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Evaluation, Exploration and Management Strategies of First Reported Case of Para-Bombay Blood Phenotype with E-Beta Thalassaemia from Bangladesh

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Abstract:

Human H/h genetic polymorphism in ABO blood group system is rare and evidenced with Bombay and Para-Bombay blood group. This patient a 27-year-old-young man having 'E-Beta thalassaemia' incidentally has been identified as a case of 'Para-Bombay phenotype' after exploring with proper blood grouping, antibody detection and saliva inhibition study. Previously he was diagnosed as 'O' RhD positive and had two events of transfusion reaction during 'O' positive blood transfusion. In literature search and browsing there is dearth information on Para-Bombay Phenotype with E-Beta Thalassaemia. So far to our knowledge, this is the first reported case from Bangladesh of patient having 'E-Beta Thalassaemia' evidenced with rare 'Para-Bombay blood Phenotype'. This study explores the laboratory work-out on Para-Bombay Phenotype and evaluates treatment strategies on this phenotype with concomitant E-Beta Thalassaemia.

Key Words: Anti-H, Bangladesh, Bombay blood type, E-Beta Thalassaemia, 'O' cell, Para-Bombay Phenotype, Rare blood group registry, Saliva inhibition Study, Secretor.

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Introduction:

Human H/h genetic polymorphism was first reported in Bombay now known as Mumbai, India in 1952 by Bhende et al. [1-3] For the genesis of blood group, the presence of H antigen, which is a precursor carbohydrate from which A and B groups are formed, its presence on the RBC membrane and in body secretions is determined by H and Se (secretor) blood group loci, respectively, which code for distinct Fucosyl transferase (FUT) enzymes-FUT1 and FUT2. [2, 3] FUT1 gene causing silences the gene or affects the efficiency of the encoded 2-alpha-fucosyltransferase. While, H antigen absent in saliva of Bombay phenotype, interestingly, in Para-Bombay type saliva may have H antigen.[4-7] Thus, the Classical Bombay group showed lack of both H and secretor gene function with genotypic expression of hh /sese; whereas, persons with hh/ SeSe or hh/Sese represented as Para-Bombay phenotype, which may have weakly reactive H transferase due to mutation at the H locus or lack H antigen on RBC, but possess it in secretions, are known as para-bombay secretors or RBC H negative secretors. [5-8] Thus after the discovery of ABO blood groups by Karl Landsteiner, various studies showed, H antigen is the precursor for the formation of A and B antigens and O antigen has full presence of H antigen, while, its absence is known as H antigen deficient phenotype and known as Bombay or Para-Bombay blood group.[2, 5, 7-9] Para- Bombay phenotype showed characteristics of deficiency of H, A and B antigens on the red cells. [8] They inherit hh/Sese or hh/SeSe genes. However, they have little or no A, B and H antigen on RBCs, while that has been found to be present in secretions with greatly reduced level of fucosyl transferase enzyme and thus known as "Para-Bombay secretors" or "red blood cell (RBC) H negative secretors". [1,5,7,9] In this way, they differ from "classical Bombay phenotype" where, individuals lack both H and secretor gene function with genotypic expression of hh/sese, while RBCs and

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secretions lack the H antigen. [5, 7, 8] Thus the relationship between ABO, Hh, Sese (secretion) and Lele (Lewis) gene play vital role in the genesis of ABO (ABH) blood group system. This case report focuses on the association of ABO (ABH) and Lewis blood group system and secretor and non secretor status to evaluate "Para-Bombay type with concomitant E-Beta Thalassemia" (Photograph- I and Photograph- II)

Case report:

