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Review Article

Transfusion related acute lung injury-TRALI: a review

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ABSTRACT

Acute pulmonary damage caused by transfusion is characterized by the sudden onset of respiratory distress in newly transfused patients within 6 hours after the transfusion, bilateral infiltrative changes in chest X-ray, PaO2/FIO2 <300 mmHg, absence of other risk factors for acute lung injury and absence of signs suggesting cardiogenic origin of pulmonary edema. Being one of the most serious complications of blood transfusion, plasma is the most involved factor, although all blood components can cause it, and is caused by antigen reactions/leukocyte antibody and lipid activity with ability to modify the biological response on primitive leukocytes. The diagnosis is based on the integration of clinical, radiological and gasometric elements, ruling out the rest of the possible causes of acute lung injury. Its differential diagnosis should include hemodynamic overload, anaphylactic reaction, bacterial contamination of transfused blood products and transfusion hemolytic reaction. The treatment is supportive measures based on the needs and does not differ from the treatment of acute lung injury secondary to other etiologies, severe cases require endotracheal intubation and mechanical ventilation while the non-severe can be managed with oxygen therapy.

Keywords: Blood products, Pulmonary injury, Plasma, Risk factor, Respiratory insufficiency, Transfusion

INTRODUCTION

Acute adverse reactions of transfusion occur within the first 4hours post transfusion, having an immune or non-immune origin; whereas the delayed reactions occur 48hours or more after the transfusion; identifying in these a mainly immune component.[1] Some old definitions of massive transfusion were; replacement of normovolemia in 24hours (7% of the ideal weight in adults, 9% in children), a replacement greater than 50% of the cardiac output in 3hours.[2] Transfusion of more than 4 units of blood in one hour, or the reception of 10 or more globular units in 24hrs[3]. Currently the definition of massive transfusion is receiving 10 units in 6hours, as this time parameter shows an increase in morbidity and mortality, taking into account that each liter of crystalloids, 500mL of colloid, or each globular unit, plasma unit or platelet apheresis, is considered a unit of resuscitation fluid.[4]

Mortality increases threefold in patients receiving more than 3 units of resuscitation fluid within 30 minutes of arrival.[3] The hemodynamic state of the patient should be considered, as well as the pathophysiological responses that are triggered when homeostasis is altered, to evolve to homeorresis and even to a state of shock, since in an acute state such as hypovolaemia, the mechanisms of adaptation cannot be activated properly, since the blood loss quickly exceeds the oxygen demand, given by the depletion of red blood cells, which begins a cascade of reactions whose trigger is hypoxia and tissue ischemia, unlike chronic situations where the body is able to compensate for the damage.[5] All hemodynamic control requires early and efficient attention; stopping the bleeding, controlling the coagulopathy, favoring a critical perfusion and minimizing the harmful responses that come with the shock state and the resuscitation fluids, because the complications of the massive bleeding are related to the consequences of the hemorrhagic shock and the massive replenishment as ischemia, tissue hypoxia, hypothermia, acidosis, thrombocytopenia, coagulopathy, hypocalcemia, hyperkalemia, fatal hemolytic reactions linked to ABO-Rh incompatibilities and transfusion respiratory distress, which have a frequency of up to 20%.[6,7] Transfusion of red blood cells is indicated solely and primarily to combat hypoxemia, that is, the need to increase oxygen delivery in patients who are unable to meet the demands through the normal cardio-pulmonary compensatory mechanisms, otherwise it can impede efficient ventricular ejection, indirectly favoring the increase in pulmonary vascular resistance, with the rapid entry of blood components; which in turn generate deleterious effects in the lung, causing acute pulmonary respiratory failure syndrome (SIRPA), circulatory overload by transfusion (TACO) and acute pulmonary injury secondary to transfusion (TRALI), which present with sudden-onset dyspnea, bilateral pulmonary infiltrates (on chest x-ray) and hypoxia less than or equal to 90%, which is a common clinical picture of these adverse responses.[8]

Methods

It has been made a bibliographic research in diverse platforms such as PubMed, Medline, Science Direct and Cochrane, using the keywords: transfusion, pulmonary injury, plasma, risk factor, respiratory insufficiency, blood products. The research was made during the year 2018 and the articles included are from years 2000 to 2018.

