Review Article

Pregnancy and sexually transmitted viral infections

P. Singhal, S. Naswa, Y. S. Marfatia
Department of Skin VD, Government Medical College & SSG Hospital, Vadodara, Gujarat, India

Address for correspondence:
Dr. Y S Marfatia, Professor & Head, Department of Skin VD, Government Medical College & SSG Hospital, Vadodara, Gujarat, India.
E-mail: ym11256@gmail.com

Abstract

Viral infections in pregnancy are a major cause of morbidity and mortality for both mother and fetus. Viral STIs occur as surface infection and then gradually infect immunologically protected sites. Therefore, these are asymptomatic, hidden and hence underdiagnosed, persistent and difficult to treat. HSV, HPV, HBV, HIV and CMV (cytomegalovirus) are the common ones. Most of these are transmitted during intrapartum period. Proper screening, identification and treatment offered during prenatal period may help in preventing their complications. Twenty five percent of women with a history of genital herpes have an outbreak at some point during the last month of pregnancy. Acyclovir is the accepted efficacious and safe therapy for HSV in pregnancy. Globally, HPV infection is the most common sexually transmitted infection. Neonatal transmission can occur in the absence of clinically evident lesions. HPV 6 or 11 may lead to Juvenile Onset Recurrent Respiratory Papillomatosis (JORRP). TCA, liquid nitrogen, laser ablation or electrocautery can be used to treat external genital HPV lesions at any time during pregnancy. Cesarean section is recommended only if the lesions are obstructing the birth canal. Mother to child transmission (MTCT) in HIV accounts for 15–30% during pregnancy and delivery, and a further 5–20% of transmission occurs through breastfeeding. HBV infection during pregnancy does not alter the natural course of the disease. In women who are seropositive for both HBsAg and HBeAg, vertical transmission is approximately 90%. Pregnancy is not a contraindication for HBV vaccination. Cytomegalovirus (CMV) is the most common intrauterine infection. Cytomegalic inclusion disease (CID) is the most severe form of congenital CMV infection. Treatment is supportive.

Key words: Pregnancy, viral STI, vertical transmission

INTRODUCTION

Immunologic changes of pregnancy may induce a state of increased susceptibility to certain intracellular pathogens, including viruses, intracellular bacteria and parasites. Viral infections in pregnancy are a major cause of morbidity and mortality for both mother and fetus. Infections can occur in the neonate transplacentally, perinatally (from vaginal secretions or blood) or postnatally (from breast milk or other sources). The risk of infection is usually inversely related to gestational age at acquisition.[1] Effect of maternal infection on fetus may range from no involvement to inapparent or apparent involvement.

HERPES SIMPLEX VIRUS

Herpes in pregnancy - Scenario

The highest incidence of Herpes Simplex Virus (HSV) infection occurs in women of the reproductive age; the risk of maternal transmission of the virus to the fetus or neonate has become a major health concern.

The recurrence rate of genital herpes appears to be higher in pregnant than in non-pregnant women, with the likelihood of recurrence increasing as the patient reaches term.[2] Twenty-five percent of women with a history of genital herpes have an outbreak at some point during the last month of pregnancy, and 11–14% have an outbreak at the time of delivery.[3]

How to cite this article:
Singhal P, Naswa S, Marfatia YS. Pregnancy and sexually transmitted viral infections. Indian J Sex Transm Dis 2009;30:71-8.
DOI: 10.4103/0253-7184.62761

Mother to Child transmission

Risk of neonatal infection is as low as 1% if mother acquires HSV in first trimester due to formation of protective antibodies. Whereas the risk significantly rises to 30–50% if mother gets infected in late pregnancy (last trimester).[4] Transmission of HSV from mother to fetus during pregnancy is uncommon; about 85% of perinatal transmission occurs during the intrapartum period.[5] In case of recurrent HSV infection in pregnancy, if the lesions are not evident during delivery, there is still a small risk of asymptomatic shedding (approximately 1%), and therefore the risk of neonatal infection can be up to 0.02% to 0.05%.[6,7] Additional risk factors for neonatal HSV infection include the use of a fetal–scalp electrode and the age of the mother less than 21 years.