This patient of 27 years, young non-diabetic Muslim male was diagnosed as a case of hereditary haemoglobinopathy "E-Beta Thalassaemia" at his age of 12 years. During that time due to history of epistaxis and pallor, he was transfused with one unit of "O Rhesus (Rh) D positive" RCC to correct anaemia and it was eventless. Since then, he was relatively well under folic acid supplement. Gradually in last 3 years he developed general weakness, fatigue, pallor and pain in the left hypochondriac region and was advised to transfuse 3 units of packed red cells. This time during blood transfusion, he developed moderate transfusion reaction with two units of blood. With the complaints of moderate anaemia and mild jaundice he admitted in Haematology Department of Bangabandhu Sheikh Mujib Medical University (BSMMU) for further management. On evaluation, He was moderately anaemic with low haemoglobin (Hb) - 7.2gm/dl (13.0-16.0 g/dl), Mean Corpuscular Volume (MCV) -61.3fl (normal range 76-96 fl), Mean Corpuscular Haemoglobin (MCH)-18.3pg (range 27-32 pg), Mean Corpuscular Haemoglobin Concentration (MCHC) -29.9g/dl (range 31.5-34.5g/dl), and increased Red cell Distribution Width (RDW): 33.3% (11.6-14.0%). However, his Erythrocyte Sedimentation Rate (ESR) was 8 mm at the end of 1st hour and Red blood cells (RBC) 3.93 x 10^{12} / L showed normal result. White blood cells (WBC) profile was within normal range with total leukocyte count: 8x 10^9/l, Neutrophil-70%, Lymphocyte: 24%, Monocyte: 04% and Eosinophil: 02%. Moreover, platelet count was normal, 170 x 10 9 /L. Peripheral blood film showed, features of haemolytic anaemia as red blood cells showed gross anisopoikilocytosis with hypochromia along with schistocyte, target cells, nRBC (nucleated red blood cell), pencil and teardrop cells; but white blood cells were mature with normal distribution and platelets were adequate with normal morphology. Haemoparasites were not seen. He was diagnosed as haemoglobin E-beta thalassaemia (Photograph- II) prior with electrophoretic pattern of HbA- 6.5%, HbF- 47.6% and Hb-E- 45.9% by Haemoglobin Electrophoresis (For normal adult : HbA- 96% to 97%, HbA2- 2.2 to 3.3 % , HbF – 0.2 to1%) method on 1% agarose gel at alkaline pH (8.6) while, Capillary Electrophoresis report showed (estimation carried out by Sebia fully automated capillary system) HbA- 4.0%, HbF- 49.7%, HbE- 41.4%, HbA2- 4.9% . His plasma glucose, Lactate dehydrogenase (LDH), ferritin, creatinine were within normal limit. His serum bilirubin was high: 3.4 mg/dl (0.2-1.0 mg/dl) but other parameters of liver function test and renal function tests were normal. Although his serum cholesterol was low 42 mg/dl (normal range: 120-200mg/dl) and his ultra-sonogram study showed splenomegaly with fatty change in liver , while his hepatitis B virus surface antigen (HBsAg) showed negative result. In this situation, for correction of his anaemia, he has been referred to the Transfusion Medicine Department of BIRDEM (*Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine and Metabolic Disorders*) General Hospital and also to the Transfusion Medicine Department of BSMMU for arrangement of new blood units. Blood grouping was performed using standard serological technique of Tube method using saline and Coomb's antiserum.[3, 4, 5]

What was the dilemma and how it was solved?

In BIRDEM, at the Transfusion Medicine Department, ABO and Rhesus blood grouping done. No agglutination was observed in forward grouping with A, B and AB antisera (BioRad, Cressier, Switzerland and Tulip Diagnostics, Goa, India), but agglutination was noticed with D antiserum. So there is no detectable ABO antigen on forward grouping or cell typing and thus mimics 'O' group at room temperature and at 4^0C. However, agglutination present in Rhesus blood with Anti-D commercial reagent (BioRad, Cressier, Switzerland and Tulip Diagnostics, Goa, India) confirmed that patient as Rhesus "D" positive. In reverse grouping, stronger (4+) agglutination occurs with 'A' cell and 'B' cell, whereas, with pooled 'O' cells (control) at room temperature showed positive reaction and seen under microscope (Table-I). So dilemma occurs to see whether it is Bombay phenotype or is it H-Deficient phenotype. When at 4^0C test result showed strong (2+) reaction with pooled 'O' cells, it suggested that, the group is not 'O' blood group (Table-I) and due to anti- H like

