0036-9330/87/0517/0101 $2.00 in USA

Scot Med J 1987;32:101-107

CLINICAL MANIFESTATIONS OF HIV INFECTIONS

D.H. Kennedy
Department of Infectious Diseases, Ruchill Hospital, Glasgow, G20 9NB

Abstract: *The wide clinical spectrum of HIV infection is reflected in the new CDC classification. Presentations range from acute infection, asymptomatic carriage and persistent lymphadenopathy through constitutional upset and neurological disease to the opportunistic infections and cancers of AIDS. AIDS is an enigmatic disease which presents special clinical diagnostic and management problems. Although any system may be affected the lungs and the brain are the most important target organs. Though the underlying disease is currently untreatable, many of the complications of AIDS are amenable to prompt therapy. Seropositive patients should be monitored to detect early signs of significant HIV disease. Skilled counselling about the avoidance of co-factors which may potentiate HIV infection, is important.*

Key words: AIDS, clinical, symptoms, signs.

'A dangerous ghost has escaped from the bottle. It appeared to us in various disguises.' — **Eva Klein.**

THE human immunodeficiency virus (HIV) type 1 is a versatile agent of disease. Furthermore, its pathogenic potential is yet to be fully realised. It is still only relatively few years since this demon was loosed into the human population. Only time and further transmission will bring into sharper focus its clinical profile. This virus, like the tubercle bacillus, produces a chronic infection affecting a variety of tissues and may thus present clinically very diverse problems. It took centuries to define the spectrum of tuberculosis; six years will hardly suffice for HIV infection.

In the meantime efforts continue to resolve many ambiguities over classification and terminology of infection. This review will largely follow the new CDC classification system (Table I). This system gives a comprehensive yet flexible outline of the protean nature of HIV infection.[1] However 'classical' AIDS is described here as Group IV subgroups C-1 and D, unlikely perhaps to replace the popular term 'AIDS'. Since it is the lethal endpoint of infection, and was historically the starting point for recognition of HIV disease, this article will start by reviewing the condition of AIDS.

The Acquired Immune Deficiency Syndrome

More than a single disease, AIDS is a kaleidoscope of many disorders. The by-word for AIDS is opportunism: opportunistic infections and opportunistic neoplasms. However, infections are, by far, the commonest problem and the AIDS patient has been described as a walking culture medium. Problems may occur in a single, multiple or sequential fashion; they may become disseminated and they can affect virtually every body system. The frequency of the commonest opportunistic diseases, which resulted in registration of the first 16,000 US cases, is shown in Figure 1.

The two commonest conditions, accounting for about 75% of presentations in gay men, are *Pneumocystis carinii* pneumonitis (PCP) and Kaposi's sarcoma (KS). In addition the AIDS patient may suffer a constellation of infections due to various protozoa, viruses, fungi and bacteria. These problems are somewhat predictable since the core defect in AIDS is malfunction of the T^4 or helper lymphocyte, the cell which sets the immune response in motion.

Other cells whose function is collaterally disturbed, include B cells (antibody defects), macrophages (infections with mycobacteria, salmonella and toxoplasma) and natural killer cells (virus infections and tumours).

TABLE I
CDC classification of HIV infection

Group	I	—	Acute infection
	II	—	Asymptomatic infection
	III	—	Persistent generalised lymphadenopathy
	IV	—	Other disease:
	Subgroup	A	— Constitutional disease
		B	— Neurological disease
		C	— Secondary infectious diseases
		C1	— as defined for AIDS
		C2	— others
		D	— Secondary cancers
		E	— Other conditions

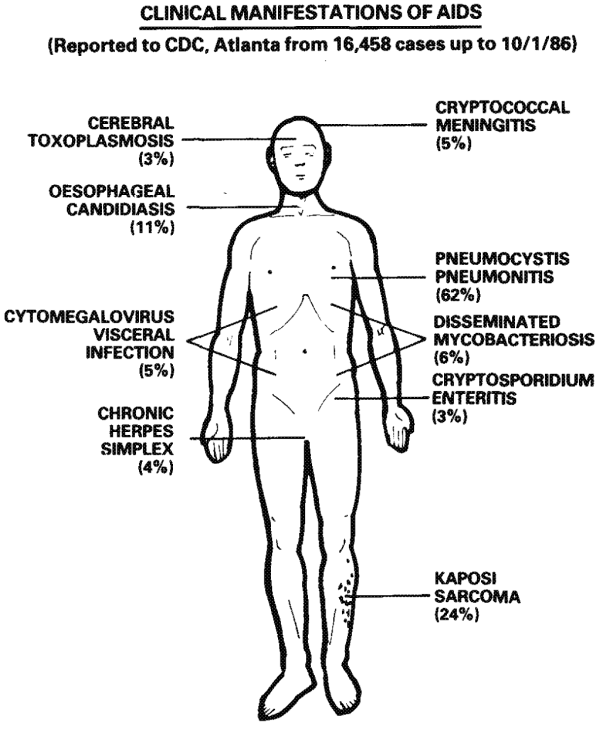

Figure 1 Clinical manifestations of AIDS

SOME GENERAL ASPECTS OF AIDS

Diagnosis

By formal definition, AIDS is the occurrence of a reliably diagnosed disease, at least moderately indicative of underlying cellular immune deficiency, in the absence of an underlying cause other than HIV infection. Reliable diagnosis often requires an invasive procedure, usually with biopsy. In some instances diagnosis of AIDS may have to be temporarily reserved and the immediate problems dealt with.

