Original article

The first Thai patient had a blood group allele that was CD36 heterozygous

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Abstract:

Introduction: *CD36, or Nak[a] (GPIV), is a glycoprotein found on platelet membranes and other cells, such as megakaryocytes, monocytes, endothelial cells, and adipocytes. It is also expressed at low levels in reticulocytes. In June 2023, the ISBT Working Party of Red Cell Immunogenetics and Blood Group Terminology (WP-RCIBGT) established CD36 as a new blood group system.* ***Objective:*** *To investigate the red blood cell (RBC) antibody, phenotype, and genotype in a thalassemia patient who required blood transfusion but could not find compatible blood that may be related to the CD36 new blood group.* ***Materials and Methods:*** *The conventional tube test (CTT) and column agglutination test (CAT) monitored red blood cell phenotype and antibody investigation. The Immucor BioArray HEA Precise BeadChipTM Kit performed genetic analyses. Next-generation sequencing (NGS) was performed using the Illumina DNA Prep with Enrichment Kit to identify variants relative to the reference sequence. The platelet antibody was performed to confirm anti-CD36 by monoclonal antibody-specific immobilization of platelet antigens (MAIPA).* ***Results:*** *The antibody investigation showed anti-E, -c, -Jk[a], -unidentified with cold autoantibody. Interestingly, the NGS result showed gene variation in CD36 (CD36) nucleotide substitution (c.380C>T), which leads to the missense amino acid [p.(Ser127Leu)] in the CD36 blood group protein. These changes were found in 191 of 409 DNA sequence reads, which are consistent with heterozygosity for c.380C>T.* ***Conclusion:*** *The new finding is that CD36 is at a heterozygous level (c.380C>T) in Thai patient. The autoantibody caused the incompatible crossmatch, not a high titer low avidity (HTLA)-related antibody.*

Keywords : ● *CD36 deficiency* ● *New blood group system* ● *Next-generation sequencing*

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นิพนธ์ต้นฉบับ

ผู้ป่วยชาวไทยรายแรกที่มีอัลลีลของหมู่เลือดเป็น CD36 heterozygous

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บทคัดย่อ

บทนำ *CD36 หรือ Nak[a] เป็นไกลโคโปรตีนที่พบได้บนผิวเซลล์เกล็ดเลือด และเซลล์อื่นๆ เช่น เมกาคาริโอไซต์ โมโนโซต์ เซลล์เยื่อบุผนังหลอดเลือด เซลล์ไขมัน และพบการแสดงออกได้ต่ำๆ บนเม็ดเลือดแดงตัวอ่อน ในเดือนมิถุนายนปี ค.ศ. 2023 CD36 ได้รับการขึ้นทะเบียนเป็นหมู่เลือดใหม่จากคณะทำงาน ISBT Working Party of Red Cell Immunogenetics and Blood Group Terminology (WP-RCIBGT) การแสดงออกของ CD36 deficiency สามารถแบ่งออกได้ 2 กลุ่มคือ CD36 deficiency type I ที่ไม่มีการแสดงออกของ CD36 ทั้งบนผิวเกล็ดเลือด และโมโนไซต์ และ CD36 deficiency type II ที่ไม่มีการแสดงของ CD36 เฉพาะบนเกล็ดเลือด* ***วัตถุประสงค์*** *เพื่อจัดหาเลือดที่เหมาะสมและปลอดภัยให้กับผู้ป่วยธาลัสซีเมีย* ***วัสดุและวิธีการ*** *การทดสอบหาแอนติบอดีและแอนติเจนบนผิวเม็ดเลือดแดงใช้เทคนิค CTT และ CAT ในส่วนการทดสอบทางลักษณะพันธุกรรม HEA ใช้ชุด Kit Immucor BioArray HEA Precise และตรวจหาลำดับเบสทางพันธุกรรมโดยใช้เทคนิค next-generation sequencing และทดสอบหาแอนติบอดีต่อแอนติเจนของเกล็ดเลือดด้วยเทคนิค monoclonal antibody-specific immobilization of platelet antigens (MAIPA)* ***ผลการศึกษา*** *พบว่าผู้ป่วยมีการสร้างแอนติบอดีต่อแอนติเจนของเม็ดเลือดแดงคือ anti-E-, -c, -Jk[a], -unidentified และ cold antibody จากการตรวจลำดับเบสของสารพันธุกรรมด้วยเทคนิค next-generation sequencing พบว่ามีการเปลี่ยนแปลงลำดับเบส ของหมู่เลือด CD36 (c.380C>T) ซึ่งนำไปสู่การเปลี่ยนแปลงกรดอะมิโน ทำให้หมู่เลือด CD36 มีการแสดงออกในรูปแบบ heterozygous* ***สรุป*** *จากการศึกษาพบว่า การที่ผู้ป่วยมีผลการตรวจความเข้ากันได้กับเลือดผู้บริจาคให้ผล incompatible อาจเกิดจาก autoantibody เท่านั้น ไม่ได้เกี่ยวข้องกับแอนติบอดีในกลุ่ม HTLA และจากการตรวจลำดับเบสของสารพันธุกรรม ทำให้พบการแสดงออกของหมู่เลือด CD36 ในรูป heterozygous*