antibody patients serum reacts at 4^0C and causes agglutination with pooled 'O' cells. So further testing with Anti-H lectin (prepared from Ulex Europaeus plant extract, Commercial, Tulip Diagnostics, Goa, India)) done and showed negative reaction with patient cells at room temperature. This revealed deficiency of H antigen on the red cell of patient. Since, a lack of A and B antigens is the 'O' type; so, the result resembled an 'O' phenotype by default but it was not a 'O' blood group and mimics as Bombay Phenotype. Then test for H-antigen done at 4^0C showed weak delayed positive result, due to H deficiency for weak H like antibody, known as anti-HI, which showed reactivity at low temperature and usually present in serum. In this situation in BSMMU same reactions got from repeat tests (Table-I). As a reference Laboratory of BSMMU, in the Transfusion Medicine Department "Secretor status" has been evaluated to see the presence of 'soluble blood group substances' in saliva. Presence of H antigen in saliva resulting the ABO blood group system of this patient as "O hm" or "O Hm" or "O Hz" phenotype of Para- Bombay blood group (Table-II) (Photograph- I). Lewis blood grouping also done in BSMMU to see the secretor status. Patients RBC for Lewis antigen showed, "Le (a-b+)", a secretor (Table-III). Further, Sensitive Adsorption –Elution test showed trace amount of ABH antigen on erythrocytes. Beside this, MN blood group of this patient was "(M-N-)" and P blood group showed "P1" positivity (Table-IV). Patients Rhesus Genotype and Phenotype test done in BIRDEM and BSMMU showed, Rhesus phenotype of "CCDee" and Probable Rhesus Genotype "CDe/CDe (R1R1)" (Table-I). So, final evaluation of the blood group of this E-Beta thalassaemia patient confirmed as "a Para –Bombay type with deficient H antigen in RBC having secretor status". His family was screened for blood group but no match with this patient.

Table-I

Reactions observed with cells and serum

Reactions	**Reagent**	**Grade of agglutination**		**Interpretation**
		At **room temperature**	At **4 ° C**	
ABO Blood Grouping (Cell Typing)	Anti-A	0	0	Mimics "O" at room temperature and at 4 ° C
	Anti-B	0	0	
	Anti-AB	0	0	
Rhesus Blood Grouping	Anti-D	4+	4+	O Rh(D) positive Rhesus phenotype of **"CCDee"** and Probable Rhesus Genotype **"CDe/CDe (R1R1)"**
ABO Blood Grouping (Serum Typing)	A_1 - cells	4+	4+	Mimics at room temperature not 'O' group ➢ Is it Bombay Phenotype ➢ **Is it H- Deficient Phenotype ? ?** Mimics at 4 ° C- not 'O' group and Anti-H like antibody detected on testing at 4 degree centigrade. ➢ **Is it "Para-Bombay"** As, H secretor with anti-H like antibody: anti-HI only reacts at 4 degree centigrade.
	B cells	4+	4+	
	O cells	1+ Positive	2+ Positive	
Test for H-antigen	Anti-H Lectin (Ulex Europaeus plant extract) (Commercial)	0 (No agglutination)	± (Weak Positive)	At room temperature revealed deficiency of H antigen on the red cell of patient at 4 ° C- **H deficient.** Weak expression of H- antigen on patient red cells showed delayed Positivity. Because, weak H like antibody, called **anti-HI** reactive at low temperature is almost always present in the serum.
	Anti-H from Oh subject	± Weak	± (Weak Positive)	

Table II

ABH in Saliva

Reactions	Reagent	Antigens in saliva		Interpretation	Report
Saliva inhibition test	Patients cell	A	-	Present in normal amount. An independent genetic system of Homozygous **zz** regulates the expression of the H gene at the site of synthesis of cellular H antigens - allows H in saliva but not on the red cells. So that is zz, Se and hence secretors.	So, ABO blood group system showed, **"O hm" "O Hm" "O Hz" phenotype (Para-Bombay)**
		B	-		
		H	+		

