ILABB Case Studies 2023

ILLINOIS ASSOCIATION OF BLOOD BANKS
The Phyllis Unger
Annual Case Studies Meeting
Thursday, November 2, 2023

This case studies meeting is dedicated to Phyllis Unger in memory of all the wonderful case studies she presented to students, technicians, technologists, residents, fellows and physicians. Phyllis was a dedicated blood banker who spent much of her time teaching and educating whether she was at Michael Reese, University of Illinois or LifeSource. She was always willing to answer questions or test a sample if you sent it to her. She inspired many blood bankers and gave them the desire to look further into a problem. Phyllis was the first Medical Technologist to be President of the ILABB. Prior to this only a physician could hold the office. She wrote many papers and contributed to a few books including “Blood Group System: MN and Gerbich.”

She had many things outside of blood banking that she enjoyed as well such as travel, music and bridge. Even these things helped give her blood banking perspective. She was known for never saying anything bad about anyone and always finding the best in them. We hope that this annual meeting will serve as a lasting memory to the knowledge she shared with all who came in contact with her over the years.

Thank you, Phyllis.

AGENDA

530pm – 615pm	Dinner
615 pm	Operationalizing a Protocol for Providing Red Blood Cells for Perfusion of Explanted Livers on a Normothermic Perfusion Device.
635 pm	A Review of Autoantibodies with Underlying Alloantibodies
655pm	Identification of Maternal Anti-Dib prior to Scheduled Repeat Cesarean Delivery
715 pm	Steroid-refractory paroxysmal cold hemoglobinuria in a 6-year-old male
735 pm	A Sticky Situation: Diagnosing Post-Transfusion Purpura in the Setting of Longstanding, Transfusion-Dependent AML
755 pm	The Challenge Is Real: Transfusion Management In A Patient With Anti-Rh17 Antibody
815 pm	Patients with Partial RHD Genotypes: Allele-Specific Relative Risks of Anti-D Alloimmunization
835pm	Sensitivity of Anti-D Reagents: Implications for Rh Typing and Clinical Management

Title: Operationalizing a Protocol for Providing Red Blood Cells for Perfusion of Explanted Livers on a Normothermic Perfusion Device.

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BACKGROUND: The OrganOx Metra® is an automated normothermic perfusion device designed to support assessment and optimization of a donor liver prior to transplanting the organ into a recipient. The liver is harvested from the organ donor, placed on the device and continually perfused with ABO-matched red blood cells (RBCs) and nutrient solutions. Utilization of the OrganOx Metra® for these explanted livers allows for optimization of the donor liver prior to transplantation, as well as testing of the perfusate to assess liver function (liver enzymes, total bilirubin, lactic acid). Prior to transplantation, the explanted liver is flushed of the perfusing solution. The challenge for the blood bank was to allocate and issue the requisite RBCs to an explanted organ in a manner that satisfied regulatory and accreditation standard, supported the organ transplant team workflow, by leveraging the laboratory information system (LIS) and electronic medical record (EMR).
CASE REPORT: A surgeon, who recently joined Loyola Medicine to lead the abdominal organ transplant team, initiated the use of the Organox Metra® as a means to increase the number of available liver allografts for transplantation. Blood bank was tasked to create a process to allocate and issue three RBC units, ABO-matched to the donor blood type, to explanted livers placed on the Organox Metra® device. His experience at a prior hospital utilized a manual downtime procedure, which was cumbersome for both the blood bank staff and the transplant team. Concerned about the high risk for error using a manual system, the Loyola blood bank team collaborated with the transplant team, patient registration, Health Informatics, and the EPIC team to develop and implement a process that leveraged use of the existing Sunquest LIS and EPIC EMR to support electronic allocation and issuing of RBC units to the OrganOx Metra®. The final process very closely mirrors the pathway used for routine blood component issuing to patients.

CONCLUSIONS: After six months' experience supporting perfusion of livers using the OrganOx Metra® device, the process for allocating and issuing RBCs to an explanted organ is streamlined, fully compliant with accreditation standards and regulatory requirements. This process additionally offers requisite documentation for blood component use for organ perfusion. 12 harvested liver allografts have been processed and successfully transplanted using the Organox Metra®, 5 of which were harvested from donors who previously would not have been considered as viable candidates. Using the LIS and EMR to eliminate a manual process improved patient safety, increased physician satisfaction, encompassed all compliance interests, and reinforced inter-departmental teamwork. The LUMC transplant team plans to expand the ex vivo protocol to harvest and optimize liver allografts from organ donors after circulatory determination of death (DCD).

