To THE EDITOR:

I would like to report a patient who was believed to suffer from a depressive reaction following the onset of cutaneous lesions of leprosy but whose cognitive impairment was found to be associated with the systemic diseases, diabetes mellitus and cerebral atrophy.

A 57-year-old Chinese male was brought to the skin clinic in March 1989 with a history of skin nodules and reactive depression of 2 years' duration. He was uncooperative but not abusive or aggressive. His appetite was good but he suffered from insomnia. There was no history of head injury, diabetes mellitus, or mental illness in the family. He was not an alcoholic but smoked about 10 cigarettes a day for 30 years. He had isolated himself and had wandered aimlessly clad in his underpants. The skin lesions were that of leprosy. He appeared in good health but was withdrawn, dull, fearful, depressed, showed a lack of emotional response, and was dressed carelessly. His speech was slow and sometimes incoherent, suggestive of impairment of intellectual functions. He had a memory defect for recent events so much so that he ate several times a day forgetting that he had taken food a few hours ago. There were no florid symptoms such as hallucinations or delusions.

His blood pressure was 110/80 mm Hg, and he had + glycosuria. A slit-skin smear for acid-fast bacilli showed a bacterial index (BI) of 5.8+ and a morphological index (MI) of 4.5%. A skin biopsy confirmed the diagnosis of lepromatous leprosy. The results of a modified glucose tolerance test were: Fasting 17.4 mmol/L (normal 3.6–6); 1 hr 26.6 mmol/L (normal 10); 2 hr 23.6 mmol/L (normal 6.7). A venereal disease research laboratory (VDRL) test was non-reactive. A radiograph of the chest and other relevant investigations were normal or within normal limits. A computerized tomography (CT) scan of the brain showed features of bifrontal atrophy (The Figure). He was treated with chlorpromazine, glibenclamide and multidrug therapy (MDT) for multibacillary leprosy. The hyperglycemia was fairly well brought under control, and he was able to sleep well. He did not talk with other patients in the ward, was difficult with medication, and did not follow instructions in his diabetic diet. There was an improvement in mood, in that he was quite cheerful and was able to smile during follow
up. His disorientation in space and time was less evident than before.

Dementia is a chronic organic brain syndrome which involves the intellect, memory, emotions, and behavior whose decline exceeds that expected of normal aging. The degree of deterioration depends upon the various causes (\textsuperscript{1}), such as cerebral arteriosclerosis, trauma, tumors, endocrine disturbances, intoxication and deficiency disorders, and a group of degenerative diseases which include Alzheimer's disease, Pick's disease, Huntington's chorea, and senile dementia. The classification of dementia is disputed and takes into account different variables (\textsuperscript{2}) such as the etiology, age at onset, pathology, etc., but the subcortical-cortical dichotomy seems to supersede that of the previously held senile and presenile dementia. Fluctuations in mood—euphoria and depression—are common in this disease. However, depression in dementia should be differentiated, which is not easy, from depressive illness and depressive reaction (\textsuperscript{3}). Presumably, diabetes mellitus might have contributed indirectly to the dementia in our patient through an arteriosclerotic mechanism. Although suicides have been recorded in leprosy patients (\textsuperscript{4}), psychosocial disorders in them are similar to that encountered in patients with other illnesses (\textsuperscript{5}). Nevertheless, overt psychiatric symptoms in these patients should be studied in depth and, if necessary, further investigated so that treatable causes of dementia are not overlooked.

"Dementia and other organic syndromes ... provide the clearest example of a relationship between disorders of the mind on one hand and physical or cerebral diseases on the other; a relationship seen in some measure throughout the medical practice" (\textsuperscript{6}).

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REFERENCES

Triethanolamine-Induced Allergic Contact Dermatitis Over a Tuberculoid Leprosy Lesion

To the Editor:

A 60-year-old man relapsed with a tuberculoid (TT) Hansen's lesion 30 years after treatment with low-dose dapsone for 4 years. Examination revealed a hypopigmented, anhidrotic, anesthetic lesion 5 cm \times 3 cm over the outer aspect of the left upper arm. He was treated with pulse doses of 600 mg of rifampin once a month and 100 mg of dapsone daily. Since he complained of dryness over the lesion, he was advised to use a topical moisturizing cream (Cotaryl\textsuperscript{®}). Six months later he complained of itching over the hypopigmented lesion. Examination revealed a mild erythema, scaling, and oozing with lichenification over the TT lesion. A biopsy showed a tuberculoid granuloma in the upper dermis with spongiosis of the epidermis and mild edema of the dermis (The Figure). A provisional diagnosis of allergic contact dermatitis (ACD) to the moisturizer was made, and the ingredients of the moisturizer (Cotaryl\textsuperscript{®}) were requested from the manufacturer. Patch tests