CHAPTER 15

SEXUALLY TRANSMITTED INFECTIONS

Abstract

Five diseases (syphilis, chancroid, gonorrhoea, Lymphogranuloma venereum, and granuloma inguinale) are known as "classical STIs". The "second generation STIs" include infections where sexual transmission is epidemiologically important and where sexual transmission is possible, and HIV infection. In the syndromic approach, the main etiological agents are classified into a group of symptoms and signs. Six major syndromes have been identified: genital ulcer (in both sexes), urethral discharge (in males), scrotal swelling (in males), vaginal and cervical discharge (in females), pelvic inflammatory disease (in females), and inguinal swelling (in both sexes). Since these six syndromes are easy to identify, flow charts have been devised for each syndrome. Each flow chart is user-friendly and depicts the decisions and actions one has to take, in a step-by-step manner. Therefore, even non-specialists at any rural or urban health care facility can initiate treatment promptly. This approach includes only those syndromes that are treatable and would lead to severe complications, if left untreated. Other STI syndromes, such as genital warts and dysuria in women are not included in this approach.

Key Words

Acyclovir, Aetiological approach, Chancre, Chancroid, Clinical approach, Genital warts, Gonorrhoea, Granuloma inguinale, Herpes genitalis, Lymphogranuloma venereum, Reproductive tract infections, Sexually transmitted infections, Syndromic approach, Syphilis

15.1 – INTRODUCTION

STIs are a group of communicable diseases that are predominantly transmitted by sexual contact. More than 20 diseases are listed in this group. These diseases were formerly known as "venereal diseases" (after Venus, the Roman goddess of love) and as "STDs". Change in nomenclature has helped in including other diseases, which are seldom transmitted by the sexual route. However, not every disease that is transmitted sexually can be considered STI. One partner may acquire an infection non-sexually (e.g. vaginal candidiasis following antimicrobial therapy) and the infection may be sexually transmitted to the other partner. Five diseases (syphilis, chancroid, gonorrhoea, lymphogranuloma venereum, and granuloma inguinale) are known as *classical STIs*, while those recently

recognised are known as *second generation STIs*. The second generation STIs comprise: (a) infections such as non-gonococcal urethritis, herpes progenitalis, genital warts, trichomoniasis, and moniliasis, where sexual transmission is epidemiologically important, (b) diseases where sexual transmission is possible (genital scabies, pediculosis, molluscum contagiosum, and hepatitis B), and (c) HIV infection (Ahuja, 1981).

The term RTI includes STIs, infections due to overgrowth of commensals in the reproductive tract of women (also called "super-infection"), and iatrogenic infections associated with insertion of intrauterine devices (IUDs) and medical procedures. The true incidence of STIs or RTIs is not known since patients tend to conceal these conditions. Moreover, many of these diseases are *not* notifiable and reporting to the health care system is inadequate. The available data suggest a rising incidence of gonorrhoea and syphilis. Many agents causing STIs are developing antimicrobial resistance. The incidence of *second generation* STIs is increasing, as compared to that of *classical* STIs.

15.2 – CO-INFECTION OF HIV WITH STIs

HIV epidemic has refocussed attention on STIs because patterns of high-risk behaviour, social contacts, and social network are similar for HIV and other STIs.

15.2.1 – Effect of STIs on HIV Infection

The risk of transmission of HIV is enhanced by the presence of other ulcerative and non-ulcerative STIs (Kreiss *et al*., 1989; Quinn *et al*., 1987; Greenblatt *et al*., 1988; Piot *et al*., 1988; Laga *et al*., 1993; Wasserheit, 1992). STIs act as a cofactor for HIV infection and there is a definite linkage between the presence of STIs and the risk of developing HIV infection. HIV has been isolated from genital secretions, tissue and mononuclear cells in patients with STIs. These cells are present in increased number in inflammatory conditions. Ulcerative STIs disrupt the integrity of the genital mucosa. HIV has been isolated from genital ulcers (Kreiss *et al*., 1989). *Epidemiological synergy* between viral STIs and HIV can be explained by STI-related molecular events (Quinn *et al*., 1987; Kreiss *et al*., 1989).

After entering the genome of the host cell, the viral nucleic acid (called "provirus") remains dormant for a long time. The dormant provirus may be activated to start rapid replication by certain infections like syphilis, gonorrhoea and diseases caused by CMV and herpes simplex virus (HSV). Once a large number of viral particles are produced, they lyse the host cell and produce immunological damage (Malaviya, 1990). Many of the AIDS-related malignancies, such as carcinoma of the uterine cervix, are the end result of STIs.

