INTRODUCTION

TRALI (transfusion related acute lung injury) is an immune-mediated event. The proposed mechanism for TRALI involves donor antibodies present in the transfused blood component reacting with cognate antigens on a recipient’s neutrophils (PMNs). The donor antibodies can be anti-HLA Class I/II or anti-HNA. Via unknown mechanisms, antibody-coated PMNs release proteases, oxygen radicals, and acidic lipids, thereby damaging the pulmonary vascular endothelium. This allows for the release of fluids into the adjacent pulmonary interstitium and alveoli, resulting in pulmonary edema and the symptoms of TRALI (image 3).

All immunizing events that may cause donors to form anti-HLA or anti-HNA antibodies include pregnancy and previous blood transfusion. Strategies implemented to prevent TRALI have decreased the number of cases overall, but the risk of TRALI remains. In the case of plasma transfusion, limiting supplies to male donors only (low risk of alloimmunization) has reduced TRALI.

PATHOGENESIS

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CLINICAL PRESENTATION

The symptoms of TRALI include hypoxemia, tachycardia, fever, mild hypotension, cyanosis, and bilateral non-cardiogenic pulmonary edema that progresses to involve the entire lung fields. Patients have a normal central venous pressure and low to normal pulmonary wedge pressure. These symptoms have a rapid onset and progression and always present within 6 hours of transfusion of plasma-containing blood components. The diagnosis of TRALI is made clinically and is confirmed by HLA typing the recipient and testing donor serum for the presence of HLA or HNA antibodies. A cognate match between a donor antibody and recipient antigen confirms the diagnosis of TRALI.

REFERENCES

2. Kleinerman, S, Kor, D.J. Transfusion-related acute lung injury (TRALI). In: UpToDate, Rose, BD (Ed), UpToDate, Waltham, MA, 2013.