The genitourinary complications of HIV infection in men

This article discusses infective and malignant complications of HIV affecting the genitourinary tract in men. Immunosuppression increases both the frequency of infections, and the range of organisms that may be involved. Cancers are common and presentations may be atypical.

The first case of acquired immunodeficiency syndrome (AIDS) was reported in 1981. The human immunodeficiency virus (HIV) was identified in 1983 as the cause of AIDS, which was then a devastating and rapidly fatal condition. The CD4 T-cell count is used to monitor disease progression. The natural history is for the CD4 count to fall, with a correspondingly increased risk of opportunistic infections and malignancies. At CD4 counts below 200 cells/mm^3 there is a significant risk of AIDS-defining illnesses. The aim of highly active antiretroviral therapy (HAART) is to fully suppress HIV replication and achieve an undetectable plasma viral load. HAART has transformed the course of HIV infection from a terminal illness into a chronic condition requiring long-term management over many decades. The optimum time for initiating treatment is the subject of ongoing research. Current guidelines recommend HAART for symptomatic HIV disease or AIDS diagnosis at any CD4 count, and in asymptomatic individuals with CD4 less than 350 cells/mm^3 (Gazzard, 2006).

Although HIV infection can involve all organ systems, the genitourinary system is primarily and secondarily affected. This article reviews the genitourinary manifestations of HIV infection and AIDS, focusing on men.

Epidemiology of HIV in the UK

By the end of 2005, 78 938 cases of HIV infection had been diagnosed in the UK since the epidemic began in the early 1980s (Health Protection Agency, 2006). In contrast, the numbers of AIDS diagnoses and deaths in HIV-infected individuals fell after HAART was introduced in the mid-1990s and has remained relatively constant since then (*Figure 1*).

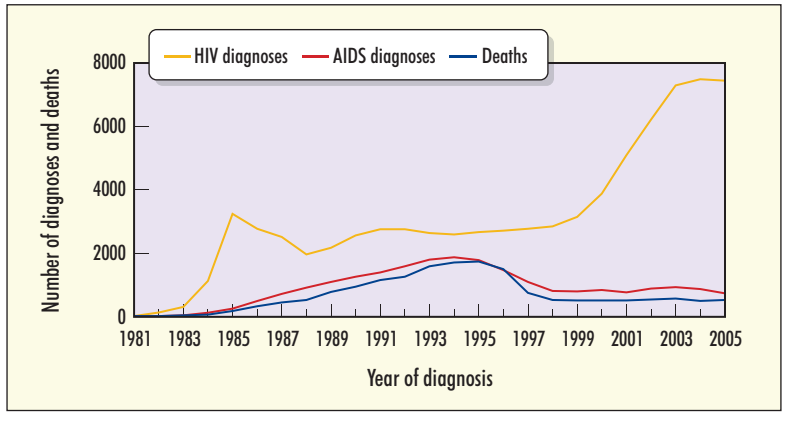
The continued rise in HIV diagnoses in the UK is the result of newly acquired infections, migration patterns, and increased uptake of HIV testing. At the end of 2005, approximately 44% of infected adults were men who have sex with men, 32% heterosexual women, 21% heterosexual men and 3% intravenous drug users (Health Protection Agency, 2006). The incidence of newly acquired infections in men who have sex with men remains high. Nevertheless, heterosexuals represented 54% of the new diagnoses in 2005. Of heterosexually transmitted infections, 85% were acquired outside the UK, and of these 89% were in Africa, the region with the highest HIV prevalence in the world.

The prevalence among injecting drug users has remained relatively low, at 3.2% in London and 1.2% in the rest of England. A small number of new diagnoses are still attributed to receipt of blood transfusions, and almost all of these individuals received transfusions in countries outside the UK (*Figure 2*). About a third of people living with HIV in the UK are unaware of their infection (Health Protection Agency, 2006).

