Emergency Medicine: Open Access
ISSN: 2165-7548

Tsalis and Blouhos, Emergency Med 2014, 4:5
DOI: 10.4172/2165-7548.1000206

Review Article Open Access

Transfusion-Related Acute Lung Injury

Konstantinos Tsalis[1*] **and Konstantinos Blouhos**[2]

[1]*Professor of Surgery, D' Surgical Department, Aristotle University of Thessaloniki, Central Macedonia, Thessaloniki, Greece*

[2]*Staff Surgeon, General Hospital of Drama, Eastern Macedonia, Drama, Greece*

[*]**Corresponding author:** Konstantinos Tsalis, MD, MSc, PhD, HPB Surgeon, Professor of Surgery, D' Surgical Department, Aristotle University of Thessaloniki, Central Macedonia, Thessaloniki, Greece Kotyoron 19, 55131, Kalamaria, Thessaloniki, Greece, Tel: +306932404130; E-mail: ctsalis@yahoo.gr

Received date: April 17, 2014, **Accepted date:** August 22, 2014, **Published date:** August 29, 2014

Abstract

Nowadays, transfusion-related acute lung injury (TRALI) is considered the leading cause of transfusion-associated mortality. TRALI is defined as new acute lung injury (ALI) during or within 6 hours after transfusion in patients with no other ALI risk factors. Immune-mediated TRALI accounts for over 80% of reported cases and is mediated by donor antibodies to human leukocyte and neutrophil antigens. Immune-mediated TRALI is mainly associated with transfusion of blood products containing large amounts of plasma from high-risk donors. Evidence-based transfusion guidelines and reduction of rich in plasma blood products transfusion from high-risk donors will likely decrease the risk of transfusion-associated morbidity and mortality.

Keywords: Transfusion; Acute lung injury; Risk factors; Prevention

Historical Background

Transfusion-related acute lung injury (TRALI) was first reported in 1951 and 1957 [1]; however, it was not until 1985, with the report of a series of 36 patients, that TRALI was recognized as a distinct clinical entity [2]. With more aggressive transfusion support and increased recognition of this syndrome, TRALI has become a common clinical complication of transfusion. Nowadays, the US Food and Drug Administration acknowledge the syndrome as the leading cause of transfusion-related mortality [3]. In the United States, the incidence of TRALI before 2007 is estimated at 1:4000 to 1:5000 units transfused (1 in 5000 units of packed RBC, 1 in 2000 plasma-containing components, and 1 in 400 units of whole-blood-derived platelet concentrates), though preventive measures may have reduced this incidence to 1:1200 by 2009 [4]. TRALI mortality has been estimated at 6%, considerable lower than the estimated mortality of other forms of acute lung injury/acute respiratory distress syndrome (ALI/ARDS) [5].

Definition

According to the American-European Consensus Conference of ARDS, the criteria for ALI are: (a) acute onset; (b) pulmonary artery wedge pressure: ≤ 18 mm Hg, or a lack of clinical evidence of left atrial hypertension; (c) bilateral infiltrates on frontal chest radiograph; and (d) hypoxemia defined as ratio of PaO2/FIO2 ≤ 300 mm Hg regardless of PEEP level [6].

TRALI is defined based on the National Heart Lung and Blood Institute Working Group on TRALI definition [7]. In patients with no ALI immediately before transfusion and no other ALI risk factor, a diagnosis of TRALI is made if there is new ALI after transfusion, and the onset of symptoms or signs is during or within 6 hours after transfusion. The definition excludes patients with ALI before transfusion; even though worsening of an existing ALI after transfusion can be attributed to the transfusion, defining this form of TRALI is problematic.

Septic shock
Sepsis syndrome without hypotension
Aspiration of gastric contents
Near-drowning
Disseminated intravascular coagulation
Pulmonary contusion
Pneumonia requiring ICU care
Drug overdose requiring ICU care
Fractures of long bones or pelvis
Burn, any present of body surface
Cardiopulmonary bypass

Table 1: ALI risk factors [56]

In patients who have other ALI risk factors (Table 1) can also develop TRALI, and thus TRALI should not be excluded from consideration in these patients. The presence of an ALI risk factor does not mean the patient will definitely develop ALI; the incidence of ALI in prospective studies of patient groups with ALI risk factors is less than 50%. New ALI in a transfused patient with an ALI risk factor could be mechanistically due to the transfusion and/or the risk factor, i.e. TRALI and/or ALI due to the risk factor. In such patients who have another ALI risk factor, the diagnosis of TRALI can be difficult. Thus, the Canadian Consensus Conference proposed the term "possible TRALI" for new ALI in a transfused patient who also has another ALI risk factor [8].