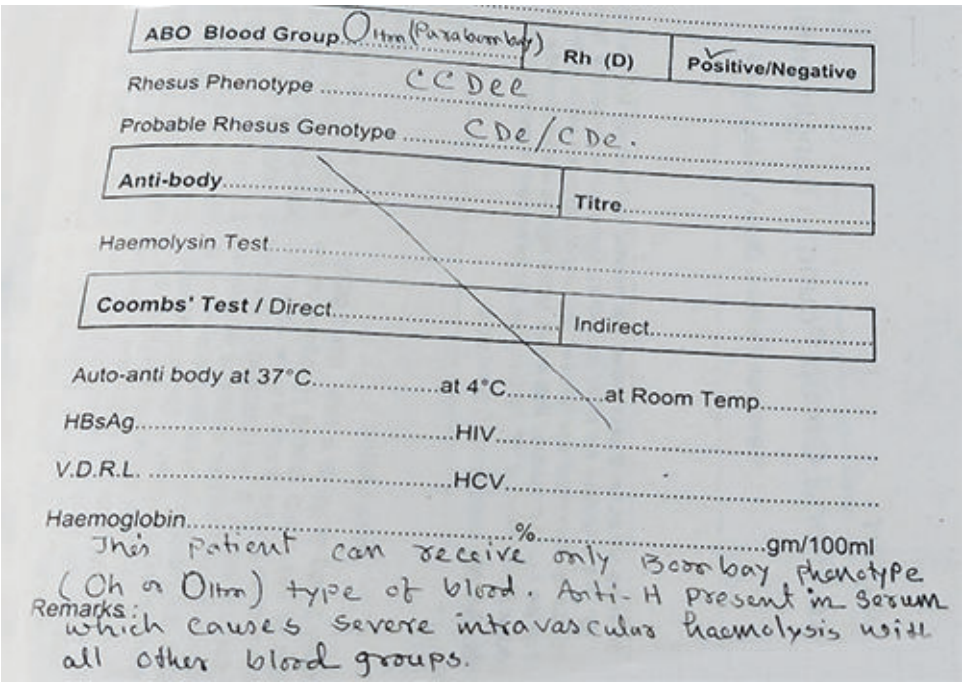

Fig.-1: *Para-bombay Phenotype of ABO Blood Group system along with Rhesus Blood Group of this patient*

Table III

Lewis blood group: secretor status

Reactions	Reagent	Phenotype	Status	Interpretation
Lewis antigen	Patients RBC	Le (a-b+)	**Secretor**	Because of their secretor status, normal levels of H substances are present in the saliva.

Table IV

Other blood groups

Minor blood group	Interpretations
MN blood group	M- N-

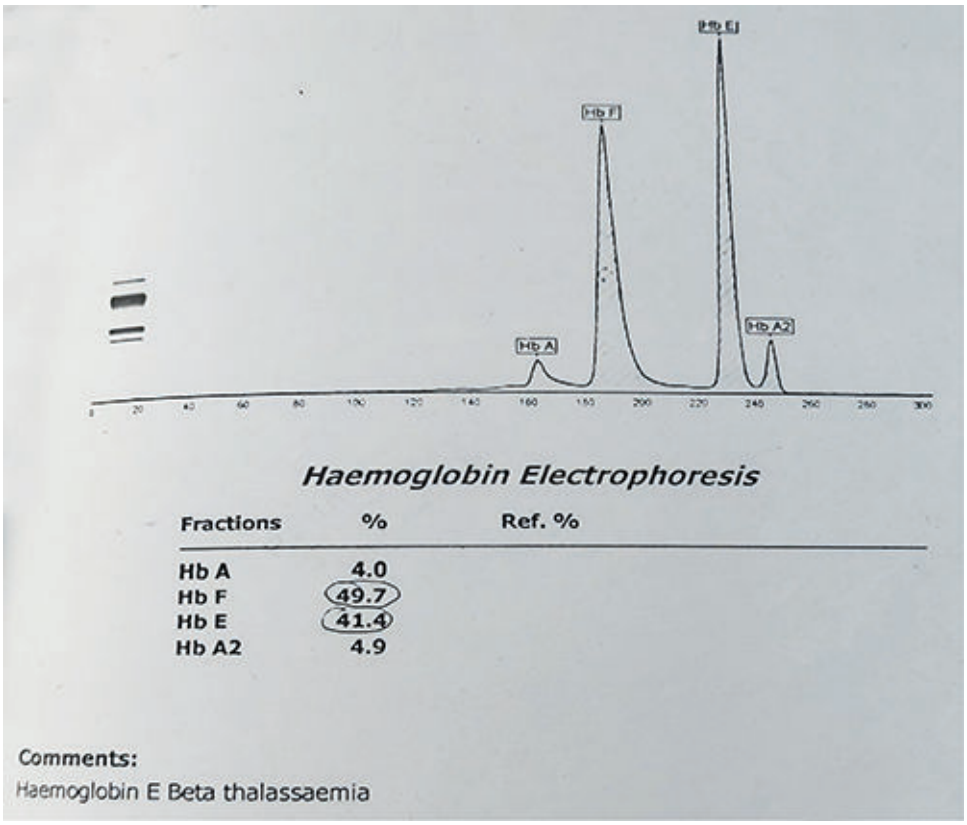

Fig.-2: *Capillary Electrophoresis showed this is a case of Haemoglobin E Beta Thalassaemia*