Urinary tract infections

In the pre-HAART era, urinary tract infections occurred in 17% of HIV-infected patients (Kaplan et al, 1987). In their analysis of patients with AIDS, Kaplan et al reported that although *Escherichia coli* is responsible for 80% of urinary tract infections in the general population, it was the causative organism in only 25% of patients with AIDS. Other bacterial causes include *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, Enterobacter, Proteus, Acinetobacter, Staphylococci, Streptococci and Salmonella. Atypical pathogens include mycobacteria, fungi (*Candida*

Figure 1. HIV and AIDS diagnoses and death in HIV-infected people. From Health Protection Agency (2006).



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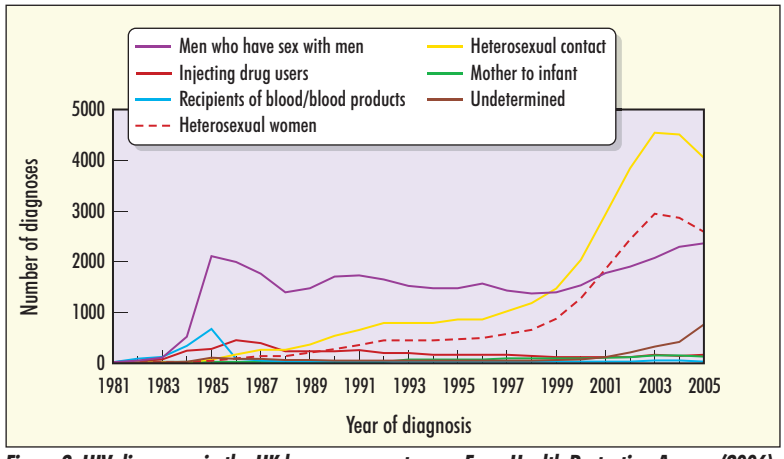


Figure 2. HIV diagnoses in the UK by exposure category. From Health Protection Agency (2006).

albicans, Aspergillus, *Cryptococcus neoformans*, *Histoplasma capsulatum*, *Pneumocystis jirovecii*), protozoa (*Toxoplasma gondii*) or viruses (cytomegalovirus, adenovirus).

Although bacterial infection may be present, cultures are often negative; patients with CD4 less than 200 cells/mm^3 take prophylaxis against *Pneumocystis jirovecii* pneumonia, most commonly with co-trimoxazole, which can result in negative urine cultures.

Uncomplicated urinary tract infections in women do not require follow up, but in men confirmed urinary tract infections should always be investigated, regardless of HIV status. Patients who have recurrent infections with an atypical organism or have evidence of pyelonephritis should be evaluated either with renal ultrasound or an intravenous urogram to exclude an abscess or urinary lithiasis. In HIV-infected patients antibiotics should be tailored to culture results and antibiotic sensitivities wherever possible. Similar to the general population, HIV-infected patients with asymptomatic bacteriuria need no treatment.

Prostatitis

The incidence of acute bacterial prostatitis is 1–2% in the general population, whereas in the pre-HAART era it was 3% in asymptomatic HIV-infected patients and 14% in patients with AIDS (Santillo and Lowe, 2006). *E. coli* is the typical causative agent for bacterial prostatitis in the general population, while HIV-infected patients are affected by typical and atypical bacteria, viruses and fungi. Other bacterial organisms that are involved in prostatitis include *Staphylococcus aureus*, *K. pneumoniae*, *Ps. aeruginosa* and *Salmonella typia*.

Gram-negative organisms cause prostatitis by way of reflux of infected urine into prostatic ducts. Fluoroquinolones for 4 weeks are recommended as first-line agents (Santillo and Lowe, 2006). Because of the ability of the prostate to harbour organisms and the poor penetration of antimicrobials into prostatic tissues, HIV-infected patients often have persistent sub-clinical foci that cause relapsing prostatitis. The incidence of prostatic abscesses in AIDS has decreased significantly with the advent of HAART, and they generally only occur in patients with very low CD4 counts.