Title: A Review of Autoantibodies with Underlying Alloantibodies

Authors: Nilson, Aleta, MLS(ASCP)CM SBB(ASCP)CM1; Howard-Menk, Chris, MS, MT(ASCP)SBB1; Bodnariuc, Jonathan, BB(ASCP)1; Yamanaka, Kayo, MT(ASCP)1; Bradford, Patricia, MT(ASCP)1
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Background: Alloantibodies are antibodies to antigens not expressing on the patient's own red blood cells (RBCs) and are produced after an antibody stimulating event. Autoantibodies are antibodies to antigens that are expressing on the patient's own RBCs and don't require exposure to produce. Immunohematology Reference Laboratories employ various methods of testing to separate out these two groups of antibodies.

Case Report 1: A 64-year-old Caucasian female with a Hgb of 7.6g/dL was transfused three and a half months ago with a history of anti-Bg and an antibody of undetermined specificity. The Ortho gel panel reactivity ranged from 1+ to 3+ and the auto control was 1+. This pattern indicated a possible underlying alloantibody. The Direct Antiglobulin Test (DAT) with anti-IgG was 2+ and anti-C3b, -C3d was negative. Immucor's Gamma ELU kit's eluate performed in gel reacted 2+ to 3+. Additionally, a select LISS panel and a no-enhancement panel tested in tube showed reactivity ranging from 0 to 3+; the auto control was 1+ (LISS) and 4+ (no-enhancement). The auto adsorption was unable to remove the reactivity of the antibody indicating possible alloantibodies. A stroma adsorption (R1R1 and R2R2 set) was performed to separate the antibodies. Also, the patient's RBCs were stripped of autoantibodies using EDTA Glycine-Acid treatment, serologically phenotyped, and that information was used to select panel RBCs. We identified a warm with underlying alloantibodies (anti-C, -K, and -s).

Case Report 2: An 82-year-old Hispanic/Latino female with a Hgb of 4.1g/dL was transfused within the last three months with a history of a warm auto antibody and anti-E. The Ortho gel panel reactivity varied from 2+ to 4+ and the auto-control was 4+. Both anti-IgG and anti-C3b, -C3d DAT were 3+. The Immucor Gamma ELU kit's eluate reacted 4+ for all the cells. A LISS and a no-enhancement select panel was tested; the R1R1 cells reacted weak positive to 2+, the rr cells were 4+, and the auto control was 2+. This tube method reduced the reactivity of the warm auto, revealing a possible alloantibody. The cold screen was positive.
Since the recently transfused patient had an autoantibody that reacted at both warm and cold phases; the stroma adsorption was incubated at 37 OC and 4 OC. We identified a warm auto and cold auto in the raw plasma, and anti-c in the adsorbed plasma.

Conclusion: In both cases Gel method was used first; however, the warm antibody obscured the reactivity of the underlying alloantibodies. When we proceeded to using tube methods with decreased enhancement strength; it helped revealed the variability of the reactivity between the weakened auto control and other panel cells indicating a possible underlying alloantibody. Thus, using the most sensitive technique is not always helpful in identifying underlying alloantibodies in a patient with an autoantibody.

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Gamma EGATM Kit. Immucor IC3023-5; revised 03/17

Title: Identification of Maternal Anti-Dib prior to Scheduled Repeat Cesarean Delivery

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Background: Antibodies against antigens from the Diego (Di) system have been intrinsically linked to HDN since the discovery of anti-Diain 1955. Presently, antibodies againt Diego A (Dia), Diego B (Dib) and Wra antigens are amongst the most clinically significant within the system1. While the frequency of Diais variable among different populations (0.01% to 36%) and its antibody has been known to cause severe HDN2-3, its counterpart, Dib, is considered a high-frequency antigen1. Anti-Dib has been associated with mild cases of HDN4.