Many neonatal infections occur because of asymptomatic cervical shedding of virus, usually after a primary episode of HSV infection. Of known infected infants, only 30% are mothers who had symptomatic HSV or a sexual partner with clinical infection.[8]

Clinical features in pregnant women

Primary HSV infection in pregnant females leads to vesicular lesions similar to those in non-pregnant state. It can result in more severe disease than that in the non-pregnant ones, in particular, gingivostomatitis and vulvovaginitis herpetica and there is a tendency towards dissemination. The acquisition of genital herpes during pregnancy has been associated with spontaneous abortion, intrauterine growth retardation, preterm labor, congenital and neonatal herpes infections.[9] Recurrent episodes of HSV infection are characterized by the presence of antibody against the same HSV type as in the first episode. The herpes outbreaks are usually mild (7–10 days) with less severe symptoms than the first episode.

Congenital HSV: morbidity and mortality

Congenital HSV infection (approximately 4% of all neonatal HSV infections) can result in an infant born with microcephaly, hydrocephalus, chorioretinitis and vesicular skin lesions.[10] Three subtypes of infection have been identified: (1) disease localized to the skin, eye or mouth; (2) encephalitis, with or without skin, eye or mouth involvement; (3) disseminated infection that involves multiple sites, including the central nervous system, lung, liver, adrenals, skin, eye or mouth. There is virtually no mortality among infants with disease limited to the skin, eyes and mouth, but mortality increases to 15% among infants with encephalitis and 57% among infants with disseminated disease, even with antiviral therapy. Long-term morbidity is common in infants who survive with encephalitis or disseminated disease, and may include seizures, psychomotor retardation, spasticity, blindness or learning disabilities.[11]

Diagnostic modalities

A suspected genital HSV infection should be confirmed with a diagnostic test. Traditionally, this has been done by viral culture of vesicular fluid. More rapid diagnosis may be obtained by direct immunofluorescent staining using fluorescein-conjugated monoclonal antibodies to HSV.[12] The sensitivity of this test is 80 to 90%, very high compared with viral culture. The polymerase chain reaction (PCR) is a more sensitive assay. An additional 9% of culture-negative women are PCR positive for HSV-2.[13]

Type-specific serologic tests for HSV

Both type-specific and nontype-specific antibodies to HSV develop during the first several weeks after infection and persist indefinitely. Accurate type-specific HSV serologic assays are based on the HSV-specific glycoprotein G2 (HSV-2) and glycoprotein G1 (HSV-1). The sensitivities of these glycoprotein G type-specific tests for the detection of HSV-2 antibody vary from 80 to 98%, and false-negative results might be more frequent at early stages of infection. The specificities of these assays are ≥96%. False-positive results can occur, especially in patients with a low likelihood of HSV infection

Management: The current ACOG guidelines

Current ACOG guidelines do not recommend routine antepartum genital HSV cultures in asymptomatic patients with recurrent disease, nor they recommend routine screening of pregnant women for HSV.[14] Suppressive antiviral therapy with acyclovir should be given to pregnant women who have a primary episode or active recurrent genital HSV at or beyond 36 weeks of gestation.[14] It should also be given to women with an active genital herpes infection, primary or secondary, near term or at the time of delivery. A recent statement by ACOG supports the use of antiviral therapy in pregnant women with outbreaks of genital herpes.[15] Acyclovir therapy started at 36 weeks of gestation may decrease viral shedding, prevent neonatal herpes, reduce the need for cesarean delivery and decrease clinical recurrences of herpes simplex virus infection. Valacyclovir is a promising substitute of acyclovir with similar efficacy, as well as the increased bioavailability of valacyclovir

and famciclovir results in their less frequent dosing to achieve the same therapeutic benefits as acyclovir.[14] All these three drugs, acyclovir, famciclovir and valacyclovir have been labeled as category B drugs. Valacyclovir is prodrug of acyclovir, which is rapidly converted to acyclovir, hence safety profile quite similar to the second one.[16]

Protocols during labor to prevent HSV transmission

Rupture of membranes for more than four to six hours before delivery increases the risk of transmission of HSV to the infant.[17] The use of fetal scalp electrode monitoring during labor, use of vacuum and forceps also provide a potential port of entry for the virus into the infant. Any patient who has a suspected active genital HSV infection, or has first-episode herpes simplex virus (HSV) infection and active genital lesions, as well as a pregnant woman with recurrent HSV and active genital lesions, or prodromal symptoms (such as vulvar pain or burning at delivery) of HSV infection should undergo cesarean section (CS).[14] Cesarean delivery is not recommended for women with a history of HSV infection but no active genital disease during labor.