Epididymitis

In young men, epididymitis usually results from spread of sexually transmitted pathogens from the urethra. *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are the most common causative organisms. In men who have sex with men engaging in unprotected anal intercourse, coliform organisms may be responsible. Urethritis may be asymptomatic, but can be detected by microscopy of urethral smear. Appropriate treatment is ceftriaxone 250 mg intramuscularly in a single dose (to cover gonococcus) plus oral doxycycline 100 mg twice daily for 10–14 days (Clinical Effectiveness Group (Association for Genitourinary Medicine and the Medical Society for the Study of Venereal Diseases), 2001a). Suppurative and/or antibiotic-resistant epididymitis may be the first presentation of a previously undiagnosed HIV infection, and may be caused by fungi or mycobacteria.

Urethritis

In recent years there has been an increase in incidence of other sexually transmitted infections in HIV-infected patients, as in the general population. Urethritis is most commonly caused by *C. trachomatis* or *N. gonorrhoeae*. Urethritis is diagnosed if ≥5 white blood cells per oil-immersion field are detected on the gram staining of urethral secretions, or if ≥10 white blood cells are seen per high-power field on microscopy of first void urine. Other infectious causes of urethritis include *Mycoplasma genitalium* (Taylor-Robinson and Horner, 2001), *Trichomonas vaginalis*, herpes simplex virus (HSV), warts, adenovirus and candida. Non-infectious aetiologies include urethral strictures and topical irritants.

Although there is no absolute gold standard for chlamydia testing, nucleic acid amplification tests, including polymerase chain reaction, have a sensitivity of at least 90%, compared with 60–70% for culture and 60% for antigen assays (Skidmore et al, 2006). Nucleic acid amplification tests also have a high specificity and are now the diagnostic test of choice. The sensitivity of nucleic acid amplification tests on urine samples from males is high and may even be higher than a urethral swab, perhaps because of the difficulty in taking an adequate urethral sample. In women the sensitivity of urine testing is lower, and a vaginal or cervical swab is preferable (Skidmore et al, 2006). Chlamydial infections can be treated with doxycycline 100 mg twice daily for 7 days or single dose oral azithromycin 1 g, with a 97% cure rate (Lau and Qureshi, 2002). Antibiotic resistance has not been an issue to date.

The presence of gram-negative intracellular diplococci establishes a diagnosis of gonorrhoea (*Figure 3*). If a microscopic diagnosis of gonorrhoea is made, individuals should also be treated presumptively for chlamydia, since concomitant infection is found in 30–50% of cases

(Centers for Disease Control and Prevention, 2006). Treatment of *N. gonorrhoeae* is complicated by widespread antimicrobial resistance. Quinolone resistance reached 22% in gonococcal isolates in England and Wales in 2005, and ciprofloxacin should only be used where antibiotic sensitivities are available. First-line treatment in the UK is usually cefixime 400 mg as a single dose orally, or ceftriaxone 250 mg as a single dose intramuscularly if pharyngeal penetrance is required (Clinical Effectiveness Group (British Association for Sexual Health and HIV), 2005).

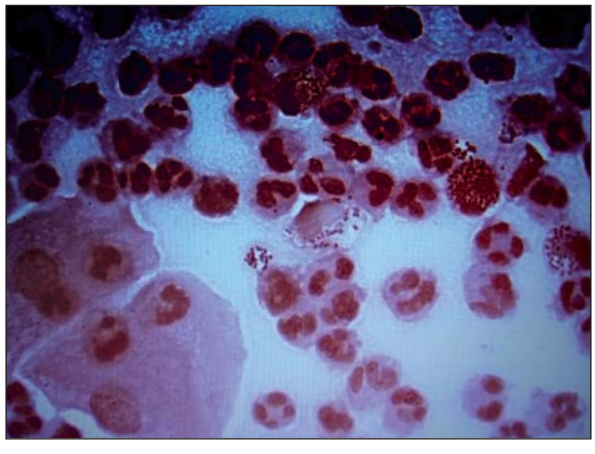
Asymptomatic urethritis should always be treated in order to prevent onward sexual transmission of pathogens. In the context of HIV infection, this is particularly pertinent, since concomitant sexually transmitted infections facilitate HIV transmission. There is good evidence that urethritis and cervicitis (Cohen, 1998) and trichomonas (McClelland et al, 2007) increase the risk of HIV acquisition. Syndromic treatment of sexually transmitted infections can reduce HIV incidence (Korenromp et al, 2005).