Case Report: A 39-year-old Hispanic female (GA 36 weeks 6 days, G5P1121, with a history of prior emergency cesarean delivery for pre-eclampsia) was admitted to the OB ED prior to a scheduled cesarean delivery at 37 WGA. The patient had been seen two weeks prior, but only an ABO/Rh was requested at that time. A type and-screen was ordered for the current visit. Both the maternal and neonate's ABO/Rh's were B Positive. The patient's plasma demonstrated variable 2-t0-3+ pan-reactivity against reagent screen-cells and panel cells. Both the polyspecific and IgG DATs were negative. The sample was then forwarded to the IRL for further investigation. Here, the patient's phenotype was serologically determined (C-E+c+e-K-S-s+Fya+Fyb Jka-Jkb+Lea-Leb+) using commercial antisera. Given the patient's ethnic background, the first suspicion was of Anti-Dib. The patient was serologically typed for Dib using unlicensed antisera. They were Dib-. Afterwards, Dib- in-house reagent red-cells and Dib untested red-cells were tested against the patient's plasma at gel and PeG AHG. Dib- cells were negative while the Dib untested cells showed 2+ reactivity in both phases. An antibody titer was performed for academic purposes and titered to 1:64. Finally, a x6 allo adsorbtion was performed using R2R2-stroma. The alloadsorbed plasma was tested against reagent red-cells to conclude rule-outs of other clinically significant antibodies.

Once the workup was complete, our IRL notified the hospital of the patient's antibody. It was requested that a sample be drawn from the patient's neonate, as there were concerns of the possibility of HDN due to the maternal Anti-Dib. A heel stick was drawn from the neonate and sent over to our IRL for workup. Testing of the plasma against reagent screen-cells showed 1+ reactivity, while both the polyspecific and IgG DATs showed 4+ reactivity in gel. An acid eluate was made using a commercial elution kit. Testing of the eluate against reagent red-cells showed 4+ pan-agglutination in gel. The same Dib- reagent red cells used for the neonate's mother were used for this workup to confirm the presence of Anti-Dibin the neonate's eluate. As expected, Dib- cells had negative reactivity, while the Dib untested cells exhibited a 2+ reactivity in gel. The neonate was then admitted to the nursery for high risk of HDN. Their total bilirubin rose from 2.9 to 7.7 mg/dL, while the indirect bilirubin peaked at 7 mg/dL, and their Hgb at 15.6 g/dL. However, none of these results were considered significant, and the neonate was discharged after three days without additional intervention.

Conclusion: Anti-Dib was identified both in the maternal plasma and the neonate's eluate. While this antibody has been associated with mild cases of HDN, in this case, however, even though the neonate

presented the antibody in her eluate and the maternal titer was markedly increased, clinically the neonate was not anemic and had only a mild bilirubin increase, which is very common in neonates. Therefore, it is important to reinforce that immunohematology tests should always be correlated with other laboratory results and clinical findings in order to diagnose HDN.

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Title: Steroid-refractory paroxysmal cold hemoglobinuria in a 6-year-old male

Authors: Nolan Donahue, DO and Kristen Krum, MD

Background: Paroxysmal Cold Hemoglobinuria (PCH) is a rare autoimmune hemolytic anemia (AIHA) that is often seen as a sequela of viral upper respiratory tract infections and immunizations in children. It is characterized by a positive Coombs test with a biphasic auto anti-P IgG known as the "Donath-Landsteiner antibody," which sensitizes red cells and fixes complement at 0-4°C, and hemolyzes red cells near body temperature. The mainstay treatment of PCH includes cold avoidance, intravenous hydration, and supportive blood transfusions. Glucocorticoids may be used in the setting of significant hemolysis, with debated efficacy. We present a case of steroid-refractory PCH in a 6-year-old boy secondary to human rhinovirus/enterovirus.

Methods: Patient clinical data was abstracted from the electronic medical record. Blood bank testing was performed through the in-house blood bank. Donath-Landsteiner testing was performed at a reference laboratory.

Findings: A 6-year-old male with a history of mild upper respiratory tract infection 1 week prior presented to the emergency department with abdominal pain, dark urine, and jaundice. Initial laboratory evaluation uncovered anemia with hemoglobinuria, hyperbilirubinemia, elevated LDH, and decreased haptoglobin, raising concerns for acute hemolytic anemia (Table 1). Follow-up blood bank testing revealed a cold autoantibody. Viral PCR was positive for human rhinoenterovirus.

Despite supportive therapy through intravenous fluids and warming, the patient’s anemia worsened significantly (Hgb 5.1g/dL) and packed red blood cell (pRBC) transfusion was required. Methylprednisolone 0.8 mg/kg IV twice daily was initiated as empiric therapy for autoimmune hemolysis without significant improvement. Blood was sent for Donath-Landsteiner antibody testing at a reference laboratory and revealed a bi-phasic hemolysin consistent with paroxysmal cold hemoglobinuria. The patient required an additional pRBC transfusion and was discharged after 4 days inpatient.

Conclusion: Our case supports a growing body of evidence that finds steroid therapy inefficacious in the treatment of hemolysis in the setting of PCH.

