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Case Report

Clinical Cases of Bacterial and Fungal Sensitization in Patients with Coxarthrosis and Gonarthrosis depending on Blood Group Differences

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ABSTRACT

Aim: To analyze bacterial and fungal sensitization in persons that have ability to absorb anti-A and anti-B antibodies incompatible with blood group type.

Material and methods: 8 people had A blood group, 2 persons had B blood group, 22 persons showed O blood group type. 9 patients with O blood group and 2 patients with B blood group absorbed anti-A antibody. 2 patients with O blood group and 5 patients with A blood group absorbed anti-B antibody. 7 persons with 0 blood group without absorbing activity of anti-A, 4 persons with O blood group and 3 persons with A blood group without anti-B absorbing activity served as control groups. Leukocyte migration inhibition reaction was performed to estimate bacterial and fungal sensitization.

Results: Persons with anti-A absorbing ability were characterized by increased antibodies, sensitization to *Penicillium, Candida Albicans, Pseudomonas aeruginosa, E. Coli and Clebsiella pneumoniae*, increased CIC as compared to those without anti-A absorbing ability. Patients with anti-B absorbing ability showed decreased spontaneous leukocyte migration inhibitory factor production, *sensitization* to *Streptococcus pneumoniae* and Staphylococcus aureus by cell type, sensitization to *Candida Lusitaniae* by humoral type. Persons with anti-A absorbing ability as compared to the persons with anti-B absorbing ability showed sensitization to *Penicillium, Candida Albicans*, while the persons with anti-B sensitization revealed sensitization to Streptococcus pyogenes. During incubation of erythrocytes with the heated polyclonal anti-A (and anti-B) and complement at room temperature the erythrocytes of the persons with corresponding anti-A (anti-B) absorbance were increased in size and agglutinated by sides.

Conclusion: Anti-A and anti-B absorbing activity of erythrocytes is associated with different fungal and bacterial sensitization.

Keywords: Absorption; Anti-A antibody; Agglutination; Erythrocyte; Sensitization

INTRODUCTION

Incompatible expression of blood group A and blood group B antigens, deletion of blood group substances and precursor blood group H antigen accumulation was described in cancer tissues. The tumors were found to express ABO antigens which were incompatible with the patient's blood type. Antigenic changes in hematological malignancies most commonly involve ABO blood group system [1]. Blood group discrepancies are defined as non-corroboration between cell grouping and serum grouping. Variable erythrocyte antigenic expressions give rise to subgroups owing to heterogeneity [2].

The phylogeny of ABH determinants shows that they appeared as tissue antigens much earlier than as red cell antigens. They are found in ectodermal and endodermal epithelial cells in lower mammals, whose red cells are completely devoid of ABH antigens. Only humans express ABH antigens on red cells.

Relatively little is known concerning differences in cell wall A, B antigens of erythrocytes and water-extractable antigens of the tissues. Similarly, a different ability exists to absorb polyclonal anti-A or anti-B and to be agglutinated by polyclonal antiserum [3]. By describing the cases of discrepancies in agglutination of erythrocytes and their absorbing ability, it hopes to contribute to

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the understanding of the difference between cell and tissue level of A,B,H antigens localization and to the elucidation of A and B antigens role in immune and host defence system.

The aim of the study: to reveal immunological parameters including specific sensitization to bacterial and fungal antigens in persons with the only anti-A or anti-B absorbing, but not agglutinating ability of erythrocytes.

MATERIAL AND METHOD

Persons with coxarthrosis and gonarthrosis aged 25-80 were examined on their ability to absorb anti-A and anti-B antibodies. Among them 8 persons had A blood group, 2 persons had B blood group, 22 persons showed O blood group type. Bacterial and fungal sensitization was analyzed in persons that have the ability to absorb anti-A and anti-B antibodies incompatible with blood group type. 9 patients with O blood group and 2 patients with B blood group absorbed anti-A antibody. 2 patients with O blood group and 6 patients with A blood group absorbed anti-B antibody. 7 persons with O blood group without absorbing activity of anti-A, 4 persons with O blood group and 3 persons with A blood group without anti-B absorbing activity served as control groups.