Decision in blood transfusion support and solution

There is no blood bank in Bangladesh for available ready stock of Bombay or Para- Bombay blood unit or no national platform for rare blood group registry. So this patient has been given oral Hydroxyurea (500 mg) once daily along with injectable Erythropoietin 10,000u subcutaneous weekly for 4 weeks and folic acid supplements once daily. This patient also received counseling about the rarity of his blood group phenotype and also advised for annual monitoring of iron overload and organ functions.

Discussion:

Spectrum of the H blood-group-deficient phenotypes showed mutation of FUT 1 (H gene) while, deletion of FUT2 (Secretory gene) found in diverse ethnic groups with Bombay Phenotype ; on the other hand, active FUT2 found among Para- Bombay .[3, 4, 9, 11] Among the Bombay phenotype in India, a high level of consanguinity has been seen.[1, 4] On the other hand, it is quite rare, 1 in 250,000 among Caucasians with 1: 10^6 prevalence in Europe, [4,9,12] while, H-deficient population in Iran is 0.0008%.[13] The incidence of the Bombay phenotype mainly confined to South-East Asian countries; [9] on the other hand, the Para-Bombay phenotypes are mostly found in Eastern Asia. [3] In India prevalence of Bombay phenotype has been estimated to be 1 in 10,000 ;[7] however, high frequency of the Para-Bombay phenotype 1 : 50 found among Lahu, ethnic minority, in China .[3] Beside this, Para-Bombay phenotype also found among Hong Kong Chinese with a frequency of 1 in 15,620, whereas, 1 in 8000 among Taiwanese population. [3] In Bangladesh, Bombay phenotype among 3 sisters (MIAH family) from Narayanganj was first reported cases documented by Rahman M et al.[14] Till now in Bangladesh, reported document showed only 40 persons had Bombay blood group. [6,15] In a conservative study over long three years in BIRDEM, among 25,119 blood donors, 0.0016% of Bangladeshi population showed H- deficient phenotypes[15] and among those three H- deficient cases one of them was a cardiac patient with Bombay Phenotype, one severely anaemic patient with Para-

