affected, and sometimes healing is associated with scar formation. Skin lesions are infrequent in mucous membrane pemphigoid. Similar oral lesions may be seen in other subepithelial disorders such as epidermolysis bullosa, dermatis herpetiformis, and linear IgA disease. Oral lesions are uncommon in bullous pemphigoid, but they are seen in most patients with pemphigus vulgaris, often preceding the appearance of skin bullae. The oral blisters of pemphigus rupture early on to leave painful and persistent irregular erosions.

Biopsy of the oral mucosa for conventional histological examination and for direct immunostaining is frequently needed to distinguish these mucocutaneous disorders, particularly to exclude pemphigus, though exfoliative cytology is of little value. Erythema multiforme of the mucous membranes (Stevens-Johnson syndrome) mostly affects young men and is usually readily distinguished on clinical grounds: the presentation is often acute or recurrent, and bloodstained crusting of swollen lips may be a helpful diagnostic sign.

Most infective mouth ulcers are caused by viral infections and are usually non-recurrent, scattered small round ulcers seen in a febrile child or young adult. Primary herpetic stomatitis is the most common viral cause of mouth ulcers. It presents with scattered vesicles and ulcers and a diffuse gingivitis. Recurrent intraoral herpes simplex virus infections are rare, though they may occur in immunocompromised patients including those with AIDS. Other herpes virus infections such as chickenpox and infectious mononucleosis may be complicated by mouth ulcers, and there may be unilateral mouth ulcers in zoster of the mandibular or maxillary divisions of the trigeminal nerve. Enterovirus may cause mouth ulcers in herpangina and in hand, foot, and mouth disease. Diagnosis of these infections is primarily clinical. Viral cultures are still the most sensitive method of diagnosing infection with herpes simplex virus, though this takes from two to four days and sometimes longer. The most rapid diagnostic technique is electron microscopy: this may take only minutes to perform and is very sensitive when intact vesicles are examined but much less so when lesions are at a later stage. Single serological assays are of little value in the diagnosis of acute herpes simplex virus infection, but seroconversion between acute and convalescent serum samples is diagnostic of primary infection.

Bacterial infections may cause mouth ulcers in tuberculosis and in all stages of syphilis, but fungal causes are rare in Britain. Culture, dark ground microscopy, and serology may be required to confirm a clinical suspicion of the chronic bacterial infections in groups at risk who have a chronic ulcer or ulcers or in those with ulcers of unusual appearance.

Clive E H Grattan

Senior Registrar in Dermatology,
General Hospital,
Birmingham B4 6NH

Professor of Oral Medicine and Oral Surgery,
University of Bristol Dental School and Hospital,
Bristol BS1 2LY

Correspondence to: Dr Grattan.

---

AIDS and insects

A question frequently put to medical experts concerns the possibility that biting insects might play a part in the transmission of the acquired immune deficiency syndrome (AIDS) in Africa and elsewhere. An answer can best be offered by a review of the epidemiological features of the local disease and the epidemic in progress in central Africa.

Much has been written on the transmission of lymphadenopathy associated virus/human T cell lymphotrophic virus type III (LAV/HTLV-III), the causative agent of AIDS. The virus has been isolated repeatedly from blood, semen, and other body fluids including saliva and tears (and possibly from breast milk), but there is no convincing evidence of infection in adults through any medium other than blood or semen (and perhaps other body fluids heavily contaminated with blood). Most cases of clinical AIDS have been reported from the United States, and of a total of 1524 patients 73% were homosexual and bisexual men, 17% were intravenous drug abusers, 1% were patients with haemophilia, 2% were recipients of transfused blood or blood components, 1% were heterosexual sex partners of patients with AIDS, and the remaining 6% have not been classified by recognised risk factors for this infection. Maternofetal transmission is believed to occur mainly in utero, but there is no evidence for transmission in breast milk. Data gathered in Africa, however, indicate that men and women there are affected in equal numbers and that transmission is predominantly heterosexual. Most Africans deny anal intercourse and oroanal or oromental contact, and promiscuity has been suggested as a particular risk factor. Transmission by unsterilised syringes and needles and non-sterile instruments used for tattooing, ritual scarification, ear piercing, and circumcision carry a high risk of infection—as is the case with hepatitis B.

In general, though transmission of AIDS within Africa is less well understood than in Western communities, there is substantial evidence against transmission by human ecto-parasites such as mosquitoes, bedbugs, and other blood-sucking arthropods. Firstly, infection is rare in children (who are bitten most frequently by mosquitoes). The incidence of infection with LAV/HTLV-III increases rapidly with sexual activity and almost all patients with the infection are in the sexually active age range. Secondly, the greatest concentration of infected people is found in urban areas, and sexual freedom is greater in urban societies than in the more traditional rural societies. Thirdly, there is close analogy between the epidemiological features of hepatitis B and
AIDS, and there is no evidence for the transmission of hepatitis B by mosquitoes. In one extensive laboratory study seven species of mosquitoes were fed artificially on human blood containing hepatitis B. Radioimmunoassay showed that all the ingested hepatitis B surface antigen disappeared in parallel with digestion of the blood meal by the mosquito. Furthermore, the infectivity of LAV-HTLV-III is less than that of hepatitis B, and the titre of circulating virus is substantially lower. Fourthly, perhaps only one in 10,000 infected (T4) lymphocytes produces complete virus (personal discussion with R C Gallo), and it seems unlikely that virus would be present in the blood meal.

