Case report

Anti-I alloantibody in an adult patient with the rare i phenotype: a case report

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Abstract:

Anti-I is commonly present in the serum of healthy individuals and is typically autoreactive, which is usually of the IgM isotype, and strongly reactive at low temperatures. It is identified by strong panagglutination with adult red cells but shows weak or no reactivity with cord red cells. Most autoanti-I antibodies are benign. In contrast, individuals with the rare adult i phenotype may develop clinically significant alloanti-I, which can cause hemolysis by destroying transfused I-positive red blood cells. This case report describes a 59-year-old male admitted to King Chulalongkorn Memorial Hospital with pneumonia, who was detected with alloanti-I and had a blood group phenotype of i using a combination of serology and molecular techniques. We present the antibody identification and crossmatching procedures and discuss transfusion strategies for the patients who produce alloanti-I and other antibodies to high-prevalence antigens.

Keywords : ● *Alloanti-I* ● *Adult i phenotype* ● *Molecular technique*

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รายงานผู้ป่วย

Anti-I alloantibody ในผู้ป่วยผู้ใหญ่ที่มีฟีโนไทป์หายาก i: รายงานผู้ป่วย

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บทคัดย่อ

Anti-I มักพบได้ทั่วไปในซีรัมของคนปกติ และโดยทั่วไปเป็น autoantibody ชนิด IgM ทำปฏิกิริยาได้ดีที่อุณหภูมิต่ำ แอนติบอดีชนิดนี้จะตรวจพบได้โดยจะทำปฏิกิริยาแบบ panagglutination ที่แรงกับเม็ดเลือดแดงของผู้ใหญ่ แต่ทำปฏิกิริยาที่อ่อนหรือไม่เกิดปฏิกิริยาเลยกับเม็ดเลือดแดงจากสายสะดือ autoanti-I ส่วนใหญ่ไม่ก่อให้เกิดความผิดปกติทางคลินิก ในทางตรงกันข้าม ผู้ใหญ่ที่มีฟีโนไทป์ i ซึ่งพบได้ยากมาก อาจสร้าง alloanti-I ที่มีความสำคัญทางคลินิก เมื่อได้รับเลือดจะก่อให้เกิดภาวะเม็ดเลือดแดงแตกจากการทำลายเม็ดเลือดแดงที่มีแอนติเจน I รายงานนี้นำเสนอผู้ป่วยชายอายุ 59 ปีเข้ารับการรักษาที่โรงพยาบาลจุฬาลงกรณ์ด้วยโรคปอดอักเสบ และตรวจพบแอนติบอดีชนิด alloanti-I และมีฟีโนไทป์ i จากการตรวจทางซีโรโลยีร่วมกับเทคนิคทางอณูชีววิทยา รายงานนี้แสดงรายละเอียด antibody identification, crossmatching ของผู้ป่วย และอภิปรายแนวทางการให้เลือดสำหรับผู้ป่วยที่สร้าง alloanti-I และแอนติบอดีต่อแอนติเจนที่มีความซุกสูงอื่นๆ

คำสำคัญ : ● *แอนติ-I* ● *ผู้ใหญ่ที่มีฟีโนไทป์ i* ● *เทคนิคทางอณูชีววิทยา*

วารสารโลหิตวิทยาและเวชศาสตร์บริการโลหิต. 2569;36:65-72.

Introduction

The I antigen is the antigen member in the I blood group system (ISBT 027), while the i antigen remains in the Ii collection (collection number 207).[1] These are carbohydrate determinants present on the surface of red blood cells (RBCs). Both I and i are high-prevalence antigens that are almost 100% expressed in the population, but they are described in a reciprocal relationship that is developmentally regulated. At birth, infant RBCs are rich i while I is almost undetectable. During the first 18 months of life, the quantity of i slowly decreases as I increases until adult proportions are reached; adult RBCs are rich in I and have only trace amounts of i antigen.[11] A linear poly-N-acetyllactosamine structure characterizes the i antigen. Postnatally, the I-branching enzyme (β-1,6-N-acetylglucosaminyltransferase) encoded by the GCNT2 gene, located on chromosome 6p24.2, adds β-1,6-N-acetylglucosamine branches. This enzymatic action converts the linear i antigen into the branched I antigen, which is expressed on adult RBCs. Some individuals fail to convert their i antigen after birth, resulting in the rare adult i phenotype (I-i+), which is an autosomal recessive trait caused by mutations in the *GCNT2* gene.[1-3] Several mutations responsible for the rare adult i phenotype have been identified.[4] The prevalence of this phenotype is estimated to be less than 1 in 10,000 worldwide. In patients of Asian ancestry, the adult i phenotype can be associated with congenital cataracts.[1-5]