Immunological investigation included the study of autoimmune antibodies (ALA), granulocytotoxic antibodies (AGA), circulating immune complexes (CIC). Leukocyte migration inhibitory factor was assessed in reaction of leukocyte migration inhibition with bacterial (*Pseudomonas Aeruginosa, Escherichia Coli, Staphylococcus epidermalis, Staphylococcus aureus, Clebsiella pneumoniae*), fungal (*Candida Lusitaniae, Penicillium*) and tissue antigens (bone, cartilago, synovial membrane).

To reveal anti-A and anti-B absorbance of erythrocytes we performed absorption of erythrocytes with polyclonal serum for 1 hour at 37°C and subsequent hemagglutination of test erythrocytes with adsorbed serum.

Incubation of erythrocytes at room temperature (RT) with heated polyclonal serum and complement (C') for 12 hours was performed to estimate the diameter difference of erythrocytes under the influence of absorbing serum.

Hemagglutination with polyclonal serum at 37°C after absorption of the serum with bacterial and fungal pathogens for 1 hour at 37°C was performed in order to estimate the activity of anti-A and anti-B serum after absorbance with bacterial and fungal pathogens. Correlation analysis was performed by Spearman. Statistical analysis was performed by Statistica 10.0. Differences at $p<0.05$ were considered as significant.

RESULTS

The cases of ability of erythrocytes to absorb anti-A and anti-B antibodies not corresponding to their blood group type were analyzed.

Persons with anti-A absorbing ability (the first group) were characterized by sensitization to *Candida Lusitaniae* (MI 0.83 ± 0.04), *Penicillum* (MI 0.83 ± 0.04), *Pseudomonas Aeruginosa* (MI 0.85±0.1), *Clebsiella Pneumoniae* (MI 0.79 ± 0.04), decreased nonspecific production of LIF (MI 1.36 ± 0.2), increased level of ALA (22.14 ± 5.8), CIC (105.68 ± 11.0), sensitization by hymoral type to serum (MI 1.19 ± 0.2) and by cell type to synovial membrane (MI 0.88 ± 0.2), bone (MI 0.82 ± 0.31), cartilago (MI 0.85 ± 0.1) (Table 1).

The first group as compared to the second group (without anti-A ability of erythrocytes to absorb anti-A) was found to show sensitization to *Candida Lusitaniae, Pseudomonas Aeruginosa, Clebsiella Pneumoniae, Penicillum, E. Coli*, increased AGA (7.25 ± 2.2 %), CIC (105.68 ± 11.0 units).

Correlation analysis by Spearman revealed positive association of MI to *Candida Lusitaniae* with MI to *Pseudomonas Aeruginosa* ($r=0.43$, $p<0.05$) and opposite association with MI to serum ($r=-0.46$, $p<0.01$), AGA ($r=-0.3$, $p<0.05$). MI to Penicillum positively correlated with MI to serum ($r=0.58$, $p<0.01$), MI to *Pseudomonas Aeruginosa* was positively associated with ALA ($r=0.8$, $p<0.01$), MI to serum ($r=0.68$, $p<0.01$), MI to *Streptococcus pygenes* ($r=0.42$, $p<0.05$), MI to *Candida Lusitaniae* ($r=0.43$), MI to *Staphylococcus epidermalis* ($r=0.64$, $p<0.05$).

Table 1: Immunological parameters of patients with anti-A and anti-B absorbing capacity of erythrocytes.