15.2.2 – Effect of HIV Infection on STIs

The usual clinical presentation and the natural history of some STIs may be altered, leading to problems in clinical diagnosis (Wasserheit, 1992). The serological tests may not be reliable in an individual with immune suppression. Vaccination against hepatitis B may not be effective if the individual has HIV-induced immune suppression. Moreover, HIV-positive individuals with STIs may not respond to the standard treatment prescribed for their HIV negative counterparts (Wald *et al*., 1993).

15.3 – SYNDROMIC MANAGEMENT OF STIs

The traditional method of diagnosing STI is through the *etiological approach*, wherein laboratory diagnosis is established by identification of the causative organisms in smears and/or cultures. Its advantage is that when the etiological agent is identified by laboratory tests, the disease or condition can be treated cost-effectively. However, this approach is expensive (needs skilled personnel and a network of advanced laboratories) and time consuming (since one has to wait for the results). Moreover, the requisite laboratory facilities are not available at primary health care level, in rural as well as urban areas, in most developing countries.

The *clinical approach* relies upon clinical diagnosis alone and does *not* involve laboratory tests. The vast majority of patients with STIs seek treatment in private or public sector clinics, which lack the required facilities and skilled personnel. It is difficult to clinically differentiate between various types of STIs, especially in the presence of mixed infections. For example, it is not possible to clinically differentiate between gonococcal and chlamydial urethritis. Studies have shown that clinical diagnosis is reliable only in 5 per cent of cases. Since the above-mentioned approaches have their own limitations, a third approach, called the *syndromic approach* has been recommended so that patients with STI can be treated quickly and cost-effectively, by non-specialists at primary health care level (NACO, 1998).

15.3.1 – Rationale

HIV epidemic has brought STIs into focus because they increase the risk of HIV transmission. Several pathogens that cause STIs have developed resistance to antimicrobials and consequently, some low-cost treatment regimens have become ineffective. The syndromic approach is based on the presumption that laboratory facilities are not available or affordable (WHO, 1997). Use of *standardised protocols* for diagnosis, treatment, and follow up ensures adequate treatment at all levels of the health care system, facilitates training and supervision of health care providers, and delays development of antimicrobial resistance.

15.3.2 – Features of Syndromic Approach

A *syndrome* is a group of symptoms (complained of by patients) and signs (found on examination). In this approach, the main etiological agents are classified into a group of symptoms and signs. Though STIs are caused by a variety of organisms, they give rise to a limited number of clinical syndromes (AIDS Prevention and Control Project, 1998). Six major syndromes have been identified

1. Genital Ulcer (in both sexes) – causative organisms are *Treponema pallidum*, *Chlamydia trachomatis*, *Calymmatobacterium granulomatis*, and Herpes simplex.
2. Urethral Discharge (in males) – due to *C. trachomatis* and *Neisseria gonorrheae*
3. Scrotal Swelling (in males) – caused by *C. trachomatis*, *N. gonorrheae* and viruses. Surgical conditions may also cause scrotal swellings.
4. Vaginal and Cervical Discharge (in females) – causative organisms are *C. trachomatis*, *N. gonorrheae*, *Candida albicans*, *Gardnerella vaginalis*, and *Trichomonas vaginalis*.
5. Pelvic Inflammatory Disease (in females) – due to *C. trachomatis*, *N. gonorrheae*, and anaerobic organisms.
6. Inguinal Swelling (in both sexes) – Causative organisms are *Hemophilus ducreyi* and *C. trachomatis*.

Since these six syndromes are easy to identify, flow charts have been devised for each syndrome. Each flow chart is user-friendly and depicts the decisions and actions one has to take, in a step-by-step manner. Therefore, non-specialists at any rural or urban health care facility can initiate treatment promptly (NACO, 1998; AIDS Prevention and Control Project, 1998).

15.3.3 – Components of Syndromic Approach

Diagnosis and Treatment of Specific Syndromes: Flow charts are used for diagnosis. If the condition is highly refractory to treatment (e.g. genital herpes, vulvo-vaginal candidiasis), the patient is to be informed likewise. However, in case of patients with low abdominal pain and scrotal swelling it is important to ensure that there is no surgical emergency (WHO, 1997).

Patient Education on Risk-reduction: During every visit to the health care facility, the patient is advised about regular treatment; follow-up, safer sex practices, partner notification, and genital hygiene. Education for prevention is an essential part of management of STIs (WHO, 1997).

