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 ([3]), 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 ([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 ([4]). 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 ([5]), psychosocial disorders in them are similar to that encountered in patients with other illnesses ([1]). 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" ([3]).

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REFERENCES

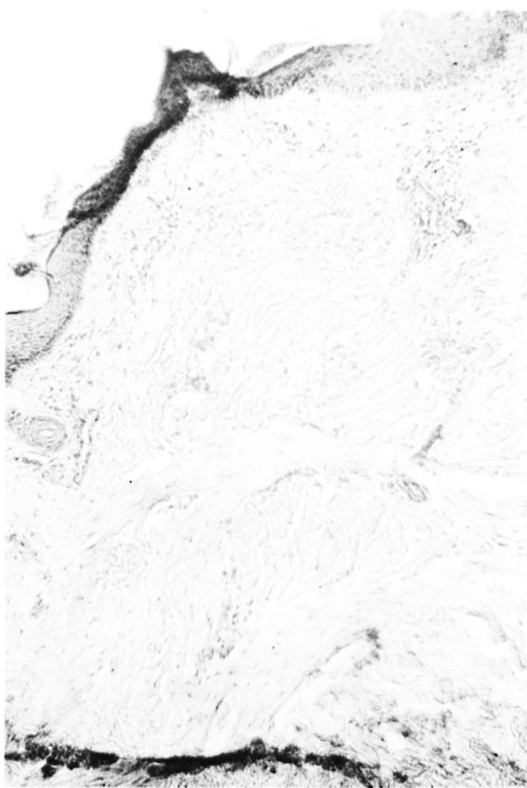
1. BAHLINGER, V. M., BRANTLEY, P. J., MADRIGAL, D. R., HEROMAN, M.W. AND VEITIA, M.C. Psychological stress in Hansen's disease: a comparison with other chronic illness patients. Int. J. Lepr. **53** (1985) 251–254.
2. CHUI, H. C. Dementia; a review emphasizing clinicopathologic correlations and brain-behaviour relationships. Arch. Neurol. **46** (1989) 806–814.
3. ROTH, M. AND MYERS, D. H. The diagnosis of dementia. In: *Contemporary Psychiatry.* Silverstone, T., Barraclough, B., eds. Ashford, Kent: Headley Brothers Ltd., 1975, pp. 87–99.
4. SLATER, E. AND ROTH, M. *Clinical Psychiatry.* London: Bailliere Tindall, 1977, pp. 188–222.
5. ZHOU, D., *et al.* [On suicide among leprosy patients.] China Lepr. J. **3** (1987) 240-243. English abstract in Int. J. Lepr. **56** (1988) 489.

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 × 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®). Six months later he complained of itching over the hypopigmented lesion.

Examination revealed a mild erythema, scaling, 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®) were requested from the manufacturer. Patch tests



THE FIGURE. Photomicrograph showing mild spongiosis, dermal edema, diffuse mononuclear infiltrate in the upper dermis, and a tuberculoid granuloma (H&E ×10).

were performed with the ingredients obtained from Chemotechnique Diagnostics AB, Sweden, using Vander Bend Chambers. Erythema and vesicles developed 48 hr following the application of 2% triethamolamine (TEA) which is used to form the emulsifier system in the moisturizing cream. TEA is an emulsifier in skin-care products and cosmetics, such as moisturizers, foundation, lipstick, shampoos, etc., ([2]) and has been reported to cause both allergic ([1, 2]) and irritant dermatitis ([1]). A 2% concentration has been recommended to avoid irritant dermatitis ([3]). Only 0.5% TEA is used along with stearic acid to neutralize it completely and to form TEA stearate soap which acts as the chief emulsifying agent for the product, Cotaryl® cream (Dr. M. G. Wagh, Medical Adviser, FDC Limited, personal communication). Thus, there is no free TEA in the marketed cream (Dr. Wagh, personal communication).

There is much debate over the role of melanocytes and melanin and functions other than protection from light are attributed to it ([4, 5]). We have reported the sparing of vitiliginous skin in parthenium-induced ACD ([5]) and the sparing of nevus depigmentosus in a case of textile dermatitis ([4, 8]). We have also successfully induced repigmentation over a TT lesion following topical ([6]) and systemic PUVASOL ([7]); thus proving that melanocytes could be activated over a TT lesion. The role of abnormal Langerhans' cells and decreased melanocytes ([4, 5]), diminished melanin ([8]), and the ability of the lesion to repigment following treatment ([6, 7]) in the induction of allergic contact dermatitis needs further investigation. We conclude that although ACD over a TT lesion is unusual, it should be recognized since there is a risk of missing the diagnosis of leprosy in such a case.

