Hodgkin lymphoma (HL), and non-Hodgkin lymphoma (NHL) have not been well documented.

Methods/Case Report: All autologous stem cell collection patients between 2017-2021 were retrospectively reviewed and categorized by primary diagnosis. Patients received a mobilization regimen of G-CSF alone, G-CSF and plerixafor, G-CSF and chemotherapy, or G-CSF, plerixafor, and chemotherapy. CD34+ stem cell collection results were recorded. All units of CD34+ stem cells were processed according to protocol and viability was assessed by Trypan blue method on a thawed aliquot 2 weeks after processing.

Results (if a Case Study enter NA): 385 patients (271 MM, 41 DLBCL, 30 HL, and 43 NHL patients; male: 242; female: 143; age range: 16-78) were identified. Binomial logistic regression demonstrated that use of plerixafor with G- CSF was negatively associated with meeting CD34+ goal collection (OR= -1.57, p= 0.003) compared to G-CSF alone. This result was particularly true for MM patients (OR= -1.62, p= 0.014). A pairwise t-test indicated that patients receiving G-CSF and plerixafor had lower CD34+ cell viabilities (t= 2.21, p = 0.028); after Bonferroni correction for multiple comparisons, MM samples also had significantly lower percentage viability than DLBCL (p= 0.003) or HL (p= 0.022) samples.

Conclusion: A higher percentage of patients mobilized on G-CSF alone were able to meet collection goal versus patients who were mobilized on G-CSF and plerixafor. Patients who received G-CSF and plerixafor had significantly lower viability than those who received G-CSF alone. We hypothesize these findings to be due to lower baseline mobilization for patients on G-CSF and plerixafor.

Two Individuals with Rare Blocked Antigen Phenomenon and Coinciding Warm Autoantibody Mimicking Alloanti-Jk3 Resolved with JK Analysis

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Introduction/Objective: Kidd antigens can bind complement (C3) as well as Kidd specific warm autoantibodies (WAAb). An 838G>A single nucleotide variant (SNV) defines JK*01 and JK*02 which codes the antithetical Jka and Jk b, respectively. Both alleles translate the high prevalence (>99%) Jk3 (JK3). The 130G>A is associated with weak Jka and weak Jkb expression. In vivo binding of non-agglutinating globulins can cause false-negative phenotypes by means of the blocked antigen phenomenon (BAP).

Methods/Case Report: Transfusions were requested for a 74-year-old Caucasian (CA) female with Evan's Syndrome, and an 85-year-old African American (AA) female with metastatic uterine cancer. Both had a history of nonspecific WAAb. Direct antiglobulin testing (DAT) detected moderate in vivo sensitization of IgG and C3. They phenotyped Jk(a- b-) with untreated and EDTA glycine-acid (EGA) treated IgG DAT-negative cells. Their serum contained anti-Jk3 reactivity, while a panreactive WAAb in the eluate reacted with Jk3- donor and EGA treated DAT-negative autologous cells. Weak anti-Jka and anti-Jkb reactivity remained in the alloadsorbed serum of the antithetical adsorbing cells.

Genetic testing of the CA revealed JK*01W.01(130A)/02 alleles, while cDNA confirmed the alleles would be transcribed into mRNA. Sequencing of the AA detected 130G/A, and 838G/A as well as other silent mutations predicting either a Jk(a+wb+) or Jk(a+b+w) phenotype. The CA received one compatible JK:-3 transfusion, and both individuals benefited from multiple least incompatible transfusions of Jk a+ and/or Jk b+ donors with expected hemoglobin increases (1 g/dL per transfusion). The CA serologically phenotyped Jk(a-b+) 132 days later following prolonged immunosuppressive therapy while a normocytic normochromic anemia and the WAAb persisted. No follow up evaluations of the AA are available.

Results (if a Case Study enter NA): NA

Conclusion: Unexpected BAP can confound immunohematology testing and lead WAAbs mimicking alloanti-Jk3 to be mischaracterized as allogeneic. By predicting phenotypes, genetic analysis can aid serological techniques in antibody characterization and help circumvent complications searching for rare JK:-3 donors.

Leukostasis aggravated by red blood cell transfusion in a chronic lymphocytic leukemia patient

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Introduction/Objective: Leukostasis/symptomatic hyperleukocytosis is commonly seen in acute leukemias and is characterized by high blast counts and symptoms of decreased tissue perfusion with a one-week mortality of 20-40%, if left untreated. It is a rare complication in chronic lymphocytic leukemia (CLL) and is seen in CLL patients with white blood cell (WBC) counts > 500x10^9/L. Studies have shown that transfusion of blood

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