate, may occur in utero transplacentally causing congenital infection or during the first few weeks of life as perinatal infection.[129] Primary CMV infection acquired during pregnancy carries a high risk of serious

congenital infection in the foetus.[130,131] About 10-20% of infants born to infected mothers develop sensori-neural deficits or developmental defects. Foetal loss may occur in about 15% cases of infected pregnant women.[124] Saigal et al reported CMV infection occurring in a pair of dizygotic twins.[132] A variety of features have been observed in neonates and infants infected congenitally with this virus. These include microcephaly, sensori-neural hearing loss, mental deficiency, CNS defects, chorio-retinitis, optic atrophy, low birth weight, neonatal hepatitis and jaundice, hepato-splenomegaly, encephalitis, thrombocytopenia, neutropenia and polycystic kidney.[124,133,134] Perinatal infection may be acquired during passage of the foetus through the infected birth canal. Most of these infants remain asymptomatic in spite of chronically excreting the virus. But in a few, interstitial pneumonia, hepatitis, exanthems and adenopathy may occur.

Hepatitis B virus infection

Viral hepatitis in pregnant women may become fulminant especially during the last trimester.[135] Transmission of the infection from the mother to the foetus may occur.[136-138] In China in the province of Taiwan, 10-15% of women are HBs Ag positive carriers and over one third of their infants acquire the infection.[139] There is little mortality in early infancy but morbidity and the risk of subsequent hepatoma need to be quantitated. In infants born to HBs Ag positive carrier mothers, active immuno-prophylaxis with the hepatitis B vaccine must be used in conjunction with hepatitis B immune globulin.[140]

Acquired immuno-deficiency syndrome (AIDS)

AIDS caused by human immuno-deficiency virus (HIV) has been reported from different parts of the world, including India. The previous view that the syndrome in infants may have resulted from a casual contact within the home with the non-parental relatives who have the syndrome or who are at risk, has been largely dispelled by the isolation of the virus from a high percentage of asymptomatic mothers of these affected infants. Spread of the infection from the mother to the foetus is now an established fact, the risk being as high as 50%. A number of cases have been seen in infants and children.[142-144] There is no evidence that AIDS can be transmitted by a routine household or social contact. A direct prenatal or transplacental transmission of the virus from the mother to the foetus is the most likely mechanism of acquiring the disease in infants. Postnatal transmission of the virus from mother to the foetus presumably through the breast milk was reported recently by Ziegler et al.[145] The virus has been detected in the breast milk in some cases.[146]

Condylomata acuminata

During pregnancy, due to increased vascularity the genital wart gets enlarged and may thus obstruct the labor. The teratogenic effects of podophyllin are well known. It is embryocidal and some women have used the drug in attempts to terminate the pregnancy. In view of the possible teratogenic effects as well as intrauterine deaths from topical use of podophyllin on genital warts during pregnancy, several authors have recommended that this medication should not be used during pregnancy. Nevertheless, one of the recent studies from Vellore showed that podophyllin is a safe and effective mode of therapy at all the stages of pregnancy.[147] Increased incidence of laryngeal papillomatosis has been reported in infants born to mothers with genital warts during pregnancy.[148,149] The possibility of caesarean section in the presence of active condyloma acuminatum must be considered.

Group B streptococcal infection

Group B streptococcus (GBS) has only recently been recognized as a sexually trans-

mittable organism that causes serious clinical disease especially in neonates and infants. The increasing incidence of GBS disease in infants with attendant high mortality and neurological sequelae in survivors parallels the increasing incidence of other sexually transmitted diseases. Many reports indicate that a large number of asymptomatic pregnant women carry GBS in their vagina.[150-152]

Although majority of the neonates born to the infected mothers harbour GBS at birth, only about 1% of them develop the disease. The disease in the neonate may be of early onset type or late onset type.[153] In one study, 67% of the infants with early onset disease were found to be bacteremic at birth, implying intrapartum acquisition of infection.[154] The clinical features of early onset GBS disease range from fulminating sepsis to asymptomatic bacteremia.[155] Acute respiratory distress, apnoea, meningitis and shock with septicemia are common in them. The disease usually manifests on the first day of life although symptoms may not appear until the fourth or the fifth day of life. About 50% of the affected neonates die despite antibiotic treatment and those who survive develop sequelae mostly of CNS. Features of late onset GBS disease include meningitis, cellulitis, arthritis, osteomyelitis and otitis. Though selective intrapartum therapy with ampicillin, benzyl penicillin or erythromycin significantly reduces the rate of transmission from mother to the foetus,[154,156] routine culturing for GBS in pregnant women and treatment of those who harbor the organism are not recommended,[151] because the risk for specific complication is too low.[157]

Trichomoniasis

Trichomonas vaginalis has been found repeatedly in a small proportion of new born babies who have been exposed to infected mothers : the infection having taken place during birth.[158] Pasyk found the parasite in 16.8% of 149 new borns and infants aged 1 day to 11 months : 92% of the infected children had leukocytes in urine.[159] *T. vaginalis* has recently been incriminated for causing neonatal pneumonia.[160] The importance of treatment for trichomoniasis during pregnancy and the necessity of at least 2 examinations for this protozoa in the new borns of non-examined mothers are stressed.[159] The results of treatment with metronidazole during pregnancy for trichomoniasis are good and morbidity is slight. Infants with symptomatic trichomoniasis or with persistent urogenital trichomonal colonization beyond the fourth week of life should be treated with metronidazole 10-30 mg/kg daily for 5-8 days.[161]

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