Promotion of Condoms: Clients need adequate knowledge about correct use and disposal of condoms. Condoms are provided free of cost, or at an affordable price, at the health care facility.

Counselling: Counselling, if done in confidence, facilitates modification of high-risk behaviour, helps in assessing chances of acquiring HIV infection due

to high-risk behaviour, and permits discussion of possibility of problems like *incurability* (herpes genitalis), *infertility* (gonorrhoea), and *infection of progeny* (congenital syphilis) (WHO, 1997).

Partner Management: The patient is encouraged to voluntarily inform his or her partners of the infection and the need for clinical evaluation and treatment. Telling the spouse or sexual partner about the diagnosis of STI may be emotionally painful issue for many patients and the counsellor is required to tackle this issue (WHO, 1997). Partner notification and management has an important role in STI risk-reduction.

15.3.4 – Advantages

The syndromic approach is a scientific and simple method of managing STIs with good compliance (NACO, 1998; WHO, 1997; AIDS Prevention and Control Project, 1998). The cure rate can be as high as 95 per cent (NACO, 1998). It is free from errors in clinical judgement even in mixed infections and HIV-positive individuals in whom the usual clinical presentation of STIs may be altered. Increase in cost-effectiveness through money saved on laboratory tests (NACO, 1998; WHO, 1997; AIDS Prevention and Control Project, 1998). Compliance is increased since it obviates the need to wait for results of laboratory investigations. Offering prompt treatment at the first visit renders the patient non-infectious quickly and can control the spread of STI. This approach is feasible for both urban and rural areas at primary health care level.

15.3.5 – Disadvantages

This approach includes only those syndromes that are treatable and would lead to severe complications, if left untreated. The other STI syndromes, such as genital warts and dysuria in women, are not included in this approach. There is a potential risk of overtreatment.

15.4 – MANAGING STIs IN HIV-INFECTED INDIVIDUALS

STIs may be asymptomatic. Therefore, a detailed sexual and life history ought to be taken and specific questions suggestive of STI-related signs and symptoms should be asked. Privacy and confidentiality be respected while eliciting sexual history. Patients with problems pertaining to genitalia tend to be cautious and evasive in giving a history (WHO, 1997). Serological tests (for syphilis and hepatitis B/C) are recommended for each case of STI. Pre- and post-test counselling is essential. A person may have more than one STI. Conversely, STI always involves more than one person. A STI may involve multiple anatomical sites. Women are to be examined gynaecologically for *Neisseria* and *Chlamydia* infection and recommend bacteriological examination of cervical or vaginal secretions and Pap smear.

15.5 – CLINICAL ASPECTS

15.5.1 – Syphilis

The causative organism is a spirochaete *T. pallidum* (subspecies *pallidum*). Though usually spread sexually, the disease can also be transmitted by blood transfusion and transplacentally to offspring. Syphilis can affect any organ in the body and can mimic any disease. The incubation period varies from 9 to 90 days, with an average of 3–4 weeks.

The disease has an *early stage*, which occurs within the first 2 years of infection, and includes – primary syphilis, secondary syphilis, and early latent syphilis. The *late stage* comprises late latent syphilis, benign late syphilis, cardiovascular syphilis, and neurosyphilis.

The *primary stage* represents the local tissue reaction at the site of entry of the organism and lasts for 3–8 weeks. The *secondary stage* represents the generalised tissue reaction, which occurs after the organism has spread to all the tissues in the body. This stage lasts for 3–9 months. In the *latent stage*, serological tests are positive, but there are no clinical manifestations. The CSF is non-reactive.

Primary Syphilis: A small, red, painless, non-tender papule is seen on the genitalia or extragenital sites such as lips, tongue, and fingers about 3–4 weeks after exposure (incubation period varies from 9 to 90 days). It develops into a well-defined hard sore (or *chancre*) in 2–3 weeks. The ulcer, which is usually single, painless, and non-tender, has an indurated base. The untreated sore heals in 6–8 weeks with a thin atrophic scar and is followed by a painless bubo in the groin. The lymph nodes in the neck, axilla, and epitrochlear region become bilaterally enlarged with rubbery consistency and are painless and non-tender. The primary sore, blood, and body fluids are *infective* in this stage (Benenson, 1990).