AIDS can be a difficult disease to diagnose since it often flouts normal diagnostic conventions. For various reasons neither serological nor culture techniques can reliably incriminate any given pathogen, particularly cytomegalovirus (CMV) so that histological confirmation is often necessary. An immunological workup is also a necessary investigation in AIDS.

Presentation and prognosis

In AIDS the nature of the presentation may partly depend on the baggage of latently acquired disorders which have anteceded it. This helps to explain the diversity of manifestations of AIDS and the variations between different risk and geographical groups. Common to all groups, however, are infections with ubiquitous environmental organisms such as pneumocystis, toxoplasma, candida and atypical mycobacteria.

The nature of the immune defect in AIDS has clinical and diagnostic implications. The impaired inflammatory response may lead to more subtle, atypical and protracted presentations than is 'conventional'. Thus the pneumonitis due to *P. carinii* may take many weeks to evolve, and the CSF cell response in cryptococcal meningitis may be completely absent. Furthermore, different complications in AIDS may register different degrees of immune compromise. Kaposi's sarcoma reflects less severe damage than disseminated CMV or atypical mycobacterial infection.[2]

AIDS may entail a protracted sequence of problems, some of which are recurrent. The illness may shift from infection to tumour to further infection, with a quickening in tempo towards the terminal phase. The prognosis will vary according to manifestation. The median survival time for KS is about 21 months, for PCP it is about 11 months, and it averages five months for other infections. Up until now this diagnosis has virtually meant a death sentence with an 80% mortality occurring by 36 months.

Management

The effectiveness of therapy in AIDS will vary with the type of complication. Some problems as with herpetic or candidal infections, are eminently treatable, whereas others, lymphoma for example, are rapidly progressive. It is important to treat potentially curable diseases at an early stage since they may prove lethal. Furthermore there is anecdotal evidence that they may worsen the patient's underlying condition especially if they are severe or protracted.

Indeed systemic infections may magnify the development of the AIDS dementia complex and this might therefore be checked by earlier intervention. Some infections such as PCP tend to recur and prophylaxis may be deemed appropriate. However many drugs are toxic to AIDS patients and must be used with circumspection. Some will commonly result in allergic reactions or marrow depression and co-trimoxazole is one good example. In the future, anti-retroviral therapy, such as with azidothymidine, is likely to repress rather than eliminate HIV infection. Thus it may only stabilise, rather than improve, the underlying immune problem which may require other means of immune reconstitution. Judicious intervention with antiviral therapy is likely to be more profitable in the pre-AIDS state.

Until new therapies prove successful, the AIDS patients may undergo, ultimately, much suffering. The appropriateness of management which continuously compounds pre-existing misery with further distress must be addressed. This should be discussed in advance with the patient. Whilst tactical decisions may be deferred to the doctor, strategic ones should perhaps be shared fraternally with the patient. Most patients are very grateful for any initially trouble-free period to set their affairs in order. We must await expectantly for therapeutic advances!

Psychosocial problems

The psychosocial problems encountered in AIDS cases can be as complex as they may be unremitting. With a diagnosis that is a prediction of death a chronic state of fear may develop. Anxiety about the terminal phase and the manner of dying may prove very disabling. Many young people have not considered the prospect of death and its contingencies. There is often fear of isolation, abandonment and powerlessness. There may be anxieties about public stigmatisation because of AIDS or of a hidden lifestyle such as homosexuality. Confidentiality is therefore important.

As physical deterioration progresses social problems emerge and this is particularly true of demented patients (and their families, too). There may be employment problems and disruption of relationships. Finally there may be difficulties with the simple, necessary tasks of life. Throughout, the patient will require the dedicated help of a multidisciplinary team including community nurses, social workers, psychologists, chaplains, home helps and volunteer 'buddies'. Intensive support in the community, rather than in hospital, is the ideal.

One Scottish hospital's experience of AIDS

By March 1987, 10 of the 11 cases of AIDS in Glasgow had died. The mean survival time was 9.4 months from diagnosis. The average duration of stay in Ruchill Hospital was 103 days, spread over 3.4 admissions. Patients underwent a mean of three invasive procedures. Overall, there was a mean of 4.4 significant diseases and 4.5 less serious problems per patient. All except one patient ultimately experienced a serious infection, whereas five cases developed a total of six neoplasms (three with KS and three with CNS lymphoma).