Genital ulcers

The commonest cause of genital ulceration in the UK is HSV, followed by syphilis. HSV migrates to the dorsal root ganglia, where it may remain latent or produce recurrent symptoms. Historically, HSV-2 caused most genital herpes, whereas orolabial herpes resulted from HSV-1. In fact both viruses can infect both sites, and in the developed world genital herpes is now caused by HSV-1 at least as often as by HSV-2 (Haddow et al, 2006). Herpes is diagnosed by viral culture or polymerase chain reaction after taking a swab from an ulcer or punctured blister (*Figure 4*). Licensed treatment regimens include acyclovir 200 mg five times daily or valaciclovir 500 mg twice daily for 5 days, although in HIV co-infection higher doses of acyclovir may be needed (Clinical Effectiveness Group (British Association for Sexual Health and HIV), 2007a).

The interaction between HIV and HSV is complex. HSV-2 increases the risk of HIV acquisition up to fourfold, particularly after recent infection (Freeman et al, 2006). Co-infection with HSV-2 also increases the probability of an HIV-positive individual transmitting HIV to his/her sexual partners (Gray et al, 2001), possibly via breaches in the mucosal integrity, increasing numbers of activated CD4 cells in herpes lesions and upregulating HIV replication in HSV co-infected cells. Both clinical outbreaks of HSV-2 and asymptomatic shedding increase HIV viral load in the plasma and genital tract (Schacker et al, 2002). Studies are ongoing to see whether suppressive treatment for HSV can impact on transmission of HIV.

Figure 3. White blood cells containing gram-negative gonococci.



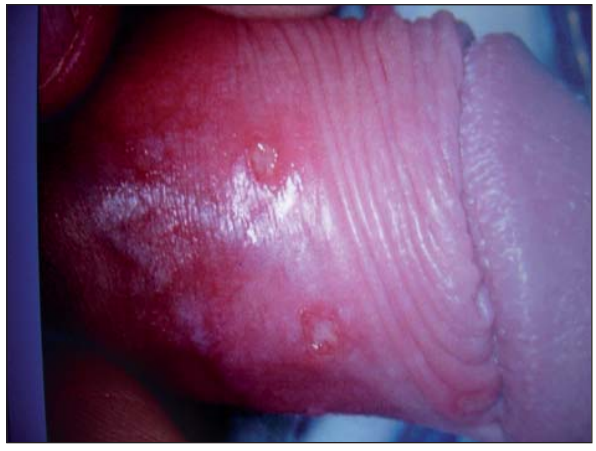
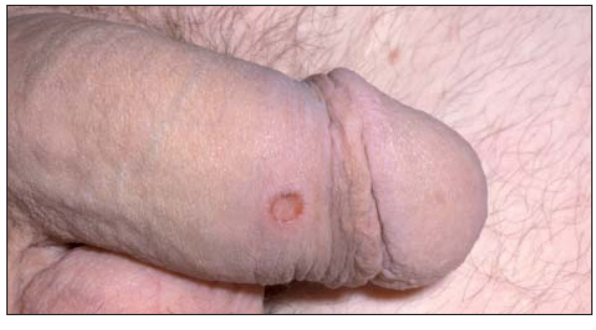


Figure 4. Penile herpes.