Neonatal care

The HSV-exposed neonate should be monitored closely for any signs of infection. Initial cultures should be performed at 24 to 48 h, and then weekly cultures of conjunctiva, nose, mouth, urine and rectum for HSV-1 or HSV-2 have been suggested. Empiric acyclovir may be instituted in infants born to mothers with suspected primary HSV infection.[17]

HPV IN PREGNANCY

Human papillomavirus - the most common STI

Globally, Human Papillomavirus (HPV) infection is the most common sexually transmitted infection.[18]

During pregnancy, the prevalence of condyloma increases from the first to third trimester and decreases significantly in the postpartum period. The risk of condyloma acuminata in pregnancy is two-fold.[19] HPV-induced lesions like cervical or vulval condyloma tend to increase in size and vascularity during pregnancy due to natural immune suppressive state and the hormonal influences. They may even obstruct reproductive passage and may cause profuse bleeding during delivery.

Risk of transmission of HPV to neonate

Neonate is exposed to the virus primarily during its passage through the birth canal.

Transmission can even occur in the absence of clinically evident lesions. Although the classic mode of transmission of HPV to the newborn is during the passage of the fetus through the birth canal and on coming into contact with infected maternal secretions;[20] however, in certain instances newborn may be infected even after being delivered by CS and it can be due to ascending infection from the vaginal canal, after a premature rupture of the amniotic membranes[21] There can even be a intrauterine transmission at the time of fertilization from sperm carrying latent HPV[22] and transplacental.[23]

Laryngeal papillomatosis

The only known disease to occur secondary to perinatal transmission of HPV is HPV 6 or 11 induced laryngeal papillomatosis. However, the reported rate of this occurrence is 1–4/100 000 births.[24] Newborn may be asymptomatic at birth but laryngeal papillomas may develop within 2–5years of life and are located on vocal cords, epiglottis and may even involve the entire larynx and tracheobronchial tree. This condition termed as Juvenile Onset Recurrent Respiratory Papillomatosis (JORRP) is one of the most common causes of hoarseness and airway obstruction in children.[25]

Neonatal infection may occasionally present as anogenital warts.

Vaginal delivery versus cesarean section

The low risk of laryngeal papillomatosis and reports of its occurrence in children born by CS, as well as the known risks of CS have promoted the recommendation that the presence of genital warts not be the sole reason for delivery by CS. Additionally, no controlled studies have suggested that CS prevents this condition. The one clinical indication for CS that involves HPV is the presence of extensive vaginal and/or introital warts blocking the birth canal. TCA, liquid nitrogen, laser ablation or electrocautery can be used to treat external genital HPV lesions at any time during pregnancy. Imiquimod is not approved for use in pregnancy. Podophyllin is contraindicated in pregnancy due to potential teratogenicity.[24]

HEPATITIS B VIRUS

Global prevalence sketch

There are 350 million individuals chronically infected with Hepatitis B Virus (HBV) worldwide. At least 50% of them acquire their infections either perinatally or in early childhood.[26]

HBV in pregnancy

The disease course in pregnancy is similar to that seen in the general population. Acute HBV infection does not have any teratogenic effects. However, a higher incidence of low birth weight and prematurity has been reported.

