ABC of Sexually Transmitted Diseases

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ACQUIRED IMMUNE DEFICIENCY SYNDROME

Acquired immune deficiency syndrome (AIDS) was originally defined as histologically proved Kaposi's sarcoma or life threatening opportunistic infection (established by biopsy or culture), or both, in a person under 60 who has no history of underlying immunosuppressive disease or treatment. In the United States of America the number of cases has increased exponentially. By November 1983 over 1600 had been notified. The original definition probably identifies only the most severe and later manifestations of the disease. The incidence of other syndromes and diseases which may be related to AIDS also seems to have increased. In addition to Kaposi's sarcoma and *Pneumocystis carinii* pneumonia, the syndrome has widened to include non-Hodgkin's lymphomas and various opportunistic viral, fungal, bacterial, and protozoal infections. AIDS has now been reported from many countries including the United Kingdom, where 27 cases had been reported to the Communicable Disease Surveillance Centre by November 1983.

The overall mortality in the USA is 41%, but half the cases have been reported only in the last six months. Mortality in cases reported two years ago approaches 100%.

Causes

Group affected (first 2000 cases in USA)		
Homosexuals	1427*	(71%)
Intravenous drug abusers	513	(17%)
Haitians	105	(5%)
Haemophiliacs	17†	(0.7%)
Miscellaneous	115	(6%)

* Includes 175 intravenous drug abusers

† Includes one homosexual and one drug addict

There are parallels between the epidemiology of hepatitis B and that of AIDS. AIDS may be spread sexually, parenterally, or perinatally. Almost three quarters of cases are in homosexual or bisexual men with a high number of casual sexual partners—an important risk factor. The disease also occurs in the female partners of heterosexual or bisexual intravenous drug abusers. Almost a fifth of patients are intravenous drug abusers. Cases in haemophiliacs may be linked to an agent in factor VIII concentrate. Cases have also occurred in adults and infants after blood transfusions. A few cases have occurred in children born to Haitian mothers or to mothers who have been promiscuous intravenous drug abusers.

The agent

- ? New agent

? Old agent (new access or amplification)

? Multiple causes

The agent implicated is still unknown. AIDS may be caused by a new agent common to all the affected groups. The new agent may be an old agent such as cytomegalovirus or Epstein-Barr virus with a new form of expression, or it may be an animal virus that has gained access to man, or a previously unrecognised human agent may have found new access and amplification in the developed world. Alternatively, there may be multiple causes of immunosuppression in the different groups affected. Multiple immunological insults in homosexual men might include recurrent episodes of sexually transmitted disease. Nevertheless, it is hard to see how multiple factors, different in different groups, would produce an identical syndrome in all groups simultaneously. Possibly an individual's response to the AIDS agent may be largely determined by host factors, which for homosexual men may include those listed above.

Clinical features

Kaposi's sarcoma—This differs from classic Kaposi's sarcoma in that young men are affected, geographical clustering occurs, and widespread skin, visceral, and lymph node disease is seen with associated opportunistic infections, immune dysfunction, and a fulminant course. The median survival for this condition alone is about 18 months.

Opportunistic infections are the commonest presenting feature of AIDS. The infections are unusual and multiple, often resulting in rapid clinical deterioration. Response to treatment is poor, with a seven to eight month median survival. The infections present largely with symptoms and signs in the lung, central nervous system, and gut: there is fever, shortness of breath, dry cough, and infiltrates on chest radiographs (*Pneumocystis carinii*, is the commonest pathogen); there are either signs of meningitis or encephalitis or focal signs; and diarrhoea. Disseminated infection, with a variety of pathogens, may present with no localising features as a pyrexia of undetermined origin.

Immune dysfunction—The opportunistic infections and the development of tumours suggest a largely cellular immune dysfunction, and a wide range of abnormalities have been described. Similar immune abnormalities have been described in homosexuals with chronic unexplained lymphadenopathy, healthy homosexuals, prostitutes, and haemophiliacs receiving indigenous as well as commercial factor VIII. A variety of factors not necessarily related to AIDS may be implicated.

Management

Since the cause of AIDS is unknown, treatment is directed largely at the major manifestations of the disease. Conventional cytotoxic therapy with single agents or combinations of drugs is being assessed for Kaposi's sarcoma. Opportunistic infection may complicate such treatment.

α -Interferons—leucocyte, lymphoblastoid, and recombinant types—are also used. It is still too early to evaluate fully these approaches to treatment.

Drugs are available for some of the opportunistic infections, but problems with diagnosis and poor response complicate the outcome. High dose co-trimoxazole or pentamidine are used for *Pneumocystis carinii* pneumonia and pyrimethamine and sulphadiazine for toxoplasmosis. Oral candidiasis may be treated with oral nystatin, amphotericin, or ketoconazole but intravenous amphotericin is the mainstay for systemic fungal infections. Acyclovir is used for severe herpes simplex but there is little evidence of benefit in other viral opportunistic infections. The atypical mycobacteria are often resistant to conventional antituberculous drugs, and gastrointestinal cryptosporidiosis is resistant to various antiprotozoal drugs.

A primary immune defect has not yet been identified, and various immunomodulatory manoeuvres are being used empirically. These include bone marrow transplantation and plasmapheresis, together with interferon, thymic hormones, and interleukin II. The place for modulating the immune system may, however, be in the very early stages of the disease, which, without a reliable marker, we cannot yet identify.

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