	Group 1 Absorbance of anti-A	Group 2 No absorbance of anti-A	Group 3 Absorbance of anti-B	Group 4 No absorbance of anti-B
LIF	1.36 ± 0.2	1.48 ± 0.2	1.24 ± 0.2**	1.71 ± 0.2
ALA, %	22.1 ± 5.8	21.1 ± 5.4	22.88 ± 5.82	16.6 ± 4.0
AGA, %	7.25 ± 2.2	5.0 ± 2.0	7.2 ± 2.3	6.58 ± 1.7
MI to *Staphylococcus aureus*	0.89 ± 0.1	0.78 ± 0.1	0.88 ± 0.04**	0.78 ± 0.04
MI to S	1.19 ± 0.2	1.23 ± 0.2	1.25 ± 0.3	1.25 ± 0.3
MI to *Clebsiella pneumoniae*	0.79 ± 0.04*	0.93 ± 0.06	0.88 ± 0.1	0.92 ± 0.2
MI to *Streptococcus pyogenes*	0.98 ± 0.2	0.91 ± 0.2	0.82 ± 0.04**	0.9 ± 0.06
MI to Penicillum	0.83 ± 0.04*	1.02 ± 0.1	1.03 ± 0.1**	0.74 ± 0.1
MI to *Candida Lusitaniae*	0.83 ± 0.04*	1.03 ± 0.06	1.00 ± 0.05**	0.81 ± 0.03
MI to *Pseudomonas Aeruginosa*	0.85 ± 0.18*	1.43 ± 0.1	0.99 ± 0.1	1.08 ± 0.2
CIC, units	105.6 ± 11.0*	77.2 ± 8.0	89.08 ± 8.4	87.3 ± 8.3
MI to *Staphylococcus epidermalis*	0.97 ± 0.2	1.12 ± 0.4	0.94 ± 0.1	0.89 ± 0.1
MI to E. Coli	0.91 ± 0.06*	1.18 ± 0.06	0.9 ± 0.3	0.88 ± 0.1
MI to synovial membrane	0.88 ± 0.2	0.98 ± 0.1	0.97 ± 0.2	0.87 ± 0.2
MI to bone	0.82 ± 0.3	0.99 ± 0.2	1.19 ± 0.1	1.27 ± 0.1
MI to cartilage	0.85 ± 0.1	0.78 ± 0.1	0.9 ± 0.1	1.35 ± 0.3

Note: MI: Migration index; LIF: Leukocyte migration inhibitory factor; ALA: Autoimmune lymphocytotoxic antibodies; AGA: Autoimmune granulocytotoxic antibodies; CIC: Circulating immune complexes; S: Serum;*: $p < 0.05$ as compared to group 2; **: $p< 0.05$ as compared to group 4

Persons with anti-B absorbing ability (the third group) not corresponding to blood group type were characterized by sensitization to *Streptococcus Pyogenes* (MI 0.82 ± 0.04), *Staphylococcus aureus* (MI 0.8 ± 0.04), *Clebsiella Pneumoniae* (MI 0.88 ± 0.1), decreased production of LIF in the presence of serum (MI 1.25 ± 0.3).

Persons with anti-B absorbing ability of erythrocytes (the 3 group) as compared to the 4 group (without anti-B absorbing ability of erythrocytes) were characterized by decreased production of LIF (MI 1.24 ± 0.2), decreased LIF production to *Candida Lusitaniae* (MI 1.0 ± 0.05), *Penicillium* (MI 1.03 ± 0.1), *Staphylococcus aureus* (MI 0.88 ± 0.04), *Staphylococcus epidermalis* (MI 0.94 ± 0.1) and increased LIF production to *Streptococcus Pyogenes* (MI 0.82 ± 0.04).

MI to *Streptococcus Pyogenes* showed direct association with MI to synovial membrane ($r=0.67$, $p<0.01$), MI to *Staphylococcus aureus* ($r=0.5$, $p<0.05$), MI to *E.Coli* ($r=0.4$), opposite association with CIC ($r=0.62$, $p<0.01$). MI to *Penicillum* was positively associated with MI to serum ($r=0.71$, $p<0.01$), MI to *Clebsiella pneumoniae* ($r=0.84$, $p<0.01$), MI to *Candida Lusitaniae* ($r=0.84$, $p<0.01$). MI to *Candida Lusitaniae* showed direct association with MI to *Staphylococcus aureus* ($r=0.64$, $p<0.01$), MI to serum ($r=0.46$, $p<0.01$), MI to *Penicillium* ($r=0.84$, $p<0.01$), MI to *E.Coli* ($r=0.65$, $p<0.01$).