คำสำคัญ : ● *ภาวะพร่อง CD36* ● *หมู่เลือดบนเม็ดเลือดแดงระบบใหม่* ● *Next-generation sequencing*
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Introduction

CD36 is a transmembrane glycoprotein (GPIV) expressed on the surface of various human cell types, including monocytes, erythroblasts, mammary glands, capillary endothelial cells, adipocytes, the membrane of the placenta, and human platelets (called Nak^a antigens), and expressed during the maturation of erythroid progenitors to red blood cells at low levels in reticulocytes and erythrocytes.[1] CD36 is working as a class B scavenger receptor.[2]

Genetic analysis of CD36 shows that it is located at 7q21.11.[1] CD36 deficiency, a genetic condition caused by a mutation in the CD36 gene that leads to reduced or absent expression of the CD36 protein. CD36 deficiency is very rare in Caucasians (0.3%), but it is more common in Chinese (2%), Japanese (3-4%), African (7.7%) populations,[3-7] and in the Thai population, the incidence is 2.28%.[8] CD36 deficiency is divided into two subgroups according to phenotype: CD36 type I deficiency is characterized by a lack of expression on platelets and all other cells. Type II deficiency CD36 is lacking from platelets only.[1] The incidences of CD36 expression in Thai populations have been reported using the flow cytometry method; CD36 positive was found in 98.3% of cases. In comparison, type I and type II deficiency rates are 0.16% and 0.84%, respectively.[9] CD36 type I deficiency (CD36 null phenotype) has been recognized as possibly leading to CD36 alloimmunization (antibody against CD36) when they receive a blood transfusion or during pregnancy and causing immune thrombocytopenic disorders, including post-transfusion purpura (PTP), platelet transfusion refractoriness (PTR)[1,10], and fetal-neonatal alloimmune thrombocytopenia (FNAIT)[11,12] caused by the anti-CD36 antibody.

In addition, transfusion-related acute lung injury (TRALI) was also reported.[13] Hydrops fetalis can be caused by a maternal alloimmune anti-CD36 antibody, as reported in two siblings' preterm cases of hydrops fetalis in Japan.[14] The anti-CD36 antibody would affect the growth and the differentiation of the erythroblasts in the neonates who showed extreme anemia with reticulocytosis at birth.[14]

Materials and Methods

Blood samples from a thalassemia patient who required blood transfusion but could not find compatible blood were sent to National Blood Centre, Thai Red Cross Society for investigations of RBC antibodies, phenotype, and genotype.