Anti-I is a common, typically benign, cold-reactive IgM autoantibody found in the serum of most healthy individuals.[2] There are two forms of the anti-I autoantibody. One which is a weak, clinically insignificant, naturally occurring antibody that may present in a healthy individual. The other one, which is a pathological anti-I detected in a cold agglutinin disease. Commonly, the antibody to I would react strongly at 4°C and may be detectable at room temperature (RT) in high titers, but is usually clinically insignificant.[2] However, activity can be transiently enhanced in patients with certain conditions, notably pneumonia caused by Mycoplasma pneumoniae, often complicating pre-transfusion testing by causing panagglutination.[1-3] In contrast, individuals with the adult i phenotype can produce clinically significant alloanti-I upon exposure to I-positive RBCs through transfusion or pregnancy. Unlike the benign autoantibody, this alloanti-I is an immune antibody that can sensitize transfused I-positive RBCs and cause severe acute hemolytic transfusion reactions.[1-3] Alloanti-I results in more severe hemolysis than autoanti-I since it is an immune alloantibody produced after exposure to I-positive red cells, resulting from its IgM combined with IgG composition leading to both intravascular and extravascular hemolysis. Whereas autoanti-I is usually of IgM isotype and generally inactive above 37°C but alloanti-I can react in thermal range up to 37°C.[1-3,6]

This report presents the exceedingly first rare adult i phenotype in a Thai patient, confirming the presence of significant alloanti-I, which leads to major difficulty in compatible blood provision.

Case presentation

A 59-year-old Thai male patient was admitted to King Chulalongkorn Memorial Hospital with pneumonia. His underlying diseases include diabetes mellitus (DM), hypertension (HT), chronic kidney disease (CKD), and bilateral blindness from congenital glaucoma. The patient developed symptomatic anemia with a hemoglobin (Hb) level of 6.1 g/dL. A request for 1 unit of leukocyte-depleted packed red cells (LDPRC) for transfusion was submitted along with the patient's blood sample. He had no prior history of transfusion at our hospital. The results of the laboratory examination, performed using the column agglutination test (CAT), ID card LISS/Coombs, IH-500, Bio-Rad, Switzerland, showed that the patient had blood type O, Rh (D) positive. The results of the antibody screening from 3 individual screening cells were all positive (3+) (Figure 1). Antibody identification using both an 11-cell panel and an enzyme-treated panel showed panagglutination (3+ to 4+) with a negative autocontrol (Table 1). Furthermore, the conventional