The index presentation of AIDS was pneumonia in six cases, but seven patients underwent an overall 14 episodes of pneumonia (six with PCP, one with CMV and seven unknown). Septicaemia occurred in five patients (with salmonella in three) and disseminated mycobacteriosis in three cases.

Other infections included CMV colitis in four and cryptosporidium enteritis in two cases. Chronic HIV encephalopathy with dementia was present in three cases and cachexia was almost invariable. In some cases the psychosocial complications were profound.

AIDS — PRESENTATION BY ORGAN SYSTEM

This section reviews AIDS by the more common forms of clinical presentation. The organ systems most frequently involved are the lungs, the nervous system, the gut and the skin. Some presentations are non-specific in nature.

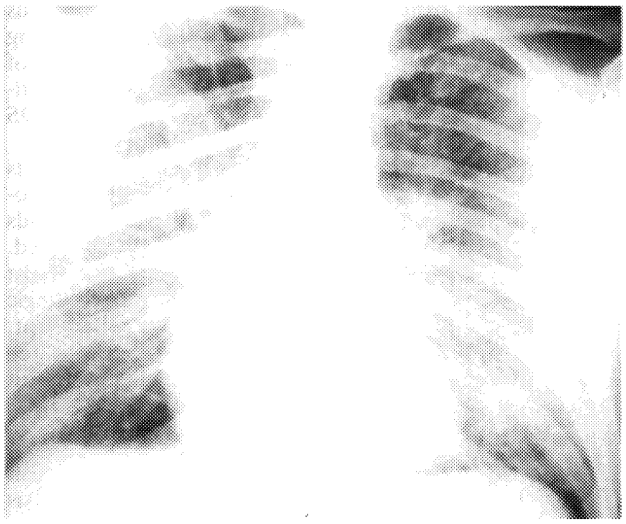

Figure 2
Chest X-ray of severe *Pneumocystis carinii* pneumonitis. Diffuse bilateral infiltrates with some sporing above the diaphragm. A five week history of cough, fever and dyspnoea. Fatal outcome some two weeks after hospitalisation.

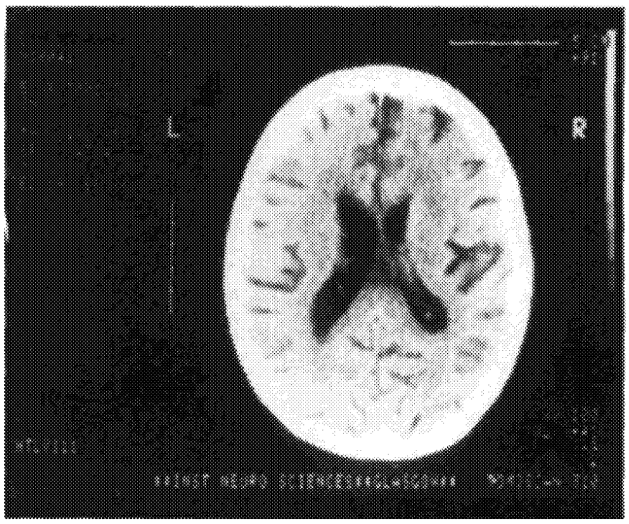

Figure 3
CT scan showing cerebral atrophy in HIV encephalopathy. This man, living alone, developed memory and personality changes.

Pneumonitis

This is the commonest disease in AIDS, ultimately affecting over 80% of patients and often occurring terminally. Many opportunistic pathogens may be involved and pyogenic infections seem to be increasing in frequency. *Pneumocystis carinii* pneumonitis (Figure 2) is the *index* presentation in about 60% of all cases of AIDS.

A serious disease, it presents as a diffuse pneumonitis of gradual onset and minimal chest signs. Many other pathogens, and even KS may produce a similar picture, especially CMV infection, which is relatively common.[3] This common virus may act alone or in synergistic combination with *P. carinii* thereby creating diagnostic and management difficulties.

Atypical mycobacterial infections, present in about 20%, occur in the more profoundly compromised patients. It is important not to miss pyogenic pneumonias which may present atypically. Since there is no effective combination therapy for AIDS pneumonitis, many argue for early aggressive efforts at diagnosis, with bronchoscopy in particular. Preliminary assessment may be done by blood gas and lung function monitoring and by gallium scans. Sputum induction by means of 3% saline inhalation is particularly useful.

Neurological manifestations

These problems are both common and diverse and may present with focal or diffuse involvement. HIV has itself neurotropic potential and as a result more than 40% of patients develop a chronic HIV encephalopathy (Figure 3). This manifests as progressive dementia, often with motor and gait problems. It may itself precede AIDS and has singular psychosocial and management implications.

