Allele Name	Ethnicity	Number Tested	Number Positive	Antigen Frequency (%)
*GYPA*Vw*	African American	2707	1	0.04
	Asian	178	0	-
	Caucasian	11,560	17	0.15
	Hispanic	1035	2	0.19
	Other	110	0	-
*GYPA*Hut*	African American	2707	14	0.52
	Asian	178	0	-
	Caucasian	11,560	2	0.02
	Hispanic	1035	1	0.01
	Other	110	0	-
*GYP*Mur*	African American	2707	2	0.07
	Asian	178	0	-
	Caucasian	11,560	0	-
	Hispanic	1035	0	-
	Other	110	0	-
*GYP*HF*	African American	2707	7	0.26
	Asian	178	14	7.87
	Caucasian	11,560	0	-
	Hispanic	1035	1	0.01
	Other	110	1	0.91
Novel	Asian	N/A	1	N/A

TABLE 1: Detection of hybrid MNS glycophorins known to be Mi(a+).

variant s antigen. The novel Mia antigen is predicted to include a portion of the GYP*Bun and GP*Mur sequences and reside within GYP*HF.

Conclusions: Numerous reports have been published detailing cases of mild to severe HDFN as well as mild to severe hemolytic transfusion reactions resulting from antibodies to antigens present on these MNS hybrid glycophorins. However, limited data exists identifying ethnic groups shown to possess the antigens. This report demonstrates the prevalence of these antigens as detected in this multi-ethnic donor population.

P-IG-4 | Soluble A Antigen Confounding Blood Type in an ABO Mismatched Stem Cell Transplant Patient: A Case Report

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Background/Case Studies: In ABO mismatched hematopoietic stem cell transplants (HSCT), the emergence of the patient's original ABO type is associated with disease relapse and/or graft loss. We describe a case in which strong re-emergence of the patient's ABO type was attributed to high levels of soluble ABO antigen in the setting of severe graft versus host disease (GVHD). Removal of red cell soluble A antigen was achieved using an older method described for Lewis antigen removal.

Study Design/Methods: Patient history and engraftment analysis came from medical chart review. Column agglutination (Gel) and serologic tube tests were used for investigation. Lewis antigen typing was performed for secretor status prediction. Neutralization tests consisted of 0.5 mL human high titer anti-A (h) mixed with 0.5 mL patient plasma and 0.5 mL saline as a control. A1 cells were incubated with doubling dilutions of neutralized plasma and saline control. To extract soluble A antigen, plasma (1 mL) from an A1 non secretor was incubated with patient and control cells (100 µL pRBCs) at 37°C for 6 days with daily plasma replacement.

Results/Findings: The patient was a Group A, RhD+ male with a history of B-ALL who received a related Group O, RhD+ haploidentical HSCT in 2021. Full donor (100%) engraftment was confirmed for lymphoid (CD3+) and myeloid (CD33/CD66b+) lines at 1 and 2 years post-HSCT with transfusion-independence. On ABO typing, his RBC were predominantly group O with trace group A RBC present. At 20 months post-HSCT, he was admitted for chronic GVHD (cGVHD) of the gut and lung, confirmed by biopsy and CT chest. He was treated with extracorporeal photopheresis (ECP) × 15, steroids, and immunosuppressives. He required 3 units of O pRBCs for ECP blood prime. During admission, there was an unexpected increase in group A reactivity (Table 1). He typed Le (a-b-). Soluble A antigen was demonstrated through neutralization of human anti-A (h) by patient plasma at titers 1:32 IgM and 1:64 IgG versus saline control (1:512 IgM and IgG). Resolution of ABO was achieved by incubating plasma from a non-secretor over 6 days with patient red cells. Appropriate reactivity was seen with control cells. A follow-up visit demonstrated a decrease in soluble A antigen reactivity once cGVHD improved.

Conclusions: A sudden course of cGVHD appears to have caused a temporary elevated A antigen reactivity in a post-HSCT patient with lung and GI cGVHD. This contributed to an unexpected forward typing problem that was resolved by exchanging soluble glycolipid lacking A antigen with patient soluble A antigen coating group O donor cells.

Days Since Admission	Method		Forward			Reverse	
	Gel	Tube	Anti-A	Anti-B	Anti-A,B	A1 cells	B cells
+3d	X		1+	0		0	2+
+7d	X		±	0		0	2+
+14d	X		1+	0		0	3+
+23d	X		2+	0		0	2+
+23d (Patient cells untreated)		X	4+ mf	0	3+mf	0	2+
+29d (Patient cells after 6-day soluble antigen exchange)		x	0	0	0		
+30d	X		2+	0		0	2+
+255d	x		1+	0		0	2+

TABLE 1: Patient's course of blood types.