About 10–20% of women seropositive for HBsAg transmit the virus to their neonates in the absence of immunoprophylaxis. In women, who are seropositive for both HBsAg and HBeAg, vertical transmission is approximately 90%. In patients with acute hepatitis B, vertical transmission occurs in up to 10% of neonates when infection occurs in the first trimester and in 80–90% of neonates when acute infection occurs in the third trimester.[27] Risk for HBV transmission at delivery is mainly due to exposure to cervical secretions and maternal blood. Minority of infections are not prevented by prompt neonatal immunization, hence a lot of transplacental transmission is also presumed. Risk factors for transplacental transmission of HBV include maternal HBeAg positivity, high HBsAg titre and HBV DNA level.[28] HBV infection during pregnancy does not alter the natural course of the disease; however, chronic infection occurs in about 90% of infected infants.[29]

Though routine prenatal screening of all pregnant women for HBsAg is the need of the hour, especially until hepatitis B vaccine is included in the scheme of compulsory vaccination of all newborns,[30] it is not practiced routinely currently.

Management

Pregnancy is not a contraindication for HBV vaccination, and pregnant females can receive three doses of the vaccine at 0, 1 and 6 months. If the female is exposed to a person with acute hepatitis B as a result of sexual contact, then a course of HBV vaccine into the deltoid along with a dose of Hepatitis B immunoglobulin (HBIG) 0.06 mL/kg IM into the contralateral arm should be given within 14 days after the most recent sexual contact. However, in cases with exposure to chronic carriers hepatitis B vaccine alone is recommended. Neonatal vaccination prevents newborn infection in about 80–95% of cases. Interferon, lamivudine, adefovir and entecavir are classified by the Food and Drug Administration as Class C, and telbivudine and tenofovir as Class B. In most cases, this is because there are insufficient data in humans to demonstrate teratogenic or embryotoxic effects. For these reasons, in most instances, it is reasonable to defer therapy until after delivery, to avoid fetal exposure to the therapeutic agents.

CYTOMEGALOVIRUS IN PREGNANCY

Most common intrauterine infection - CMV

Cytomegalovirus (CMV) is the most common intrauterine infection.[31] Congenital CMV infection occurs in 0.2 to 2.2% of live births worldwide. It may result from transplacental acquisition of either a primary or recurrent (i.e., cytomegalovirus infection that occurs in the context of preconceptual immunity) maternal infection.[32] The average rate of transmission to the fetus in primary maternal infection during pregnancy is 40%; and approximately 65% of these infants have CMV disease at birth. With recurrent maternal infection the risk of transmission to the fetus is lower, ranging from 0.5 to 1.5%; most of these infants appear normal at birth (i.e., silent infection).[33]

CYTOMEGALIC INCLUSION DISEASE

Many women who become infected with CMV during pregnancy are asymptomatic, but some develop mononucleosis-like illness.[32] Cytomegalic Inclusion Disease (CID) is the most severe form of congenital CMV infection. Approximately 10% of infants with congenital infection have clinical evidence of disease at birth. CID is characterized by intrauterine growth retardation, hepatosplenomegaly, hematological abnormalities (particularly thrombocytopenia) and various cutaneous manifestations, including petechiae and purpura (i.e., blueberry muffin baby). The most significant manifestations of CID involve the CNS, manifesting as microcephaly, ventriculomegaly, cerebral atrophy, chorioretinitis and sensorineural hearing loss. Most infants who survive symptomatic CID have significant long-term neurological and neurodevelopmental sequelae, even 10% of asymptomatic neonates eventually develop neurologic sequelae. It has been estimated that congenital cytomegalovirus may be second only to Down syndrome as an identifiable cause of mental retardation in children.[33] Symptomatic neonates have a mortality rate of up to 30%, and 70 to 90% of survivors have some morbidity in the form of neurologic impairment, including hearing loss,

mental retardation and visual disturbances.[32]

Diagnosis and treatment

Symptomatic congenital CMV infection must be distinguished from other congenital infections, including toxoplasmosis, rubella and syphilis (TORCH infections). Diagnosis in mother can be made by serologic testing and in neonates; the primary diagnostic tool is viral culture. Treatment is supportive.[32] Nucleosides like ganciclovir and cidofovir are the only true antiviral agents active against cytomegalovirus. Both are Class C drugs for pregnancy. In infants, antiviral therapy with ganciclovir may be of benefit in reducing the prevalence of neurodevelopmental sequelae, in particular sensorineural hearing loss. Its use still remains controversial.[33] Prevention is an important tool to save the neonate from this deadly virus.

