

Pulmonary tuberculosis in the elderly: a different disease?

Since the early 1980s there has been concern at the increasing incidence of pulmonary tuberculosis in people over 65. Part of the concern has been the difficulty in making the diagnosis in this population¹⁻³ and the ease with which, in enclosed environments such as institutions and homes for the elderly, the disease may reach epidemic proportions among the susceptible aged.^{4,5} The predisposition to develop tuberculosis in those immunocompromised by age, drugs, disease, or malnutrition is well recognised.

The basic classification of tuberculosis into primary infection (and disease) and postprimary (or reactivated) disease is well established. The radiological features of the primary infection (small mid zone peripheral lesion or segmental inflammatory lesion, or both, with hilar adenopathy) and of postprimary disease (apical fibrosis, pulmonary opacities, cavitation, and apical pleural thickening) are well known. Why then is there difficulty in diagnosing the disease in the elderly? Is it because it presents differently in primary and in classical postprimary disease?

A few studies have reported the atypical radiological picture seen in pulmonary tuberculosis in the elderly^{6,7} and some authors have commented on the haematological and biochemical findings.⁸ A recent survey in 93 elderly patients of the radiological, haematological, and biochemical features of pulmonary tuberculosis¹ showed that the manifestations of the disease in this age group are different from those seen in classical primary or postprimary disease.⁹ Only 7% of the radiographs in this elderly population showed isolated apical opacification. Of the rest, half had pulmonary shadows in the mid and basal zones only and half in a combination of apical, mid, and basal zones. Cavitation was apparent in only one third of radiographs and in half of these it occurred in the mid and lower zones. A basal pleural reaction (effusion or thickening, or both) was present in association with pulmonary shadowing in half the cases. This is an unusual finding in postprimary tuberculosis, though apical pleural fibrosis is common. Basal effusions are sometimes associated with primary disease. Thus the pattern of radiological appearances overall differs from those of primary or postprimary tuberculosis. (They are similar to those seen in patients with decreased or absent cell mediated immunity, such as patients with AIDS.)¹¹ Changes in biochemical and haematological indices in elderly patients with tuberculosis differ from those seen in postprimary disease in young adults.⁹ About two thirds of our elderly patients had increased liver enzyme activities (compared with one third in the younger group) and more patients had hypoalbuminaemia, hyponatraemia, and hypokalaemia. About two thirds of the elderly patients had normochromic normocytic anaemia, polymorphonuclear leucocytosis, and a raised erythrocyte sedimentation rate. These features were generally more severe than is usual in the younger adult group with postprimary disease.⁹ The reason for these differences is not clear but they may reflect dissemination or reactivation at other sites, especially the liver, in older people.

Thus, although the clinical presentation of pulmonary disease in elderly people does not differ significantly¹⁹ from that in younger patients (cough 80%, weight loss 70%, haemoptysis 8%, and fever 55%), there are pronounced differences in the radiological appearance and more abnormalities in haematological and biochemical indices. These changes are not diagnostic but their presence without

obvious cause should alert clinicians to the possibility of tuberculosis.

The means of acquiring the disease may also be different in elderly people, as not all postprimary disease in the elderly is due to reactivation. When clusters of cases occur, as in homes for the aged, an index case has caused either an exogenous initial primary infection or, importantly, reinfection.^{4,12} In the latter case previous acquired immunity would be presumed to have waned, that individual thus becoming vulnerable to reinfection and disease. Possibly endogenous acquired infection may play a part. Today's elderly people contracted the disease at a time when not only was tuberculosis more prevalent but effective treatment was non-existent. Most people born in the early twentieth century became infected¹³ and are therefore liable to reactivation of infection. It is postulated that with decreasing immunity breakdown of a dormant (apical) lesion containing viable bacilli results in the release of organisms into the airways, endogenously infecting the rest of the lungs by bronchial spread. Forty per cent of our patients¹ had apical fibrosis compatible with previous disease, though only about 16% had obvious apical cavitation. We would not, however, expect to see small cavities on chest radiographs. Breakdown of an endogenous lesion may also result in dissemination of infection via the bloodstream, thereby affecting many organs, including the lungs. With an effective host cell mediated immune response this dissemination results in granulomas and produces a miliary picture; but in an areactive host the disease is diffuse and not easily detectable. "Cryptic tuberculosis" occurs predominantly in people over the age of 60, usually with a normal chest radiograph, pyrexia of unknown origin, and a negative tuberculin reaction. It is notoriously difficult to diagnose.¹⁴

From a clinical and diagnostic viewpoint it is of great importance to be aware that primary and postprimary pulmonary tuberculosis may present differently in children and young adults and in the elderly. Whether the differences are such that tuberculosis in the elderly should be regarded as a separate entity deserving a separate classification or as part of the range of the disease is a matter for debate (see table). In favour of a new classification is the fact that it would reinforce the differences between this atypical form of the disease and the classical disease pattern seen in young patients, and would emphasise the difficulties of diagnosis in the elderly. This is particularly impor-

Range of radiographic appearances of pulmonary tuberculosis in elderly and immunocompromised patients (the atypical form being common, the classical unusual, and the disseminated rare)

Classification	Chest radiographs
<i>Atypical</i> Pulmonary tuberculosis in the elderly	Lower zone opacities with basal pleural effusions or thickening and few cavities found equally in apex or lower zones.
<i>Classical</i> Postprimary or reactivated	Apical interstitial fibrosis and pleural thickening, cavitation and opacities.
<i>Disseminated</i> Miliary (reactive) Cryptic (areactive)	Miliary nodularity Normal

tant in developed countries, where the incidence of the disease is low in the general population and any variation from the textbook description of the classical disease reduces the chance of diagnosis.

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