WEAKENING OF ANTIGEN D AND DEVELOPMENT OF ANTI-fl IN A PATIENT WITH ACUTE MYELOID LEUKAEMIA

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Blood groups are inherited in accordance with the principle of Mandel[1] and they remain unchanged throughout the life[2]. Change in blood group antigen or antibody content may occur in cases of malignancy so that grouping of such patients occasionally present some problems. Sometimes there is a lack of the autoantibodies (Anti A and/or Anti B) because the patient has little or no gamma globulin in his serum. Occasionally more antibody is formed, e.g. anti-B has been found in A, B. Sometimes an excess of blood group substances occur in the serum which may lead to the neutralisation of the anti A and B grouping sera, unless the patient's red cell are carefully washed several times. Also it is common to find antibodies especially in case of leukaemia[3]. The commonest change in red cell is that they become poly agglutinable due to the exposure of the T antigen on the cell antiT occurring in all adult human sera. Other changes which may occur are reduction in strength of the A agglutinogen, the acquisition of pseudo B antigen by a cell and alternation in strength of the Rh — antigen[4]. It is also possible to find antibodies specific for D antigen in person whose red cells are D positive. Such antibodies are negative with a very few D positive person including the individuals in whom the antibodies are formed. Itis suggested the D antigen is a complex one and these antibodies are specific for different part of it. It is, when a part of D antigen is missing in a particular individual that a antibody to the missing fraction may be formed. So far four different fractions with corresponding antibodies have been found RhA, Rh and RhD. Most D positive individuals possess all the fractions so they do not form the antibodies[5].

CASE REPORT

A 20 year old boy was diagnosed to have acute myeloid leukaemia, his blood group was O + vein June 1988. From June 1988 to December 1988 he had received eleven transfusion of positive blood. Each time he was tested and found to be O + ye. In the end of December 1988 his blood failed to agglutinate with anti-D and thus was declared 0-ye. On 16/1/1989 his blood was tested at blood transfusion service, JPMC for blood grouping where it was found non-reactive with potent anti-D sera on slide agglutination technique but on test tube technique the cell appeared sticky microscopically. Patient's red blood cell treated with anti-D showed weak agglutination with anti human globulin serum. Serum of the patient demonstrated cold antibodies which appeared at 4°C and 12°C but disappeared at 37°C. Antibodies specificfor D red cellswere also foundintheserum of the patient which were non-reactive to patients own red cell. The rhesus genotype of the patient appeared cde/cde. Patient's father's blood was 0-ye with rhesus-genotype cde/cde and mother's blood group was B +ve with most likely genotype cDe/cde or cDe/cde.

DISCUSSION

Change in blood group antigen or antibody content in diseases may present particular problem in blood grouping. Therefore blood grouping and cross matching particularly of patients with malignancies must

be done carefully on each transfusion. All requests for blood grouping must accompany a brief account of the patient's illness and report of previous blood grouping test if any.

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