HUMAN T CELL LYMPHOTROPIC VIRUS TYPE I

Human T cell lymphotropic virus type I (HTLV 1), in some cases type II, is other STI likely to affect pregnant woman, being transmitted perinatally and sexually. This infection causes a serious form of spastic paralysis or human T cell lymphotropic-associated myelopathy, as well as T cell lymphoma or leukemia.[34]

HUMAN IMMUNODEFICIENCY VIRUS

Global Scenario

Globally, about 50% of all adults living with Human Immunodeficiency Virus (HIV) are women and the prevalence of HIV positive children is 2.5 million. In 2001, the United Nations General Assembly Special Session on HIV/AIDS committed countries to reduce the proportion of infants infected with HIV by 20% by 2005 and by 50% by 2010.[35]

Indian scenario

Twenty seven million new pregnancies occur per year in India of which 97 000 pregnancies occur in HIV +ve mothers (prevalence - 0.36%). There are 30 000 HIV-infected babies (25–30% transmission rate) born every year. Still, significant number of pregnant women needs to be covered under the umbrella of HIV testing and preventive medicine.

Mother to child transmission (MTCT)

HIV infection from an HIV-positive mother to her child can occur during pregnancy, labor, delivery or breastfeeding.[36] Without treatment, around 15–30% of babies born to HIV positive women become infected with HIV during pregnancy and delivery. A further 5–20% become infected through breastfeeding.[37] In 2008, an estimated 430 000 children became newly infected with, the majority of them through MTCT.[36]

The risks associated with perinatal transmission of HIV-1 are multifactorial. Known risk factors include high maternal plasma viremia, advanced clinical HIV-1 disease, reduced maternal immunocompetence, vaginal delivery and a lengthy interval between rupture of the amniotic membrane and delivery. In addition, direct exposure to maternal blood, presence of ulcerative genital infection in the maternal vaginal tract at the time of delivery, illicit drug use during pregnancy, prematurity, and low birth weight have all been associated with increased mother-to-child transmission.[38]

Clinical features in pregnant mother and child

HIV transmission to the fetus can occur as early as the 15th week of pregnancy. Prenatal infection may cause a HIV-specific embryopathy in the majority of infected children. It is characterized by a small forehead, short flat nose, pronounced philtrum, microcephaly, thick lips and hypertelorism. There is evidence suggesting that pregnancy also favors the progression of the HIV disease in the mother. The most important determinant is the virus load present in the mother.[39]

HAART, ARV prophylaxis and regimens

In all HIV-infected pregnant women, initiation of ART for their own health is recommended if their CD4 cell count <350 cells/mm^3, irrespective of WHO clinical staging.[36] While those with higher CD4 counts warrant short courses of ARV drugs started in late pregnancy or during labor reduce the risk of in-utero and peripartum HIV transmission two- to three-fold [Table 1].

REGIMENS FOR BREAST-FED AND NON BREAST-FED INFANTS

More than 200 000 of the 500 000 new human immunodeficiency virus (HIV) infections that occur each year in children are the result of transmission of the virus through the mother's breast milk.[40] In resource-constrained settings, current policies with respect to breastfeeding by mothers who are infected with HIV are guided by observational evidence that exclusive breastfeeding for the first 4 to 6 months of life reduces the risk of transmission of HIV as compared with mixed breastfeeding (i.e., feeding both breast milk and formula) and may have survival benefits at 18 to 24 months that are similar to those for exclusive formula feeding. Other measures that

Table 1: ARV prophylaxis regimen

	Mother presents Early during pregnancy	Mother presents Late in 3rd trimester	Mother presents At labor
Mother	AZT from 28 wks	AZT + 3TC as soon as possible	
	NVP during labor	NVP during labor	AZT + 3TC + NVP during labor
	AZT for 7 days post-natally	AZT +3TC for 7 days post-natally	AZT + 3TC for 7 days post-natally
Child	NVP within 72 h + AZT for 1 wk	NVP within 72 h + AZT for 4 wks	NVP within 72 h AZT + 3TC for 4 wk

AZT - Zidovudine, NVP - Nevirapine, 3TC - Lamivudine

may minimize risk of transmission through breast milk are:

