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Blood transfusions and infected lungs-Worse outcomes?

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With an estimated incidence of 0.8 to 2.6 per 10,000 transfused units, TRALI remains the leading cause of transfusion-related mortality.¹ It is defined as acute lung injury that develops during or within 6 hours after transfusion of one or more units of blood or blood components.² A research group at UCSF and the Mayo Clinic performed a prospective case study with controls, from 2006 through 2009, by using an active surveillance system, that utilized blood gas information to identify patients who might have TRALI when they had a PaO₂/FiO₂ ratio of <300 mmHg within 12 hours of receiving a transfusion of any blood component¹. Eighty-nine patients from the sample of patients receiving 463,207 units of blood and blood components transfused, were identified as having TRALI by two physician experts using predetermined criteria. One hundred and sixty four other patients were the transfused controls. The factors that were found that might explain why some patients develop TRALI compared to those patients who received blood from the same donor but did not develop TRALI were: 1] patients were found to have received more units of every component and so probably received larger quantities of antibody; 2] the HLA class of the cognate antibody appeared to matter; 3] the strength of the anti-HLA-class II reaction and 4] the presence or absence of patient factors that increased the risk for TRALI.¹ Patient risk-factors for TRALI in this study were: shock, chronic alcohol abuse, patients with intravascular fluid overload, and current smoking¹.

These same authors are now publishing data from this study regarding the clinical outcomes of the patients studied. Patients who were intubated within 24h before or 24h after transfusion of the first unit were evaluated for the duration of mechanical ventilation [MV].³ Duration of hospital and intensive care unit stays were also evaluated, as were deaths. The principal findings of this new analysis were that : 1] TRALI produced vital sign changes suggestive of a systemic inflammatory response {SIRS} [increased body

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temperatures, tachycardia, tachypnea, decreased blood pressure and need for mechanical ventilation]; 2] TRALI patients had a decrease in platelet counts and an increase in neutrophils and pro- and anti-inflammatory cytokines in their peripheral blood; 3] TRALI patients had significant in-hospital morbidity, requiring longer duration of mechanical ventilation and longer ICU stays; 4] possible TRALI cases had higher morbidity and mortality compared to TRALI cases.³

The study is limited in that the control patients were not matched for baseline characteristics and comorbidities and matching might have led to different findings in terms of clinical course and outcomes. However, matching would not have allowed the investigation of TRALI incidence and risk factors.

The new analysis [3] does suggest that it is difficult to separate TRALI from acute infections and is associated with a mortality rate of 17%. These results suggest that infected patients may be more susceptible to TRALI; this could be tested by limiting transfusions to septic patients or to patients with known infections and comparing outcomes to similar infected patients who receive transfusions.

There is increasing data that limiting transfusions is not inferior to more liberal transfusion practices⁴⁻⁵. The decision to transfuse blood products should be guided by individual patient characteristics and symptoms, and the benefit of treating anemia or coagulopathy needs to balance with the desire to avoid unnecessary transfusion and the associated side effects. Recipient risk factors (e.g. liver transplantation, alcohol abuse, tobacco use, shock and positive fluid balance)¹ should be carefully considered, and characteristic clinical presentation of TRALI should lead to immediate discontinuation of the transfusion. Given the many similarities between clinical symptoms of TRALI and SIRS, as reported by Looney and colleagues³, it is possible that the TRALI diagnosis can be delayed in patients with pre-existing systemic inflammation before transfusion. The reported finding of higher morbidity and mortality among patients with *possible TRALI* (i.e. those with ALI or ARDS risk factors present at the time of transfusion) as compared to TRALI (with no ALI or ARDS present at the time of transfusion) is also in accord with the above hypothesis, and may in part be attributed to a delay in its diagnosis and management. Clinicians should, therefore, remain especially vigilant for signs of TRALI in patients with pre-existing SIRS or pulmonary symptoms. Perhaps limiting transfusions to patients with infections will be a superior strategy.

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