Definition

Acute pulmonary damage caused by transfusion or TRALI is defined as the appearance of an acute respiratory distress in a newly transfused patient is one of the most dangerous adverse reactions to the use of blood products. It is a clinical syndrome characterized by acute respiratory failure and non-cardiogenic pulmonary edema during or after a transfusion of blood products[1] (Table 1).

Table 1: Definition of acute pulmonary injury related to transfusion (TRALI).

Definition	Criteria
Suspicion of TRALI	• Acute onset, 6 hours after the blood transfusion • PaO2 / FiO2 <300 mmHg or worsening of the P to F ratio • Bilateral infiltrative changes in chest radiography • No signs of hydrostatic pulmonary edema (PcP ≤ 18 mmHg or central venous pressure ≤ 15 mmHg) • No other risk factor for acute lung injury
Possible TRALI	• Same criteria that we suspect of TRALI + risk factor present for acute lung injury
Late TRALI	• Same criteria as possible TRALI + Appearance within 6-72 hrs. After the blood transfusion

It was described by Barnard (1951), and in 1983, it was recognized as a distinct clinical entity receiving the term of acute pulmonary injury related to transfusion (TRALI). 2 years later Popovsky and Moore (1985) established the minimum criteria for the diagnosis of TRALI: acute respiratory distress and bilateral pulmonary infiltrations on chest radiography within the first 6 hours after transfusion and absence of volume overload or heart failure , being TRALI the main cause of diseases related to transfusions, their diagnostic criteria are shown in Table 2.[9]

Table 2: Criteria for the definition of acute lung injury caused by transfusion (national heart lung and blood institute working group on transfusion related acute lung injury).

	Criteria
North American European Consensus Conference	1. Acute start 2. Pulmonary artery occlusion pressure <18 mmHg or without evidence of increased pressure in the left atrium 3. Radiology of the thorax: bilateral infiltrates. 4. PO2 / FiO2 <300 mmHg regardless of the level of PEEP applied or SaO2 <90% of air from the breathing room
Additional criteria for TRALI	1. Start in the first 6 hours of the transfusion of blood products 2. Non-existence of LPA prior to transfusion 3. TRALI is possible even though there is another LPA risk factor 4. Massive transfusion should not exclude the possibility of TRALI
APL = acute lung injury; TRALI = Transfusion Related Acute Lung Injury (acute lung injury caused by transfusion). * Added by the working group to recognize TRALI in situations in which arterial blood gas was not obtained.[21]	

Epidemiology

Its incidence is not clear, due to its difficult diagnosis and discordance in definitions. Previously, the prevalence of TRALI was estimated at approximately 1 in 5,000 transfusions. According to a prospective identification of a case recently published in a US study. The risk would be a little less than 1 case of the 12,000 units transfused, being one of the most serious complications of blood transfusion given its mortality (1-10%).[10] Despite the decrease in cases since 2007, it remains the leading cause of death related to transfusions in the United States (according to 2015 data) and the second leading cause (behind post-transfusion circulatory overload, or TACO).[11]

Before 2005, mortality reports for patients receiving massive transfusions were 55% to 65%, in 2007 they decreased from 45 to 50%, due to the protocols that were implemented (MPT).[2] Currently, recent studies in 25 countries showed that the rate of adverse reactions due to transfusion of blood products was 660 per 100,000 individuals; where 3% of these were categorized as severe.[5] The mortality associated with the transfusion was 0.26 deaths per 100,000; Sixty percent of the deaths were due to a circulatory excess associated with post-transfusion circulatory overload (TACO), transfusion-related lung injury (TRALI), and transfusion-related injury.[10]

