

A few considerations should be highlighted: (i) in chronically multitransfused patients, HPA alloantibodies might be responsible for some of the FNHTRs; (ii) in our series rare HPA specificities were found, involving the HPA2 alloantigens. In this context, it could be hypothesized that the mechanisms of recognition in multitransfused patients might be different: alloantigens expressed on the CD42 protein (HPA2) might be more immunogenic in multitransfused patients than alloantigens expressed in the CD41/61 complex (HPA1 or HPA3) or in the CD49b-related antigen (HPA5), which are more frequently involved in neonatal alloimmune thrombocytopenia or in post-transfusional purpura.⁷⁻¹⁰

Key words

Alloimmunization, β -thalassemia, human platelet antigen (HPA), multitransfused patients

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Spontaneous decrease of spleen size in a patient with type 1 Gaucher's disease

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We present a patient with type 1 Gaucher's disease in whom the spleen size during 34 years of follow-up reached a maximum of 6 cm. below the costal margin, but in 1993 began to decrease spontaneously and presently can no longer be felt by abdominal palpation.

Gaucher's disease is an autosomal, recessive storage disease due to glucocerebrosidase deficiency; the spleen may increase to ten times the normal size.¹ We have treated more than 30 patients with this condition, but the patient presented here is the only one in our series in whom a spontaneous regression of the spleen size was noted.

A.M. is a 62-year-old nurse of Ashkenazi origin, a mother of two sons. In 1959, when she was 37 year-old, she was diagnosed as having Gaucher's disease following the appearance of mild purpura, petechiae and hepatosplenomegaly of 2 and 4 cm. below the costal margins. Blood examinations showed: hemoglobin 12.2 g/dL, white blood cells $4.7 \times 10^9/L$ and platelets $330 \times 10^9/L$. Serum acid phosphatase 5.5 U (normal range 0-0.8 U). Bone marrow aspiration biopsy revealed Gaucher's cells. X-ray examination was remarkable for Erlenmeyer flask deformity of the femora.

One son is a heterozygous carrier of the disease.

During the years of follow-up the size of the spleen progressively increased, reaching a maximum of 6 cm. below the costal margin. Bone biopsies performed in 1966 and 1973 showed the presence of Gaucher's cells. However, beginning in 1993, the size of the spleen progressively decreased by about one cm per year until 1996, when the spleen could be not palpated at all. Two abdominal ultrasounds and a ⁹⁹Technetium sulfur colloidal scan, showed a spleen size of 10 cm. A Doppler examination of the spleen vessels was without pathological findings. Examination of her peripheral white blood cells showed that she is a homozygote for the 1226 G variant of Gaucher's disease.

The onset of the disease, the clinical and laboratory findings and the family history of the patient are consistent with the diagnosis of adult, type 1 Gaucher's disease. The long course and almost asymptomatic presentation of the illness, exclude the possibility of myeloproliferative disorders in which pseudo-Gaucher's cells may be found. Our patient should be distinguished from those with asplenomegaly (cryp-

tic) Gaucher's disease that is a rare form, of which only a few cases have been reported. Morrison *et al.*² described 2 patients with asplenomegalic Gaucher's disease in whom the presenting symptoms were purpura and anaemia respectively and the diagnosis was established only after bone marrow examination. The authors cited two more cases of Gaucher's disease without splenomegaly. In a series of 34 patients, Matoth *et al.*³ treated only one 9-year-old patient in whom the spleen was not palpable. In our series of more than 30 cases with Gaucher's disease, we had one asplenomegalic patient. Since there is a correlation between the size of the spleen and the severity of the symptoms including the hematological findings, it is possible that some asymptomatic patients with Gaucher's disease remain undiagnosed. With the introduction of enzyme replacement therapy, a reduction of the spleen size has been reported in both type 1 and 2 Gaucher's disease.^{4,7}

In our patient, causes of the decrease in spleen size, such as infarctions and obstruction of the splenic blood vessels, were excluded on the basis of lack of clinical symptoms and normal ultrasound and isotope scan examinations. Absence of red cell pitting, Howell-Jolly and Heinz bodies excluded the possibility of hyposplenism in which the spleen function is impaired. On the contrary, although the spleen in our patient decreased to a normal size, its function remained increased, judging from the mild peripheral blood pancytopenia.

Key words

Gaucher's disease, spleen, splenomegaly

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Acute leukemias after treatment with radioiodine for thyroid cancer

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Leukemia is an uncommon complication of exposure to radioiodine (¹³¹I), used in treatment of thyroid cancer, because low doses are now used. We report two cases of acute myelogenous leukemia developed after the treatment of a thyroid carcinoma with a small dose of ¹³¹I.

Radioiodine (¹³¹I) has been used in the treatment of thyroid cancer in order to eliminate residual thyroid tissue after thyroidectomy and to treat metastatic disease.^{1,2} Leukemia is one of the most prominent late effects of exposure to ionising radiation,³ but is an uncommon complication of exposure to ¹³¹I.⁴ Most cases reported in the literature, have occurred after cumulative dosages higher than 800 mCi, but we report two cases of acute myelogenous leukemia after a small dose of ¹³¹I.

A 34-year-old woman was admitted at our hospital for anemia and a bone marrow aspirate revealed M2 acute myeloid leukemia. Chemotherapy was ordered and achieved complete remission. Three years later, the myeloid disorder relapsed and she received an autologous bone marrow, remaining in remission on date. Two years before of the leukemia, she was diagnosed papillary thyroid carcinoma, which was surgically intervened. A post-operative total body iodine scan showed cervical uptake, so she received a single dosage of 150 mCi ¹³¹I. Eight months later she had a negative whole body ¹³¹I scan. The patient's condition remained stable thereafter on a suppressive dose of thyroid hormone.

A 43-year-old female was admitted at our department for thrombocytopenia. An acute promyelocytic leukemia was diagnosed and she was treated with ATRA (all-trans-retinoic acid) and chemotherapy. She remained in remission 2 years but died later during the relapse treatment. Five years before of the leukemia, she was diagnosed of a papillary thyroid carcinoma and a partial thyroidectomy was performed. A post-operative total body iodine scan showed residual thyroid activity but no metastatic lesions. She received a dose of 150 mCi ¹³¹I and 3 subsequent follow-up scans which were normal.

It is believed that ionizing radiation can be leukemogenic. Acute leukemias have been reported after radioiodine therapy for thyroid cancer.^{3,4} The bone marrow should not receive a total dose which exceed 1000 mCi, and there should have an interval at least