Table 1

	Unit	Value	Reference Range
Hemogram			
Hb	g/dL	8.2	10.9-14.9
WBC	K/μL	20.3	4.9-14.5
Urinalysis			
Blood		large	negative
Protein		3+	negative
Urobilinogen	mg/dL	4.0	< 2.0
RBCs	cells/HPF	1	1-2
Biochemical Parameters			
Total Bilirubin	mg/dL	14.0	0.2-1.4
Direct Bilirubin	mg/dL	1.0	0.0-0.3
LDH	U/L	929	165-308
Fibrinogen	mg/dL	516	200-400
Haptoglobin	mg/dL	< 30	32-173
Complement C3	mg/dL	174	79-152

Title: A Sticky Situation: Diagnosing Post-Transfusion Purpura in the Setting of Longstanding, Transfusion-Dependent AML

Authors: Constantine Kanakis, MD and Kristen Krum, MD

Background/Case Studies: A 73-year-old female patient presented to the emergency department with acute, worsening fatigue, lethargy, pallor, and shortness of breath. She was found at home by emergency medical services with bright red blood covering her legs, originating from the rectum, several longstanding pressure ulcers, and oxygen saturation in the 80s. The initial physical exam revealed a cachectic, ill-appearing woman who was tachypneic, wheezing, with new and old blood at anus, generalized weakness, fatigue, shortness of breath, cough, myalgias, and diffuse bruising of various sizes. There was no loss of consciousness or history of anticoagulant therapy. The patient had a complex medical history which included acute myeloid leukemia with concurrent chemotherapy complicated by longstanding, transfusion-dependent pancytopenia, lung adenocarcinoma status-post radiation therapy now in remission, congestive heart failure, chronic obstructive pulmonary disease on continuous home oxygen, chronic intermittent bilateral lower extremity rashes secondary to adult-onset Still's disease, and multiple visits to the emergency department, inpatient admissions, and numerous outpatient infusions for the management of pancytopenia.

Study Design/Methods: The laboratory evaluation of this patient focused on the clinical concern for refractory multilineage pancytopenia secondary to her history of acute myeloid leukemia. Further examination of available medical records, concurrent ancillary diagnostic testing, and serological send-out testing was performed. Appropriate investigation and evaluation of repeated transfusion reactions with clinical symptomatology in accordance with available biovigilance surveillance guidelines aided in the diagnosis.

Results/Findings: The patient's initial laboratory workup in the emergency room setting demonstrated clear, previously known, pancytopenia along with severe acidosis, marked anemia, and derangements of coagulation testing. With numerous visits to both the emergency department and outpatient infusion centers, the patient had previous transfusion reactions which ranged from mild allergic to repeating patterns of fulminant weakness, lethargy, and confusion relating to the timing of transfusions. With increasing skin manifestations of purpuric skin lesions, severely acute thrombocytopenia regardless of HLA matched or compatible platelet units, and results of send-out serology demonstrating 100% reactivity to all platelets and specific HPA antibodies, the diagnosis of post-transfusion purpura was made.

Conclusions:
Despite this patient succumbing to diffuse hypoxic anemia and suffering a fatal cardiac dysrhythmia, the results of reference laboratory send-out serology revealed positivity in 100% of platelets crossmatched and the presence of antibodies to HLA Class I and HPA-2b, which satisfy the clinical and serologic criteria for post-transfusion purpura and represent a rare diagnostic finding.

Title: The Challenge Is Real: Transfusion Management In A Patient With Anti-Rh17 Antibody

Authors: Noah Mehr, MD, Fatima Aldarweesh, MD, Timothy Carll, MD, Rahaf Alkhateb, MD, Christine Fuja, DO

Background: The Rh system is a highly immunogenic, complex and polymorphic blood group system with 56 currently characterized Rh antigens1. Antigens are located on 2 proteins, RhD and RhCE, encoded by two genes, RHD and RHCE, that are closely linked on chromosome 1 (1p36.11).1 Rh17, also known as Hr0, is a high frequency antigen composed of several epitopes on the RhCE protein. Anti-Rh17 antibodies can be made by individuals with missing or varied C/c, E/e antigens such as the phenotypes: D- -, D··, Dc-, DCW- .2 The anti-Rh17 antibody has been reported in a variety of populations and there have been case reports of hemolytic disease of the fetus and newborn (HDFN) ranging from mild to severe3,4. Finding compatible blood for patients with anti-Rh17 can be particularly difficult given that only 1 in 100,000 people are Rh17 negative5. Literature is sparse on the transfusion of incompatible blood to non HDFN patients with anti-Rh17 antibodies. Here we report a case of transfusion of incompatible pRBC to a 54- year-old woman with anti-Rh17 antibodies in the setting of worsening critical anemia.

