The Turkish Journal of Pediatrics
Volume 23, No. 2, April - June 1981

ACQUIRED B-LIKE ANTIGEN*

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Key words: acquired (B-like) antigen, bacterial infection, blood group antigen

In 1959, Cameron et al[1] described the first examples of acquired B antigen. Since then, some 50 cases of acquired B antigen in elderly individuals of group A blood type have been published[2-10]. This paper presents an additional case of acquired B antigen in a young woman with intestinal perforation, peritoneal abscesses and sepsis due to E. coli.

Material and Methods

A standard tube technique was employed using 2% prewashed red cell suspensions. Incubation periods for titration and absorption tests were two hours at room temperature. Titration results were scored with the values recommended by Dunsford and Bowley[11] (++++ or C, = 12, +++ = 10, ++ = 8, + = 5, (+) = 3, gw = 2, w = 1). A regular heat elution method was performed for the elution studies. For inhibition tests, various salivas were added to equal volume and 1/2 diluted sera and eluates. After 15 minutes at room temperature, suitable red cells were added in parallel.

Case Report

G.E., a 27-year-old married woman, was first admitted to Hacettepe University Hospital on March 15, 1971, with abdominal pain and sterility. Pelvic inflammatory disease and anemia were diagnosed. She was grouped as A Rh positive and one unit of A Rh positive blood was transfused with no ill effects.

On September 11, 1973, she was admitted to the hospital for the second time with nausea, vomiting, abdominal pain and distension. An urgent laparotomy was indicated and a blood transfusion needed. Difficulty occurred during routine preoperative blood grouping tests. She was grouped as AB Rh positive, with anti-B in her serum. After additional tests, she was accepted as being primarily A Rh

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positive, with an acquired B antigen in her red cells. Six units of group A Rh positive blood were transfused during and after laparotomy. Intestinal perforation, peritonitis and multiple abscesses in the pelvis were observed during the operation, and an ileal resection and anal ileostomy were performed. The patient died 5 days after the operation. A. aerogenes and E. coli were grown from the pus from the peritoneal cavity, and E. coli was grown from the hemoculture.

Results

Serological studies: The patient's red cells reacted with anti-A, anti-B and anti-AB from a group-0 donor and weakly with her own serum at 4°C. Reaction was negative at room temperature. This was attributed to a weak non-specific cold autoagglutinin. Reactions with four group-AB sera showed a slight degree (+) of polyagglutinability. Direct antiglobulin test was negative (Table I).

TABLE I

REACTIONS OF THE PATIENT'S RED CELLS

	Anti-A	Anti-B	Anti-AB	D. Coombs
G.E. Red Cells	+++	++	+++	—

The patient's serum reacted with B and AB red cells, but not with A and 0 cells (Table II).

TABLE II

REACTIONS OF THE PATIENT'S SERUM

	A cells	B cells	0 cells	AB cells
G.E. Serum	—	+++	—	+++

In order to show B-like antigen on the patient's red cells, an anti-B test serum (from Dade Reagent Inc.) in the titre 1/512 with normal B cells was absorbed with the patient's red cells. The same anti-B serum was absorbed with normal A and B cells in the same fashion. Absorptions were carried out with equal volumes of sera and cells. After absorptions, each specimen of serum was titrated against normal B cells. Because of the inadequate specimen from the patient, only one absorption could be carried out (Table III).

It is seen that the patient's red cells and normal B cells reduced the titration scores of anti-B serum. After absorption of anti-B serum with the patient's cells, a heat elution method was carried out. Eluted antibody from the patient's cells reacted

TABLE III

RESULTS OF THE ABSORPTION STUDIES OF ANTI-B SERUM WITH G.E. RED CELLS AND CONTROL RED CELLS

	Anti-B serum	
	Titration with normal B cells	Scores
Before absorption	512	76
After absorption with G.E. cells	128	44
After absorption with normal B cells	64	37
After absorption with normal A cells	512	74

with B and AB cells, but not with A and 0 cells. These absorption and elution tests showed that the patient's cells reacted specifically with anti-B serum and yielded an eluate with anti-B specificity.

Inhibition studies: As will be seen below, inhibition effects of the different salivas on the reactions of anti-B serum, the patient's serum and the patient's eluate with the different cells were detected during these tests.

As can be seen in Table IV, reactions between anti-B and the patient's (G.E.) cells and normal B cells were inhibited by B-containing saliva.

TABLE IV

INHIBITION OF THE REACTIONS BETWEEN ANTI-B AND G.E. CELLS AND B CELLS BY DIFFERENT SALIVAS

	G.E. cells	B cells
Anti-B serum	+ +	+ + +
Anti-B + B secretor saliva	—	—
Anti-B + A secretor saliva	+ +	+ + +
Anti-B + B non-secretor saliva	+ +	+ + +

Tables V and VI show that reactions between G.E. serum and eluate and B and AB cells were strongly inhibited by B secretor saliva, but not inhibited by A secretor or B non-secretor saliva. Accordingly, red cells of the A-group patient reacted with anti-B sera, absorbing the anti-B to their surface and then yielding an anti-B eluate. It is obvious that red cells of the group-A patient had an additional and acquired

TABLE V

INHIBITION OF G.E. SERUM WITH A AND B SECRETOR AND B NON-SECRETOR SALIVAS

	A cells	B cells	0 cells	AB cells
G.E. serum	—	+ + +	—	+ +
G.E. serum + B secretor saliva	—	—	—	—
G.E. serum + A secretor saliva	—	+ + +	—	+ +
G.E. serum + B non-secretor saliva	—	+ + +	—	+ +

TABLE VI

INHIBITION OF ELUTED ANTIBODY FROM G.E. CELLS AFTER ANTI-B ABSORPTION

	A cells	B cells	0 cells	AB cells
G.E. eluate	—	+ +	—	+ +
G.E. eluate + B secretor saliva	—	—	—	—
G.E. eluate + A secretor saliva	—	+ +	—	+ +
G.E. eluate + B non-secretor saliva	—	+ +	—	+ +

B-like antigen on their surface. Serological studies of the patient were cut short by her death a few days later. Saliva samples from the patient and a comprehensive family history for genetic studies could not be obtained. Incubation of normal group-A cells with concentrated E. coli suspension for 24 hours at 37°C did not produce any change in the serological behavior of the group A cells.