Pathophysiology

Two theories have been proposed to explain the pathophysiology of TRALI. The first suggests that an antibody-mediated reaction after transfusion of anti-granulocyte antibodies into patients who have leukocyte that express the cognate antigens is responsible for TRALI. The second postulates that TRALI is mediated by an interaction between biologically active mediators in banked blood products and the lung [9].

It is consensus opinion that neutrophil activation is the key mechanism by which antibodies to human neutrophil antigens (HNAs) and to HLA class I mediate TRALI by direct binding to the cognate antigen on the surface of the neutrophil or, in the case of HLA class I antibodies, possibly by binding to HLA class I molecules on pulmonary endothelial cells, which leads to neutrophil trapping by the neutrophils Fc receptors and subsequent neutrophil activation. Consequently neutrophil-dependent ALI is characterized by neutrophilic inflammation of the lung and disruption of the lung alveolar-capillary permeability barrier [10].

HLA class II molecules are usually not expressed on neutrophils and endothelial cells. HLA class II molecules can be found on alveolar macrophages, which are unlikely to be reached by a transfused antibody before the breakdown of the blood-lung barrier and on some blood leukocytes, including monocytes [11]. (Kopko et al., 2003) was the first to demonstrate that incubation of test monocytes with serum containing anti-HLA class II antibodies increases the percentage of cytokine-positive cells whenever the corresponding antigen was present [12]. This finding was corroborated by a subsequent report on the release of leukotriene B4 (LTB4) from antigen-positive monocytes being incubated with a serum containing HLA class II antibodies, indicating that in HLA class II antibody–induced TRALI, HLA class II antibodies bind to monocytes that express the cognate antigen and thereby induce the release of potent cytokines and LTB4 [13]. Currently available data from large series and published hemovigilance reports indicate that HLA class II antibodies matching the recipient's antigens are present in 50% of all TRALI cases and thus represent the most frequently detected matched antibodies in implicated donors [14].

The biologically active mediator model postulates that TRALI is the result of two events. The first event can be caused by a variety of insults to the pulmonary vascular endothelium such as sepsis, cardiopulmonary bypass, hematologic malignancy, thermal injury, and trauma, resulting in pulmonary endothelial activation and neutrophil sequestration, and the second event is the transfusion of biologically active mediators (lipids, cytokines) that activate adherent neutrophils leading to endothelial damage, capillary leak, and TRALI [15]. Such lipids have been shown to be breakdown products of cell membrane phospholipids that form during prolonged storage of cellular blood components. In particular, Lysophosphatidylcholine has been identified as a component of such blood products that can prime neutrophils [16]. However, recent large case control studies in general transfused patients failed to demonstrate an association between such biologically active lipids and an increased risk of TRALI [17-19]. In addition, non-polar lipids in the plasma of stored leukoreduced red blood cells can also prime neutrophils in vitro, but the clinical relevance of this observation remains to be determined [20].

Trali Risk Factors

High-risk donors

The donor is defined as implicated in TRALI if antibodies to HLA class I or II antigens or antibodies to HNAs with specificity against an antigen present on the recipient's white cells are detected [21]. The high-risk donors may include all female donors or only those previously pregnant; all transfused donors; and donors with previously demonstrated WBC antibodies. Discussion of laboratory evaluations of donors and acceptability of various blood fractions of these donors require further consideration and is well depended on availability of blood resources [22].

Transfusion risk factors

All blood components have been implicated in TRALI, but those containing large amounts of plasma are mainly responsible. The risk of developing delayed TRALI is greater with transfusion of plasma-rich blood products, fresh frozen plasma, and platelets than transfusion with packed RBCs [23]. TRALI has also been described after intravenous immunoglobulin administration, a pooled plasma derivative rich in **polyvalent**-IgG **antibodies** [24]. In the study by (Khan et al., 2005) the odds ratio for developing delayed TRALI was 2.48 (95% CI, 1.29-4.74) in patients receiving fresh frozen plasma, 3.89 (95% CI, 1.36-11.52) in those receiving platelets, and 1.39 (95% CI, 0.79-2.43) in those who received RBC transfusions alone [25].

Transfusion of multiple units of RBCs has long been considered a risk factor for ALI [26]. Massive transfusion has been defined as transfusion of >10 units of RBC's or whole blood within a 12-hr period or 15 units within a 24-hour period. The incidence of ARDS among those massively transfused patients may be as high as 57% [27]. Miller et al studied risk factors for developing ARDS in a cohort of 4,397 patients who sustained blunt trauma. In this study, the 24-hour transfusion of >10 packed red cells was an independent predictor for developing ARDS [28].