The UK has seen a resurgence in the incidence of syphilis (Frauenfelder, 2006), and syphilis and HIV share epidemiological features. Genital ulcer disease (Wawer et al, 2005) and syphilis (Reynolds et al, 2006) both increase the risk of acquisition of HIV. All patients diagnosed with syphilis should therefore be counselled and tested for HIV. Primary syphilis manifests as an ulcer at the site of inoculation, which is typically solitary and painless (*Figure 5*). The diagnosis can be made by visualizing the spirochetes *Treponema pallidum* by dark-field microscopy, or a dry swab can be sent to the reference laboratory for detection of treponema antigens by polymerase chain reaction. Syphilis serology is the most important diagnostic tool, although it can be difficult to interpret and is beyond the scope of this review. Rapid simple dipstick treponemal tests have been developed (Mabey et al, 2006) which may increase the coverage of syphilis screening programmes by allowing testing in settings without laboratory facilities. In early primary infection serology

Figure 5. Primary syphilis chancre.



may be negative, and in HIV infection false negative results and the delayed appearance of seroreactivity have been reported. HIV-infected patients have higher rates of neurological complications early in the disease and experience more treatment failure. It is recommended that individuals with syphilis be referred to genitourinary medicine specialists for management and follow up.

Lymphogranuloma venereum

Lymphogranuloma venereum is a systemic disease caused by the invasive serovars L1, L2 or L3 of *C. trachomatis*. Lymphogranuloma venereum had been a rare occurrence in industrialized countries from the 1960s until 2003, when outbreaks began to occur among men who have sex with men, most of whom were HIV positive.

The primary lesion is transient and often imperceptible, in the form of a painless papule, pustule or shallow erosion. It is may be found on the penile coronal sulcus, and in women on the posterior vaginal wall, fourchette or on the vulva. After receptive anal sex, rectal infection may develop, and men who have sex with men usually present with proctitis. Spread to regional lymphatics causes lymphadenopathy after a few days to weeks. Penile infection leads to inguinal and/or femoral lymphadenopathy that is typically unilateral. Involvement of lymph nodes above and below the inguinal ligament results in the classical 'sign of the groove'. Vaginal or rectal inoculation leads to retroperitoneal lymphadenopathy which is not clinically obvious. Untreated disease can lead to genital elephantiasis, strictures or fistula formation. Diagnosis of lymphogranuloma venereum requires a high index of suspicion, and is made by nucleic acid amplification tests followed by serovar typing in a reference laboratory. Treatment requires prolonged courses of antibiotics such as doxycycline 100 mg twice daily for 3 weeks (Clinical Effectiveness Group of the British Association for Sexual Health and HIV, 2006).

Genital warts

Genital warts (condyloma acuminata) are caused by human papilloma viruses (HPV). Ninety per cent involve HPV type 6 or 11 (*Figure 6*). Warts occur significantly more commonly in HIV-infected patients than the general population (Rosen, 2006). Treatment for genital warts remains unsatisfactory, with all treatments having initial response rates of 50–90%, and recurrences are common. Small areas of warts can be treated by topical podophyllotoxin, trichloroacetic acid or cryotherapy (Clinical Effectiveness Group (British Association for Sexual Health and HIV), 2007b). An immune response modifier, imiquimod 5% cream, has similar response rates to other treatments and may reduce the likelihood of recurrences. Extensive areas may require surgical excision. Vaporization with carbon dioxide lasers may be hazardous because of the dispersion of virus in the aerosol.

Figure 6. Penile wart.

Molluscum contagiosum

Molluscum contagiosum is caused by a pox virus (*Figure 7*). It is a common and transient infection in immunocompetent children or adults, and can be left alone, or treated in the same ways as genital warts (Tyring, 2003). In untreated HIV disease lesions may be giant (>1 cm), persistent and fail to respond to treatment. Improvements in immunity associated with HAART have made extensive molluscum much less common.