Case report: A 54-year-old group OPos female was transferred for management of a new diagnosis of Philadelphia chromosome positive acute lymphoblastic leukemia. The referring hospital provided reference laboratory results which showed anti-Rh17 antibodies and no other clinically significant alloantibodies. Blood was requested from the American Rare Blood Donor Program (ARDP). On admission the hemoglobin was 7.1 g/dL. Antibody screen was positive, and workup showed 4+ pan-reactivity in gel and tube testing. Following chemotherapy initiation and despite daily erythropoietin therapy, hemoglobin dropped to 2.8 g/dL and the patient was placed on an oxygen supplement. Transfusion was strongly requested at this time. Multiple units with varying Weiner haplotypes were all 4+ incompatible. Based on the patient's preliminary phenotype reported by American Red cross, the decision was made to transfuse one split pRBC unit of rr, K and Fya antigen negative blood while pending Hemopure (cell free hemoglobin product) approval. The patient was premedicated with methylprednisolone and IVIG. After approximately 55mL was transfused the patient developed chills and the transfusion was immediately stopped. The immediate transfusion reaction evaluation, including hemolysis markers, was unremarkable and signed out as underlying disease. Post transfusion hemoglobin was 2.5 g/dL. ARDP could not identify donors. The patient received Hemopure per protocol. Finally, monocyte monolayer assay suggested accelerated clearance of antigen positive red blood cells and the RBC genotype revealed low signals with all Rh antigens depicting possibly D - - phenotype.

Conclusion: Overall difficulties in this case included the challenge of finding compatible units, dilemma of transfusing incompatible units in a patient with severe anemia and the obtaining of alternatives to blood products. The search for Rh17 negative units is still ongoing and the possibility of repeat transfusion with incompatible units remains. An overview of the approach to unavoidable incompatible transfusion and management of patients receiving hemoglobin-based oxygen carriers is provided.

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Title: Patients with Partial RHD Genotypes: Allele-Specific Relative Risks of Anti-D Alloimmunization

Authors: Bianca Carter, MD, Christina M. Barriteau, MD, MPH, and Glenn Ramsey, MD.
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Background: In patients with partial RHD genotypes exposed to conventional D+ RBCs through pregnancy or transfusion, allele-specific risks of anti-D alloimmunization are uncertain. Our 5-year experience with hospital-based RHD genotyping was reviewed for D+ patients with anti-D. US literature was examined for other informative data.

Methods: We performed RHD genotyping for adult patients with weak or discrepant microplate or tube RhD phenotypes and D+ adults with anti-D (Barriteau 2022). Records were reviewed for D+ patients with anti-D. Hemizygous-plus-homozygous prevalences for partial RHD alleles in US Blacks were calculated from published RH gene surveys in Black blood donors or sickle-cell disease (SCD) patients (Ramsey & Barriteau, AABB 2023). The Chicago relative rates of anti-D for each allele were expressed as the number of anti-D cases per 1 percentage point of US partial-RHD prevalence (e.g., 1 DIIIa anti-D/0.81% DIIIa prevalence; 2 DOL anti-D/0.14% DOL prevalence). In 690 pediatric SCD patients in Philadelphia with partial-RHD allele frequencies and anti-D information (Takasaki 2023), we expressed their anti-D rates as the number of variant anti-D cases per variant allele in the cohort (e.g., 1 DIIIa anti-D/23 DIIIa alleles; 1 DOL anti-D/3 DOL alleles). Anti-D rates were normalized to DIIIa for comparison.

Results: Eleven Chicago patients were D+ with anti-D reactivity. Seven Blacks and 1 White patient had partial RHD genotypes: DOL1 or DOL2 (n=2), DIVa (n=2), DIIIa, DAU3 (more likely) or DAU11, RHD*39 (307C) and DNB (White). Two cases had conventional RHD genes in DNA array testing; one was managed as auto-anti-D (with autoanti-e and allo-anti-C and -E) and one may have had an unidentified RHD variant. One patient with weak D type 1 had remote transient anti-D, likely to have been autoantibody. All had normal 3-4+ D phenotypes in microplate typing except the type 1 case (negative direct agglutination). Three cases had weak-D phenotypes in tube typings (type 1 antiglobulin+ only; DNB and DOL 2+). Five cases had other alloantibodies: anti-C (2); anti-C- and E; anti-E and -K; and anti-G in RHD*39 (G-negative). The RHD*39 case was also hrB-negative and DIVa was likely linked to ceTI (partial c and e). The DIIIa case also had an S-, s-, Uvar predicted phenotype.