While comparison of two groups with different anti-A and anti-B absorbing ability sensitization to *Candida Lusitaniae, Penicillium* was found in persons with anti-A absorbing ability of erythrocytes and sensitization to *Streptococcus Pyogenes* (MI 0.82 ± 0.1) was found in persons with anti-B absorbing ability of erythrocytes. The majority of values of MI in patients with anti-A absorbance were found less than in control group, while in persons with anti-B absorbance MI to the studied antigens exceeded the values of control group.

In the study of erythrocyte diameter change after loading with heated polyclonal antibody and complement the diameter of erythrocytes of the person with anti-A absorbing activity: 5.1, 5.5, 5,7 μm in 0.9 % NaCl (Figure 1), whereas after loading with the heated anti-A and complement: 6.5, 7.0, 8.0 μm (Figure 2). Without anti-A absorbing activity diameter of 0 erythrocytes: 5.9, 5.8, 7.3, 8.6 μm in 0.9 % NaCl (Figure 3) and at room temperature 6.9, 6.4, 6.1 μm (Figure 4) after contact with heated anti-A with complement (no increase in size).

The diameter of erythrocytes of the person with anti-B absorbing activity at room temperature: 5.9, 6.6, 7.4 μm in 0.9% NaCl (Figure 5); at the room temperature after loading with heated anti-B and

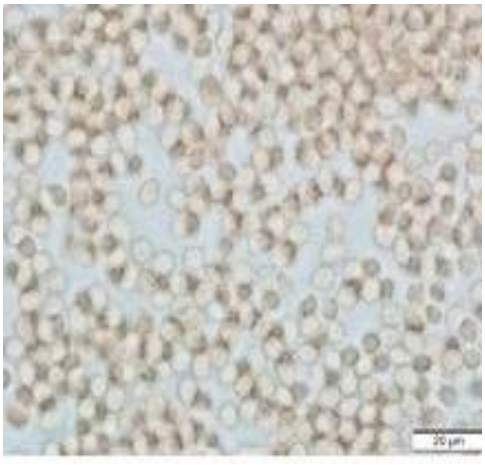

Figure 1: B er., 0.9% NaCl at 37°C (B person with anti-A absorbance).

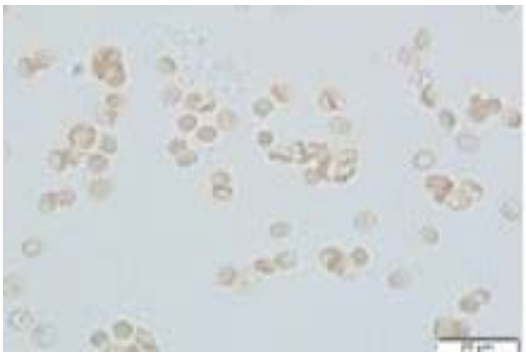

Figure 2: S anti-A heated, B er., C at 0.9% NaCl at 37°C (B person with anti-A absorbance).

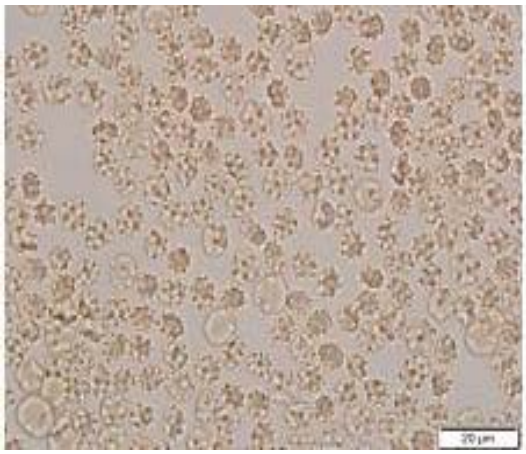

Figure 3: 0 er., 0.9% NaCl at RT (0 person).