Secondary Syphilis: This stage is characterised by sore throat, anaemia, skin rashes, lymphadenitis, and swelling in bones and joints. Arthralgia may be worse at night. Systemic symptoms like headache, fever, and malaise may be present. *Cutaneous lesions* are symmetrically distributed, polymorphic, non-pruritic and are abundant on the central part of the body, as compared to the extremities (called "centripetal distribution"). These skin lesions may manifest as rose-coloured macules, coppery papules, pustules, or ulcers. *Condyloma lata* (Latin: *lata* = broad; plural: condylomata) are flat, moist, raised, warty papule found on moist surfaces like groin, anus, vulva, and infra-mammary region. All the lymph nodes are enlarged, but painless and non-tender. *Snail-track ulcers* (white mucous patches or plaques which erode and form ulcers) may develop in the throat, mouth, prepuce and vulva. In this stage of syphilis, blood, body fluids, condylomata, and "snail-track" ulcers are *infective* (Benenson, 1990).

Early Latent Syphilis: The visible manifestations disappear with or without treatment. If the patient has had the disease for less than 2 years, it is called "early latent syphilis". In the latent stage, blood and body fluids are *not* infective (Benenson, 1990).

Tertiary Syphilis: This stage is seen in inadequately treated or untreated patients. *Benign tertiary syphilis* occurs about 8–10 years after the primary stage. *Gumma* (plural: gummata) is the characteristic lesion. Gummata are painless nodules, which may progress slowly, suppurate and form ulcers on the skin. Cutaneous gummata are usually asymmetrical. Gummatous ulcers have punched-out edges and are covered with "wash leather" (or "chamois leather") slough. They heal with a thin non-contractile scar. Gummata may be seen on tongue, soft palate, pharynx, lips, nose, bones (sternum, clavicle, vertebrae, long bones), and viscera (stomach, liver, spleen, intestines, testes). In the tertiary stage of syphilis, blood and body fluids are *not* infective. A broken and ulcerating gumma in the throat or skin may be infective ((Benenson, 1990).

15.5.1.1 – Diagnosis

Dark-field microscopy is the simplest and most rapid method of diagnosing syphilis. However, the organisms cannot be detected after the primary stage, when the skin lesions have healed. Hence, the main method for diagnosing syphilis is by testing a patient's serum for antibodies to *T. pallidum*. Serological tests (RPR and Treponema pallidum particle agglutination (TPPA)) are used for the diagnosis of late primary, secondary, and late syphilis. The *RPR test* is advised for any patient with STI to identify asymptomatic cases. If positive, the test should be repeated after 3–6 months of treatment.

15.5.1.2 – Syphilis and HIV infection

HIV infection (the most recent STI) and syphilis (the oldest known STI) share common features: (a) multiple modes of transmission, including the tragedy of mother-to-child (transplacental) transmission, (b) uncertain and prolonged asymptomatic phase, and (c) unpredictable clinical manifestations of varying severity affecting every organ system (Ramachandran, 1990). The primary lesion is an ulcer, which increases the risk of transmission of HIV. In HIV-infected individuals, syphilitic ulcers may be multiple, larger and may persist for longer periods. The symptoms of tertiary syphilis (e.g. meningitis, ocular complications, and gummata) may occur during the primary, secondary, and latent phases (Wald *et al*., 1993). Serological tests for syphilis may not be reliable. Seroconversion for syphilis occurs more rapidly in HIV-infected individuals (Wald *et al*., 1993). The effectiveness of past treatment determines the occurrence of reactivation or relapse. Secondary and tertiary stages of syphilis may occur earlier in HIV-positive patients (Wald *et al*., 1993). Neurological syphilis is also more frequently seen (Musher *et al*., 1990). For treatment of syphilis in HIV-positive individuals, injection procaine penicillin should be preferred over benzathine penicillin.

15.5.2 – Chancroid

(Synonym: SOFT SORE). A slender rod-shaped organism, *Haemophilus ducreyi* (or Ducrey's bacillus), causes this disease. The average incubation period is 2–5 days, up to 14 days. The incubation period is only about 24 hours if there are

abrasions on the genitalia. Though the sexual route primarily transmits the disease, it can also spread by auto-inoculation in persons with poor personal hygiene. The disease is infective till the lesions heal. The lesion appears on the genitalia as a papule or vesicle, which ulcerates rapidly. The ulcers are painful, tender, multiple, shallow, and ragged with undermined edges (unlike the hard sore of syphilis). The floor of the ulcer is covered with necrotic slough. Extragenital lesions may be seen on lips, tongue, chin, breast, and umbilicus. There is unilateral enlargement of regional lymph nodes, which are tender, firm, and matted (unlike that of syphilis). The lymph nodes soften, suppurate, forming "buboes" and get fixed to the skin. The bubo ruptures with a single opening on the skin (unlike the bubo of LGV). Phimosis is a common complication in males. Extensive ulceration may lead to destruction of prepuce and shaft of penis. Swabs taken from the base of the ulcer are used for microscopic examination and culture.