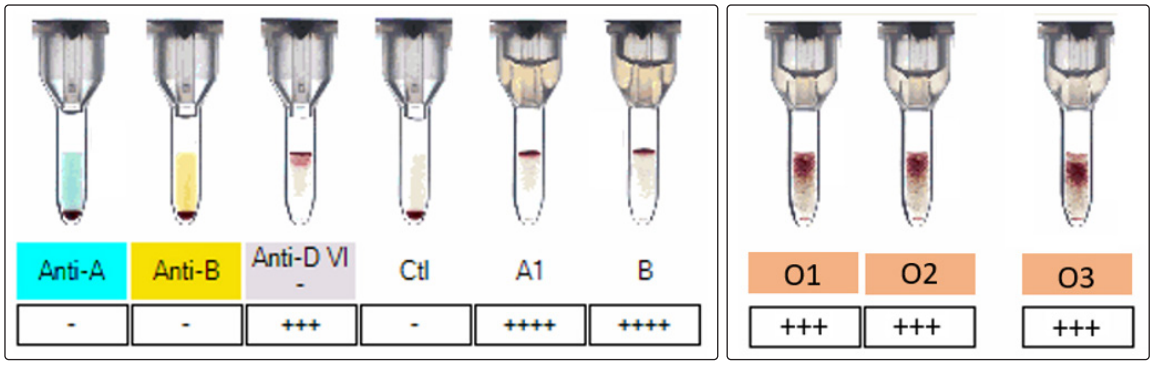

Figure 1 ABO/RhD typing and antibody screening by column agglutination test

Table 1 Antibody identification result using EDTA plasma

No	Rh					MNSs				P	Lewis		Mi[a]	Kidd		Duffy		Kell		Diego		CTT			CAT	
	D	C	E	c	e	M	N	S	s	P1	Le[a]	Le[b]		Jk[a]	Jk[b]	Fy[a]	Fy[b]	K	k	Di[a]	Di[b]	RT	37°C	IAT	IAT	Enz
1	+	+	0	0	+	+	0	0	+	0	0	0	+	+	0	+	+	0	+	+	+	4+	3+	2+	3+	4+
2	+	+	0	0	+	+	0	+	+	0	+	0	0	0	+	+	0	0	+	+	+	4+	3+	2+	3+	4+
3	+	+	0	0	+	+	0	0	+	+	0	0	+	+	0	+	0	0	+	0	+	4+	3+	2+	3+	4+
4	+	+	0	0	+	+	0	+	0	0	0	+	0	+	0	0	+	0	+	0	+	4+	3+	2+	3+	4+
5	+	+	+	0	+	+	+	+	+	0	+	0	0	0	+	+	+	0	+	0	+	4+	3+	2+	3+	4+
6	+	+	+	+	+	+	0	0	+	0	0	0	0	+	+	+	0	0	+	0	+	4+	3+	2+	3+	4+
7	+	+	+	+	+	+	0	0	+	+	0	+	+	0	+	+	+	0	+	0	+	4+	3+	2+	3+	4+
8	+	0	+	+	0	0	+	0	+	0	+	0	0	+	+	+	0	0	+	0	+	4+	3+	2+	3+	4+
9	0	0	0	+	+	+	+	+	+	0	0	+	0	+	0	+	+	+	+	0	+	4+	3+	2+	3+	4+
10	0	+	0	+	+	0	+	0	+	+	0	+	0	0	+	+	0	0	+	0	+	4+	3+	2+	3+	4+
11	0	0	+	+	+	+	+	0	+	0	0	+	0	0	+	+	0	0	+	0	+	4+	3+	2+	3+	4+
																			Auto control			0	0	0	0	0
																			Cord blood			W+	W+	W+	W+	W+

RT = room temperature; IAT = indirect antiglobulin test; Enz = enzyme

Table 2 Patient's blood type

Red cell phenotype (serology)	C+ E-c+e+ M+N-S+s Mi(a-) P1+ Le(a-b+) Jk(a+b+) Fy(b-) Di(a-) K-H+ PP1Pk+
Predicted phenotype (DNA analysis)	C+ E-c+e+M+N-S+s+U+Jk(a+b+) Fy(a+b-) Di(a-b+)K-k+Lu(a-b+) Lw(a+b-)Do(a+b+) Kp(a-b+)Co(a+b-)Js(a-b+)

tube test (CTT) confirmed strong panreactive results in all test phases (4+ at room temperature (RT), 3+ at 37°C, and 2+ at indirect antiglobulin test (IAT); the autocontrol remained negative). These findings strongly suggested the presence of an antibody directed against a high-incidence antigen with IgM and possible mixed with IgG pattern.

Therefore, an antibody to a high-prevalence antigen was suspected. Extended phenotyping was performed. The patient demonstrated common antigen profiles with no rare phenotypes identified, effectively ruling out corresponding alloantibodies. Further testing of the patient's RBC phenotype confirmed H and PP1Pk positive, excluding anti-H and anti-PP1Pk (Table 2). Allogeneic adsorption with R1R1 and R2R2 cells at 4°C for 5 cycles, followed by testing of the adsorbed plasma against panel cells and enzyme-treated panel cells, yielded negative results with all panel cells, confirming no other underlying alloantibodies and may possess antibodies to high-incidence antigens (Table 3). The plasma showed weak positivity with cord blood (i+I- phenotype) in all phases. While this serologic profile was consistent with anti-I, the possibility of an autoantibody with depressed I antigen expression due to critical illness was