Hemagglutination tests were performed using the conventional tube test (CTT) and column agglutination test (CAT) (LISS/Coombs' Polyspecific IgG/C3d, Ortho Clinical Diagnostic, NJ, USA) according to the manufacturer's recommendation. For antibody investigation, the in-house panel cells, including saline cells and papainized cells, National Blood Centre, Thai Red Cross Society, Bangkok, Thailand were used. The clinically significant red cell phenotypes were performed using CTT, anti-C, -E, -c, -e, -S, -s, -Jk^a, and -Jk^b (Merck Millipore, The Millipore Corporation, Merck KGaA, Darmstadt, Germany), and anti-k (Immulab, ParagonCare, Victoria, Australia) were used according to the manufacturer's instructions. In-house anti-M, -N, -Mi^a,-P1, and -Di^a monoclonal antibody reagents (National Blood Centre, Thai Red Cross Society, Bangkok, Thailand). Genomic DNA was extracted from EDTA-whole blood samples using a QIAsymphony DSP DNA midi kit (Qiagen, Hilden, Germany), and HEA was performed using the Immucor BioArray HEA Precise BeadChip^{TM} Kit (Immucor, Warren, NJ, USA). Next-Generation Sequencing (NGS) was performed using the Illumina DNA Prep with Enrichment Kit (Illumina, San Diego, USA) and a custom-designed panel that enables comprehensive genotyping for 43 blood group systems and transcription factors KLE1 and GATA1. Sequencing occurred on the Illumina Miseq platform, and then the DNA sequence reads were aligned against the human reference sequence (*GRCh38*) to identify variants relative to the reference sequence.

Table 1 CBC parameters of a patient requiring blood transfusion*

CBC parameter	Results	Reference Range
Hb (g/dL)	5.4 (Critical)	12.0-16.0
Hct (%)	20.7 (Critical)	36.0-48.0
RBC (cells/cu.mm^3)	2.88 x 1,000,000 (Low)	4.00-5.50
WBC (cells/cu.mm^3)	13,450 (High)	5,000-10,000
Platelet (cells/cu.mm^3)	150,000	140,000-450,000
MCV (fL.)	71.9 (Low)	80.0-99.0
MCH (pq.)	18.8 (Low)	27.0-31.0
MCHC (g/dL.)	26.1	-
RDW (%)	31.9 (High)	11.5-14.5

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Case presentation

A 23-year-old Thai female with beta-thalassemia has been receiving regular blood transfusions every two months. Mild allergic transfusion reactions following every blood transfusion were also noted. She was admitted to the hospital for a blood transfusion the physician requested 2 units of leukocyte-poor packed red cells (LPRC). The admission laboratory results (Table 1) revealed severe anemia before transfusion. And the history of antibody identification was anti-E, -c, -Jk^a, and -I.

A routine serology workup was performed. Her blood group is O, Rh(D) positive. The antibody investigation showed anti-E, -c, -Jk^a, and unidentified with cold antibody (Tables 2 and 3) using the in-house panel cells, including saline cells and papainized cells (National Blood Centre, Thai Red Cross Society, Bangkok, Thailand) by the CTT and CAT. The EDTA plasma and allogeneic adsorbed plasma were used for investigation. The direct antiglobulin test (DAT) was positive (2+) with monospecific anti-human globulin IgG. An auto-cell elution test was performed using an acid glycine solution. The result was negative, with no antibody detected in the elution. The phenotype results are C+E-c-e+M+N+S-s+Mi(a-) P1+Jk(a-b+)Di(a-)k+. This reactivity pattern is agreeable with the antibody investigation results above. The compatibility test results of the patient EDTA plasma and 3 units of phenotype-matched RBCs gave an incompatible result by CAT (3+).

Then, the patient's genomic DNA was extracted from EDTA-whole blood samples. The Immucor BioArray System™ analyzes the individual SNV results to determine the predicted phenotypes, as shown in Table 4.