Amongst other forms of encephalitis is that due to CMV infection. Blindness may result from a CMV chorioretinitis but this may respond to the anti-CMV drug DHPG. In 5% a subacute meningitis occurs due to the fungus *Cryptococcus neoformans*. This should be specifically sought in all CNS presentations since it is treatable. Mass lesions, manifesting with focal signs, are likely to be due either to lymphona or to toxoplasmosis. The latter causes characteristic ring shapes on CT scan and is also treatable.

Gastrointestinal tract manifestations

These are almost universal in AIDS and may involve all levels from mouth to anus. The most frequent problem is oral candidiasis. This can extend into the oesophagus to cause severe dysphagia which may also result from CMV, herpes or Kaposi lesions. So diagnosis may require endoscopy to exclude treatable conditions.

Diarrhoea is a recurring problem in over 70% of patients. A specific HIV enteropathy can result in malabsorption and cachexia. The protozoon cryptosporidium produces a relentlessly torrential diarrhoea which is resistant to therapy. This is a slow and undignified way to die.

Chronic salmonella infections are common and often result in recurrent bacteraemia. A CMV colitis is characterised by fever, abdominal pain and bloody diarrhoea. About 50% of KS patients develop GI involvement, especially of the mouth, but these are often clinically silent.

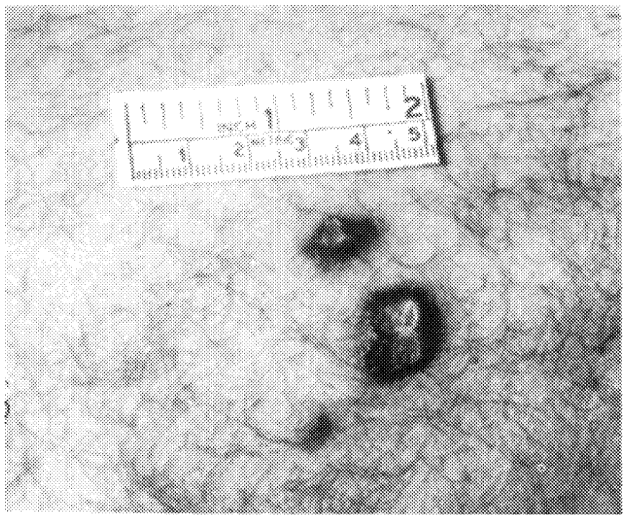

Figure 4
Typical lesions of Kaposi's sarcoma. Within six months of his presentation with KS, this man developed a seminoma, disseminated mycobacteriosis and died of cerebral problems, possibly due to cerebral lymphoma.

Skin manifestations

The major skin problem in gay men with AIDS, is Kaposi's sarcoma. The lesions are palpable, purple-red plaques (Figure 4) which may develop multifocally, anywhere on the skin. They may then coalesce. Herpetic lesions are also common and progressive perianal herpes is a very painful, though treatable condition. Various fungal infections and allergic drug rashes are frequently encountered.

Constitutional upset

Fever commonly heralds the onset of AIDS and often recurs during its progression. Sometimes it has no clear explanation but may reflect covert disease of a focal or disseminated nature. Ultimately bone marrow or lymph node biopsy and lumbar puncture may be required.

Cachexia is a striking, almost invariable feature in AIDS. It may result from anorexia, dysphagia or malabsorption and the body weight may drop by 25% to 50%. This may compound the immune compromise so it is important to maintain nutrition.

COMMON INFECTIONS IN AIDS

In this section, infections will be reviewed in respect of their presentation, diagnosis and treatment.

Protozoal infections

Pneumocystis pneumonitis has a mortality of 35% and therefore early diagnosis is important. It usually has an insidious development over two to 10 weeks with fever and a hacking, non-productive cough. Progressive dyspnoea develops as the illness quickens. The chest X-ray is abnormal in 80% showing diffuse interstitial markings. However, this can be a capricious disease especially when onset is acute or the chest X-ray is normal. The parasite must be demonstrated in stained pulmonary secretions, obtained by bronchoscopy or by sputum induction with 3% saline. Therapy, with high dose co-trimoxazole should produce response within five days.

Treatment for the required three weeks often proves toxic and Dapsone-trimethoprim or pentamidine are alternatives. Prophylaxis against relapse is being currently evaluated.

Toxoplasma gondii may involve various tissues, but principally the CNS resulting in encephalitis or brain abscess. The patient develops headache, confusion and often focal signs. Ring lesions on CT scan are highly suggestive, perhaps thus avoiding brain biopsy. Treatment is with fansidar, which is continued long-term to prevent relapse.

