Tuberculosis and HIV: blind man’s buff

The year 1985 might be seen as a watershed for tuberculosis. In that year tuberculosis notifications both in the United States and in England and Wales began to deviate from the previous exponential decline and, in the last few years, there has been a small but steady increase. It is now apparent that notifications were increasing in several other developed countries, including Japan, Scandinavia, Italy, and Spain, from about the same time. The simple explanation was thought to be HIV infection. By compromising the cell-mediated immunity of individuals also infected by the tubercle bacillus HIV activated tuberculosis infection and caused disease, and a worldwide increase in tuberculosis was occurring as a consequence. Analysis of the age and sex distribution of cases, showing a rapid increase in notifications in young men, initially suggested that HIV infection was the reason for this increase in the United States. Subsequent data have suggested that some other factors, such as social deprivation, immigration, and a disintegration of the tuberculosis control programme, may be just as important.

An age-sex analysis of the notifications in England and Wales since the upturn shows that the biggest initial increase was in the elderly. Though the proportion of younger men with disease has increased so has the proportion of younger women. From these indirect data HIV seems unlikely to be implicated yet in the rise in notifications we are experiencing in England and Wales (notifications in Scotland continue to decline).

The paper by Watson et al. in this issue of Thorax (p 199) tries to look more directly at the relation between tuberculosis and HIV infection in England and Wales on the basis of three methods: by calculating the incidence of tuberculosis in reported cases of AIDS and the incidence of AIDS reported in cases notified during the two Medical Research Council tuberculosis notification surveys of 1983 and 1988, and by a questionnaire sent to doctors in the 1988 survey asking how many of the notified patients were known to be HIV positive. The first and second methods attempted to identify patients with dual infection by a system of register matching.

Their conclusion, based on the fact that only one patient was known to have tuberculosis and AIDS in the 1983 survey and nine in the 1988 survey, is that tuberculosis related to HIV infection is increasing but has yet to have any appreciable impact on tuberculosis notifications. One important finding from the survey of doctors was that a higher proportion of patients with AIDS developed tuberculosis among those of non-white ethnic origins—about 2.5 times as many in Asian and three times as many in black as in white patients. This is because tuberculosis infection is much more prevalent in the ethnic minorities in the age group most at risk from HIV infection than in the white population of the UK.

What is much more problematical is how HIV related tuberculosis is likely to affect notifications in the future. As Watson et al. point out, we have virtually no data on the prevalence of tuberculosis infection in the population. The only data we do have are in schoolchildren aged 12–14, who are routinely tuberculin tested before BCG vaccination. Though the prevalence is low, under 2%, we have no idea what happens, in Britain as a whole, to the tuberculin response as tuberculin test negative children grow up.

The voluntary reporting of cases of AIDS means that there is no statutory obligation for clinicians to report their cases as is required for tuberculosis. National monitoring of HIV related tuberculosis is therefore difficult. For this reason the 1993 survey of tuberculosis notifications, being coordinated by the Public Health Laboratory Service Communicable Disease Surveillance Centre, is asking clinicians to perform HIV testing of notified patients on a voluntary or anonymous basis.

HIV related tuberculosis appears to be as infectious as disease not related to HIV and therefore poses a health hazard for the community, even among those at no risk from HIV infection. The magnitude of the problem in parts of sub-Saharan Africa, where case rates have risen threefold in a decade as a result of HIV infection, is all too apparent. The scale of the problem will not be as great in most developed countries, but may appreciably affect some sections of the community where tuberculosis infection is prevalent. The present system of monitoring and reporting cases of AIDS seems unnecessarily secretive and cumbersome. If we are to plan resources appropriately a better way of monitoring AIDS and HIV infection is necessary. The current restrictions of confidentiality in reporting require those who perform our health surveillance to play a sort of blind man’s buff to answer Watson et al.’s point out, we have virtually no data on the prevalence of tuberculosis infection in the population. The only data we do have are in schoolchildren aged 12–14, who are routinely tuberculin tested before BCG vaccination. Though the prevalence is low, under 2%, we have no idea what happens, in Britain as a whole, to the tuberculin response as tuberculin test negative children grow up.

To plan the resources needed to combat the potential increase in tuberculosis due to HIV infection in Britain we need to know the answers to three further questions. Firstly, what is the prevalence of tuberculosis infection in the population? Secondly, what is the prevalence of HIV infection in the population? Thirdly, what are the changes in the prevalence of these two infections? The extraordinary fact is that we probably know more about the prevalence of HIV than of tuberculosis infection.

A reasonably certain guess is that the rate of decline of tuberculosis infection, particularly with the current increase in notifications, cannot compensate for the rate of increase in HIV infection. The risk of dual infection will therefore increase in the foreseeable future, with a
consequent increase in the risk of tuberculosis in the community. This effect will first be seen in ethnic minorities, where the prevalence of tuberculosis infection is highest. The prevention of HIV related tuberculosis in the indigenous white population will depend on our ability to control tuberculosis as much as HIV infection. Continued maintenance of our tuberculosis control programme is therefore vital.

One fact may at least give encouragement: spread of tuberculosis between different ethnic groups in the UK has not been described.

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