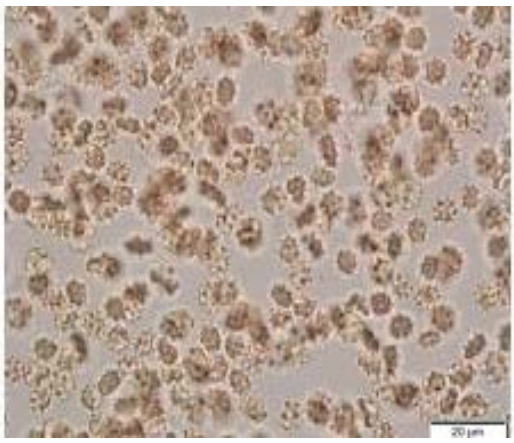

Figure 4: 0 er., heated, anti-A,C at RT (0 person).

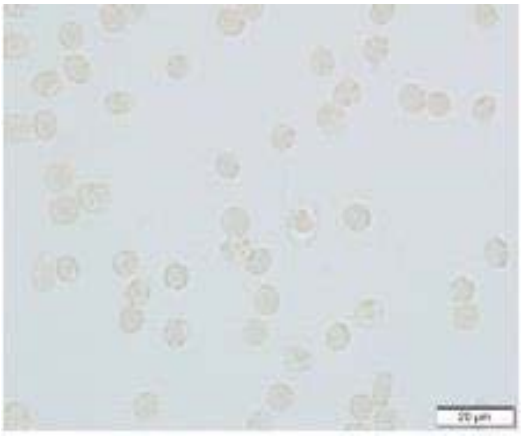

Figure 5: A er., 0.9% NaCl at RT (A person with anti-B absorbance).

complement: 6.7, 6.2, 7.6, 8.4 μm (Figure 6), erythrocytes are increased in size.

During incubation of erythrocytes with the heated polyclonal anti-B and complement at room temperature the erythrocytes were increased in size and agglutinated by sides. Diameter of erythrocytes without anti-B absorbing activity after loading with heated anti-B and complement: 3.7, 4.5, 4.7 μm.

As positive control for anti-A absorbing ability diameter of AB erythrocytes in 0.9 % NaCl at 4°C: 6.8, 7.2 μm and after loading with heated anti-A, B and complement at 4°C: 7.2, 8.9, 10.5 μm and 5.3, 8.4, 8.7 μm at room temperature.

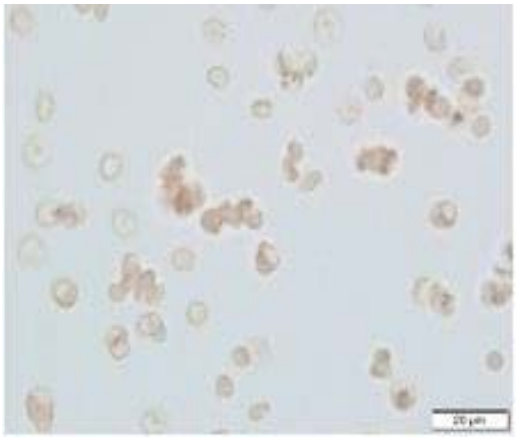

Figure 6: A er., heated, anti-B,C at RT (A person with anti-B absorbance).

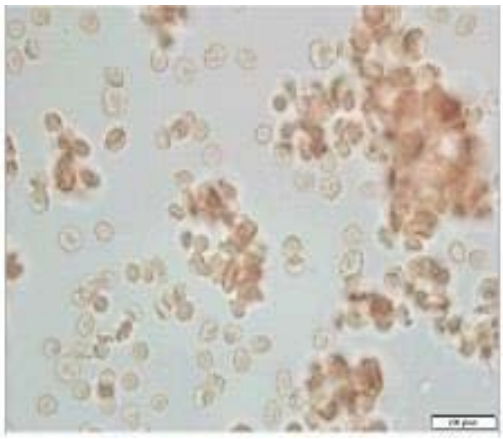

Figure 7: S anti-A heated, 0 er., C at Room Temperature (0 person with anti-A absorbance).

