AIDS-related malignant disease

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Abstract

Immunodeficiency, whether congenital or acquired, iatrogenic (e.g. allograft recipients) or infectious (e.g. human immunodeficiency virus (HIV)), is associated with an increased risk of malignancy. In the case of HIV infection, most cancers are associated with oncogenic virus infection. Although the overall risk of any cancer is increased 2–3-fold in people living with HIV, there are three acquired immune deficiency syndrome (AIDS)-defining cancers whose relative risk is dramatically higher. These three AIDS-defining illnesses are Kaposi's sarcoma, high-grade B cell non-Hodgkin's lymphoma (including primary cerebral lymphoma) and invasive cervical cancer. Since the introduction of combination antiretroviral therapy, the incidence of the AIDS-defining malignancies has declined in populations with access to these medications. In contrast, the effect on the incidence of other cancers has been small; however, the increased longevity of people living with HIV and the ageing of this population mean that there has been a rise in the number of cases of non-AIDS-defining malignancies. Recent advances in the management of malignancy in people with HIV have led to similar outcomes to those for the general population.

Keywords AIDS; anal cancer; cervical cancer; HAART; HIV; Hodgkin's lymphoma; Kaposi's sarcoma; MRCP; non-Hodgkin's lymphoma

Key points

- People living with HIV are at increased risk of cancers
- Human oncogenic viruses, such as Epstein–Barr virus, human herpesvirus 8, human papillomavirus (HPV), hepatitis C virus (HCV) and hepatitis B virus (HBV) play a major role in the pathogenesis of HIV-associated cancers
- In the era of highly active antiretroviral therapy, the outcomes for cancer are similar for HIV-seropositive people and the general population as long as the same anti-cancer therapy is used
- All patients' treatment must include combination antiretroviral therapy and prophylaxis for opportunistic infection as well as specific chemo- and/or radiotherapy
- Screening programmes and HPV vaccination are key to reducing the incidence of HPV-related malignancies

Acquired immune deficiency syndrome (AIDS)-defining malignancies

Systemic non-Hodgkin's lymphoma (NHL)

Epidemiology and histology: systemic high-grade B cell NHLs occur 60–100 times more commonly in people living with human immunodeficiency virus (HIV) than in the age- and gender-matched general population. The incidence has fallen since the introduction of combination antiretroviral therapy (cART), although this fall is more marked for primary cerebral lymphoma (PCL) than for systemic NHL.[1] Around one-third of these tumours are Burkitt's lymphomas, and two-thirds are diffuse large cell lymphomas. A minority are rarer plasmablastic and primary effusion lymphoma variants rarely diagnosed in immunocompetent individuals.

Clinical presentation: half the cases of HIV-associated systemic NHL present with nodal disease, a third with gastrointestinal disease and the remainder with disease at other extranodal sites. Finally, a very few present as effusions without a nodal mass of disease; this variant, called primary effusion lymphoma, is associated with Kaposi's sarcoma herpesvirus (KSHV).

Clinical management and prognosis: the treatment of systemic AIDS-related NHL involves administering combination chemotherapy with intrathecal chemotherapy for individuals at risk of meningeal relapse. This should be given with concomitant cART and prophylaxis against *Pneumocystis jirovecii*, *Mycobacterium avium* complex, recurrent herpes simplex and fungal infections.[2] This combined management approach results in durable complete remission in 65% of patients, who will in effect be cured of their lymphomas.

Primary cerebral lymphoma

PCL is lymphoma that is confined to the cranio–spinal axis without systemic involvement. It is associated with advanced immunosuppression and has a particularly poor prognosis. The incidence of PCL has declined dramatically since the introduction of cART.

Toxoplasmosis and PCL are the most common causes of cerebral mass lesions in HIV, and the differential diagnosis can prove difficult; both occur at low CD4 counts (<50 cells/mm^3) and present with headaches and focal neurological deficits. Clinical features that favour PCL include a more gradual onset over 2–8 weeks and the absence of a fever. Computed tomography (CT) and magnetic resonance imaging (MRI) usually reveal solitary or multiple ring-enhancing lesions with prominent

