Case report

Autoimmune hemolytic anemia in Philadelphia positive chronic myeloid leukemia with t(7;14) anomaly after 5 years of interferon alpha treatment

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Abstract—A 41-year-old woman, Philadelphia positive chronic myeloid leukemia patient, had progressive decline in hemoglobin levels. She had been receiving interferon alpha treatment for five years. Autoimmune hemolytic anemia was diagnosed. Subsequent bone marrow examination revealed translocation t(7;14). She was placed on prednisone treatment. The patient responded to prednisone as treatment for the hemolytic process. The result of a direct Coombs’ test remained positive. The patient died shortly after the diagnosis of autoimmune hemolytic anemia. Autoimmune hemolytic anemia should be considered in the evaluation of chronic myeloid leukemia patients with a sudden decrease in hemoglobin levels.

Key words: Autoimmune; chronic myeloid leukemia; cytogenetic abnormality; hemolysis.

INTRODUCTION

Autoimmune hemolytic anemia (AIHA) can be seen during the clinical course of a wide variety of diseases, including connective tissue disorders, infections, benign and malignant tumors and lymphoproliferative disorders [1]. AIHA is rarely seen in chronic myeloid leukemia (CML) and has been described in only a few cases [2–5]. We herein report a new case of AIHA in a Philadelphia positive CML patient associated with the cytogenetic abnormality of t(7;14) in bone marrow examination.

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CASE REPORT

A 41-year-old woman was admitted to our hospital in February 1995 with the chief complaints of abdominal pain and vaginal bleeding. She was first seen at the gynecology clinic where pelvic examination, ultrasonography and endometrial biopsy were all found normal. Complete blood count revealed leukocytosis, for which she was referred to the hematology clinic. Physical examination revealed that the liver and spleen were 5 and 3 cm palpable below the costal margin, respectively. Hemoglobin was 10 g/dl and the white blood cell count was $122 \times 10^9/l$ with 58 polymorphonuclear leukocytes, 14 metamyelocytes, 12 promyelocytes, 9 lymphocytes, 3 basophils, 2 bands and 2 eosinophils. The platelet count was $399 \times 10^9/l$. The leukocyte alkaline phosphatase score was zero. Bone marrow examination showed myeloid hyperplasia and Philadelphia chromosome was positive. A diagnosis of CML was reached.

The patient was placed on hydroxyurea and interferon alpha treatment. Her condition remained stable until February 2000, when she developed progressive weakness. She had icteric sclerae and pale mucous membranes. The spleen and liver were both 7 cm below the costal margin. Hemoglobin was 5.5 g/dl, the white blood cell count was $7.6 \times 10^9/l$ and the platelet count was $56 \times 10^9/l$. Peripheral smear was again compatible with chronic phase CML and anisopoikilocytosis was prominent in erythrocyte morphology. The corrected reticulocyte count increased to 4.2%, haptoglobin level was 13 mg/dl, indirect bilirubin was 5 mg/dl and the lactate dehydrogenase level was 2000 IU/l. A direct Coombs’ test result was positive. Bone marrow examination showed both myeloid and erythroid hyperplasia. Cytogenetic studies performed at this time revealed t(7;14) (p22;q22) at one of the 9 metaphases in the bone marrow (Fig. 1). Complete karyotype of the patient is seen in Fig. 2. The patient was placed on prednisone (1 mg/kg/day), hydroxyurea and interferon alpha (9 000 000 U/day) treatment. Five units of 500 ml washed red blood cells were transfused. One week after the start of prednisone treatment, hemoglobin level stabilized around 9 g/dl, corrected reticulocyte level decreased to 1.2%, indirect bilirubin and lactate dehydrogenase levels returned to normal. The result of Coombs’ test was still positive. Two weeks later, the

Figure 1. Cytogenetic analysis of the bone marrow of the patient disclosing t(7;14) (p22;q22).