NGS results showed that the patient had nucleotide substitutions (c.59T>C and c.71_72delAGinsGT) leading to missense amino acids [p.(Leu20Ser) and p.(Glu24Gly)] in the GPA glycoprotein. These changes were found in 33 of 119 and 45 of 138 DNA sequence reads, consistent with heterozygosity for c.59T>C and c.71_42delAGinsGT. These variants define the *GYPA*01* allele and M+ phenotype.

In addition, DNA sequencing of the patient's *GYPB* (MNS) gene showed nucleotide substitution (c.173C>G), leading to missense amino acid (p[Pro58Arg]) in GPB glycoprotein. This change was found in 17 of 55 DNA sequence reads, consistent with heterozygosity for c.173C>G. This variant defines the *GYPB*23* allele and sD+ phenotype. Together with the above-predicted genotypes *GYPA*01/02* and *GYPB*04/23* with the predicted phenotypes of M+, N+, S-, s+, and sD+, it is in concordance with HEA using the Immucor BioArray HEA Precise BeadChip™ Kit.

Interestingly, the NGS result showed gene variation in *CD36* (CD36) nucleotide substitution (c.380C>T), which leads to the missense amino acid (p.[Ser127Leu]) in the CD36 blood group protein. These changes, found in 191 of 409 DNA sequence reads, are consistent with

Table 2 The antibody identification and result using plasma EDTA

Panel Cells for antibody identification

Screening Cells | Lot No : 66120 | Expiry Date : 15/01/2024 | Sample : Plasma EDTA

	No	Rh								MNSs					Lewis		P1Pk	Kidd		Duffy		Kell		Diego		Xg^a	CTT					CAT	
		D	C	E	c	e	f	CE	C^w	M	N	S	s	Mi^a	Le^a	Le^b	P1	Jk^a	Jk^b	Fy^a	Fy^b	K	k	Di^a	Di^b		18℃	37℃	IAT	CCC	Enz	IAT	Enz
Screening Cells	01	+	+	0	0	+	0	0	0	0	+	+	+	0	+	0	+	+	0	+	+	0	+	+	+	0	/	/	/	/	/	3+	3+
	02	+	+	0	0	+	0	0	0	+	0	+	+	0	0	+	W	0	+	+	0	+	+	0	+	+	/	/	/	/	/	3+	3+
	03	+	0	+	+	0	0	0	0	+	0	0	+	+	0	+	0	+	+	+	0	0	+	0	+	+	/	/	/	/	/	3+	4+
Panel cells	1	+	+	0	0	+	0	0	0	+	+	+	0	0	0	0	0	+	+	+	0	0	+	0	+	0	2+	1+	3+	/	3+	3+	3+
	2	+	+	0	0	+	0	0	0	+	+	+	+	0	0	+	0	+	+	0	+	0	+	0	+	0	2+	1+	3+	/	3+	3+	3+
	3	+	+	0	0	+	0	0	0	+	0	0	+	+	+	0	+	0	+	+	0	0	+	0	+	0	4+	3+	3+	/	3+	3+	2+
	4	+	+	0	0	+	0	0	0	0	+	0	+	0	0	0	+	+	0	+	0	0	+	+	+	+	2+	1+	3+	/	3+	3+	3+
	5	+	+	0	+	+	+	0	0	+	+	0	+	+	0	+	+	+	+	+	0	0	+	0	+	+	4+	3+	3+	/	3+	3+	3+
	6	+	+	+	0	+	0	+	0	+	0	0	+	0	+	0	0	+	+	+	0	+	+	0	+	+	2+	1+	3+	/	4+	3+	3+
	7	+	+	+	+	+	0	0	0	+	0	0	+	+	0	+	0	+	0	+	0	0	+	0	+	+	3+	1+	3+	/	3+	3+	4+
	8	+	0	+	+	0	0	0	0	+	+	0	+	0	0	+	+	0	+	+	0	0	+	0	+	0	3+	1+	4+	/	3+	4+	4+
	9	0	0	0	+	+	+	0	0	0	+	0	+	0	0	+	+	+	+	+	0	0	+	0	+	0	3+	1+	3+	/	3+	3+	3+
	10	0	+	0	0	+	0	0	0	+	0	0	+	0	0	+	W	+	+	+	0	0	+	+	+	0	2+	1+	3+	/	3+	3+	3+
	11	0	0	+	+	+	+	0	0	+	+	0	+	0	+	0	0	+	+	+	0	0	+	0	+	+	2+	3	3+	/	3+	4+	3+
Cord blood																											2+	1+	2+	/			
H1 deficiency																											1+	1+	3+	/			
H2 deficiency																											1+	1+	3+	/			
Autocontrol		+	+	0	0	+				+	+	0	+	0				0	+				+	0			0	0	0	1+		2+	