Bombay phenotype having E-Beta Thalassaemia and one healthy Para-Bombay blood donor.[15] To our knowledge from the literature search, this was the first report of a 27-year-young man having E-Beta Thalassaemia along with rare Para-Bombay blood phenotype not only from Bangladesh but also not documented from any other countries of the world. Interestingly, ABH antigens on erythrocytes of all Bombay and Para-Bombay individuals were not detected by the ordinary method of hemagglutination; [2, 5, 6] so that, using a more sensitive method, the adsorption-elution test need to perform to determine trace amount of ABH antigens on erythrocytes. [2,3,7] On the other hand, Anti-H in their serum may cause severe intravascular haemolysis with all other blood groups.[2, 9] (Photograph- I) Studies showed Para-Bombay group individuals usually retain some H antigen on RBC and weak anti-H activity which is often demonstrable only at 4º C or by adsorption and elution technique. [5, 7, 8] Reverse grouping (serum typing) with O cell control, anti-H lectin and detection of secretor status all are important tools for detecting the Bombay and Para – Bombay blood type.[2, 5, 7] This case point out that, without the use of anti-H lectin, this particular E- Beta thalassaemic patient might have been labeled as group 'O', rather than Para-Bombay phenotype. So, this case emphasizes the importance of judicious use of anti-H in blood group detection.[2, 5, 7] In addition to Para-Bombay phenotype, this patient also suffering from concomitant haemoglobinopathy, known as, haemoglobin (Hb) E-beta thalassaemia , which is common in this part of world [16, 17] Hemoglobin E/beta-thalassemia results from the co- inheritance of a hemoglobin E mutation from one parent and a beta-thalassemia mutation (either beta 0 or beta +) from the other parent, although its natural history remains poorly defined. Hb E is caused by a substitution of glutamic acid by lysine at codon 26 of the ß-globin gene and these mutations correlates with the severity of the disease.[16] Worldwide, beta thalassaemia major and haemoglobin E-beta-thalassaemia (Hb E/â-thalassaemia) are about 50 per cent of those affected with severe beta thalassaemia.[16] The highest prevalence showed in the subcontinent , in India and Bangladesh; while in Southeast Asian countries like Thailand, Laos and Cambodia this is very common.[16- 18] In Hb E/â-thalassaemia, alleles inherit from haemoglobin E (Hb E) and beta-thalassaemia both.[16] As a result, throughout these regions, Hb E/â-thalassaemia stand out as an alarming public health problem.[16, 18] A conservative world health organization (WHO) report estimates that, in Bangladesh about 3.0% of populations are carriers of Beta-thalassaemia and 4.0% are carriers of Haemoglobin-E. [17, 18] The expected births of babies with Hb E beta thalassaemia are about 6, 443 while beta thalassaemia major were 1040 in Bangladesh. [19] Farhana et al [18] showed 20.72% of haematological diseases were haemoglobinopathies and among them 21.74% had beta thalassaemia major, 47.83% beta thalassaemia trait, while haemoglobin E- trait was 17.39% and haemoglobin E-disease 13.04%. [18] A study done in Bangabandhu Sheikh Mujib Medical University Dhaka showed, 30.47% cases were diagnosed as HbE/beta thalassaemia.[20] Recent studies showed treatment of these patients with Hb-F modulating agents hydroxyurea indicate 40% patients showed clinical improvement while, hydroxyurea and erythropoietin combination also showed good result. [16, 21] Here, Hb F-modulating agent causes moderate changes in hemoglobin with marked improvement in phenotype. [16, 21, 22] Till now, there is no database for the rare blood group in Bangladesh. [23] In 2021, a study of Afroz etal, found that the incidence of Bombay phenotype was 0.006% in two centres of Dhaka city.[24] Recent report showed that, in Bangladesh about 40 persons had Bombay blood group, but no report or document found on the Para Bombay phenotype in this country [6, 15, 23] Arrangement of compatible units of blood for this critical and rare E-Beta thalassaemia patient having Para –Bombay phenotype is really challenging. [2,7, 8 21] From the above evaluation this patient should be transfused with only Bombay phenotype (Oh or OHm) type of blood (Photograph- I), Para Bombay or autologous blood for transfusion, if allo-anti –H or anti- HI in their serum is clinically significant (i.e. reacting at 37^0 C). [25] There is evidence that, anti –HI act as clinically insignificant.[25] So, the patient with anti-H / anti-HI reacting at lower temperatures (4^0 C -22^0 C), if there is no availability of the Para-Bombay blood group, Anti- Human Globulin (AHG) compatible units of ABO blood groups can be transfused. [25] Various studies showed that, ABO compatible blood units tested up to indirect anti-globulin test (IAT) with pre-warmed technique which may be helpful, when Para-Bombay blood is not available. [5,7, 8, 25] But the problem happened with this patient that, he

was not suitable as an autologous candidate or none of compatible blood unit was available for him. Finally, as there is no available Para-Bombay blood donor or any compatible unit for this patient, for the benefit and better management, we treated this E-Beta thalassaemia patient with concomitant Para –Bombay phenotype prescribing oral hydroxyurea (500mg) once daily, Injectable Erythropoietin 10,000u s/c weekly for 4 weeks in addition to folic acid supplements once daily. Beside the therapeutic management, this patient had received counseling about the disease, the rarity of this phenotype, scarcity of blood products and advice was given for annual monitoring of iron overload and organ functions.

Conclusion:

This case emphasizes the importance of proper blood grouping incorporating cellular and serum typing including 'O' panel cell and Coomb's test. Use of Anti-H reagent and secretor status detection has enormous role for identification of Para-Bombay phenotype. To confirm Para-Bombay phenotype adsorption and elution test have an important role. Proper Blood group database, molecular studies, rare blood group registry and haemovigilance may solve the discrepancies and blood transfusion related complications.

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