Viral infections

Cytomegalovirus infection is common in homosexuals and especially in those with AIDS. For example, intermittent viraemia is present in about 50%. However, its role is not always clear cut. It may simply be a 'passenger' virus or else act in synergy with other pathogens. This virus itself seems to have immunosuppressive effects. The diagnosis of CMV disease really requires histological evidence of inclusion bodies in addition to virus isolation. In AIDS cases with pneumonitis CMV is implicated in about 20%. It may also present as a severe colitis, oesophagitis, chorioretinitis and meningo-encephalitis.

Terminally overwhelming CMV infection may occur with viraemia and multi-organ involvement. Treatment with the nucleoside analogue DHPG is being assessed and appears to have a role.

Herpes simplex virus may cause severe ulcerating mucocutaneous lesions. It may also involve most internal organs. Timely therapy with acyclovir should be effective.

Fungal infections

Candidiasis is ubiquitously encountered in AIDS patients. In fact, oral candidiasis is often a harbinger of the early onset of AIDS in seropositive patients. Candidal oesophagitis usually presents with dysphagia though it may be silent. Both conditions may lead to malnutrition and thus treatment should be energetic with local and/or systemic therapy. Ketoconazole is commonly used in prophylaxis.

The meninges are the particular target of *Cryptococcus neoformans* but in 25% of infections other sites, such as the lungs, are involved. This is a subacute atypical meningitis with subtle symptoms of headache and fever. Meningeal signs and CSF changes may be absent and, in a confused patient, fluid should be examined by India ink stain. Treatment requires amphotericin, perhaps combined with 5-Fluorocytosine.

Bacterial infections

AIDS patients are at special risk from facultative intracellular parasites. These include the mycobacteria, salmonella and legionella species. Disseminated *M. avium-intracellulare* infection, extremely rare prior to AIDS, is now commonly encountered. It is isolated from many sites, including the blood, and is associated with fever and debilitation. Classical histological changes are usually not observed and resistance to conventional antituberculous therapy is very common.

There is considerable concern currently in the USA about *M. tuberculosis* infection. Pyogenic infections due to pneumococci and staphylococci may also occur.

AIDS-RELATED NEOPLASMS

With time, a broad range of malignancies are emerging in seropositive patients, which is not surprising in view of the past experience with immunodeficiency. Those tumours indicative of AIDS in HIV infection, are Kaposi's sarcoma (the second commonest manifestation of AIDS in gay men), cerebral lymphoma and high grade, B cell non-Hodgkins lymphoma. These tumours tend to be more aggressive in AIDS. Other neoplasms encountered in the seropositive state, include various oral and anorectal carcinomas and Hodgkin's lymphoma.

Kaposi's sarcoma

This is a neoplasm of vascular endothelium. It does not metastasise, but instead develops in a widespread multifocal fashion. It is usually seen only in the homosexual sub-population with AIDS, presumably in association with a sexually transmitted oncogenic virus. When visible, it becomes the brand of AIDS, the modern leper's stigma. Typical lesions are red and purple nodules, one or two cms in size, occurring on any part of the skin or mouth and painless. They are locally invasive and may coalesce. They may follow the lines of skin creases. Lymph node involvement is common and may lead to local oedema. Most patients are relatively symptom free.

Systemic manifestations do occur but most patients succumb to infection rather than KS. Gut lesions, though common, are usually silent. However, pulmonary involvement is an ominous development and may mimic PCP. Diagnosis is made by skin biopsy and any unusual lesion in a seropositive patient should be so investigated. A staging

assessment of the disease should be undertaken for treatment purposes.

No therapy is entirely satisfactory and the natural history of KS varies widely. Local treatment with radiotherapy or chemotherapy may be employed. Cosmetic aspects may be important. For extensive disease alpha interferon, single agent or combined chemotherapy may be used.

Non-Hodgkins lymphoma

About 5% of AIDS patients develop a lymphoma. This is usually of B cell origin and often presents in extranodal sites especially in the CNS (about 40% of cases) or bone marrow. About two-thirds of these tumours are high grade, aggressive tumours and they respond poorly to chemotherapy.

Other HIV-related conditions

Whereas most manifestations in AIDS result from immune deficiency, many features in the seropositive patient relate directly to HIV infection. Nevertheless, with progression to AIDS, minor opportunistic problems become more notable.

The state of debilitation that usually precedes AIDS has been termed AIDS-related complex (ARC). This diagnosis requires certain specified clinical and investigational abnormalities. It implies significant immune compromise such that progression to AIDS occurs in 20% to 35% within two years. These conditions can sometimes be more distressing than even AIDS itself and may require hospitalisation. For example the ratio of AIDS to ARC patients in hospital may be about 10 to one.

Even apart from AIDS, there is a broad spectrum to HIV disease and this includes an acute febrile illness, acute or chronic neurological or haematological conditions, lymphadenopathy, enteropathy and constitutional disease. Consequently patients may initially present — with HIV disease or undiagnosed AIDS — to a wide variety of specialists.