Table 3 Antibody identification result using allogeneic adsorbed plasma

No	Rh					MNS				P	Lewis		Mi^a	Kidd		Duffy		Kell		Diego		R1R1		R2R2	
	D	C	E	c	e	M	N	S	s	P1	Le^a	Le^b		Jk^a	Jk^b	Fy^a	Fy^b	K	k	Di^a	Di^b	CAT IAT	CAT Enz	CAT IAT	CAT Enz
1	+	+	0	0	+	+	0	0	+	0	0	0	+	+	0	+	+	0	+	+	+	0	0	0	0
2	+	+	0	0	+	+	0	+	+	0	+	0	0	0	+	+	0	0	+	+	+	0	0	0	0
3	+	+	0	0	+	+	0	0	+	+	0	0	+	+	0	+	0	0	+	0	+	0	0	0	0
4	+	+	0	0	+	+	0	+	0	0	0	+	0	+	0	0	+	0	+	0	+	0	0	0	0
5	+	+	+	0	+	+	+	+	+	0	+	0	0	0	+	+	+	0	+	0	+	0	0	0	0
6	+	+	+	+	+	+	0	0	+	0	0	0	0	+	+	+	0	0	+	0	+	0	0	0	0
7	+	+	+	+	+	+	0	0	+	+	0	+	+	0	+	+	+	0	+	0	+	0	0	0	0
8	+	0	+	+	0	0	+	0	+	0	+	0	0	+	+	+	0	0	+	0	+	0	0	0	0
9	0	0	0	+	+	+	+	+	+	0	0	+	0	+	0	+	+	+	+	0	+	0	0	0	0
10	0	+	0	+	+	0	+	0	+	+	0	+	0	0	+	+	0	0	+	0	+	0	0	0	0
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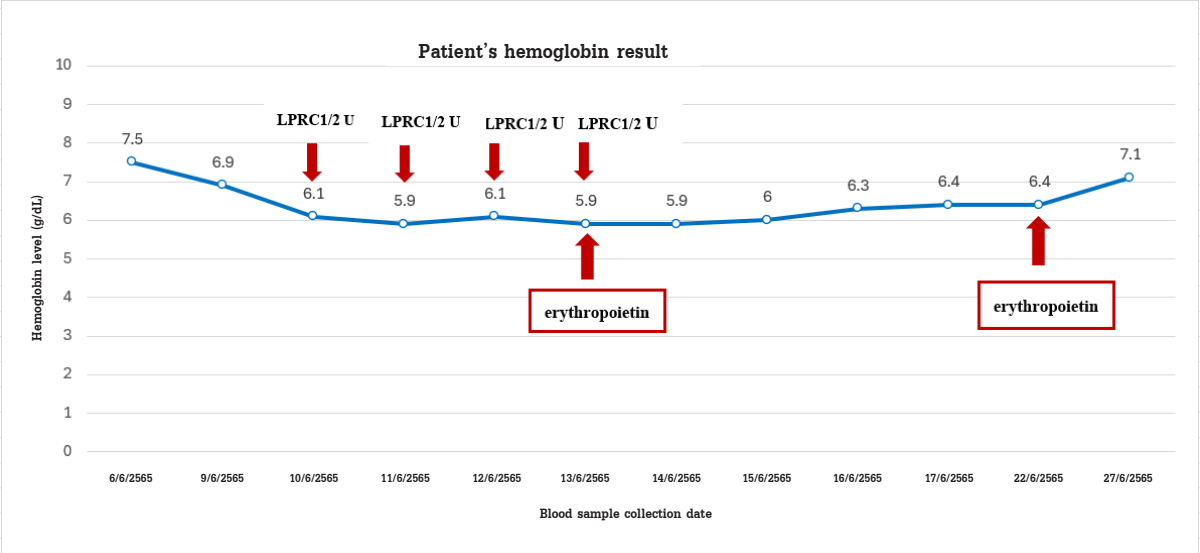

Figure 2 Timeline of the patient's hemoglobin level at the first admission

considered. However, a prewarmed crossmatch with a phenotypically matched unit remained positive (IAT 2+), supporting the presence of a clinically significant alloantibody.

Crossmatching of multiple phenotypically matched donor units with the patient's plasma demonstrated incompatibility by CAT (3+). Confirmatory CTT showed strong reactivity at RT (4+), 37°C (3+), and IAT (2+), in all units. The laboratory informed the doctor of the crossmatch result. Given the patient's increasingly anemic status, a transfusion of a half unit of blood was recommended to be given at a time, and the blood must be infused through a blood warmer. The patient's clinical signs and symptoms must be closely monitored after the blood transfusion. After transfusion of a half unit of blood, the patient had no transfusion reaction and his Hb level remained unchanged at Hb 6.1 g/dL. An additional half unit was administered daily until a total of 2 units had been transfused. As his Hb level still did not improve, transfusions were discontinued, and alternative treatments, erythropoietin (2 doses), were initiated, which resulted in a rise in Hb level to 7.1 g/dL within two weeks. A timeline of the patient's hemoglobin levels is shown in Figure 2.