Although massive transfusion has long been identified as a risk factor for ALI/ARDS, transfusion of a smaller blood volume has until recently not been well studied and has not generally been considered a risk factor for ALI. However, several studies, reported over the last 5 years, have demonstrated that even a single unit of blood increases the risk of patients with other risk factors for ALI for developing ALI/ARDS. In these studies, the transfusion of blood or blood products was an independent risk factor for the development of ARDS with a pooled odds ratio (OR) of 2.13 (95% confidence interval, 1.75-2.52) [29]. The observation that blood transfusion increases the risk of ALI in critically ill patients is supported by the results from the Canadian Critical Care Trials Group study. In this study, a liberal transfusion strategy was associated with an increased risk of ALI/ARDS (OR 1.5; 95% CI, 0.97-2.49) [30]. Similarly, Zilberberg et al observed that patients who received small numbers (<3 units) of packed red cells had an over two-fold increase in the risk for developing ARDS (OR 2.19; 95% CI 1.41-3.41) relative to patients receiving no transfusion [31].

Clinical manifestations and diagnosis

It is noteworthy that definitions of ALI are based solely on clinical symptoms. Therefore, careful observation of the patient is crucial in diagnosing TRALI. TRALI commonly develops well prior to the 6-hour time-point, often during the first hour of a transfusion [32].

Clinical hallmarks of TRALI include dyspnea, tachypnea, hypoxemia, bilateral pulmonary opacities on chest radiograph (Figure 1a and 1b) [33,34], and absence of evidence of volume overload or cardiac dysfunction. In mechanically ventilated patients, the diagnosis should be considered whenever there is an acute, unexplained worsening in respiratory status that is temporally associated with a transfusion [35].

Echocardiography, measurement of the pulmonary artery occlusion pressure, the brain natriuretic peptide (BNP), the atrial natriuretic peptide (ANP), and analysis of the pulmonary edema fluid, are complementary tests that can be used to exclude cardiac dysfunction and volume overload.

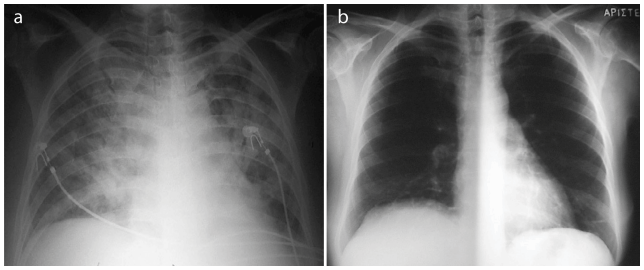

Figure 1: Chest radiograph of a patient: a) immediately after the onset of the transfusion-related acute respiratory distress episode showing bilateral patchy alveolar infiltrates with a normal cardiac silhouette consistent with TRALI; and b) 4 days after the episode showing complete resolution of TRALI (From Tsalis K, Ganidou M, Blouchos K, Vasiliadis K, Betsis D. Transfusion-related acute lung injury: a life threatening transfusion reaction. Med Sci Monit 2005; 11:19-22 with permission).

Echocardiography can be particularly helpful by providing insight into both cardiac function and volume status. Pulmonary artery catheterization and determination of pulmonary artery occlusion pressure provides additional information regarding volume status; however, routine use of this invasive procedure is not warranted [36]. Analysis of the pulmonary edema protein content can aid in the exclusion of circulatory overload. A ratio of 0.75 or more between the protein in the edema fluid and the plasma is consistent with increased permeability, whereas a ratio of 0.65 or less is characteristic of hydrostatic edema [37].

BNP is a polypeptide released by the ventricles and atria in response to volume or pressure overload. Zhou et al demonstrated 81% sensitivity, 89% specificity, 89% positive predictive value, 81% negative predictive value, and 87% accuracy of BNP in diagnosing volume overload. When interpreting BNP levels, it is important to compare post-transfusion with pre-transfusion levels; a normal BNP level may exclude volume overload and post-transfusion increases in the BNP level favor volume overload [38]. ANP is another major peptide that has been postulated as a superior marker for volume overload because of its rapid release into the blood following atrial stretch and its shorter half-life of 2 to 4 minutes. However, the role of BNP and ANP in TRALI remains to be determined [39].

Differential diagnosis

Transfusion-associated circulatory overload is chief among entities that cause acute respiratory distress in critically ill patients [40]. Anaphylactic transfusion reaction, acute hemolytic transfusion reaction, transfusion of a contaminated blood product must also be considered and ruled out because signs and symptoms may include respiratory distress. Sepsis usually manifests as hypotension, fever, and even circulatory collapse, often accompanied by respiratory distress [41].

