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Letters to the Editor

CONCOMITANT OCCURRENCE OF LEPROSY AND TUBERCULOSIS: LEPROSY VACCINE, A MYTH OR REALITY?

Sir,

S, a 40-year-old man, presented complaining of nasal blockage, crusting and loss of sensation over the hands and feet for $2\frac{1}{2}$ years. He had apparently been well 3 years before when he had a problem of a blocked nose with crust formation in the anterior nares. This was initially neglected, but he was alarmed when he noticed streaks of blood in the nasal discharge. Subsequently he noticed that the sensation in his hands and feet had diminished, and he often inadvertently burned his finger tips when holding hot objects. His attention was drawn to an ulcer over the sole of his right foot by the foul smell emanating from it. Meanwhile, his wife noticed a change of appearance in his face, which had become oily and coarse, with loss of eyebrows. He sought advice in a hospital where he was put on treatment with monthly doses of rifampicin (600 mg) and clofazimine (50 mg) and dapsone (100 mg). He was, however, irregular in taking his medicines. A few weeks before reporting for treatment he developed fever, malaise, an evening rise of temperature and a productive cough. He revealed that he had had similar symptoms 5–6 years before which had been diagnosed and confirmed as pulmonary tuberculosis. He had been adequately treated with short-course intensive therapy with rifampicin (600 mg) isoniazid (300 mg) and streptomycin (1 g daily) for 3 months after which streptomycin was discontinued, and treatment continued with rifampicin and isoniazid for a further 6 months to complete a 9-month course. His response to treatment was favourable and he was pronounced cured and the treatment was discontinued. He remained symptom-free until he once again had a recurrence. The symptoms and signs were indicative of activation of pulmonary tuberculosis.

Examination revealed that he was febrile, with a temperature of 100°F. The vital and general physical examination was unrewarding. Respiratory examination was marked by a few diffuse fine bilateral crepitations. Cutaneous examination was marked by hypopigmented shiny macules with ill-defined margins merging imperceptibly into the surrounding skin. There were innumerable lesions that were extensively distributed over his extremities and trunk. Facial examination demonstrated atrophic wrinkled skin, atrophic earlobes, a depressed bridge of the nose and a lateral loss of one-third of the eyebrows. Nasal examination was indicative of haemorrhagic crusting resulting in an oooze and crust formation. Other mucosae were normal. The sensation of temperature, touch and pain were impaired over the palms and soles. The greater auricular, radial, ulnar, common peroneal and anterior and posterior tibial nerves were thickened. A trophic ulcer was present over the base of the great toe on the right side. The laboratory investigations were marked by haemoglobin 11·5 gm/dl, total leukocyte count 6800 cu/mm, differential leukocyte count polymorphs 50, lymphocytes 34, eosinophils 16, erythrocyte sedimentation rate (Westergren) 134 mm/1st hour. Liver and kidney function tests were within normal limits. A Mantoux test was non-reactive. Sputum for acid-fast bacilli was negative. The results of sputum culture on Lowenstein–Jensen median did not yield any growth after 5 weeks. The enzyme-linked immunosorbent assay (ELISA) for IgG to *Mycobacterium tuberculosis* was 162/ml (suspect positive 161–199). The immunoglobulin/profile assay revealed raised levels of antibodies, IgM 74·83 g/l (0·5–2 g/l), IgA

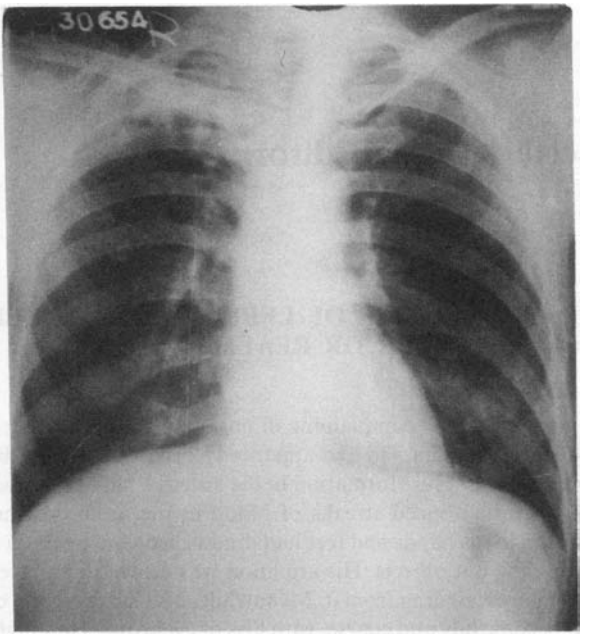

Figure 1.

5·73 g/l (0·6–2), and IgG 737·7 g/l (8–16 g/l). An X-Ray of the chest was marked by intense infiltration of lung parenchyma, reflecting in the form of soft opacities in the right upper zone, suggestive of pulmonary tuberculosis (Figure 1). The trachea was central, and the heart was normal in size. The bony cage was within normal limits. Histopathological examination of a tissue section revealed nonspecific dermal inflammatory infiltrate indicating resolving granuloma.

Leprosy, a chronic infective disease, has shown a steady decline across the globe[1] apparently because of an overall amelioration of the socioeconomic status, improved health care delivery systems, the advent of multidrug therapy (MDT) and its compliance.[2] However, with the advent of the acquired immunodeficiency syndrome (AIDS), a resurrection of mycobacterial diseases, including leprosy, has been observed. A similar trend has been noticed in systemic tuberculosis that has once again been recorded in places where it had been eradicated. The compromised cellular immunity probably reactivates the dormant bacilli that had become quiescent following adequate chemotherapy.

The interaction between leprosy and tuberculosis is rather intriguing. Initial reports suggest that leprosy encouraged the development of tuberculosis. It could be speculated that both mycobacterial diseases probably shared a common genetic predisposition.[3] Besides the low socioeconomic factors that conferred higher susceptibility to both diseases, it was observed that BCG vaccination of mice inhibited subsequent multiplication of *M. leprae* in footpads.[4] This initiated the exploration of crossimmunity between both diseases and the possibility of a mycobacterial vaccine offering protection from leprosy.[5–8] In the ensueing years, BCG emerged as a candidate that was used in trials for the immunoprophylaxis and immunotherapy of leprosy. However, many studies[9,10] on the protective efficiency of BCG vaccine have provided equivocal results. The initial euphoria has been further offset by an interesting revelation that delayed type hypertensitivity (DTH) and protective cellular immunity are directed to separate mycobacterial antigens and DTH may be augmented by MDT, repeated lepromin testing, cytokines *per se* and

antigenic challenges that induce cytokine activation. These have cast a doubt on the potential of mycobacterial vaccines to achieve immunological upgrading.[11,12] The coexistence of leprosy and tuberculosis[13,14] once again compels us to explore this myth.

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