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-

thyroglobulin antibodies, cryoglobulins and false-positive serology for syphilis ([8]). Thus, it is possible that autoantibodies against erythroid progenitors or erythropoietin were the cause of the pure red cell aplasia in this patient.

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