Treatment

There is no specific treatment for TRALI. As with other forms of ALI, management of TRALI requires proper supportive care. In most cases, TRALI is self-limited and carries a better prognosis than other causes of ALI. If the patient is still being transfused, the transfusion should be stopped. For mild cases, supplemental oxygen and routine supportive care may be sufficient. For severe cases, mechanical ventilation, restrictive fluid policy, invasive hemodynamic monitoring, and vasopressors may be required [42]. In rare cases, the hypoxemia resulting from TRALI can be so severe that extracorporeal oxygenation may be required as a temporizing measure while the lungs heal [43]. In patients who require mechanical ventilation, a low tidal volume strategy, as would be used in other cases of ALI, should be employed [44]. While several case reports describe the treatment of TRALI with glucocorticoids, no randomized, controlled trials have studied this therapy in TRALI [45].

Primary prevention

In 2006, the American Association of Blood Banks clarified the TRALI risk reduction requirements: (1) blood collecting facilities should implement interventions to minimize the preparation of high plasma volume components from donors known to have or be at risk for leukocyte alloimmunization; (2) blood transfusion facilities should work toward implementing appropriate evidence-based hemotherapy practices to minimize unnecessary transfusion; (3) blood collection and blood transfusion facilities should monitor the incidence of reported TRALI and TRALI-related mortality [46].

The American Association of Blood Banks recommended the reduction of the transfusion of plasma and platelets from probable high-risk donors. Receipt of female plasma is a strong risk factor; decrease in the incidence of TRALI after conversion to male predominant plasma containing products, such as frozen plasma, fresh frozen plasma (FFP), cryosupernatant plasma, apheresis plasma and liquid plasma, support the effectiveness of this approach [47]. Despite the move toward predominantly male plasma for transfusion, plasma from female donors continues to be important to support the needs of transfused patients. To further reduce TRALI risk in female plasma-rich components, clinical evidence support the suggested screening for strong anti-HLA-Class II in platelet donors and the development of high-throughput granulocyte immunofluorescence test by flow cytometry for antibody to HNA methods to screen for known and unknown human neutrophils antigens. However, the majority of plasma from female whole blood donors can be sent to fractionators to be processed into plasma protein products such as intravenous gamma globulin and albumin [48].

Evidence-based transfusion guidelines will likely decrease the number of transfusions and the risk of associated morbidity and mortality. A restrictive RBC transfusion strategy is at least as effective as a liberal one [49]. Most commonly, plasma is used to correct coagulopathy in bleeding patients or patients being prepared for invasive procedures. However, (Abdel-Wahab et al., 2006) prospectively examined coagulation screening test results before and

after FFP transfusion. The international normalized ratio normalized in only 0.8% of the patients and decreased to at least halfway toward normalization in only 15% [50]. Adoption of plasma transfusion strategies based on these and other studies would undoubtedly reduce the amount of FFP transfused [51]. Prophylactic platelet transfusion in thrombocytopenic patients has been a mainstay of therapy for many decades. Historically, a platelet count of 20 × 103/μL was used as the trigger prompting platelet transfusion in patients in clinically stable condition. Research in the last decade has indicated that a transfusion trigger of 10 × 103/μL does not result in an increased risk of bleeding or RBC transfusion but does result in a reduction in the number of platelet transfusions. Clearly, adherence to lower platelet transfusion thresholds and reserving them for treatment of clinical bleeding can reduce the number of patient exposures [52].

Strategies to minimize the risk of transfusing biologically active lipids and cytokines have also been presented. Because these substances accumulate in cellular components during storage, the use of RBCs less than 14 days old and platelet concentrates less than 2 days old would mitigate the effects of these compounds. However, clinical evidence is lacking to support a fresh blood policy; moreover given current requirements regarding serologic and bacterial detection testing, providing platelets less than 2 days old is impractical in a clinical setting [53].

Secondary prevention: Management of donors associated with TRALI

A donor is definitely implicated if shown to have: a positive cross match with the recipient's leukocytes or an antibody with specificity corresponding to a known recipient cognate antigen or an antibody with HNA-3a specificity. A donor is possibly implicated if shown to have: a leukocyte antibody but is cross match untested with the recipient or antibody specificity is not defined or is untested or recipient cognate antigen type is not known [54]. All donors who are definitely implicated are permanently deferred and in-date components withdrawn. Washed RBCs may be used if the person is a rare blood type. These red cells are frozen and/or washed. Their whole plasma can be sent for fractionation. Large volume plasma products such as frozen plasma, cryoprecipitate and platelets from these donors are discarded. All in-date components are withdrawn [55].

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