Letter to the Editor

Molecularly confirmed para-Bombay (Ah) secretor phenotype with *FUT1* c.328G>A and *FUT2* c.357C>T mutations: first case report from India

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Dear Editor,

The H antigen is essential for ABO expression and is synthesized by *FUT1* on red cells and *FUT2* in secretions. Mutations in both genes cause the Bombay phenotype. Para-Bombay individuals exhibit absent or weak red cell H antigen due to mutation in the *FUT1* gene; para-Bombay secretors have H substance in their secretions due to an active *FUT2* gene, whereas nonsecretors lack it entirely. Both variants can cause blood group misinterpretation and pose life-threatening transfusion complications due to clinically significant anti-H antibodies.

We have received a request for one unit of packed red blood cells (PRBC) for a 35-year-old multigravida patient with severe anemia, documented as blood group O RhD positive. A blood group discrepancy was identified during pretransfusion testing. The patient was O RhD positive on cell grouping and A group on reverse grouping using the column agglutination technique (CAT) (Tulip Diagnostic, Panaji, Goa, India). A detailed history revealed that her historical blood group was the Bombay phenotype, and she has received six units of Bombay group PRBCs without experiencing any adverse transfusion reactions in the past.

On performing advanced immunohematological tests, blood group was interpreted as para-Bombay (Ah) secretor with clinically insignificant anti-H antibody. Results of immunohematology workup are summarized in **Table I**. Based on the immunohematology workup results, one unit of saline and AHG phase compatible O RhD-positive PRBC was transfused without any adverse reaction. This is consistent with a previous report indicating that, in the absence of Bombay group PRBCs, transfusion with ABO-compatible units can be undertaken safely when anti-H is clinically insignificant[1]. To test the inheritance pattern, available family members were tested, and none had the para-Bombay phenotype.

Further patient's blood samples were sent to the National Institute of Immunohematology (NIIH) in Mumbai for genotyping. Genomic DNA extracted from peripheral blood and coding region *FUT1* and *FUT2* genes were amplified by the Polymerase Chain Reaction method and further analysed by Sanger sequencing, revealing homozygous mutation of c.328G>A in *FUT1* and c.357C>T in *FUT2*. A homozygous *FUT1* c.328G>A mutation was associated with weak H antigen expression in red blood cells and the FUT1*01w.02 allele[2]. The patient also had a homozygous *FUT2* c.357C>T mutation, which does not impact the enzyme's amino acid sequence and preserves ABH secretion[3]. These results confirmed the para-Bombay secretor phenotype. This *FUT2* variant has been observed in general

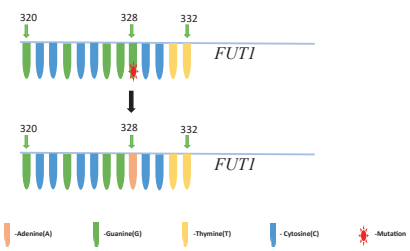
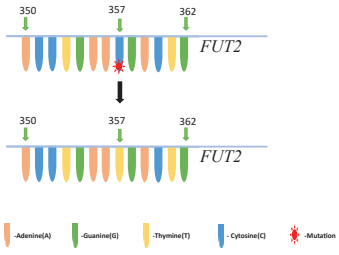
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populations from Southeast Asia; however, the associated allele for *FUT2* c.357C>T mutation is not listed in the ISBT 018 H allele database[2]. Similar studies include those of Anukul *et al.*, who identified four individuals

Table I - *Summary of immunohematological workup*

Blood grouping											
		Cell grouping						**Serum grouping**			
Method		**Anti-A**	**Anti-B**	**Anti-AB**	**Anti-D**	**A1 lectin**	**H lectin**	**A cell**	**B cell**	**O cell**	**Auto control**
CAT		Neg	Neg	Neg	4+	-	-	Neg	4+	Neg	Neg
CTT	**IS**	Neg	Neg	Neg	4+	Neg	Neg	Neg	4+	Neg	Neg
	4°C	-	-	-	-	Neg	Neg	2+	4+	2+	Neg
	37°C	-	-	-	-	Neg	Neg	-	-	-	Neg
Inference		**O Rh D positive**						**A group**			
Coombs test											
Method		DCT						ICT			
CTT		Negative						Negative			
Inference		No sensitization of RBC						No allo antibody			
Adsorption and elution test											
Method		A antigen						H antigen			
CTT		No agglutination						No agglutination			
Inference		A antigen not detected						H antigen not detected			
Test for specificity of cold autoantibodies											
Method		Adult O cell (I)		Cord O cell (i)		Adult A1 cell		Bombay cell		Autologous cell	Enzyme treated adult O cell
CTT	**RT**	Neg		Neg		Neg		Neg		Neg	Neg
	4°C	2+		Neg		2+*		Neg		Neg	Neg
Inference		Anti-H antibody reacts at 4°C									
Salivary inhibition test											
Method		A substance				B substance				H substance	
CTT		No agglutination				Agglutination present				No agglutination	
Inference		A substance present				Absence of B substance				H substance present	
Lewis phenotyping											
Method		Anti -Le^a						Anti -Le^b			
CAT		Negative						Positive			
Inference		Le(a-b+) indicating ABH secretor status									
Molecular genotyping											
Method		*FUT1* gene						*FUT2* gene			
Sanger sequencing		Homozygous mutation in c.328G>A 						Homozygous mutation in c.357C>T 				
Inference		H+ weak						Secretor			
Interpretation: Ah Rh D positive para-Bombay secretor. *Reaction due to cold reactive anti-H antibody.											

Neg: negative, RT: room temperature.

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with similar *FUT1* and *FUT2* mutations in Thailand, as well as additional reports by Zelan Gu *et al.* in China and Kim *et al.* in Korea, in an Indonesian patient[3-5].

In conclusion, we report the first molecularly confirmed para-Bombay secretor phenotype with homozygous mutation of c.328G>A in *FUT1* and c.357C>T in *FUT2* from India, which highlights the importance of incorporating molecular blood typing into routine serological testing for patients with rare blood groups and to detect these novel alleles with mutations.

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The Authors declare no conflicts of interest.

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