Relative to DIIIa (normalized to 1.0), the estimated rates of anti-D in Chicago/Philadelphia were 2.3/0.8 for DIVa, 0.7/2.1 for DAU3 and 14.6/7.7 for DOL. Other relative rates in Philadelphia were 1.0 for DAU5, 1.1 for type 4.0, 3.9 for DAR and 7.7 for DAU4. These estimates were unadjusted for likelihood of management as normal D+ phenotypes prior to anti-D formation. In Chicago 54 type 4.0 cases and 32 DAR cases were found through weak or discrepant RhD phenotypes; none presented with anti-D.

Conclusions: D+ patients with anti-D frequently have other RBC antibodies and genetic risks for more complex alloimmunization. In adult transfusion-service patients in Chicago and pediatric SCD patients in Philadelphia, DIIIa and DIVa carried relatively lower risks per patient for anti-D and DOL carried a relatively higher risk.

Title: Sensitivity of Anti-D Reagents: Implications for Rh Typing and Clinical Management

Authors: Robert Boehm1; Catherine Lazo1; Mingmar Sherpa, DCLS1; David Allision, D.O.2 Institution:
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Background: The RhD antigen, a vital determinant of the Rh blood group system, is encoded by the most polymorphic RHD gene, with over 600 alleles resulting in D antigen variants. The detection of these variants is crucial in transfusion medicine and obstetric care. With the advent of highly sensitive monoclonal blend Anti-D reagents, there is a potential concern about the over-detection of partial D antigens, leading to patients being classified as RhD-positive. Misclassifying weak D or partial D individuals has significant implications for transfusion practices and Rh immunoglobulin (RhIG) prophylaxis in obstetric care.

Case Report: A 39-year-old female presented with symptomatic anemia (Hemoglobin 5.2 g/dL), and history of uterine fibroids causing heavy menstruation. She had no prior history in the hospital's medical record. Type and screen and check ABO were ordered in preparation for red blood cell transfusion. Initial type and screen performed at the University of Illinois Hospital (UIH) blood bank lab using Ortho Vision instrument (method gel) identified her as RhD-positive (reaction strength with anti-D gel card was with a 4+). Check ABO was performed using Ortho's Bioclone anti-D antisera in tube, which marked the patient as RhD-negative. Since a discrepancy between gel and tube method was identified, the patient's sample was sent out for RHD molecular genotyping, which was performed using the Immucor's RHD BeadChip assay, and RHD Zygosity determined by hybrid Rhesus box detection. Results for the RHD genotyping showed c.602G (p.201Arg) and c.667G (p.223Val) alleles resulting in RHD weak partial 4.0 and predicted RhD phenotype as weak D partial. Hybrid Rhesus box came back negative. The patient's results raised concerns about the sensitivity of the initial Anti-D reagent and the potential for misclassification of her RhD status.

Discussion: The increasing sensitivity of Anti-D reagents poses challenges. While ensuring that weakly reacting D individuals without D variant are not misclassified as RhD-negative, there is a risk of over classifying individuals with partial Das RhD-positive. This has two primary implications:
1. Transfusion Concerns: Misclassification can lead to inappropriate blood product selection, potentially causing hemolytic transfusion reactions in cases where D variant individuals receive RhD-positive blood.
2. Obstetric Care: RhD-negative mothers require RhIG prophylaxis to prevent Rh alloimmunization. However, mothers with clinically significant D variants, if misclassified as RhD-positive due to sensitive reagents, might not receive RhIG, putting future pregnancies at risk of hemolytic disease of newborn. Given these concerns, there should be a discussion on whether blood banks should employ a combination of both more and less sensitive testing methods to ensure strongly reacting partial D individuals are not misclassified as RhD-positive.
Conclusion: The sensitivity of Anti-D reagents plays a pivotal role in determining Rh status and guiding clinical management. Blood banks may need to consider using a combination of Anti-D reagents or multiple methods to ensure accurate RhD typing. Further studies are warranted to establish guidelines addressing the challenges posed by the varying sensitivities of Anti-D reagents, especially in transfusion medicine and obstetric care.