Table 3 The antibody identification result using allogeneic adsorbed plasma

Panel Cells for antibody identification

Screening Cells | Lot No : 66120 | Expiry Date : 15/01/2024 | Sample : Adsorbance plasma

	No	Rh								MNSs					Lewis		P1Pk	Kidd		Duffy		Kell		Diego		Xg^a	No.1 (R2R2)		No.2 (R.R.)	
		D	C	E	c	e	f	CE	C^w	M	N	S	s	Mi^a	Le^a	Le^b	P1	Jk^a	Jk^b	Fy^a	Fy^b	K	k	Di^a	Di^b		IAT	Enz	IAT	Enz
Panel cells	1	+	+	0	0	+	0	0	0	+	+	+	0	0	0	0	0	+	+	+	0	0	+	0	+	0	3+	3+	2+	2+
	2	ı	ı	0	0	ı	0	0	0	ı	ı	ı	ı	0	0	ı	0	ı	ı	0	ı	0	ı	0	ı	0	3ı	2ı	2ı	2ı
	3	+	+	0	0	+	0	0	0	+	0	0	+	+	+	0	+	0	+	+	0	0	+	0	+	0	3+	0	2+	0
	4	+	+	0	0	+	0	0	0	0	+	0	+	0	0	0	+	+	0	+	0	0	+	+	+	+	3+	3+	2+	2+
	5	+	+	0	+	+	+	0	0	+	+	0	+	+	0	+	+	+	+	+	0	0	+	0	+	+	3+	2+	3+	3+
	6	+	+	+	0	+	0	+	0	+	0	0	+	0	+	0	0	+	+	+	0	+	+	0	+	+	2+	2+	3+	3+
	7	+	+	+	+	+	0	0	0	+	0	0	+	+	0	+	0	+	0	+	0	0	+	0	+	+	3+	3+	3+	4+
	8	+	0	+	+	0	0	0	0	+	+	0	+	0	0	+	+	0	+	+	0	0	+	0	+	0	2+	2+	4+	4+
	9	0	0	0	+	+	+	0	0	0	+	0	+	0	0	+	+	+	+	+	0	0	+	0	+	0	2+	2+	3+	3+
	10	0	+	0	0	+	0	0	0	+	0	0	+	0	0	+	W	+	+	+	0	0	+	+	+	0	2+	2+	2+	2+
	11	0	0	+	+	+	+	0	0	+	+	0	+	0	+	0	0	+	+	+	0	0	+	0	+	+	3+	2+	3+	4+
Adsorbed cells	1	+	0	+	+	0	0	0	/	+	+	+	+	0	0	+	0	0	+	+	0	0	+	0	/	/				
	2	+	+	0	0	+	0	0	/	+	0	+	+	0	0	0	+	+	0	+	+	0	/	0	/	/				

