FAILURE OF PASSIVELY TRANSFERRED LEPROSY LYMPHOCYTES TO DEMYELINATE PERIPHERAL NERVE

Sir,

Recent studies on the interaction between *Mycobacterium leprae* and cells of the immune system have raised the question whether peripheral nerve damage in leprosy is (a) incidental to the cell-mediated immune response to the bacilli in a neural milieu, 1,2 (b) the result of specific humoral and cellular mechanisms directed against neural components, 3,4 or (c) a combination of the 2 factors. We passively transferred human leprosy lymphocytes intraneurally to peripheral nerves of guinea-pigs and looked for cell-associated demyelination.

The donors of circulating lymphocytes were 8 patients with BT leprosy (2 of them undergoing Type I reaction) and 4 normal individuals. Fifty microlitres of the lymphocyte cell suspension in RPMI 1640 (containing 3.75–9 × 10⁶ cells), or RPMI 1640 alone were injected into the sciatic nerves of guinea-pigs. (Cell separation, counting and viability testing were kindly performed for us by Dr Sita Naik and colleagues at the K.E.M. Hospital Bombay.) The animals were sacrificed on day 4 and the nerves collected for light microscopy. (In an analogous study in Guillain–Barre polyneuropathy, Feasby et al. 5 found no evidence of host rejection of intraneurally injected heterologous (human) lymphocytes up to 4 days post-injection.) The histopathologic features were rated semi-quantitatively.

Normal endoneurial contents with a few or moderate numbers of lymphocytes in or outside the perineurium . . . 1

- Localized demyelination without cells (attributed to injection damage) . . . 2
- Normal endoneurial contents in the presence of lymphocytes . . . 3
- Demyelination in the presence of lymphocytes . . . 4

Statistical comparison between the ratings awarded to nerves injected with leprosy lymphocytes and normal lymphocytes and RPMI 1640 alone showed no evidence that demyelination was significantly correlated with the presence of leprosy cells. These observations are compatible with the reported *in vitro* absence in leprosy of circulating T lymphocytes specifically responsive to peripheral nerve myelin. Neither has there been evidence of anti-myelin antibodies in leprosy. 3,4 Specific anti-axonal antibody has been detected in the sera of some lepromatous and tuberculoid leprosy patients but there was no correlation with the degree of nerve damage, and hence no pathogenetic significance. 4 The probability that the ‘by-stander’ effect causes major nerve fibre damage in non-lepromatous leprosy appears strong.

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References


AN APPRAISAL OF MALIGNANT PLANTAR ULCER

Sir,

There are few reported cases of malignant change in plantar ulcer in leprosy. The number dwindles if lesions not commencing on the sole are excluded. It is suggested that the remarkably low incidence of malignant change in these ulcers is due to the fact that the plantar surface of the foot is minimally exposed to sunlight. Published case reports support this notion.

Reports in English of malignant change in plantar ulcer in leprosy are remarkably few. This may be because the condition is truly rare, because it is unrecognized, or because it appears to have no particular features warranting publication, or indeed for all 3 reasons.

In Papua New Guinea squamous carcinoma arising in old tropical ulcer scars is very common, and the principal function of the Artificial Limb Factory is to provide prostheses for patients whose amputations were for ‘S.C.C. leg’. Against this background the regular appearance of malignant change in plantar ulcers in my own practice has not seemed remarkable, and a score or more of cases seen since 1965 remain inadequately documented. We have even had 2 in the ward at the same time.

However, when every surgical unit on our coast has cases of ‘S.C.C. leg’ in the ward nearly all the time, so that students pass them by in search of something more interesting, 1 or 2 malignant plantar ulcers a year seem a very small yield given the large number of chronic ulcers presumably at risk.

The scars from tropical ulcer, and from burns, which become malignant, are those which have healed by granulation so that the eventual scar is an area of intense local albinism. Examination of the malignancies arising in such scars almost always clearly reveals their origin in the most depigmented area.

For this reason it was long ago suggested that sunlight was an important aetiological factor.\(^1\) This should not be surprising, given the photobiological axiom that keratoses and epitheliomata may be regarded as normal processes that occur in white skin if exposure to sunlight is continued for long enough.\(^2\) It seems unlikely that malignancy arising in chronic ulcers or scars on the front of the leg and those a few centimetres lower, on the sole, should have differing aetiology.

An examination of published case reports gives some support to this suggestion. Job & Riedel\(^3\) reported 4 cases. Their case 1 seems not to have been a plantar ulcer in the anatomical sense, having occurred on the lateral side of a varus foot, in an area obviously exposed to sunlight.

Their case 2, of which they publish a photograph, shows an ulcer on the lateral side of the heel and lower leg, once again exposed to sunlight. Their other 2 cases were true plantar ulcers, but are not illustrated.

Riedel\(^4\) subsequently published details of another case in which the ulcer was on the plantar aspect of the heel and was connected to the lateral malleolus by a sinus. In the absence of more information it is not possible to speculate on the extent to which the depigmented scar was exposed to the sun in this case.

Srinivasan & Desikan\(^5\) described 13 cases of cauliflower growths, only 3 of which were frankly malignant, all low grade. The other 10 were reported to show pseudo-epitheliomatous hyperplasia, and their good response to local excision confirms this diagnosis.

This condition is occasionally seen in the mouths of betel-nut chewers, and sometimes at the site of an old tropical ulcer, but has not been identified on the sole in my patients.

The fact that Srinivasan\(^5\) was able to collect only 3 malignant plantar ulcers in many years of very busy practice in leprosy suggests that malignant change is fairly rare in plantar ulcer in India.