All blood components are involved, but those containing plasma are more frequent. Platelet concentrates obtained from whole blood have caused the highest number of reactions, followed by fresh frozen plasma, packed red blood cells, whole blood, platelet concentrates by apheresis and granulocytes.[9]

In Mexico, statistical data is not available to assess the complications caused by transfusion. For this reason, haemovigilance networks such as the British network SHOT (Serious Hazards of Transfusion) where we find their data accumulated from 1996 to 2007 (n = 4.334). Shown 5.1% (n = 219) of TRALI cases. In 2007, 561 adverse effects were registered, of which 4.3% (n = 24) correspond to TRALI. Five of these were produced by transfusion of red blood cells, another 5 by transfusion of platelets, one by another type of products and in 13 cases could not be determined.[4] This is how TRALI went from being considered an infrequent complication of transfusion therapy to being currently the main cause of mortality by transfusion, according to haemovigilance systems in Europe and North America.[10]

Pathophysiology

The pathological findings in patients who died from TRALI are consistent with early acute respiratory distress syndrome (ARDS), which shows interstitial and intra-alveolar edema and extravasation of neutrophils. There is a positive correlation between the degree of capillary leukostasis and the amount of proteinaceous flow in the alveolar air spaces. That is, during the first stages of TRALI, neutrophils and endothelial cells of the pulmonary microvasculature establish close contacts, leading to a firm cell adhesion that causes the retention of neutrophils, which immobile, activate microbicidal mechanisms inducing endothelial damage and capillary leakage, that allows the transit of proteinaceous fluid from the vessels to the air spaces, which translates into acute pulmonary edema[9] (Figure 1).

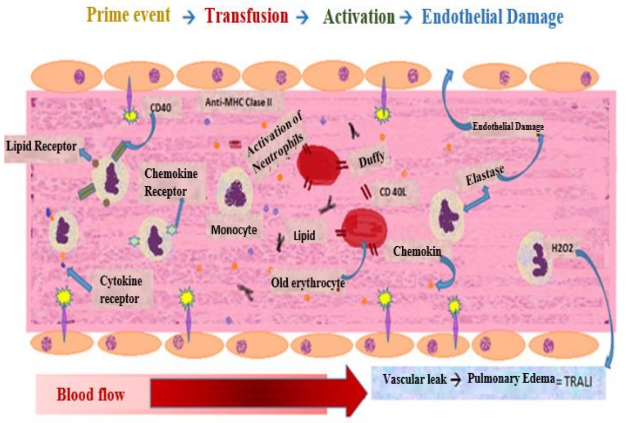

Figure 1: Pathology of endothelial damage.

Pathophysiological classification of TRALI

Immune TRALI

The antibodies involved in TRALI are directed against HLA and human neutrophilic alloantigens (HNA) among others. Being the most documented and known ones directed against HNA-1a, HNA-1b, HNA-2a, HNA-3a (5b) and HLA-A2.[12]

The antibodies from the donor cause the majority of cases of TRALI, although it is documented by antibodies in the blood of the recipient, where it is caused by whole blood or non-depleted red cells of leukocytes.[12]

Multiple mechanisms of neutrophil activation have been proposed in TRALI. Like, antibodies against human leukocyte antigen (HLA) Class I and human neutrophil antigens (HNA) that can bind to neutrophil receptors and trigger their activation (Bux J, 2007, donor women, due to exposure to fetal alloantigens during pregnancy, they have a much higher prevalence of anti-HLA antibodies (19). While anti-HNA antibodies represent less than 5%, antibodies directed against HNA 3a antigen have been associated with severe or fatal cases.[13]

It is also associated with antibodies from donors directed against HLA Class II, in antigen presenting cells, which can bind HLA Class II antigens in monocytes, causing the release of cytokines that activate primed neutrophils.[14]