What about other human ectoparasitic insects? J Maudner (personal communication) considers that the mouthparts of fleas might be better adapted than most for the mechanical transmission of the infection, but the habits of the flea, including host fidelity and infrequent feeding, make it an unlikely vector. The habits of bedbugs are such that these are not efficient vectors of disease in the community. Lice are even less likely to transmit AIDS, partly because of the effective one way valves of the mouthparts, partly because the mouthparts are unweatherable and are emptied and cleaned after use, and partly because lice have not been implicated in the transmission of viral diseases.

Nevertheless, research is needed into the survival of virus introduced by blood meals and by inoculation into mosquitoes, and this work is in progress at the London School of Hygiene and Tropical Medicine. Equally careful surveillance and collection of data on AIDS must continue in all countries. At present, however, it is evident that the time picture of the epidemiology of AIDS does not include transmission by blood sucking insects.

Arie J Zuckerman
Professor of Microbiology,
London School of Hygiene and Tropical Medicine,
London WC1E 7HT


Sarcoid heart disease

The first case of sarcoidosis affecting the heart was reported by Bernstein et al in 1929, and since then many case reports and reviews have been published. The most recent of these, in the Mayo Clinic Proceedings, highlighted the characteristic features. Analysis of a large series has provided a comprehensive picture of the variations of this fascinating disease. In our own series we now have 290 cases seen in Britain, 100 with necropsy confirmation.

Though cardiac lesions may occur in any patient with sarcoidosis and at any stage, sarcoid heart disease most often presents with cardiac symptoms—and only when sarcoidosis is suspected and sought is the aetiologic diagnosis established. This is done by seeking clinical evidence of sarcoidosis, preferably with histological confirmation by biopsy of the skin, a lymph node, bronchus, or lung or a Kveim test. Endomyocardial biopsy is diagnostic if the result is positive, but the patchy distribution of the lesions makes a negative finding of absolutely no value. At necropsy the characteristic pattern is massive heart lesions with minimal sarcoidosis in other organs. The classic giant cell granulomas with fibrosis may be confluent and occupy enormous areas of the myocardium or may be microscopic and diffuse. Haemodynamically important valve lesions are rare, and the pericardium is more commonly affected than is clinically apparent. The frequency of extensive replacement of the conducting system by granuloma is consistent with the clinical presentation of heart block, sudden death, and complex arrhythmias.

The common patterns of presentation in order of frequency are complete heart block, ventricular extrasystoles or ventricular tachycardia, myocardial disease with failure, sudden death, first degree heart block or bundle branch block, especially right, supraventricular arrhythmia, mitral valve dysfunction simulating myocardial infarction, and pericarditis. Many of these features may appear in any one case, and sarcoidosis should be suspected in any patient younger than the usual with complete heart block or unexplained heart failure, particularly with complex arrhythmias difficult to control.

The age range of patients is wide—in our experience from 18 to 88 with a peak between 25 and 55. We now know that the prognosis in the individual case is not necessarily bad, particularly with energetic treatment. Many patients have survived serious illness for 10 years and a few for even 20 years.

Of the many methods of investigation, 24 hour electrocardiographic monitoring and echocardiography are perhaps the most rewarding. The efficacy of the treatment of arrhythmias must be carefully monitored. Echocardiography may show left ventricular dysfunction, localised dyskinesia, mitral valve dysfunction, septal thickening, and bright echoes—particularly from the ventricular septum and the left ventricular free wall—consistent with fibrogranulomatous infiltration.

Treatment for the cardiac condition will include the whole gamut of drugs. Amiodarone has proved helpful in cases of resistant arrhythmia. Heart block must be treated by pacing. In all groups energetic use of steroids can be very beneficial—which explains the importance of the physician making the aetiological diagnosis.

The widely quoted paper of Roberts et al deserves further attention. Regrettably, it omitted reference to the two earlier reports of large numbers of cases and in its analysis therefore perpetuated the myth that this disease is much more common in blacks. In our series of 290 only 21 were not white. The suggestion that steroids might encourage development of left ventricular aneurysm has inhibited their use. We have frequently found these aneurysms in patients who have received no treatment at all, and we believe the possibility of aneurysm formation need not be considered before deciding to treat with steroids. The prognosis is by no means as bad as suggested by Roberts et al and the outcome is not necessarily related to the extent of the sarcoidosis of the heart. Hearts massively affected by transmural granulomas may continue to perform astonishingly normally, while microscopical lesions can lead to sudden death or, if widespread, to congestive heart failure.

Several transplant centres have found previously undiagnosed sarcoid of the heart in occasional cases of ‘cardiomyo.