APPARENT CHANGE OF RHESUS BLOOD GROUP TYPING IN A CASE OF ULCERATIVE COLITIS

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ABSTRACT

An interesting case of ulcerative colitis with an apparent change of Rhesus blood group typing is described. To our knowledge, this has not been reported before. We postulate that during the initial active phase of ulcerative colitis, an unknown D-like antigen, possibly bacterial in origin, could temporarily give rise to a Rhesus D-positive blood group typing in a patient with Rhesus D-negative blood type. Interestingly, with continuous immunosuppressive therapy for ulcerative colitis, the patient did not develop anti-D antibodies despite multiple transfusions with D-positive blood.

Keywords: Rhesus D-antigen, ulcerative colitis

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INTRODUCTION

The effect of some diseases in altering the existing antigens or acquiring a new antigen in the ABO blood group system is well known. In leukaemia an apparent weakening of the A, B and H antigens have been described[1]. The acquired B antigen has been observed in some A_1 individuals with carcinoma of the colon[1]. However, a change of Rhesus D antigen status in diseases is relatively rare. We describe an interesting case of ulcerative colitis with an apparent change of Rhesus D antigen typing.

CASE REPORT

A 27-year old single, female Sikh was diagnosed to have ulcerative colitis in April 1990 by Barium enema, sigmoidoscopy and rectal biopsy. She presented with frequent bloody diarrhoea and anaemia which necessitated blood transfusion.

She had no previous record of her blood group and had no blood transfusion in the past. Her blood group was typed as group B by forward and reverse typing using the slide test[2]. Her Rhesus blood group was typed as D-positive by slide testing[2] using Rhesus anti-D reagent from Biotest Diagnostics with auto (reagent), positive and negative controls. The direct Coombs test was negative. In addition, she had anti-Le^a, anti-Le^b and anti-P_1 antibodies. She received a total of 13 units of group B Rhesus D-positive, Le^{a-b-} blood over a period of 4 months. Her blood group was typed on 8 separate occasions and each time the blood group was counterchecked. She was consistently typed as group B Rhesus D-positive.

Upon diagnosis in April 1990 she was started on treatment with high dose prednisolone of 60 mg daily and Tab sulphasalazine 1 gm three times a day. Tab azathioprine 100 mg daily was added in July 1990 when she failed to achieve remission. By September 1990 she was in remission, and her prednisolone and azathioprine were reduced to 20 mg daily and 50 mg daily respectively. Sulphasalazine was continued at the same dosage.

In October 1990 she presented with anaemia (haemoglobin 7.2 gm/dl). Her blood group this time was consistently typed as group B Rhesus D-negative on 3 separate occasions using the same type of anti-D reagent and method as before. The possibility of a Rhesus (D^u) variant, a weak form of D antigen, was excluded by a negative D^u test. This was performed by an indirect anti-globulin technique after incubating the patient's cells with anti-D serum[2]. Direct Coombs test was negative. Her Rhesus genotype was cde/cde. Interestingly, her anti-D anti-body was not identified despite previous multiple transfusions with Rhesus D-positive blood. The anti-Le^a and anti-P_1 has now disappeared, leaving only anti-Le^b. The Rhesus genotype of her parents were CDe/cde and cDe/cde, both D-heterozygous.

DISCUSSION

There was an apparent change of Rhesus D-positive to a Rhesus D-negative blood type in this patient, 6 months after the diagnosis of ulcerative colitis. The frequency with which her blood group was typed and checked virtually ruled out any human error. However, she has never had her blood group typed before. It will therefore be difficult to ascertain whether she was originally Rhesus positive and became Rhesus negative consequent to her disease or that she was actually Rhesus negative but became positive during the active phase of her disease until she was treated. Both her parents were heterozygous for the D antigen, and this did not help to resolve the issue.

Change of Rhesus D-positive to D-negative blood group has been reported in myelofibrosis[3] and myeloid metaplasia[4]. This change in Rhesus status has been attributed to chromosomal mutation in chromosome 1, on which the Rhesus gene is known to be located. Drugs given to this patient, namely prednisolone, sulphasalazine and azathioprine, were not shown to cause chromosomal mutation in man. The disease itself, ulcerative colitis, is also not known to be associated with mutation in human chromosomes.

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In ulcerative colitis, a host of bacteria organisms were incriminated in its pathogenesis, including Escherichia coli, Aerobacter aerogenes, Proteus, Alcaligenes fecalis, the Friedlander bacillus, staphylococci, Pseudomonas aeruginosa, and fecal streptococci[5]. Numerous Gram-negative bacteria, especially Enterobacteriaceae, contain immunogenic blood group substances[6]. Human erythrocytes were shown to be readily sensitised in vitro by blood group active protein-lipopolysaccharides from E. coli[7]. A possible mode by which erythrocytes of this patient with severe ulcerative colitis acquire new antigens is through the fixation of bacterial substances to their surfaces. Apart from bacteria organisms, various protein antigens and macromolecules are absorbed from the gastrointestinal tract in inflammatory bowel disease[8,9] which could possibly coat the red cells. It is therefore possible that some of these antigenic substances could mimic rhesus D antigen. Anti-erythrocyte antibodies have been reported in ulcerative colitis[10]. However, the Coombs test will be positive, and may cause a false positive reaction with anti-D reagent in the slide test. This is not the case in this patient as her Coombs tests were repeatedly negative.

We postulate that in this patient, during the initial active phase of ulcerative colitis, an unknown D-like antigen, possibly bacterial in origin, reacted with the anti-D typing reagent, giving rise to the Rhesus D-positive blood group. When the disease was vigorously treated by immunosuppressive therapy including prednisolone, azathioprine and sulphasalazine, the D-like antigen disappeared after 6 months and the patient became Rhesus D-negative. Interestingly, as she was on continuous immunosuppressive treatment, she did not develop anti-D antibodies despite previous multiple transfusions with Rhesus D-positive blood. Incidentally, out of her initial 3 natural occurring anti-bodies, namely anti-Le^b, anti-Le^a and anti-P_1, the latter two disappeared after 6 months of treatment.

This case brings to the awareness that an apparent change in Rhesus blood group typing may occur in a case of ulcerative colitis.

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