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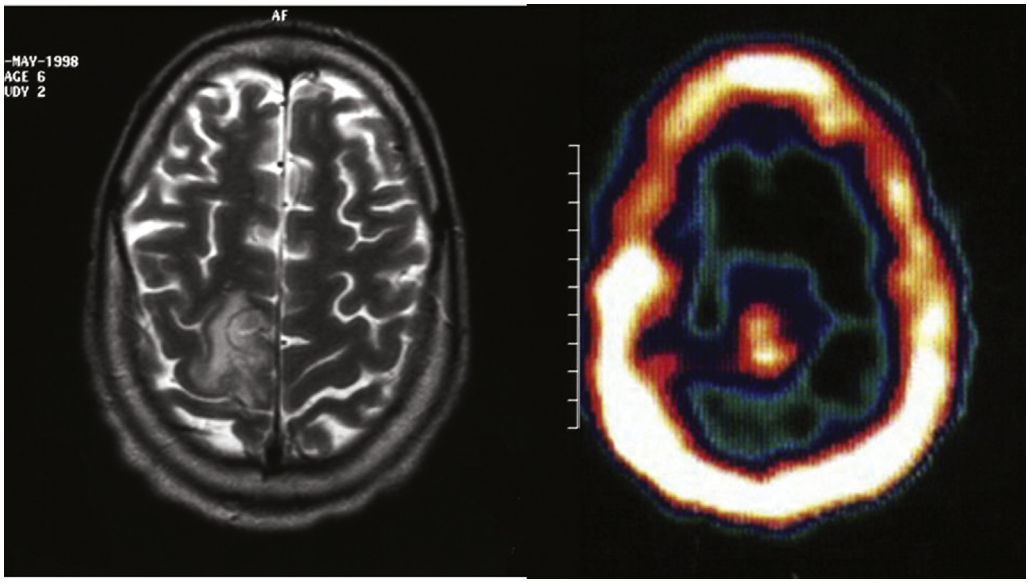

Figure 1 Paired MRI and FDG-PET scans of a patient with AIDS-related PCL.

mass effect and oedema (Figure 1). Again, these features occur in both diagnoses, although PCL lesions are usually periventricular, whereas toxoplasmosis more often affects the basal ganglia.

More than 85% patients with cerebral toxoplasmosis respond clinically and radiologically to 2 weeks of anti-*Toxoplasma* therapy, and this has become the cornerstone of the diagnostic algorithm for cerebral masses in severely immunodeficient patients. Immunodeficiency-related PCLs are Epstein–Barr virus (EBV)-related tumours, and the detection of EBV DNA in the cerebrospinal fluid by polymerase chain reaction (PCR) has become established as a diagnostic test with a high sensitivity and specificity.[3] ^{18}F-Fluorodeoxyglucose positron emission tomography (FDG-PET) also helps to differentiate between PCL and cerebral toxoplasmosis.

Clinical management and prognosis: the standard treatment modality for PCL in HIV patients is palliative whole-brain

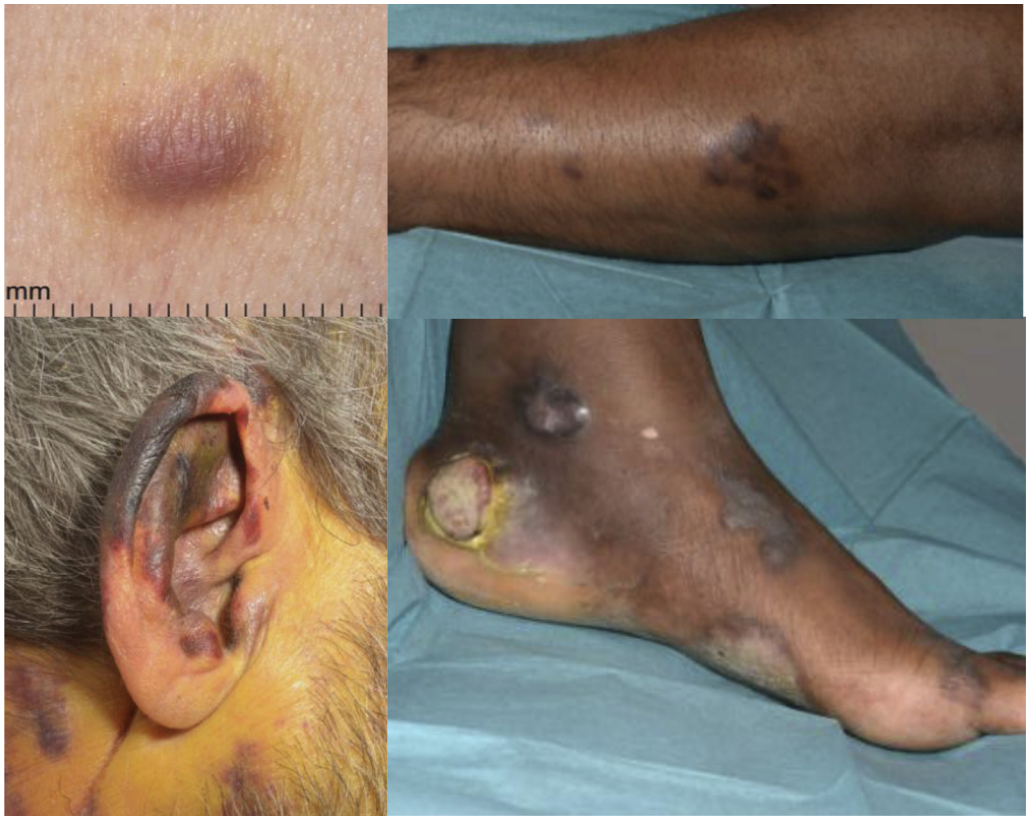

Figure 2 Cutaneous KS lesions.

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