Home grown heterosexually acquired HIV infection

Still difficult to predict

The public and the health professions might well feel confused about the heterosexual spread of HIV in the United Kingdom. Press headlines swing from dire warnings of a major epidemic to arguments based on the available data that the risks are minuscule, the public education campaigns erroneous, and the policy makers guilty of scare mongering.

Two articles published by the Communicable Disease Surveillance Centre last month help to clarify the situation, if not to resolve the uncertainty about future heterosexual spread.12 Evans et al reported that by the end of last year 417 cases of AIDS and 1620 cases of HIV-1 infection probably acquired through heterosexual intercourse had been reported in people in England, Wales, and Northern Ireland. Between 1986 and 1991 the proportion of cases of AIDS attributable to heterosexual transmission increased from 2% to 14% and diagnosed HIV infections from 4% to 23%.3 Similar increases have been reported from Edinburgh, southern Europe, and the United States, particularly in association with concurrent epidemics among injecting drug users.45

Most of the heterosexual acquired cases of HIV infection and AIDS in England, Wales, and Northern Ireland, however, are attributable to sexual contact in parts of the world where heterosexual transmission predominates (largely sub-Saharan Africa).1 The recent public debate has focused on the lack of prominence given to this epidemiological fact in public health campaigns and an ill defined feeling that infection acquired abroad can be discounted because it does not involve heterosexual transmission within the United Kingdom.

Evans et al defined three categories of heterosexual transmission.1 “First generation transmission” describes heterosexual transmission of HIV infection from a partner infected by other means—for example, injecting drug misuse, blood products, or sex between men. “Second generation transmission” describes heterosexual infection by partners who were themselves infected through heterosexual intercourse. Cases of second generation transmission are subdivided according to where infection is presumed to have occurred—either abroad or in the United Kingdom.

Ten per cent of cases of AIDS acquired heterosexually (17% of HIV infections) were due to first generation transmission, with injecting drug misuse the commonest route of infection in the source partners. Four out of five cases of heterosexually acquired AIDS (74% of HIV infections) were categorised as due to second generation transmission abroad. Second generation transmission in England, Wales, and Northern Ireland accounted for 11% (n=47) of cases of heterosexually acquired AIDS and 9% (n=131) of cases of heterosexually acquired HIV infection, showing a consistent annual increase.4

From detailed interviews Gilbart et al have provided 15 case histories of people infected by heterosexual intercourse whose partners had no history of major risk factors or of sexual contact abroad.2 Although in some cases the chain of transmission is not entirely clear, the authors argue that the weight of evidence points to second generation transmission in the United Kingdom. As these and other observers emphasise available reports considerably underestimate heterosexually acquired HIV infection because only people coming forward for testing are included, and many cases are disclosed only when symptoms occur.127

These papers and a recent report from Edinburgh provide the first systematic evidence of second generation transmission in the United Kingdom.125 But even given the caveats of underestimation, the problem currently remains small. Preoccupation with second generation transmission reflects concern about the possible size of a purely heterosexual epidemic in the British population who do not inject drugs. But getting caught up with this issue gets us nowhere. We cannot regard the heterosexual British population as isolated from the drug injecting population or people from other countries. Many sexually transmitted diseases have been introduced from elsewhere (syphilis from continental Europe in the fifteenth century, penicillinase producing gonococci from parts of Asia in the 1970s, HIV from the United States in the 1980s) and have subsequently thrived locally. As HIV spreads in parts of the world where we have close sexual and other contact there is no reason why the United Kingdom should be spared further second generation transmission. We are observing a series of interacting epidemics and after only 10 years it is not surprising that we can still trace chains of transmission to the early epicentres.

The suggestion that previous forecasts have foretold indiscriminate spread in the heterosexual population is at odds with the conclusions of two national working parties on predicting the course of the epidemic.14 These emphasised the great uncertainty concerning future heterosexual spread, which may develop over a much longer time course than the epidemics seen in gay men and drug misusers. Herein lies the key to our difficulties. It is as erroneous to arrive at conclusions in the early 1990s about the eventual prevalence of HIV as it would have been to expect fifteenth century epidemiologists in the decade after the introduction of
Diagnosing pulmonary embolism

If the lung scan is non-diagnostic attention should turn to the proximal leg veins

The importance of establishing the diagnosis in a case of suspected pulmonary embolism is beyond doubt. Clinical trials in the early 1960s established the untreated condition's high mortality (30%) and the effectiveness of giving anticoagulants. Continuous intravenous heparin provides immediate and almost complete protection against recurrence of embolism and should therefore be started as soon as the diagnosis is suspected. On the other hand, anticoagulants are not without risk: heparin given by continuous intravenous infusion for seven days carries a 5% risk of major haemorrhage; warfarin given for three months an 8% risk. A vigorous attempt should therefore be made to substantiate the diagnosis before anticoagulants are continued long term.

Although reviews traditionally conclude that pulmonary angiography should be used much more widely to diagnose pulmonary embolism, little evidence exists that this advice is taken. A recent British survey found that only one third of acute hospitals could provide the service, and in those pulmonary angiography was performed on average four times a year for suspected pulmonary embolism.

Since its introduction in the 1960s the radioisotope ventilation-perfusion lung scan has dominated the investigation of this disease: a simple, minimally invasive test that is cheap and quick to perform and has reasonable specificity. A steady flow of retrospective studies in the 1970s comparing this technique with the definitive investigation of pulmonary angiography has recently been refined by two prospective studies. The have identified two groups in whom a therapeutic decision can be made. The first is patients with a "high probability" scan result (10-20% of patients), which means the presence of multiple segmental or lobar defects in perfusion that are unmatched by defects in ventilation. Patients with such a scan result have a near 90% chance of embolism and should be given anticoagulants unless there is a history of previous embolism—probably the commonest cause of a false positive result—in which case comparison with old scans is desirable.

The second group consists of patients with a normal or near normal scan (15-40% of patients), in whom the diagnosis can be virtually excluded. In these patients another cause for the symptoms should be sought. In all other cases (40-70% of patients) the scan result should be regarded as non-diagnostic. Although scans from such patients may be categorised into various subsets with different probabilities of embolism, this does not solve the clinician's problem because the overall rate of angiographically proved embolism in this group is about 40%.

These findings have not, apparently, greatly influenced current clinical practice. Doctors are still relying on results of lung scans to decide patients' management without resorting to pulmonary angiography to clarify the diagnosis. The reasons for this are clear: angiography is relatively expensive, time consuming, and perceived to be risky. The last of these is a misconception. In skilled hands the procedure carries little risk.