Chancroid and HIV Infection: Being an ulcerative STI, it increases the risk of transmission of HIV. In HIV-positive patients, the ulcers tend to be larger, more prominent and do not respond well to the standard single dose regimen (Wald *et al.*, 1993).

15.5.3 – Gonorrhoea

This disease is caused by *N. gonorrhoeae*. The incubation period is 2–5 days. Non-sexual routes, such as sharing infected towels, can also transmit this disease. The acute stage is characterised by inflammation of the urethra (in males) and of urethra, cervix, and vagina (in females). Usual manifestations in *males* are – thick, purulent, greenish yellow discharge from external urethral meatus, severe pain during micturition, and frequency and urgency of micturition. Symptoms are more marked if the posterior urethra is inflamed. In *females*, the symptoms are dysuria, frequency, and urgency. Epidemiologically, females are more liable to transmit the disease since they have few symptoms or obvious signs. In *female children*, the lesion is essentially *vulvo-vaginitis* and the infection may be acquired from infected towels, infected parents, or sexual assault. Babies born to infected mothers may develop gonococcal eye infection, which is acquired during passage through the birth canal. The condition is known as *ophthalmia neonatorum*. The eyelids are oedematous and stuck together with a purulent discharge. The conjunctiva is red. In severe cases, involvement of the cornea leads to keratitis and corneal ulceration.

Complications: In males, the infection may extend to prostate, seminal vesicles, bladder, renal pelvis, or rectum by contiguity or by lymphatics. Stricture may occur at bulbous urethra due to fibrosis of the urethral mucous membrane. In females, the infection may spread to uterus, fallopian tubes, peritoneum, and Bartholin glands. About 30 per cent of women with salpingitis may develop secondary sterility. In both sexes, blood-borne spread to joints, muscles, tendons, and eyes may occur. Meningitis and endocarditis are rare.

Laboratory Diagnosis: Smears taken from urethral discharge (in males) or cervix uteri (in females) are used for microscopic examination under Gram's stain to demonstrate Gram-negative kidney-shaped intracellular organisms. Enriched media such as chocolate agar are used for culturing *N. gonorrhoeae*.

Gonorrhoea and HIV Infection: Inflammation and formation of micro-ulcers in the genital mucosa may increase the risk of HIV transmission (Wald *et al*., 1993). The disease may be co-transmitted with HIV in case of unprotected sexual intercourse. In HIV-infected women, gonorrhoea may progress to painless pelvic inflammatory disease or tubo-ovarian masses. These women are less likely to have leukocytosis or abdominal pain (Wald *et al*., 1993; Hoegsberg *et al*., 1990).

15.5.4 – Granuloma Inguinale

C. granulomatis (or *Donovania granulomatis*) causes this disease. The disease is also known as "donovanosis" after Major Donovan, who discovered *Donovan bodies* in India in 1905. The incubation period is unknown, and is probably between 8 and 80 days. The lesion starts as a hard papule or nodule, which soon ulcerates. The floor of the ulcer has "beefy red" or "velvety red" granulations and bleeds on touch. The edge is rolled out. Satellite ulcers develop in the periphery of the initial lesion due to auto-inoculation and gradually the edges overlap. The regional lymph nodes are *not* enlarged. However, the lymphatics may get blocked subsequently due to fibrosis resulting in pseudo-elephantiasis of the genitals. The lesion is usually seen on the genitalia. Extragenital lesions may occur on moist and warm areas like folds between the scrotum and thighs (in males) or labia and vagina (in females). Blood-borne spread may occur to liver, spleen, bones, and lymph nodes. The disease is infectious till wet lesions are present on the skin or mucosa. Smears are taken by scraping the edge of the ulcer. Giemsa stain reveals *Donovan bodies* (Gram-negative, large, oval, intracytoplasmic bodies inside large mononuclear cells).