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REFERENCES

1. HANNUKSELA, M., KOUSA, M. and PIRILÄ, V. Contact sensitivity to emulsifiers. Contact Dermatitis **2** (1976) 201–204.
2. JONES, S. K. and KENNEDY, C. T. Contact dermatitis from triethanolamine in E45 cream. Contact Dermatitis **19** (1988) 230.
3. MAIBACH, H. I., AKERSON, J. M., MARZULLI, F. N., WENNINGER, J., GREIF, M., HJORTH, N., ANDERSEN, K. E., and WILKINSON, D. S. Test concentrations and vehicles for dermatological testing of cosmetic ingredients. Contact Dermatitis **6** (1980) 369–404.
4. NORLUND, J. J., FORGET, B., KIRKWOOD, J. and LEVNER, A. B. Dermatitis produced by applications of monobenzone in patients with active vitiligo. Arch. Dermatol. **121** (1985) 1141–1144.

5. SINGH, K. K., SRINIVAS, C. R., BALACHANDRAN, C. and MENON, S. Parthenium dermatitis sparing vitiliginous skin. Contact Dermatitis **16** (1987) 174–178.
6. SRINIVAS, S. R. Photoactivated topical 8-methoxypsoralen in repigmentation of a tuberculoid leprosy lesion. Int. J. Lepr. **56** (1988) 109–110.
7. SRINIVAS, C. R., NAIK., R. P. C., KRUPSHANKAR, D. S. R. and MENON, S. K. Photoactivated 8-methoxypsoralen in repigmentation of tuberculoid leprosy. Int. J. Lepr. **55** (1987) 159–160.
8. SRINIVAS, C. R., SINGH, K. K. and BALACHANDRAN, C. Textile dermatosis sparing nevoid depigmentation. Contact Dermatitis **16** (1987) 235.

Association of Pure Red Cell Aplasia and Lepromatous Leprosy

TO THE EDITOR:

Pure red cell aplasia is a widely used name for a type of anemia characterized by an isolated depletion of the erythroid tissue. It may be acute and self-limited or chronic, a form which may be constitutional or acquired ([5]). The acquired chronic type occurs predominantly in middle-aged adults, and immunological rejection of the erythroid tissue may be the underlying cause. This is suggested by a) the association with systemic lupus erythematosus ([2]), chronic lymphoid leukemia ([1]), and rheumatoid arthritis ([10]) and b) its responsiveness to treatment with immunosuppressants ([3]). Frequently, antibody against erythroid progenitors ([6]) or erythropoietin ([9]) can be detected, and antibodies which react with erythroblasts occur in 50% of the cases ([7]). Approximately 30% to 50% of the cases are associated with a thymoma ([11]).

Anemia occurring in leprosy patients may be due to chronic disease and, in some cases, hemolysis is detected, associated with the use of sulfones ([4]) or due to the presence of autoantibodies ([8]). We describe a case of leprosy associated with pure red cell aplasia. No reports of this association were found in the literature.

Case report. A 45-year-old man presenting with severe anemia was seen at our service. Fourteen months before, lepromatous leprosy was diagnosed by the presence of *Mycobacterium leprae* in a liver biopsy, nasal mucus, and earlobe lymph. He was treated with rifampin, thalidomide and dapsone. Anemia had appeared 6 months after diagnosis and had become worse, even after discontinuation of treatment. Hemoglobin was 5.9 g/dl; red cell count, 2.1 × 10^6 μl; hematocrit, 18.7%; mean corpuscular volume, 89 fl; mean corpuscular hemoglobin, 28.1 pg; mean corpuscular hemoglobin concentration, 32.4 g/dl; reticulocytes, 0.2%; leukocyte count was 17,300 with 6% band forms, 79% neutrophils, 12% lymphocytes, and 3% monocytes; platelet count was 188,000/μl. A bone-marrow biopsy demonstrated normal cellularity with rare elements of the erythroblastic series. The granulocytic series and megakaryocytes were normal. Blood group alloantibodies and autoantibodies were negative, and a chest investigation showed no evidence of thymoma.

Thus, the diagnosis of pure red cell aplasia was made, and the treatment consisted of the oral administration of 80 mg of prednisone per day. The patient improved, and 6 weeks later his hemoglobin was 13.7% g/dl. Prednisone was then gradually reduced, but when the dose reached 40 mg, his anemia returned (Hb = 6.8 g/dl). The patient needed high doses of prednisone for maintenance of a good hemoglobin level. He died 2 months later from pneumonia.

Several drugs may cause pure red cell aplasia ([4, 5]), possibly due to toxic interference in the metabolism of erythroblasts. Frequently, however, these features are reversible after suspension of the drugs ([6]). To our knowledge there are no previous reports on pure red cell aplasia induced by the drugs used in this study. Besides, the anemia persisted after the discontinuation of these drugs. Therefore, in the present case, it is unlikely that the red cell aplasia was caused by drugs. Since lepromatous leprosy may be associated with autoimmune phenomena ([8]), this could be the cause of the red cell aplasia observed. Autoimmunity in leprosy can be demonstrated by the positiveness of tests such as antinuclear antibody, lupus erythematosus cell, rheumatoid factor, anti-