Why there should be such variation in the clinical expression of HIV is unknown; nor is the ultimate prognosis known for seropositive individuals. However, after three years of infection, about 8% to 10% of gay men will develop AIDS per year for an unknown number of years.

The rate in respect of drug users is unknown and the incidence of other serious problems is also unknown. Co-factors may play an important role in progression. Thus repeated infections and exposure to antigens may activate the replication of HIV, leading to an increased burden of infection with more recruitment, and eventual depletion, of target cells. These co-factors include other sexually transmitted infections (perhaps all infections!), continued intravenous drug use and pregnancy. Thus patients must be forcefully counselled about avoidance of co-factors.

As effective, non-toxic therapy emerges, it will become important to diagnose prodromal states before there is irreversible immune paralysis. The range of non-AIDS conditions, listed in the CDC classification system in Table I, will now be considered.

Acute HIV infection (Group I)

Transient manifestations may accompany acute infection within two to five weeks. The acute retroviral syndrome resembles glandular fever and occurs in about 10% of cases. Characteristically the patient has a two-week febrile illness with headache, arthralgia, lymphadenopathy and a maculopapular rash. Seroconversion usually follows. Various acute CNS problems can occur such as aseptic meningitis (which may relapse) and encephalitis.

Asymptomatic infection (Group II)

Most patients are asymptomatic when infected and seroconvert some four to 12 weeks later. However, rarely this can take up to nine months. This period can last for many years and perhaps may be indefinite. Immunological and haematological tests may, or may not, be normal.

Persistent generalised lymphadenopathy — PGL (Group III)

PGL is a common finding. It is detectable in about one-third of seropositive patients, many of whom are asymptomatic. By definition, lymph nodes in PGL must be enlarged by 1 cm at least, at two or more extra-inguinal sites and without other explanation. The presence of symptoms, but not lymphadenopathy alone, infers a poorer prognosis and AIDS progression occurs in 15-25% within three years.

The posterior cervical group are usually involved and these may go unnoticed. Histologically there is usually a benign, reactive follicular hyperplasia due to B cell proliferation. However, lymph node biopsy is only indicated if there is marked constitutional upset, asymmetrical or painful node enlargement or hilar lymphadenopathy which is unusual. Infection or lymphoma may be present. Disappearance of nodes may imply a poor prognosis but benign resolution of PGL is also well-described.

Other disease in Group IV

This group defines the more chronic and severe expressions of HIV disease. It also includes the various manifestations of AIDS. These have already been reviewed and will be omitted.

The constitutional diseases in subgroup A require the occurrence of fever or diarrhoea present for over one month, or of weight loss which exceeds 10%. Whether

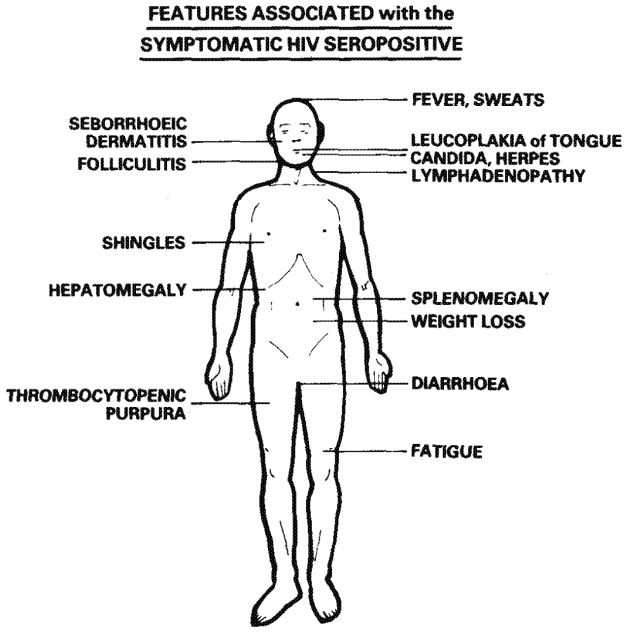

Figure 5
Features associated with the symptomatic HIV seropositive.

these problems reflect covert opportunistic diseases, or illustrate more advanced HIV infection, is uncertain.

In subgroup B, neurological disease reflects the occurrence of disabling problems such as dementia, myelopathy or neuropathy in patients who do not have AIDS. There is good evidence that the damage here is directly mediated by HIV infection.

Subcategory C2 covers those opportunist infections which do not meet the diagnostic criteria of AIDS (as in subcategory C1). These include oral candidiasis, tuberculosis and recurrent salmonella bacteraemia.

Subgroup E covers various other problems of infection and neoplasm in HIV positive patients. One well-described problem is an immune thrombocytopenic purpura due to peripheral platelet destruction. Platelet counts may be under $20x10^9/1$ and bleeding problems are sometimes encountered.