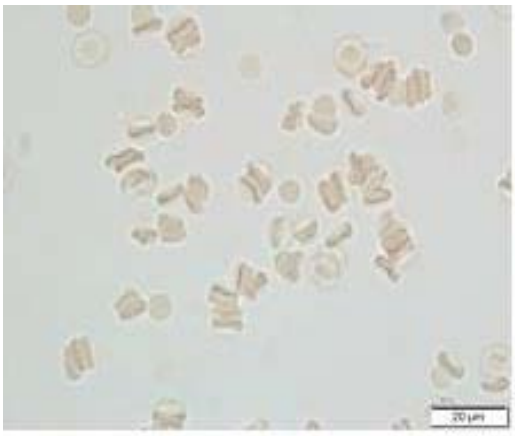

Figure 8: S anti-A,B heated, 0 er., at Room Temperature and complement (0 person with anti-A absorbance).

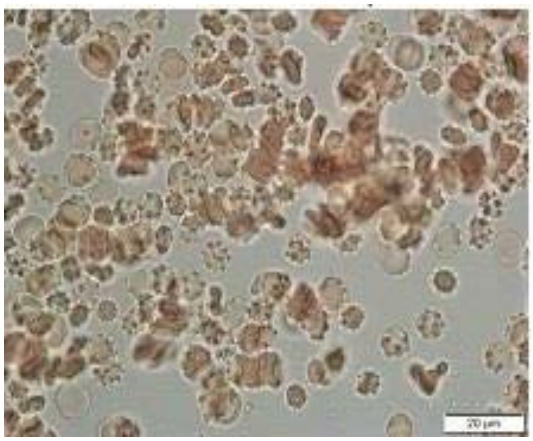

Figure 9: S anti-A, heated,B er., C at RT and complement (B person with anti-A absorbance).

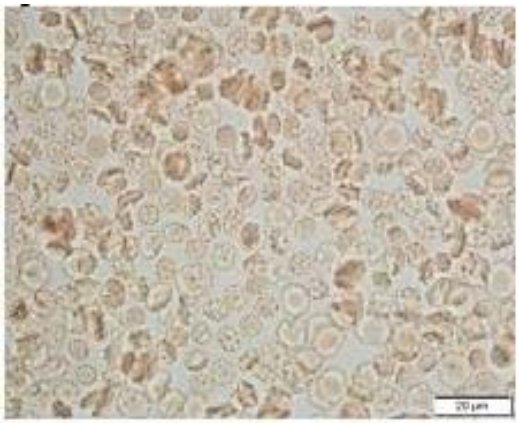

Figure 10: S anti-B, heated, A er., C at Room Temperature (A person with anti-B absorbance).

The size of erythrocytes increases and shades of erythrocytes appear.

Diameter of 0 erythrocytes with heated AB serum and complement as for negative control: 5.5,5.2,7.3 μm (no increase in size). Diameter of A erythrocytes in 0.9% NaCl: 6.4,5.6,7.2 μm, with heated anti-A and complement at room temperature: 4.8, 6.7,7.5,8.3 μm (increase in size), with heated anti-B and complement: 4.5,5.0,7.0,7.4 μm with heated serum without anti-A and anti-B and complement: 5.3,4.6 μm (no increase size). Diameter of another A erythrocytes at room temperature in 0.9% NaCl: 5.4, 5.0, 5.2 μm, after heated anti-A and complement: 8.0, 7.4, 7.6 μm (the size is increased).

Thus, the heated serum and complement with concordant specificity increases the erythrocyte size.

The erythrocytes of the persons after contact with the heated polyclonal antibodies at 37°C and at room temperature are presented in figures.