- Good lactation management so that breastfeeding problems such as cracked nipples, engorgement and mastitis are prevented.
- Where the mother does develop mastitis or abscesses, she must express milk from the affected side frequently, discard it and continue feeding from the unaffected side.
- Condoms must be used throughout the lactation period.
- If the infant has oral thrush, it must be treated promptly

WHO recommends that 'where replacement feeding is acceptable, feasible, affordable, sustainable and safe HIV-infected women should avoid breast feeding.' Although peripartum prophylaxis with a single dose or a short course of antiretroviral agents effectively reduces intrapartum HIV transmission, its effect does not extend much beyond 4 to 6 weeks in breastfeeding populations.

WHO also recommends that infants born to HIV-infected women receiving ART for their own health should receive:

a) breastfeeding infants: daily NVP from birth until 6 weeks of age
b) non-breastfeeding infants: daily AZT or NVP from birth until 6 weeks of age.

In case of breastfeeding infants of HIV-infected pregnant women who are not in need of ART for their own health, maternal ARV prophylaxis should be coupled with daily administration of NVP to the infant from birth until one week after all exposure to breast milk has ended.

In non-breastfeeding infants, maternal ARV prophylaxis should be coupled with daily administration of AZT or NVP from birth until 6 weeks of age.[36]

ELECTIVE CESAREAN SECTION VERSUS VAGINAL DELIVERY

An elective cesarean section (CS) substantially reduces vertical transmission among untreated or highly active ART (HAART) treated pregnant women. However, CS has higher post-partum morbidity than vaginal delivery especially in HIV-infected women, compared with their non HIV-infected counterparts. ACOG thus recommends, ''the decision regarding mode of delivery must be individualized'. In the HAART era, vaginal delivery should be considered if woman is treated with HAART and has a viral load before labor of below 1000 copies/mL.[41] A meta-analysis of 15 prospective cohort studies also suggested that elective CS reduces vertical transmission of HIV-1 independent of zidovudine therapy. Although not recommended in the United States, elective CS is routinely recommended in some European countries for HIV-1-infected pregnant women after 36 weeks of gestation.

CONCLUSION

Sexually transmitted infections (STIs) are a major public health problem, especially in developing countries. The current syndromic approach focuses on curable STIs like trichomoniasis, syphilis or gonorrhea, whereas viral STIs like HSV, HPV and HIV affecting pregnant women are on a rise. Hence, where resources allow, routine screening and treatment of STIs/RTIs in the antenatal care setting should be offered. All pregnant women and their sex partners should be asked about STDs, counseled about the possibility of perinatal infections, and ensured access to treatment, if needed. Being non-curable, prevention and early diagnosis with prompt treatment and prevention of grave consequences, sequelae and complications remain the key tool to curb maternal and perinatal morbidity. Future research and public health preventive efforts should target not only the classical bacterial RTIs but also viral STI.