Subsequently, the patient's blood sample was sent to the Australian Red Cross Lifeblood for antibody identification and DNA sequencing through the National Blood Centre of the Thai Red Cross Society. DNA

extraction from the red cells in the SAGM sample was performed using QIAGEN Symphony automation, and genotyping was performed using Immucore BioArray HEA precise Beadchip kit, showing C+c+E-e+V-VS-K-k+Kp(a-b+)Js(a-b+)Fy(a+b-)Jk(a+b+)M+N-S+s+U+Lu(a-b+)Di(a-b+)Co(a+b+)Hy+Jo(a+)LW(a+b-)Sc(1+,2-) with negative HbS screening. The next-generation sequencing (NGS) using Illumina was performed and found the rare nucleotide substitution (c.1154G>A) at the homozygous level in exon 3, leading to a missense amino acid p. [Ag385His] in the I blood group system. The variant defines the GCNT2*01N.02/*01N.02 allele, this nucleotide substitution (c.1154G>A) leads to missense amino acid p. [Arg385His] in the I blood group protein. The mutation was found in 479 of 487 DNA sequence reads, consistent with homozygosity for c.1154G>A. This variant defines the GCNT2*01N.02 allele and the I phenotype (ii phenotype). Interestingly, the NGS result showed the patient has UI(a+) phenotype, which is a rare low-incidence antigen in the Kell blood group system. Antibody identification using saline methods at RT, 37°C, IAT, and papain-IAT confirmed that only alloanti-I was present, concordant with previous results.

Six months later, the patient was re-admitted with dyspnea and leg edema for two days and was subsequently diagnosed with cellulitis of the left leg. During hospitalization, his Hb level declined from 7.4 g/dL to 5.8 g/dL over three days, and he developed signs of anemia, necessitating transfusion of one unit of LDPRC. Pre-transfusion laboratory findings were consistent with previous results, demonstrating panreactive antibody identification (3+), incompatible crossmatches (3+ by CAT) with a negative autocontrol result. Notably, even a prewarmed crossmatch with a phenotypically matched unit persisted as positive (IAT 2+). After unsuccessful attempts to locate compatible blood, a half unit of incompatible blood was transfused as a lifesaving measure and the blood must be infused through a blood warmer. The patient's clinical signs and symptoms were closely monitored after the blood transfusion. Approximately three hours post-transfusion, the patient developed hypotension.

A transfusion reaction investigation was promptly initiated. Post-transfusion laboratory analysis revealed plasma partial hemolysis and a weakly positive direct antiglobulin test (DAT), while the acid-elution test (DiaCidel, Bio-Rad, Switzerland) was negative. Other laboratory results, including antibody screening and crossmatching were identical to the pre-transfusion findings. Supporting laboratory data included a falling haptoglobin from 209.6 mg/dL to 87.4 mg/dL (RR 14-258 mg/dL), lactate dehydrogenase (LDH) was markedly elevated from 210 U/L to 1,931 U/L (RR 125-220 U/L), and total bilirubin rose from 0.34 mg/dL to 2.10 mg/dL (RR 0.20-1.20 mg/dL) with a concurrent rise in direct bilirubin increase from 0.22 mg/dL to 0.64 mg/dL (RR 0.00-0.50 mg/dL). The reticulocyte count remained within the reference range at 1.7% (RR 1.0-2.0%). Concurrently, hemoglobin increased marginally from 5.8 g/dL to 7.1 g/dL. A diagnosis of acute hemolytic transfusion reaction (AHTR) was established. Consequently, all further transfusions were discontinued. Despite supportive care, the patient's condition deteriorated rapidly, culminating in a fatal outcome due to hospital-acquired pneumonia with type 1 respiratory failure and septic shock.