Persons with anti-A absorbing ability at room temperature: Figures7-9. Persons with anti-B absorbing ability at room temperature: Figures 10-12.

The study of inhibition of hemagglutination by bacterial and fungal pathogens with anti-A polyclonal antibody showed inhibition of hemagglutination of A erythrocytes by *Candida Lusitaniae* (on 2 degrees), *Pseudomonas Aeruginosa* (on 1 degree) (Table 2).

In a study of inhibition of hemaglutination with anti-B by bacterial and fungal pathogens *Candida Lusitaniae, Clebsiella pneumoniae* and *Bacillus cereus* (*B. cereus*) showed the strongest anti-B absorbing ability (Table 3). *Clebsiella pneumoniae* (on 2 degrees), *Streptococcus*

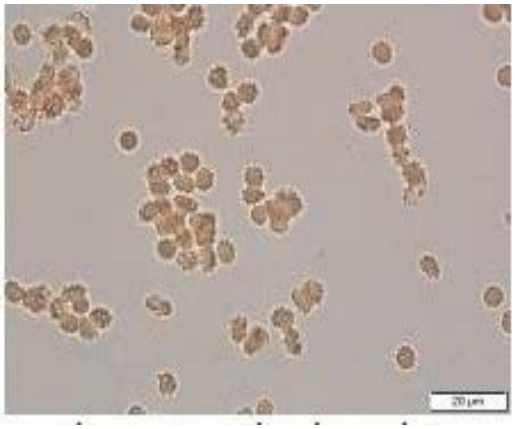

Figure 11: S anti-B, heated, A er., C at Room Temperature (A person with anti-B absorbance).

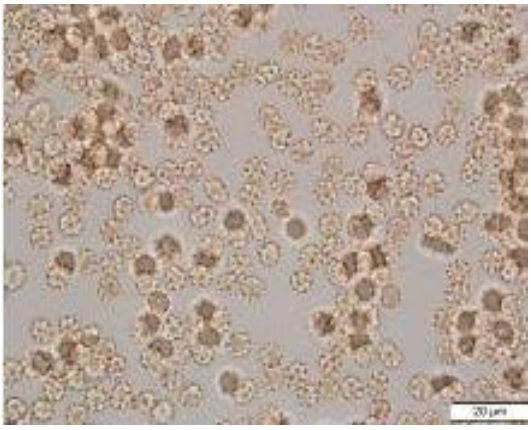

Figure 12: A er., 0.9% NaCl.

Table 2: Inhibition of hemagglutination with anti-A by bacterial and fungal antigens.

Titer of the serum	1:2	1:4	1:8	1:16	1:32	1:64	1:128
S anti-A and A er.	+	+	+	+	+m	-	
S anti-A after *Candida Lusitaniae* and A er.	+	+	+m	+m	-	-	-
S anti-A after *Pseudomonas Aeruginosa* and A er.	+	+	+	+m	-	-	-
S anti-A after *E. Coli* and A er.	+m	+m	+m	+m	-	-	-
S anti-A after *B. cereus* and A er.	+	+	+m	+m	-	-	-
S anti-A after *St. aureus* and *A er.*	+	+	+	+m	-	-	-
S anti-A after *St. epidermalis* and *A er.*	+	+	+m	+m	-	-	-
Note: S: Serum; St.: Staphylococcus							

Table 3: Inhibition of hemaglutination with anti-B serum by bacterial and fungal antigens.