Discussion

The acquired B antigen is in fact a pseudoantigen and is found exclusively in patients of the A blood group. It is most often observed in the course of a bacterial infection, frequently associated with digestive tract pathology, and disappears after recovery.

In spite of its rarity, acquired B antigen is one of the various problems encountered in blood grouping. The peculiarity of the blood group in these patients is recognised during processing before a possible transfusion. A and B antigens are known to be

affected and weakened by some blood diseases, but such damage to an existing antigen is easier to understand than the acquisition of an antigen.

Some cases that may have been examples of inheritance of a weak B antigen in group A_1 subjects have been reported[12]. But in acquired B antigen cases, B-like antigen is transient and of an acquired nature. All the reported patients were group A with a normal anti-B in their serum, with the main peculiarity being that the red cells were agglutinated by anti-B sera. This reaction of the red cells with the anti-B serum is not as strong as those of normal group B cells.

Marsh et al[3] and Marsh[4], who published an acquired B-antigen case, were able to induce a similar change in vitro by treating A_1 cells with a powerful T-activating bacterial enzyme. In this manner, T-activating enzyme-treated normal red cells would take up anti-B and then yield an anti-B eluate. Marsh and his colleagues concluded that the B-like antigen was probably acquired through the action of a bacterial enzyme. They called the antigen pseudo-B.

It is known that extractions from some organisms can make the red cells agglutinable by appropriate bacterial antibody, and a substance from some micro-organisms can transform the red cells and render them agglutinable by normal sera[13].

Again some organisms — including pneumococci, streptococci, Staph. albus, aureus and citreus — can render the red cells polyagglutinable by activating T-receptors on the red cells. Furthermore, B.cereus and pseudomonas can cause false positive antiglobulin reactions[13].

It has been shown that there is a close serological relationship between E. coli 086 and blood group B antigen[14]. Although attempts have been made to locate this organism in the feces of some patients with acquired B antigen, they have not been successful[14].

Stratton and Renton[5] suggested that the change might be due to absorption of B-like bacterial polysaccharides on to the red cells. Later, Springer and Ansell[6,7] were able to show that some purified lipopolysaccharides and protein-lipopolysaccharides from E. coli 086 and some other organisms could be absorbed on to the A and 0 red cells, giving them a B-like antigen and rendering them agglutinable by anti-B sera.

There is a question concerning this reaction. If E. coli 086 polysaccharide is responsible, why does it adhere to the red cells in vitro but not in vivo? An answer to this question has not yet been found.

It is generally accepted that the acquisition of a B-like antigen by group A people may be associated with age or disease, since the phenomenon has not been reported previously in younger patients. There is no doubt that the change could not occur in young or middle-aged healthy people, or it surely would have been recognized long ago in blood donors[1-3]. Most of the published reports are of

patients over 60 years of age, and all the patients were group A. The absence of group 0 persons among the cases is highly significant[15].

Gerbal et al[8] suggested that the fact that the acquired B phenomenon is observed only in group A individuals may be explained by supposing that it is due to deacetylation of N-acetylgalactosamine on the red cells of group A people.

A limited number of coli samples are capable of causing the acquired B phenomenon. This capability may be the same for the other bacterial varieties.

Occurrence of the B reactivity in a group A patient may be the result of a bacterial esterase activity. Probably, the activity of a specific bacterial deacetylase transforms the A reactive N-acetylgalactosamine into galactosamine: this sugar would give a cross-reaction with galactose, the immunodeterminant sugar of the B specificity[9,10]. Such a deacetylase has been shown by Yamamoto and Iseki[16] in Clostridium Tertium A, and by Roseman[17] in the E. coli K12.

Two possibilities may be considered for the rarity of the observed cases of acquired B antigen: a) Most of the A persons are able to destroy the modified red cells having acquired B antigen, and b) Most of the coli strains involved in infections, especially of the digestive tract, probably lack suitable deacetylase[18].

To our knowledge, our patient is the youngest reported in the literature. This first example suggests that the phenomenon can happen as a result of disease to young people as well. E. coli may cause a B-like antigenic change in vivo. The present case adds a further example of this phenomenon associated with perforation in the intestinal system, peritoneal abscesses and sepsis due to E. coli.

Weak reactions with anti-B sera in a sample of red cells may be explained by transfusion of group AB blood into group A persons. Akeroyd and O'Brien[19] have shown that small numbers of AB cells may survive for some weeks after transfusion into group A individuals.

As can be seen from the history, our patient had not received a doubtful transfusion before admission.

Summary

A young woman of the A blood group, with digestive tract pathology and sepsis due to E. coli, showing the acquired B antigen peculiarities is reported.

(Editor's note: Although the case in question does not belong to the pediatric age group, its significance lies in the fact that it exemplifies the condition in a young adult, with the possibility that it may occur in adolescence as well. For this reason the paper is presented in this journal.)

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