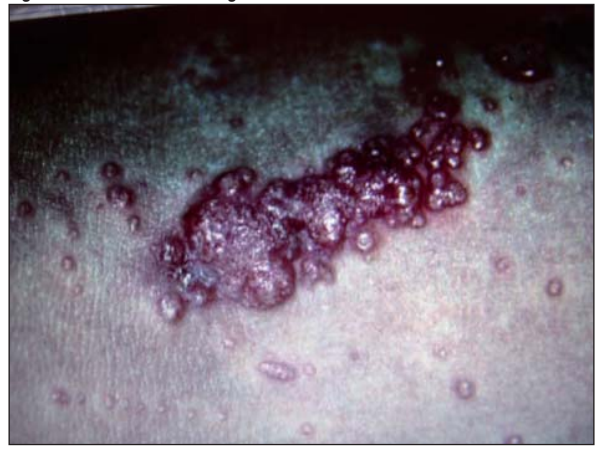
Candidiasis

Approximately 75% of HIV-infected women have at least one episode of vulvovaginal candidiasis. Candida balanitis is less common. The diagnosis is usually made on clinical grounds (*Figure 8*). Treatment is with topical antifungals such as clotrimazole or miconazole cream, or oral fluconazole 150 mg as a single dose (Clinical Effectiveness Group (Association for Genitourinary Medicine and the Medical Society for the Study of Venereal Diseases), 2001b). Longer courses and higher doses may be needed in HIV-positive patients, for example oral fluconazole 200 mg once daily for 7 days.

Urolithiasis

Nephrolithiasis has been found in up to 40% of kidneys of AIDS patients examined at autopsy, but this does not appear to be clinically significant (Seney et al, 1990). Renal colic was reported in 2–7% of patients taking the protease inhibitor drug indinavir (Clayman, 1998).

Figure 7. Molluscum contagiosum.



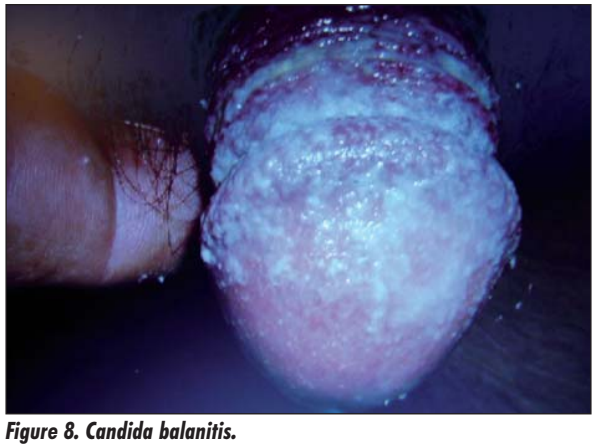


Figure 8. Candida balanitis.

Indinavir is 20% excreted unmetabolized in the urine, where it precipitates to form crystals. Indinavir is rarely used nowadays, but kidney stones have also been reported with the protease inhibitors lopinavir and atazanavir.

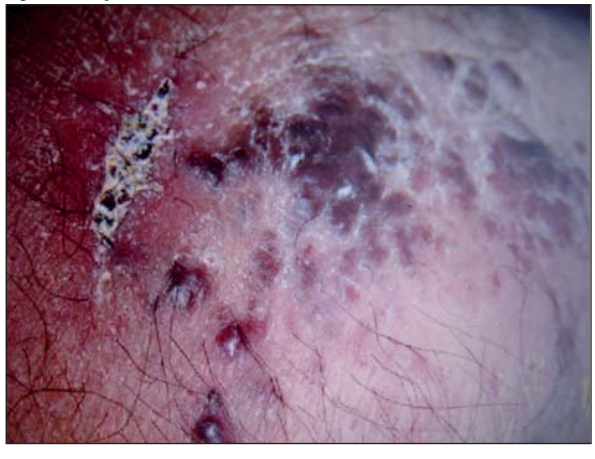
Malignancy

HIV-positive individuals are at an increased risk of cancer, especially malignancies caused by viruses, such as human herpesvirus-8-related Kaposi's sarcoma and Epstein–Barr virus-associated non-Hodgkin's lymphoma (Grulich et al, 2007).