Titer of the serum	1:2	1:4	1:8	1:16	1:32	1:64	1:128
S anti-B and B er.	+	+	+	+	+m	-	
S anti-B after *Clebsiella pneumoniae* and B er.	+	+	+m	-	-	-	-
S anti-B after *Streptococcus pyogenes* and B er.	+	+	+	+	~	-	-
S anti-B after *Candida Lusitaniae* and B er.	+m	+m	-	-	-	-	
S anti-B after *Pseudomonas aeruginosa* and B er.	+	+	+	+m	-	-	-
S anti-B after *B. cereus* and B er.	+	+	+m	-	-	-	-
S anti-B after *Proteus vulgaris* and B er.	+	+	+m	+m	~	-	-
Note: er: Erythrocytes; S: Serum; B: Bacillus							

pyogenes (on 1 degree), *Candida Lusitaniae* (on 3 degrees), *Pseudomonas aeruginosa* (on 2 degrees), *Proteus vulgaris* (on 2 degrees), *Staphylococcus epidermalis* (on 1 degree), *B. cereus* (on 2 degrees), *E. Coli* (on 1 degree) showed inhibition of anti-B hemagglutinating activity.

DISCUSSION

Microbiota has been found to regulate antogony of transferases acting on production of blood group substances [3]. Aberrant fucosylation plays important role in all aspects of biology [4,5]. Different strategies are used to detect blood group type, including absorbtion studies [6,7].

Association of bacteria and fungi with A and B antigens absorbing ability is of interest for the discussion of microbial influence on A and B antigens of erythrocytes. Thus, beta-D-galactosidase of *E.Coli* is known to affect on D-galactose, which is a substrate of B antigen. The researchers have discovered destruction of group antigens by enzymes of bacterial and fungal origin [8,9]. A-blood group activity of *Pseudomonas Aeruginosa* and *Clebsiella* was reported by authors [10].

In *E.Coli*, *Proteus vulgaris*, *Str. Pyogenes* the molar ratio of galactosamine (A antigen) has been calculated very small, however in *Staphylococcus aureus* 5% of hexosamine occurred as galactosamine. Galactosamine (the main component of A antigen) has been found in the cell walls of *Ps. Aeruginosa*. The ratio of muramic acid to galactosamine in Ps. Aeruginosa was found to be 1.0:0.7 and that might indicate that in this species galactosamine may be a major structural component of the cell wall [11-14].

In the study we also obtained sensitization by *Ps. aeruginosa* and not by *Proteus Vulgaris*, *Str. Pyogenes, E. Coli* and *Staphylococcus aureus* in patients with anti-A absorbing capacity of erythrocytes.

Interestingly, *Streptococcus faecalis* was shown to have galactosidase activity, which degrades A substance, thus converting N-acetylgalactosamine into D-galactose [15]. In our study majority of persons with anti-B absorbing ability had A blood group and sensitization to *Streptococcus Pyogenes*.

Spontaneous destruction of group antigens occurs in saliva and feces at 37°C. The question has not been settled whether this

destruction is due to bacterial enzymes or to an enzyme produced by the organism. Two conceptions have been suggested. In the first instance phenomenon is due to organic enzymes and saliva provoked by irritation of parasympathicus contains very little enzyme as against saliva provoked by irritation of sympathicus. Another conception is based on microbial influencer.

There is evidence that the host's AB0 blood type and secretor status affects the specificity of blood group-degrading enzymes produced by the host's fecal bacteria in vitro. Fecal cultures containing blood group substances are a feasible source for purifying blood group antigen-degrading enzymes.

The destruction of blood group specific substances by microorganisms has been recently described. A mycobacterium was found effective against the group specific substance A. Schiff observed the destruction of blood group specific substances by B. Welchii. There is notion, that bacteria produce blood group ABH-degrading enzymes. Chain degradation appeared to be dependent on extracellular but not cell-bound b-galactosidase or b-N-acetylglucosaminidase [14,15].

CONCLUSION

The presence of anti-A absorbing ability of erythrocytes was associated with sensitization to *Candida Lusitaniae*, *Penicillium*, *Pseudomonas Aeruginosa*. *Pseudomonas Aeruginosa* has been reported to contain A antigen (galactosamine). Candida Lusitaniae showed anti-A absorbing ability. Anti-B absorbing ability was associated with sensitization to *Streptococcus pyogenes, Staphylococcus aureus, Clebsiella Pneumoniae.Clebsiella Pneumoniae* showed anti-B absorbing ability.

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