15.5.5 – Lymphogranuloma Venereum (LGV)

This disease, caused by *C. trachomatis* (types L-1, 2, and 3), affects the lymphatics and lymph nodes. The incubation period varies from 7 to 21 days. The lesion begins as a vesicle on the external genitalia, which ulcerates rapidly and heals without a scar. The primary lesion may not be noticed. After 1–3 weeks, the disease recurs as "climatic bubo" (so called because it is found mainly in tropics and subtropics). The inguinal lymph nodes, which are unilaterally enlarged, firm, and tender, get fixed due to periadenitis. When both the femoral and inguinal lymph nodes are enlarged, the inguinal ligament that separates the two masses appears as a groove. This is called the "grooving sign". This mass suppurates and breaks down, with seropurulent discharge from multiple sinuses (unlike the bubo of chancroid). During recurrence, systemic manifestations (fever, bodyache, malaise, joint pains, and splenomegaly) may be present. The disease is infective till the lesions are clinically active.

Complications: In both sexes, multiple abscesses may form along the lymphatics resulting in their destruction. The lymphatics are destroyed over a period of few weeks to 20 years or longer and their destruction results in elephantiasis of genitalia. Anorectal stricture may occur due to polypoid growth in rectum. This is more common in females. Urethral fistulae may occur.

Laboratory Diagnosis: Demonstration of intracellular "Halberstaedler- Prowazek inclusion bodies" with Giemsa stain.

15.5.6 – Herpes Genitalis

HSV type II (HSV-2) causes recurrent episodes of sores or ulcers in the genital area. Prevalence of HSV-2 infection is generally higher in women than in men and is associated with increasing age and sexual activity in both the developing and developed countries (Smith & Robinson, 2002). In the developed world, HSV-2 has emerged as the leading cause of genital ulcers (Maynaud & McCormick, 2001). The usual pattern of manifestation consists of a first episode, followed by recurrences. The incubation period varies from 7 days to months. The disease is *infective* even in the asymptomatic stage. The disease starts with appearance of very small vesicles, which break down forming superficial ulcers. These ulcers heal by themselves and recur again. There is intense burning sensation over the vesicles and ulcers.

Laboratory diagnosis involves the demonstration of IgG and IgM antibodies to HSV-2. The *first episode* of herpes genitalis infection is treated by oral administration of acyclovir 200–400 mg, six times a day, for 7 days or until remission occurs. Topical application of 5 per cent acyclovir cream is also advised. *Frequent* recurrent episodes are treated with orally administered acyclovir 400 mg twice a day (or 200 mg orally thrice a day). The WHO recommends acyclovir as a first-line treatment in countries where the prevalence of HSV-2 infection is greater than 30 per cent. Daily suppressive treatment with acyclovir is advised for patients who experience more than six episodes of genital herpes per year (WHO, 2003). Severe disease requires intravenous administration of the drug. Acyclovir-resistant infection usually responds to intravenous administration of foscarnet.

Herpes Genitalis and HIV Infection: Herpes genitalis is an independent risk factor for transmission of HIV. Activated lymphocytes including CD4 cells, are often recruited to sites of inflammation and are primed to receive or present HIV at the site of ulceration. Thus, genital ulcers can be a portal of entry or exit for HIV. HIV infection increases the frequency of reactivation of HSV-2 and the episodes become more extensive and persistent with the decline in CD4 cell counts. HSV-2 increases HIV replication by directly affecting HIV transcription (Margolis *et al*., 1992) or by indirect mechanisms involving cytokines (Clouse *et al*., 1989). In HIV-positive individuals, ulcers may persist indefinitely and the recurrences may be more frequent. Atypical sites, such as oesophagus

may be involved. Chronic mucocutaneous infection may manifest as painful ulcerating lesions in genital, perianal, or perioral regions. Disseminated infections, myelitis and encephalitis are rare (Wald *et al*., 1993). Persistence of herpes for more than 1 month is considered as AIDS-defining illness. HIV-infected patients may not respond to treatment with acyclovir.

15.5.7 – Genital Warts

(Synonym: Condyloma accuminata). Caused by HPV, the infection may be subclinical or may present with a broad spectrum of clinical manifestations from warts to neoplasms. This disease is not included in syndromic management.

Genital Warts and HIV Infection: The treatment is more difficult and the lesions may recur frequently (Wasserheit, 1992; Hoegsberg *et al*., 1990). Neoplastic changes may occur in the lesions in cervix uteri or anus. Hence, Pap smear should be done annually in all women suffering from genital warts. If the CD4 count drops below 200 cells per μL, the Pap smears should be taken six monthly.

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