REFERENCES

1. Marino T. Viral infections and pregnancy. Available from: http://www.emedicine.medscape.com. [updated in 2010].
2. Brown ZA, Vontver LA, Benedetti J, Critchlow CW, Hickok DE, Sells CJ, *et al*. Genital herpes in pregnancy: Risk factors associated with recurrences and asymptomatic viral shedding. Am J Obstet Gynecol 1985;153:24–30.
3. Arvin AM, Prober CG. Herpes simplex virus infections. Pediatr Infect Dis J 1990;9:765–7.
4. Centers for Disease Control and Prevention Website. Sexually transmitted disease guidelines. Available from: http://www.cdc.gov/std/treatment/2006/rr5511.pdf. [cited in 2010].
5. Enright AM, Prober CG. Neonatal herpes infection: Diagnosis, treatment and prevention. Semin Neonatol 2002;7:283-91.
6. Prober CG, Sullender WM, Yasukawa LL, Au DS, Yeager AS, Arvin AM. Low risk of herpes simplex virus infections in neonates exposed to the virus at the time of vaginal delivery to mothers with recurrent genital herpes simplex virus infections. N Engl J Med 1987;316:240–4.
7. Brown ZA, Benedetti J, Ashley R, Burchett S, Selke S, Berry S, *et al*. Neonatal herpes simplex virus infection in relation to asymptomatic maternal infection at the time of labor. N Engl J Med 1991;324:1247–52.
8. Fleming DT, McQuillan GM, Johnson RE, Nahmias AJ, Aral SO, Lee FK, *et al*. Herpes simplex virus type 2 in the United States, 1976 to 1994. N Engl J Med 1997;337:1105–11.
9. Brown ZA, Selke S, Zeh J, Kopelman J, Maslow A, Ashley RL, *et al*. The acquisition of herpes simplex virus during pregnancy. N Engl J Med 1997;337:509-15.
10. Jones CL. Herpes simplex virus infection in the neonate: clinical presentation and management. Neonatal Netw 1996;15:11–5.
11. Jacobs RF. Neonatal herpes simplex virus infections. Semin Perinatol 1998;22:64–71.
12. Pouletty P, Chomel JJ, Thouvenot D, Catalan F, Rabillon V, Kadouche J. Detection of herpes simplex virus in direct specimens by immunofluorescence assay using a monoclonal antibody. J Clin Microbiol 1987;25:958–9.
13. Cone RW, Hobson AC, Brown Z, Ashley R, Berry S, Winter C, *et al*. Frequent detection of genital herpes simplex virus DNA by polymerase chain reaction among pregnant women. JAMA 1994;272:792–6
14. American College of Obstetricians and Gynecologists (ACOG). Management of herpes in pregnancy. Washington (DC): American College of Obstetricians and Gynecologists (ACOG); 2007.
15. ACOG practice bulletin. Management of herpes in pregnancy. Number 8 October 1999. Clinical management guidelines for obstetrician-gynecologists. Int J Gynaecol Obstet 2000;68:165–73.
16. Serdar HU. Genital herpes in pregnancy. Available from: http://www.emedicine.medscape.com. [updated in 2010].
17. American Academy of Pediatrics. Committee on Infectious Diseases. In: Pickering LK. 2000 Red book: report of the committee on infectious diseases. 25th ed. Elk Grove Village, Ill.: American Academy of Pediatrics; 2000. P. 309–18.
18. Available from: http://www.cdc.gov/STD/HPV/STDFact-HPV.htm. [updated in 2010].
19. Robert VH. Condyloma acuminatum. Available from: http://emedicine.medscape.com. [updated in 2009].
20. Tseng CJ, Liang CC, Soong YK, Pao CC. Perinatal transmission of human papillomavirus in infants: relationship between infection rate and mode of delivery. Obstet Gynecol 1998;91:92-6.
21. Tenti P, Zappatore R, Migliora P, Spinillo A, Belloni C, Carnevali L. Perinatal transmission of human papillomavirus from gravidas with latent infections. Obstet Gynecol 1999;93:475-9.
22. Lai YM, Yang FP, Pao CC. Human papillomavirus deoxyribonucleic acid and ribonucleic acid in seminal plasma and sperm cells. Fertil Steril 1996;65:1026-30.
23. Tseng CJ, Lin CY, Wang RL, Chen LJ, Chang YL, Hsieh TT, *et al*. Possible transplacental transmission of human papillomaviruses. Am J Obstet Gynecol 1992;166:35-40
24. Human papilloma virus. Available from: http://www.asccp.org/hpv_diagnosing.shtml. [updated in 2010].
25. John EM. Recurrent respiratory papillomatosis. Available from: http://www.emedicine.medscape.com. [updated in 2008].
26. Alter MJ. Epidemiology of hepatitis B in Europe and worldwide. J Hepatol 2003;39:64-9.
27. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 86: Viral hepatitis in pregnancy. Obstet Gynecol 2007;110:941-56.
28. Xu DZ, Yan YP, Choi BC, Xu JQ, Men K, Zhang JX, *et al*. Risk factors and mechanism of transplacental transmission of hepatitis B virus: A case-control study. J Med Virol 2002;67:20-6.
29. Centers for Disease Control and Prevention, Workowski KA, Berman SM. Sexually transmitted diseases treatment guidelines, 2006. MMWR Recomm Rep 2006;55:997.
30. Capar M, Kosić-Andrasević V, Popić G, Raguz L, Mahuĺja-Stamenković V, Zuzek A, *et al*. The need and value of routine screening of all pregnant women for hepatitis B surface antigen. Acta Med Croatica 1995;49:161-4.
31. Rahav G, Gabbay R. Cytomegalovirus in pregnancy: Primary versus recurrent infections: a Prospective cohort study on 208 pregnancies. Abstr Intersci Conf Antimicrob Agents Chemother Intersci Conf Antimicrob Agents Chemother 2003;43:176.
32. Congenital and Perinatal Cytomegalovirus Infection (CMV). The Mercks manual online medical library. Available from: http://www.merck.com/mmpe/sec19/ch279/ch279b.html. [cited in 2010].
33. Mark RS. Cytomegalovirus infection: Treatment and medication. Available from: http://www.emedicine.medscape.com. [updated in 2009].
34. Sevgi OA, Mead O. Sexually transmitted infections. NCBI Bookshelf-Disease control priorities in developing countries, 2006. 2nd ed. Available from: http://www.ncbi.nlm.nih.gov/bookshelf/br.fcgi?book=dcp2andpart=A1920. [cited in 2010].
35. Antiretroviral drugs for treating pregnant women and Preventing HIV infection in infants guidelines on care, treatment and support for women living with HIV/AIDS and their children in resource-constrained settings. Available from: http://www.who.int/hiv/pub/mtct/en/arvdrugswomenguidelinesfinal.pdf. [cited in 2010].
36. RAPID ADVICE- Use of antiretroviral drugs for treating pregnant women and preventing HIV infection in infants. Available from: http://www.who.int/hiv/pub/mtct/rapid_advice_mtct.pdf. [cited in 2010].
37. Preventing mother-to-child transmission of HIV (PMTCT). Available from: http://www.avert.org/motherchild.htm. [cited in 2010].
38. Chen F, Pau AK, Piscitelli SC. Update on preventing vertical transmission of HIV type 1. Am J Health Syst Pharm 2000;57:1616-23.
39. Clinical features of human immunodeficiency viruses infection. Available from: http://virology-online.com/presentations/index.htm. [cited in 2010].
40. AIDS epidemic update: Special report on HIV/AIDS: December 2006. Geneva: Joint United Nations Programme on HIV/AIDS (UNAIDS) and World Health Organization; 2006.
41. Suy A, Hernandez S, Thorne C, Lonca M, Lopez M, Coll O. Current guidelines on management of HIV-infected pregnant women: Impact on mode of delivery. Eur J Obstet Gynecol Reprod Biol 2008:139:127–32.