Table 4 Predicted phenotypes using Immucor BioArray HEA Precise BeadChip™ Kit

Blood group system	Antigen	Predicted phenotype
Rh	C	+
	c	0
	E	0
	e	+
	V	0
	VS	0
Kell	K	0
	K	+
	Kp^a	0
	Kp^b	+
	Js^a	0
	Js^b	+
Duffy	Fy^a	+
	Fy^b	0
Kidd	Jk^a	0
	Jk^b	+
MNS	M	+
	N	+
	S	0
	S	+
	U	+
Lutheran	Lu^a	0
	Lu^b	+
Diego	Di^a	0
	Di^b	+
Dombrock	Do^a	0
	Do^b	+
	Hy	+
	Joa	+
LW	Lw^a	+
	LW^b	0
Scianna	Sc1	+
	Sc2	0
HbS	HbS	0

heterozygosity for c.380C>T. This variant is listed in the UCSC genome browser under rs201765331 with a frequency of 0.000.

From the result of the gene variation heterozygous for the CD36 phenotype, the platelet antibody was performed to confirm anti-CD36 in the plasma. We tested the patient's plasma with platelets that were CD36+ by monoclonal antibody-specific immobilization of platelet antigens (MAIPA) (commercial kit plus MAIPA, Immucor, Norcross, GA, United States). This result was negative. It means that the serum does not contain anti-CD36.

Discussion

From the red cell serology, platelet antibody, and molecular technique results, we believe the extra reaction is likely just the autoantibody, not the high titer low avidity (HTLA) related antibodies from tested with common HTLA negative cells such as Chido/Rodgers, Cromer, or others still reactive. Elution test from auto cell was unsuccessful with no antibody detected in eluate, possibly related to drug-induced hemolytic anemia but the patient has no history of medications. From elution results can exclude autoimmune hemolytic anemia (AIHA) and delayed hemolytic anemia.

Consequently, the suggestion for future blood transfusions should be a red blood cell phenotype-matched for the clinically significant blood group system. In addition, the next-generation sequencing results of *GYPB* (MNS) gene variation define the *GYPB*23* allele and the s^{D}+ (MNS23) phenotype. The genotype frequency for *GYPB*sD* in a selected Thai blood donor population is 2.2%. Hemolytic disease of the fetus and newborn caused by anti-s^{D} antibody in a GP.Mur/Mur has been reported.[15] Interestingly, based on the new finding that CD36 heterozygous (c.380C>T) in a Thai patient, CD36 was expressed at low levels in reticulocytes and erythrocytes. This finding has led to its inclusion as a new blood group system (ratified as a blood group in June 2023 by the ISBT Red Cell Working Party).[1]

According to the case report from Canals et al.,[16] anti-CD36 was reported to cause a weak panagglutination in routine RBC antibody testing. More recently, Peyrard et al. also reported anti-CD36 in the plasma of more than 100 patients during antibody identification and showed that the antibody could be neutralized with soluble recombinant CD36, giving unequivocal support for CD36 expression on normal RBCs. In addition, in the severe hydrops fetalis case by Okajima, et al.,[14] it seems very likely that the maternal anti-CD36 antibody was related to the observed. Erythroblasts are known to be CD36-positive, whereas mature erythrocytes have low levels of CD36. Therefore, it is understandable that the DAT was negative, and the examination of maternal CD36 expression or evaluation of anti-CD36 antibody in maternal serum should be considered in screening of hydrops fetalis.

In case of an antibody to CD36 being suspected in a patient, it is necessary to use a red cell CD36-negative donor for transfusion. At present, in red cell serology, there is no test to detect CD36 antigen on RBCs, but typing for CD36 on platelets by flow cytometry can be used. Therefore, integration of laboratory techniques may be required.

Conclusion

In summary, our case report presents the new finding of CD36 in a heterozygous level (c.380C>T) in a Thai patient, which was confirmed by next-generation sequencing. The incompatible crossmatch was caused by an autoantibody, not an HTLA-related antibody.

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