HYPERTHYROIDISM AND AUTOIMMUNE HEMOLYTIC ANEMIA — A CASE REPORT

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INTRODUCTION

IT IS well known that autoimmune hemolytic anemia (AIHA) may occur in patients who have suffered from, or are suffering from diseases such as systemic lupus erythematosus, rheumatoid arthritis, ulcerative colitis and lupoid hepatitis which are thought to be of autoimmune origin. Wasastjerna reported a case of AIHA and Hashimoto's thyroiditis. Pernicious anemia has been strongly associated with hypothyroidism, hyperthyroidism and thyroiditis. Here we report a case of hyperthyroidism manifesting some six years after the onset of AIHA.

CASE REPORT

In August 1972, a 39 year old Chinese woman was admitted with a two month history of amenorrhoea and generalised weakness and two weeks of giddiness and palpitations. She had marked pallor, mild jaundice and was in cardiac failure. Lymph nodes, spleen and liver were not palpable. Erythrocyte sendimentation rate (ESR) was 140 mm in the 1st hour, hemoglobin (Hb) 6.8g/dl, leucocyte count 4 x 10^9 /1 (4,000/mm^3), platelet count 370 x 10^9 /1 (390,000/mm^3) and reticulocyte count 1.5%. Peripheral blood showed red blood cells moderately hypochromic, microcytic with a few well hemoglobinised normocytes. Bone marrow picture was consistent with hemolysis. Direct Coomb's test initially negative, was positive two months later. Rheumatoid factor was weakly positive and no LE cells were detected. IgG antibody of no known specificity was detected.

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She improved with blodd transfusion, prednisolone and folic acid. However she required further admissions for hemolytic crises easily controlled by stepping up the maintenance dose of steroids. In October 1975, she complained of generalised weakness and clinically had proximal myopathy. Her Hb 8.4g/dl, ESR 113 mm in 1st hour, serum creatine phosphokinase, serum aldolase, blood urea and serum electrolytes were within normal. She remained weak and lost weight. In May 1978, she was readmitted with severe hemolysis and thyrotoxicosis. Her thyroid gland was firm, diffusely enlarged but she had no eye signs or acropachy. Radioiodine thyroid uptake studies confirmed hyperthyroidism. Antinuclear factor, antimicrosomal antibody and antithyroid antibody were negative. A transient pancytopenia with Hb 5.6g/dl, leucocyte count 2.6 x 10^9 /1 (2,600/mm^3) and platelet count 20 x 10^9 /1 (20,000/mm^3) was noted. Reticulocytes were 10%. The bone marrow showed megaloblastosis though serum folate and vitamin B levels were normal. She was given therapeutic radioiodine.

COMMENT

Our patient probably has Graves' disease, an autoimmune disease, and its coexistence with AIHA is not surprising. Pirofsky (a) found 24 patients with clinically demonstrable thyroid disease in 234 patients with AIHA of whom only four had hyperthyroidism. Twenty-one of these 24 patients were females with a median age of 40 years at the time of onset of detectable thyroid disease. AIHA preceded the thyroid disease by 4, 15 and 191 months in three patients; the reverse occurred in 14 patients while in seven patients both diseases were simultaneously discovered. Seventeen of these 24 patients already with these two pathologies had additional disease states representing a cross section of immunologically related disease.

It is highly probable that, in our case, hyperthyroidism, while still clinically inapparent and

untreated, made control of AIHA brittle as decreasing the steroid dosage to maintenance doses appeared to precipitate hemolytic crises. Although steroids are used to treat autoimmune conditions, whether they are beneficial in Graves' disease remains conjectural.

As regards the pancytopenia, it is unlikely that our patient has pernicious anemia as serum vitamin B_{12} level is normal. However pancytopenic type of AIHA has been documented. (Pirofsky (b))

Thus besides excluding the well known secondary causes of AIHA (Dacie) for any deterioration in the patient's health, other diseases of autoimmune aetiology could easily coexist and must be considered.

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