Discussion

Usually, autoanti-I is commonly found in healthy individuals or in patients with viral or bacterial infection, known as a clinically insignificant IgM antibody. Among infectious causes, *Mycoplasma pneumoniae* is well known to induce autoanti-I production via cross-reactivity with I-like antigens on the bacterial surface. Anti-I antibodies are generally weak, but potent examples may be found as autoantibodies in patient with cold agglutinin disease (CAD) or following *Mycoplasma pneumoniae* infection. In the rare inherited adult i phenotype, red cells express very low I antigen.[1-3, 6] Alloanti-I is typically found at a high titer in i adult sera, usually as a low-thermal-range IgM antibody. Rare examples are hemolytic with a

thermal range up to 37°C[7] and even those active below 37°C can cause shortened survival of transfused I-positive red cells.[8] In vivo survival studies clearly indicate the antibody capacity to provoke a dangerous transfusion reaction, showing less than 1% of radiolabeled I-positive red cells survived 15 minutes after injection into an i adult with anti-I.[9] However, not all ii adults have alloanti-I in their serum.[10]

From a case report by Chaplin, et al. about the alloanti-I patient with massive hemorrhage. This article describes a 37-year-old man who had never been transfused was admitted with a ruptured spleen and massive hemorrhage. All crossmatches were incompatible. His serum contained alloanti-I and his red cells were I negative and strongly i positive. An in vivo chromium survival study demonstrated almost complete destruction of I positive red cells within 15 minutes. Afterwards, survival study using Ii phenotype red cells from his daughter showed near normal survival. This case report demonstrates that alloanti-I may have more serious clinically significant for transfusion than auto-anti-I. The possibility that Ii red cells may represent a safe and effective for transfusion in the presence of alloanti-I deserves further investigation.[9]

This case report describes the first report in the Thai population of an exceedingly rare adult i phenotype (I-i+), confirmed by molecular techniques, with alloanti-I. This finding presents a critical transfusion challenge as the I antigen is a high prevalence antigen present in nearly all donors (almost 100%), making compatible blood virtually unavailable. Management in such cases necessitates a multidisciplinary approach focused on patient stabilization, minimizing transfusion exposure, and exploring alternative therapeutic strategies. Interestingly, our patient developed alloanti-I despite having no prior history of blood transfusion, suggesting that the antibody may have occurred naturally. Additionally, while the adult i phenotype is typically associated with congenital cataracts, this patient was diagnosed with congenital glaucoma. However, the diagnosis was made many years ago, and further details could not be retrieved, representing a limitation of this report.

For unavoidable transfusions, the administration of least-incompatible, phenotype-matched RBC units is recommended under strict monitoring, utilizing an in-line blood warmer, as performed in our case. Transfusing small aliquots of blood with close clinical observation and serial hemoglobin monitoring mitigates the risk of acute hemolysis. Furthermore, erythropoietin may stimulate endogenous erythropoiesis, thereby limiting transfusion dependence. In this case, two doses of erythropoietin were administered, resulting in a demonstrable hematologic response with a noticeable increase in Hb from 5.9 g/dL to 7.1 g/dL within two weeks. The subsequent, unavoidable transfusion of a second incompatible unit as a lifesaving measure directly precipitated a confirmed AHTR.

This case highlights the critical importance of considering alloantibodies against high-prevalence antigens, such as anti-I, in the differential diagnosis of panagglutination with a negative autocontrol. A combination of advanced serological techniques, phenotyping, and genotyping is often required for a definitive diagnosis. For patients with confirmed anti-I, the procurement of I-negative blood is the cornerstone of safe transfusion practice. Blood from a family member who also tested I-negative may be used. When this is not feasible, clinicians must be aware of the significant risks and manage transfusion with extreme caution, recognizing that it may not provide therapeutic benefit and the possibility of lethal outcomes.

Conclusion

This patient's report showed alloanti-I, which is common in adult i phenotype, and blood group genotyping confirmed that the patient had genotype I- (i+I-). Alloanti-I causes panagglutination of all adult cells with a negative autocontrol. Crossmatching blood to a patient will result in an incompatible reaction. Because of alloanti-I, which is strongly reactive at low temperatures,

transfusions should be administered slowly through an inline blood warmer, and the patient should be closely monitored. Patient blood management is important to manage alternative treatment for the patient to avoid unnecessary transfusion of incompatible units and lead to hemolysis from hemolytic transfusion reaction. Due to the antisera for I antigen were not available commercially, molecular technique is important to conclude the phenotype to confirm antibody presence in patient plasma.

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