ASSESSMENT OF THE SEROPOSITIVE PATIENT

The features associated with the more progressive forms of HIV disease are shown in Figure 5. Symptoms may vary from mild to very debilitating but their presence is often of significance. At clinical examination abnormalities of the mouth may be especially revealing. Oral candidiasis suggests AIDS is likely to develop within a year. Oral hairy cell leucoplakia equally signals an adverse prognosis. It presents as raised white areas, often giving a ribbed appearance on the lateral aspects of the tongue. Unlike candidiasis it cannot be scraped away.

A history of shingles or recurrent herpes may also be significant. Various skin features may be strikingly evident. Seborrhoeic dermatitis and folliculitis of the face may sometimes be florid. Tinea versicolor and other fungal and bacterial infections may be noted. Thrombocytopenic purpura may present with petechiae and bruising. Splenomegaly is not uncommonly palpable.

Routine investigations are usually unremarkable. A minority may have a mild reduction in white cell, platelet or haemoglobin counts. However, immunological abnormalities are common and include depression of the T^4 lymphocyte count, perhaps with elevation of the T^8 count and partial cutaneous anergy. Some of the adverse factors found in the prodromal stages of AIDS are shown in Table III. These should prompt closer observation so that intervention may be early if AIDS develops. However there is a need for a more systematic system of clinical monitoring. The Walter Reed staging classification is one clinically useful method of charting deterioration by means of clinical and immunological changes.[4]

MANAGEMENT OF THE SEROPOSITIVE PATIENT

Intensive support of the AIDS patient is necessary on both practical and humanitarian grounds. However this should not result in neglect of the seropositive patient who, though less conspicuously ill, has still a potentially life-threatening condition. It is in this group, numerically much greater than AIDS, that preventive efforts may prove more effective. These patients will require regular medical and counselling support.

Medical review may allow early detection and intervention if serious complications are developing. There should be screening for chronic, treatable infections such as syphilis, gonorrhoea and chlamydial disease. Considerable skills in counselling are needed to persuade patients engaging in risk activities to effect major behavioural changes. This essentially means 'safer sex' for heterosexuals, and 'safer drug use' for abusers who will not abstain.

Here the interests of both the patients and the public health coincide. The psychological devastation experienced by the newly diagnosed seropositive should not be underestimated. In the drug user this commonly leads to an intensification of the drug habit, often despite the realisation that this may well aggravate HIV infection. The doctor's role here must be one of steady patience in trying to effect harm reduction and ultimately rehabilitation.

The management of the seropositive patient is summarised in Table III. The frequency of outpatient review will depend on the patient's clinical and psychological status and need for counselling. Those more severely affected may require review every month, those with PGL every three months, and those who are asymptomatic every four to six months. The history should include a comprehensive systematic enquiry and questions on continued risk activities. Physical examination should be careful and the patient's weight should be documented. Intercurrent infections should be screened for and the blood count monitored. If abnormal or if the patient has symptoms then T Cell subsets and tests for cutaneous anergy should be performed. In broad terms clinical upset correlates with lowered T^4 lymphocyte counts and diminished skin test reactivity.[5] If symptoms are more pronounced then more extensive investigations may be undertaken.

Efforts should be made to ensure the patient has a balanced understanding of the implications of HIV infection, including knowledge about transmission. Counselling about co-factors, harm reduction and health boosting may give the patient a more positive outlook and some limited sense of influence over the outcome.

TABLE II
Factors associated with subsequent AIDS development

Development of oral thrush, leucoplakia
Involution of lymphadenopathy
Splenomegaly
Herpes zoster
Unexplained fever
Constitutional symptoms
Rising ESR
Peripheral cytopenia

TABLE III
Management of HIV seropositive patients

CLINICAL EXAMINATION FOR:
- Lymphadenopathy, hepatosplenomegaly
- Oral check for monilia, herpes and leucoplakia
- Skin check for KS, eczema, purpura and ringworm
- Gum infection (GUM Physicians)

LABORATORY TESTS FOR:
- FBC + diff., platelet count, ESR
- LFTs, immunoglobulins
- HBsAg, herpes simplex, chlamydia, syphilis

IN SYMPTOMATIC PATIENTS — as appropriate:
- Chest X-ray
- Lymphocyte subsets + tests for cutaneous energy
- Pulmonary function tests, Gallium scans
- Serological screen — CMV, EBV
- Biopsy

COUNSELLING ABOUT:
- Nature of HIV infection
- Avoidance of infections and co-factors
- Health boosting and stress reduction
- Protection of others

HIV infection in children

HIV infection has somewhat different characteristics in children, especially babies, than in adults. AIDS may develop and progress more rapidly, than in older patients. A high proportion of infected babies (perhaps 50%) will develop AIDS. They are more likely to have severe bacterial infections and CNS abnormalities. They are prone to develop a lymphoid interstitial pneumonitis. Infected babies may fail to thrive and a distinctive congenital HIV syndrome has now been described. Between 30% and 50% of seropositive women will transmit the virus to the fetus. Since about 25% of all known seropositives in Scotland are young females, we can expect to acquire considerably more experience in this, the most tragic of all the consequences of HIV infection.

