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HIV and gynaecological infections

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Human immunodeficiency virus (HIV) infection primarily affects women during their reproductive years, and the co-existence of gynaecological infections is not surprising, given the fact that HIV is mainly acquired via heterosexual contact. Most gynaecological infections are themselves sexually acquired, and have the potential to increase the risk both of acquiring and transmitting the HI virus. As most sexually transmitted infections are asymptomatic, there is a need to improve methods of diagnosis and algorithms for early detection of sexually transmitted infections. HIV infection, however, particularly advanced disease, may alter the clinical presentation, course and response to conservative treatment for some of the sexually transmitted infections.

Key words: sexually transmitted infections; vaginitis; pelvic infections; syndromic management.

OVERVIEW OF SEXUALLY TRANSMITTED INFECTIONS AND HIV

There are a large number of studies that examine the different aspects of interaction between sexually transmitted infections (STIs) and HIV infection. In a systematic review, Rottingen et al found that the presence of a genital ulcer disease increases susceptibility to HIV in both men and women, with the effect being 1.6 times higher in men.[1] Non-ulcerative infections also increase susceptibility, but the effect was 60% of that observed for genital ulcer disease. Infections are thought to increase susceptibility to HIV infection by resulting in either loss of protective H_2O_2-producing lactobacilli, disruption of the normal epithelial barrier; recruitment and stimulation of susceptible cells, or enhanced HIV replication.

The effects of an STI on HIV infectiousness have been studied indirectly by investigating the effects of the former on biological markers for increased transmission (herpes simplex virus and HIV shedding). The presence of cervical pus or vaginal inflammation has been associated with increased local HIV shedding. The presence of

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an STI may lead to a transient increase in viral load, and since viral load is correlated with survival, STIs could theoretically enhance HIV disease progression. Though studies showed increased viral load in the presence of an STI, which could be a marker of already advanced disease, there is no clear evidence that transient increase in viraemia affects HIV disease progression. On the other hand, HIV is thought to increase susceptibility to STIs, as well as increase the infectiousness of diseases such as herpes simplex virus (HSV). In addition, HIV seems to prolong the duration of infectiousness as well as alter the clinical presentation and laboratory response of some diseases e.g. syphilis.

Ulcerative lesions

Herpes simplex virus

This is the commonest cause of infectious genital ulcer disease in both developed and developing countries.[2,3] Its prevalence has been increasing in parallel with that of HIV infection. Herpes simplex virus infections are caused by HSV type 1 (HSV 1) or type 2 (HSV 2), but 60–95% of recurrences are due to HSV 2. Infection causes mucosal disruption as well as the aggregation of large numbers of activated CD4-bearing lymphocytes, which are target cells for HIV attachment. This results in an increased risk of HI virus acquisition by the exposed host. The presence of HSV 2 (clinical and subclinical) is associated with a 2–4-fold increase in the acquisition of HIV infection.[1] Both clinical and subclinical HSV infection lead to increased replication of HIV at mucosal surfaces, and this results in increased transmission of the virus. It is possible therefore that suppression of HSV infection by antiviral agents may reduce the rate of HIV transmission.[4] HSV 2 infection may also enhance HIV disease progression.

HIV also alters the course of HSV infection. Most HSV reactivations are subclinical, and occur at all levels of CD4 counts; however, HIV-infected women have more frequent reactivation rates, and are more likely to shed HSV than HIV-uninfected women.[5] HIV-infected women with HSV may be treated with any of the three antiviral agents available—aciclovir, valaciclovir and famciclovir, all with equal efficacy. Treatment of acute episodes limits the duration of symptoms and reduces the chance of recurrence, and may be achieved by any of the antiviral agents, over 5 days (e.g. aciclovir 200 mg 5 times daily for 5 days), whereas recurrent episodes may be treated over a shorter duration. If new lesions develop whilst receiving treatment with aciclovir, the dose may be increased to 800 mg 5 times a day, and the course prolonged to 7 days, or the antiviral agent should be changed.[6] At least 10% of HIV-infected women have aciclovir-resistant HSV.[7] Chronic suppressive therapy may be considered for people with poorly controlled HIV disease, or frequent HSV reactivation (six or more recurrences per year). A randomized controlled trial showed that both clinical and subclinical recurrences can be reduced by famciclovir given at a dose of 500 mg twice daily.[8]

Syphilis

Syphilis is a systemic disease, regarded as a great mimicker, with variable clinical manifestations, depending on the stage of the disease. Though the prevalence of syphilis is declining in most developed countries, it remains almost steady in developing countries, despite adequate methods of diagnosis and treatment.[9] As an ulcerative disease, syphilis is associated with a 2.5 increased effect in the acquisition of HIV