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Hematologic Malignancies are Associated with Lowered Red Cell Alloimmunization

Nikolic Turnic*

Department of Internal Medicine, Jose Maria Morales Meseguer University General Hospital, Murcia, Spain

***Corresponding author:** Nikolic Turnic, Department of Internal Medicine, Jose Maria Morales Meseguer University General Hospital, Murcia, Spain; Email: Nikolicturnic@gmail.com

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Introduction

Transfusion of red cells exposes recipients to non-self-antigens, which can lead to the production of alloantibodies. Although earlier alloimmunization necessitates the use of only donor blood that is antigen-free, accidental re-exposure can result in severe hemolytic transfusion responses. Transfusion of ABO/RhD compatible units to all red cell recipients helps to prevent alloimmunization and its effects. Furthermore, because the number of transfusions is highly associated with the chance of alloimmunization, matching beyond those antigens is indicated for select individuals thought to be at high risk of alloimmunization due to recurrent exposure. As a result, patients with hemoglobinopathies and Myelodysplastic Syndrome (MDS), who are frequently transfused over lengthy periods of time, receive red cell units matched for the most immunogenic and clinically significant antigens C, c, E, e, and K in various high-income nations.

The recipient's immune system's ability to elicit a humoral alloimmune response in response to red cell alloantigen exposure is likely influenced by his or her clinical state. While oncological patients were thought to have a similar alloimmunization risk to the general transfused population, certain investigations found that MDS patients had a high rate of alloimmunization. These studies did not take into consideration cumulative red cell exposure, which is commonly high in oncological patients and is a major factor in alloimmunization. As a result, the impact of disease-specific characteristics has yet to be determined. Furthermore, cancer types differ in terms of their intrinsic immunobiological properties and the immunosuppressive nature of their treatments. As a result, alloimmunization rates found in a diverse group of oncology patients cannot be generalised to specific diseases.

Malignancies and Red Cell Alloimmunization

In this nested case-control study, we looked at whether patients with hematologic malignancies and solid tumours had a higher likelihood of developing red cell alloantibodies than the overall transfused patient population. The incidence of clinically relevant alloantibodies against red cell alloantigens was reduced threefold in patients with acute leukaemia (of either myeloid or lymphoblastic origin) and mature (B- or T-cell) lymphomas. The alloimmunization rate among patients treated for various hematologic malignancies or solid tumours, on the other hand, was comparable to that of the non-malignant patient population. Although previous research found similar or even higher rates of red cell alloimmunization in oncological patients, these prevalence-based studies did not account for the large number of transfusions these patients often undergo. The cumulative transfusion dose, on the other hand, is a well-known factor of alloimmunization. As a result, the found positive relationships could be related to the extensive red cell transfusion assistance that is commonly required in the treatment of some cancers, rather than disease-specific characteristics. There have been no studies that have compared the hazards of alloimmunization in distinct oncological disorders. Our data imply that immunosuppressive therapy, particularly dose-intensive immunosuppressive therapy, has an impact on alloimmunization. This appears to be biologically feasible. Cyclophosphamide, purine nucleoside analogues, and anthracyclines, among other traditional cytotoxic drugs commonly employed in the treatment of acute leukaemia and lymphoma, are known to cause sustained (primarily nave) CD4 T-cell and B-cell depletion. Furthermore, corticosteroids, a type of immunosuppressant that we previously observed to protect against red cell alloimmunization, are frequently used in chemotherapy regimens. Patients who received anti-lymphocyte targeted medicines had a much lower rate of red cell alloimmunization (*i.e.* ATG, alemtuzumab, and rituximab). ATG is well-known for its potent and long-lasting T-cell depletion properties. Antibodies against many B- and even plasma cell-specific antigens are also seen in ATG preparations. In agreement with this, rituximab-induced B cell eradication has been linked to reduced primary and re-calls vaccination responses. Finally, in the setting of HSCT, whether autologous or allogeneic, we observed significantly decreased alloimmunization rates, which looked to be sustained at least for the first six months following transplant. Even though we can't rule out the possibility that the 8 cases of alloimmunization after an allogeneic HSCT were caused by donor-recipient red cell antigen mismatches (in addition to transfusion exposure), these findings are in line with previous research that found anti-D formation to be uncommon in RhD-negative HSCT recipients exposed to RhD. Adaptive immune cell reconstitution takes 6–12 months after HSCT, depending on age-related thymic function, kind of stem cell harvest, and intensity of T-cell depletion techniques, but humoral immunity may remain weak even after many years. Although treatment-induced immunosuppression appears to be the most likely explanation for our findings, additional unmeasured factors associated with therapy (such as co-morbidities and illness stage) may have interacted with disease-specific immune responses. As a result, we can't rule out the potential that some of the reported effects are due to the diseases themselves, such as the production of an immunosuppressive but tumor-tolerant state by malignant cells exploiting host immune evasion systems.

MDS who received treatment, like those with acute leukaemia and mature lymphoma that were aggressively treated, had a lower rate of alloimmunization. As a result, the decision to transfuse extended donor-matched products to this patient population should be based on other variables associated with an enhanced alloimmune response, such as a high transfusion burden, rather than the MDS diagnosis itself. Finally, independent of treatment, the alloimmunization RR in patients with chronic lymphocytic leukaemia (CLL) appeared to be higher than in lymphoma patients, while we recognise that the number of CLL patients in the current study is insufficient to support such a notion. CLL, on the other hand, is characterised by severe immunological problems, such as the non-clonal production of IgG

autoantibodies directed against blood cell antigens. The condition appears to disrupt normal regulatory potential, according to observations. Antimicrobial vaccine responses are frequently compromised in CLL patients, which appear to contradict these findings.