ACKNOWLEDGEMENTS: I wish to thank Miss Edith H. Simpson and Mrs Doreen O'Keefe for secretarial assistance and Miss Sheena McDonald for artwork.

REFERENCES

1 Centres for Disease Control (CDC). Classification system for human T lymphotropic virus type III/lymphadenopathy — associated virus infections. MMWR 1986, 35, (20): 334-339.
2 Pinching AJ. Immunology: Immunological profile, pathogenesis and the role of diagnostic tests. IN: The management of AIDS patients (eds. Miller D, Weber J and Green J). London. Macmillan 1986; 35-52.
3 Hopewell PC and Luce JM. Pulmonary manifestations of the acquired immunodeficiency syndrome. Clin Immunol. Aller 1986; 6: 489-519.
4 Redfield RR, Wright DC, Tramont EC. The Walter Reed staging classification for HTLV III/LAV infection. New Eng J Med, 1986; 314: 131-132.
5 Lang W, Anderson RE, Perkins H *et al.* Clinical immunologic and serologic findings in men at risk for acquired immunodeficiency syndrome. J Amer Med Assoc 1987; 257: 326-330.

0036-9330/87/0197/0107 $2.00 in USA

Scot Med J 1987; 32: 107-109

HUMAN IMMUNODEFICIENCY VIRUS INFECTION IN HOMOSEXUAL MEN

A. McMillan
University Department of Genito-Urinary Medicine, The Royal Infirmary of Edinburgh

IT WAS the occurrence amongst homosexual men in the United States of America of Kaposi's sarcoma and *Pneumocystis carinii* pneumonia that first focused the world's attention on the emergence of a severe acquired immunodeficiency syndrome. Indeed as the first cases were amongst homosexuals the disease was initially termed the Gay Related Immune Deficiency Syndrome (GRIDS). Subsequently, it became clear that other population groups could be affected and the name was changed to the acquired immune deficiency syndrome (AIDS). The majority of AIDS patients in Western Europe and the USA, however, are still homosexual men, the majority being young (median age 35 years).

Although the prevalence of human immunodeficiency virus infection in the population is unknown, the testing for HIV antibodies of blood from patients attending genito-urinary medicine clinics shows clearly the increased prevalence of the virus amongst homosexual men (Table I). The table also illustrates geographical differences in infection rates.

Serological data suggest that the virus was introduced into the European homosexual community from the USA during the late 1970s. Retrospective testing of sera showed that in 1981, 9% of 250 Danish homosexual men had been infected with HIV, and that seropositivity correlated strongly with sexual contact with men from the USA.[10] In the UK, indigenous spread during the early 1980s is indicated clearly by the increasing proportion of seropositive homosexual men who attended genito-urinary medicine clinics in London.[1] A cohort study in Amsterdam showed that the attack rate of HIV infection increased markedly between 1980 and 1982.[11] The homosexual spread of HIV infection during this time is emphasised by the report from London of clustering of cases linked by sexual contact.[12]

The homosexual community is heterogeneous and in a discussion on an individual's risks of HIV infection, it is helpful to consider the typology of sexual behaviour (Table II). Those most at risk are the functional, dysfunctional and open-coupled, these groups accounting for some two-thirds of homosexual men.

Sexually active homosexual men participate in various activities, that if undertaken with an infected partner, predispose to HIV infection. Most studies have shown a clear correlation between seropositivity, large numbers of sexual partners and anal intercourse, the penoreceptive partner being at greater risk.[2, 9, 25] There is a significant

TABLE I
Prevalence of antibody against human immunodeficiency virus (HIV) in the sera of individuals attending genito-urinary medicine or screening clinics

City (Country) and year	Percentage of individuals seropositive for HIV (number studied)		Reference
	Homosexual/ bisexual men	Comparison group	
London (UK)			
— 1982	7 (153)	0 (109)*	(1)
— 1984	22 (153)	0 (111)*	(1)
— 1984/85	32 (304)	NS	(2)
Sheffield (UK) 1986	3 (NS)	NS	(3)
Paris (France) 1984	18 (44)	1† (100)	(4)
Munich (West Germany) 1984	27 (48)	0 (18)⁰	(5)
Rome (Italy) 1984	9 (70)	NS	(6)
Amsterdam (The Netherlands) 1984/85	31 (714)	NS	(7)
San Francisco (USA) 1978	5 (290)	NS	(8)
1984	67 (435)	NS	(8)
New York City (USA) 1982	53 (66)	NS	(9)

* Heterosexual men who attended the clinic. ⁰ Laboratory Workers.
† Blood Donor. NS Not studied.