Kaposi's sarcoma is a disease of the reticuloendothelial system that can involve any organ in the body, but predominantly the skin. The genitalia are affected in up to 20% of cases (Lowe et al, 1989). Cutaneous lesions present as purple, papular, plaque-like or ulcerated lesions that are usually painless (*Figure 9*). When Kaposi's sarcoma presents on the penis, it is usually indicative of widespread disease. The diagnosis is confirmed on histology. Treatment is with HAART, with or without intralesional chemotherapy or radiotherapy. When Kaposi's sarcoma is disseminated to organs such as the gut or lungs, systemic chemotherapy is required, with a 3-year overall survival of over 80% (Nasti et al, 2003).

In the pre-HAART era, non-Hodgkin's lymphoma was about 60–200 times more common in HIV-infected patients than in the matched HIV-negative population (Stebbing et al, 2004). HIV-positive patients still present with more symptomatic and advanced disease, but with HAART, the survival of patients with AIDS-related non-Hodgkin's lymphoma has improved (Palmieri et al, 2006). Non-Hodgkin's lymphoma of the testis is more common in HIV-positive men and is more aggressive and more likely to be bilateral than in the general population. At diagnosis, the disease has often metastasized widely. Lymphoma also affects other parts of the genitourinary system including the kidneys (6–12%) (D'Agati and Appel, 1998), where it commonly presents as bilateral enlarged kidneys with diffuse infiltration, or as single or multiple renal parenchymal masses. Non-Hodgkin's lymphoma is treated with HAART and combination chemotherapy using a regimen similar to those used in the general population (Powles et al, 2002). Complete response rates of about 50–75% are seen in HIV cohorts, with a 2–3-year overall survival of 40–60% (Behlar and Kaplan, 2006).

Figure 9. Kaposi's sarcoma.



Germ cell testicular tumours are 20–50 times more prevalent among immunocompromised individuals (Powles et al, 2004). HIV-related germ cell testicular tumours are not more aggressive than germ cell testicular tumours in the general population, and have identical treatment response rates and tumour-free survival. Orchidectomy and surveillance is a safe management policy in stage 1 germ cell testicular tumours in HIV-positive patients if they are fully compliant with the follow-up protocol. Metastatic germ cell testicular tumours may respond to chemotherapy (Powles et al, 2004).

Anal cancer is associated with oncogenic HPV, predominantly types 16 and 18. It is more common in men who have sex with men than heterosexuals, and the risk is further increased by HIV infection. A meta-analysis suggests that HIV infection results in a 1.5-fold increased risk of renal cancer, but that there is no increased incidence of bladder or prostate cancer (Grulich et al, 2007).

Conclusions

The advent of HAART, and prophylaxis against opportunistic infections, have dramatically altered the prognosis of HIV infection, which is now managed as a chronic disease. Increasing numbers of HIV-infected patients will present to clinicians with infections, urolithiasis and malignancies. The epidemiology of sexually transmitted infections and HIV are intimately intertwined, and sexually transmitted infections need to be recognized and treated promptly to reduce morbidity, and transmission of HIV. **BJHM**

Conflict of interest: none.

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KEY POINTS

- HIV prevalence in the UK continues to increase. Contributing factors include sustained levels of newly acquired infections in men who have sex with men, further diagnoses among heterosexuals who acquired their infection in Africa, and earlier and increased HIV testing.
- HIV-infected patients with symptomatic urinary tract infections should be treated with antibiotics that are culture-specific, while patients with asymptomatic bacteriuria need no treatment.
- The UK has seen a resurgence in the incidence of syphilis and all patients diagnosed with syphilis should be counselled and tested for HIV.
- Sexually transmitted infections should always be identified and treated promptly, to reduce the risk of both acquisition and onward transmission of HIV.
- HIV infection significantly increases the risk of many cancers, including Kaposi's sarcoma and non-Hodgkin's lymphoma, although the prognosis is greatly improved by highly active antiretroviral therapy.