THE TABLE. *A comparison of compliance in patients treated with loose MDT and MDT/BCP.*

	Loose MDT patients tested	Compliance (%)	MDT/BCP patients tested	Compliance (%)
Paucibacillary				
Males	21	67	16	75
Females	30	70	12	83
Total	51	68	28	79
Multibacillary				
Males	115	76	136	76
Females	88	75	94	85
Total	203	76	230	81
Combined total	254	72%	258	80%

the correct amount of loose dapsone on the previous clinic day. Thus, MDT/BCP offers the additional advantage of ensuring that the patient receives the exact quantity of MDT drugs required for treatment.

It has been estimated that MDT presented in BCP adds only US$0.46 and US$0.21 per month for the treatment of MB and PB leprosy, respectively ([1]). This is a small price to pay for the benefits of compliance and acceptability by patients and the ease with which it can be dispensed to patients, even by general health workers. As the resolve of leprosy control experts that leprosy control be integrated into the general health services becomes realized, one further benefit of MDT/BCP will become more significant, i.e., the diversion (by health workers) of rifampin meant for leprosy patients to (especially) tuberculosis patients. This fear already is being confirmed in at least one program running combined tuberculosis and leprosy services in Nigeria.

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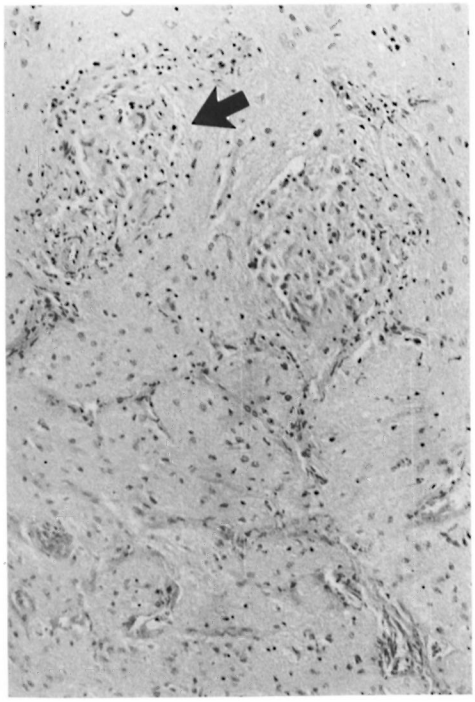
Tuberculous Optic Neuritis Histologically Resembling Leprous Neuritis

TO THE EDITOR:

We wish to report an interesting histopathological observation in the optic nerve of a case of ocular tuberculosis. A 5-year-old female underwent enucleation of the blind left eyeball for suspected retinoblastoma following detection of a pupillary white reflex, shallow anterior chamber, iris neovascularization and secondary glaucoma. On clinical examination right axillary lymphadenitis was found. This was diagnosed to be tuberculous on fine-needle aspiration cytology.

Histological examination of the eyeball revealed widespread tuberculosis involving uveal tissue and the retina. A section through



THE FIGURE. Granulomas (arrow) in the optic nerve. The optic nerve is distinguished by its glial appearance in histological sections (hematoxylin and eosin × 100).

the resected end of the optic nerve (The Figure) showed epithelioid cell granulomas in the substance of the nerve, reminiscent of neuritic leprosy. Tuberculous infection of the optic nerve is rare. It spreads from tuberculous lesions in the eye, brain or the adjacent orbital structures ([1, 2]).

Leprosy, on the other hand, is a disease of the peripheral nerves. Demonstration of *Mycobacterium leprae* or granulomas in nerves are the mainstay of histopathological diagnosis. Granulomas in a nerve have the same diagnostic significance as the presence of *M. leprae* ([8]). This is explained by the fact that *M. leprae* are the only known neurotropic mycobacteria ([4]). Cranial nerves with a peripheral component, i.e., the facial and the trigeminal, are known to be affected in leprosy. The optic nerve escapes probably because it is devoid of Schwann cells ([3]).

Other mycobacteria, not being neurotropic, do not primarily cause neuritis. However, granulomatous inflammation is destructive of pre-existing tissue ([7]), and nerves caught up in the inflammatory process may be involved. This fact probably explains the sporadic case reports of evidence of nerve involvement in nonleprosy granulomatous dermatoses ([5, 6]) with accompanying sensory impairment.

We have reported earlier our observations of different patterns of dermal nerve involvement in lupus vulgaris, granulomatous secondary syphilis and other nonleprosy granulomas ([9]). A granuloma within a dermal nerve was found in a case of lupus vulgaris. The present case documents granulomatous involvement of a large cranial nerve with tuberculosis.

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Protective Effect of BCG Against Leprosy in South Sulawesi, Indonesia

TO THE EDITOR:

A protective role of BCG against leprosy has been documented in several areas in the world ([1]). As yet, there have been no studies reported from Indonesia. We conducted a case-control study in South Sulawesi Province of Indonesia, an area with a registered leprosy prevalence of 13 per 10,000. Home visits were made to 115 children (< 15 years) with known leprosy. An experienced leprosy control officer confirmed the diagnosis of the patients and the classification into paucibacillary (PB) or multibacillary (MB) leprosy, using the criteria recommended by the Indonesian Ministry of Health (i.e., patients with six or more characteristic lesions are MB; others are PB). Two health workers assessed independently the presence of a BCG scar on the deltoid area of the patients and of 326 controls living in the same neighborhood, who were matched for sex, age, and place of birth.

The presence of a household member with leprosy should be considered a risk factor for the development of leprosy. Patients and controls were asked whether any members of their households had ever been diagnosed with leprosy. The information on leprosy in household members was also verified by checking the patient card.

BCG vaccination gave an overall protection, calculated as (1 − OR) × 100%), against leprosy of 76% [95% Confidence interval (CI) = 39%–90%; matched analysis, controlled for type of leprosy and contact status, p = 0.003]. Analysis of the data separately for MB and PB patients showed a protective effect of BCG vaccination for MB disease (OR = 0.18) but not for PB disease (OR = 1) (The Table).

However, further analysis of the data leads us to postulate that BCG vaccination also protects against PB disease, but that this protection is masked because of the design of the study. Firstly, we observed that PB patients with a case of leprosy among their households appeared to have a high vaccination coverage of 57.1% (8/14). The vaccination of PB patients without leprosy in their households was only 14.3% (3/21). The difference did not reach a significant level (Mantel-Haenszel, controlled for age, p = 0.09), but this could be due to the small numbers involved. Secondly, we found that the BCG vaccination status influenced the development of the type of disease in patients. In the BCG-vaccinated patient group who had a MB leprosy patient in their households, 71% (5/7) acquired PB disease compared to 23% (6/26) in the nonvaccinated patients of this group (p = 0.027). Six patients had a PB patient in the household: three of them were BCG vaccinated and developed PB disease; three were not BCG vaccinated and developed MB disease.

These observations suggest that after exposure to leprosy through a household member, a BCG-vaccinated person may develop the paucibacillary form rather than the multibacillary form of the disease. Then, in these cases, the number of PB patients with BCG vaccination will be increased.

Our results are consistent with the theory that BCG vaccination brings about a shift in the immune response to a higher level of cell-mediated immunity and, thereby, offers protection especially against the more severe multibacillary form of the disease ([2]). When this phenomenon is operative, case-control studies on the protective effect of