Source of Support: Nil. **Conflict of Interest:** None declared.

Multiple Choice Questions

Q. 1. Indication for cesarean section in pregnant woman affected with HPV is
- Presence of genital warts
- Giant warts obstructing the introitus
- To prevent JORRP
- H/O HPV in previous pregnancy

Q.2. Vaccination schedule of Hepatitis B virus in pregnant female is-
- 0, 6 months
- 0,1,2 months
- 0,1,6 months
- Vaccination is contraindicated in pregnancy

Q.3. If HIV positive mother presents at labour with CD4 count 400/μL, the recommended ARV prophylaxis for the infant is
- AZT for 4 weeks
- Nevirapine SD within 72 hours
- Nevirapine SD within 72 hours + AZT for 4 weeks
- Nevirapine SD within 72 hours + AZT and 3TC for 4 weeks

Q.4. Transmission rate from mother (not on HAART) to child during pregnancy and delivery is-
- Up to 5%
- 5-10%
- 10-15%
- 15-30%

Q.5. Suppressive therapy with acyclovir should be given to pregnant woman with primary episode or active recurrent genital HSV from _____ weeks of gestation
- 12 weeks
- 16 weeks
- 28 weeks
- 36 weeks

Answers-
A.1 Giant warts obstructing the introitus
A.2 0,1,6 months
A.3 Nevirapine SD within 72 hours + AZT and 3TC for 4 weeks
A.4 15-30 %
A.5 36 weeks