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Acute Myeloblastic Leukemia with Erythrophagocytosis and ABO Blood Type Change

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Abstract A 61-year-old Japanese male developed acute myeloblastic leukemia in which ABO blood type change and erythrophagocytosis by leukemic myeloblasts were simultaneously observed. His blood type was AmB at the time of diagnosis, and then changed to AB in association with hematological remission. Erythrophagocytosis by leukemic blasts also disappeared. Accompanied with relapse of AML, erythrophagocytosis reappeared, though his blood type remained AB. We speculate that these rare phenomena occured incidentally and they are causally unrelated each other.

Key Words : Acute myeloblastic leukemia, ABO blood type change, erythrophagocytosis

Introduction

It is well known that erythrophagocytosis by malignant cells is one of characteristic features in malignant histiocytosis. This rare phenomenon is also documented in lymphoma,[1] leukemia,[2-5] and plasma cell dysclasia.[6-8]

ABO blood type is known to be changed in some malignant cases.[9-15] We have already reported a case of acute myeloblastic leukemia (AML), in which these phenomena were simultaneously observed.[5] Recently, we experienced another case of AML (M2) with similar phenomena. The simultaneous appearance of these two phenomena seemed to be incidental.

Case report

A 61-year-old Japanese male with chief complaints of fever, nonproductive cough and petechiae was admitted to the Onoda Red Cross Hospital in September 22, 1987. He had been healthy and had not received blood transfusions previously. Physical examination revealed no significant findings except for petechiae on legs and trunk. There were neither hepatosplenomegaly nor lymphadenopathy. His peripheral blood examination showed hemoglobin 12.9 g/dl, platelet count $19 \times 10^9/_1$, and leukocyte count $8.9 \times 10^9/1$ with 67% leukemic myeloblasts, 2.0% promyelocytes, 1.0% myelocytes, 10.0% band neutrophils, 2.0% segmented neutrophils, 4.0% eosinophils, 3.0% monocytes and 11.0% lymphocytes. The bone marrow aspiration revealed a markedly hypercellular marrow with 34.4% leukemic myeloblasts, 4.8% promyelocytes, 2.0% myelocytes, 2.6% metamyelocytes, 4.8% band neutrophils, 1.2% segmented neutrophils, 3.0% eosinophils, 6.8% lymphocytes and 0.4% monocytes. The megakaryocytes were reduced in number and erythroid precursor cells were still reserved. Although

morphological features of erythroid precursor cells showed no abnormalities, the degranulated mature neutrophils were occasionally seen in the bone marrow. The blasts were uniformly positive with peroxidase stain and negative with PAS stain. They were stained with naphthol AS-D chloroacetate esterase but not with alpha-naphtyl butylate esterase stain. In Wright stain, the blasts had large nuclei with a fine chromatin pattern and a few nucleoli. Cytoplasm of the blasts was stained basophilic, occasionally containing azurophilic granules and Auer rods. Erythrophagocytosis by leukemic blasts was seen in about 0.1% of blasts in the bone marrow. Each blast contained one mature erythrocyte in their cytoplasm (Fig.1), but neither platelets nor granulocytes were engulfed.

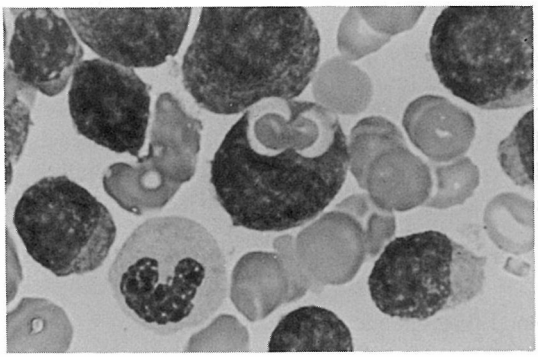

Fig.1 Erythrophagocytosis by a leukemic myeloblast and a degranulated neutrophil in the bone marrow are shown.

His blood cells agglutinated normally with anti-B sera, anti-A+B sera and anti-H sera, but not with anti-A sera. His sera did not agglutinate A, B and O blood type cells at all. Unexpected antibodies were not detected. His saliva was a non-secretory type. A family study of the blood type disclosed that his wife, son and daughter were all A type. From these findings, it was concluded that his blood type was very close to AmB. The level of lysozome in serum and urine was within normal limits. Direct and indirect Coombs' tests were negative. Analysis of karyotype revealed 46+XY.

BHAC-DMP combination chemotherapy (behenoyl-arabinofuranosylcytosine, daunorubicin, 6-mercaptopurine and prednisolone) resulted in complete remission at 36th hospital day. During induction therapy, he was given nine units of AB type packed red cells without hemolytic reaction. Erythrophagocytosis by leukemic blasts disappeared in association with hematological remission. His blood type was AB at 55th day after the final blood transfusion. Three months later, erythrophagocytosis by leukemic blasts reappeared in the bone marrow with a relapse of AML. His blood type remained AB, though agglutinated particles of his erythrocytes with anti-A sera were small in size. Thereafter, the patient has been taking a course of smouldering leukemia, and needed frequent blood transfusions of red cells and platelets.

Discussion

The blood group change is a rare phenomenon, observed in some cases of leukemia.[2-4,16] These changes are acquired, and occured often in the A antigen of ABO blood groups.[16] Our patient's blood type was AmB at the time of diagnosis, and then changed to AB in association with hematological remission. His original blood type was estimated to be AB. Briefly, his blood type change occured with the development of leukemia, and then reverted to the original blood type in association with hematological remission. The mechanism of the blood group changes still remains unknown. There are, however, a few interesting reports which proposed a chromosome abnormality,[16] a genetic mutation,[17] or alteration of serum glycosyltransferase enzymes[18] as a possible mechanism of this rare phenomenon. Kuhns et al. demonstrated that activities of H enzyme were low in patients with acute leukemia prior to chemotherapy and reverted to normal activity at the time of clinical remission, but it became low again with clinical relapse.[18] Unfortunately, serum glycosyltransferase enzymes were not determined in our case. The determination of these enzymes might contribute to resolving the mechanism of the blood group changes.

Erythrophagocytosis by leukemic cells was noted in our patient. Gori-Bergamini and Spremolla emphasized the rarity of this phenomenon which was observed only in one of 54 cases with acute leukemias.[2] Phagocytic activity in vitro has been demonstrated in various kinds of leukemic blasts, mainly in monocytic origin.[19,20] Erythrophagocytosis by hematological malignant cells is recog-

nized in a limited number of cases of malignant lymphoma,[1] acute myeloblastic leukemia,[2,5] acute lymphoblastic leukemia,[3] prolymphocytic leukemia,[4] plasma cell leukemia[5] and multiple myeloma.[7,8] The mechanism of erythrophagocytosis by leukemic cells is not clarified at all. Some authors have suggested that erythrophagocytosis is related to prolonged chemotherapy,[3] or acquired ability through process of dedifferentiation of neoplastic cells in lymphoproliferative disorders.[4] The underlying mechanism of this phenomenon in our case was not clear.

In conclusion, ABO blood type change and erythrophagocytosis by leukemic blasts were simultaneously noted in a patient with AML (M2), but we could not find a cause-and-effect relationship between these phenomena.

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