To make the diagnosis of TRALI mediated by antibodies, antibodies directed against HNA and/or HLA antigens must be detected in the blood donor (s) involved.[15]

TRALI not immune or of two events

In this model, the first event is an aggression that activates the pulmonary endothelium and favors the recruitment and adherence of neutrophils to the capillary endothelium, involving sepsis, trauma and surgery as possible causes. The second event is caused by some trigger that activates the neutrophils that causes the release of cytotoxic factors and endothelial damage with capillary damage that is caused by exposure to biologically active agents or with the ability to modify the biological response present in the blood transfused and produced by blood cells during storage probability of developing transfusion reactions.[9]

TRALI is preceded by an increase in the concentration of IL-8, IL-6 and the α1-antitrypsin elastase complex. It has been postulated that endothelial cells produce IL-8 producing neutrophil attraction to the lung compartment. The conformational changes occurring in the β2 integrins allow close contact of the neutrophils with the endothelial cells, followed by the adherence of neutrophils in the capillaries of small diameter of the lung. L-selectin binds to neutrophils partially, after which E-selectin, P-Selectin (derived from platelets) and intracellular adhesion molecules (ICAM1) facilitate firm adhesion. This adherence is the first event in the pathogenesis of TRALI.[16] The increase of thrombin-antithrombin complexes and the reduction of plasminogen activator activity indicate the activation of coagulation.

The transfused factors responsible for host neutrophil activation can include antibodies to the blood component directed against receptor antigens, or soluble factors such as bioactive lipids that can activate neutrophils. Bioactive lipids and other soluble factors in the transfused blood component can act as biological response modifiers (MRB).[9] Some of the biological response modifiers that have been implicated in the activation of neutrophils are lysophosphatidylcholines (white blood cells and platelets), neutral lipids (from the decomposition of red blood cell membranes), ceramides and soluble CD40 ligand (which accumulates in stored platelet concentrates).[17] In the "threshold model," the threshold is formed by the level of activated pulmonary neutrophils and the ability of transfusion mediators to activate primed neutrophils.

Other factors

The induction of TRALI does not always start from the primed neutrophil, but can be triggered by an activated pulmonary endothelium.[9] In most cases, antibodies or neutrophil priming agents present in the blood component are responsible for the pulmonary reaction, but TRALI has been described in alloimmunized patients receiving blood components containing neutrophils, indicating relevance in patients receiving leukocyte transfusions.[9]

Both, leukocyte antigen/antibody reactions and those derived from the lipid activity with the capacity to modify the biological response on the primo-activated leukocytes require the presence of neutrophils in the receptor. However, there are rare cases of TRALI in neutropenic patients where it is due to the transfusion of biologically active substances, such as vascular endothelial growth factor (permeability factor) and CD40 ligand (synthesis and release of IL-1b, prostaglandin E2 (PGE2) and tumor necrosis factor alpha (TNF-a) of lung macrophages, endothelial cells and fibroblasts, which intensify vascular permeability and inflammation. In addition, HLA type II has been found in the vascular endothelium, so that the transfusion of anti-HLA type II antibodies would be able to produce endothelial damage.[18]

Clinical presentation and diagnosis

TRALI has a clinical presentation indistinguishable from any other acute respiratory distress, except for its temporal relationship with the transfusional act.[9] It appears more frequently between the first and second hour of the transfusion, although the "late" TRALI is mentioned, which does not appear beyond 6 hours after the patient has been transfused, with few authors even allowing up to 72 hrs.[16]

Table 3: Main differential diagnoses and their clinical presentation against TRALI.

Clinical entity	Symptomatology	Clinical evolution
Taco	High pulmonary pressure, hypertension, pulmonary edema (crepitating rales, gallop s3)	Within the first 6 to 8 hours, post transfusion
Trali	Dyspnea, hypoxia, hypotension, pulmonary edema (crackles of rales), fever.	During the first 6 hours of the transfusion
Tad (dyspnea with trasfusion)	Dyspnea of isolated presentation, without pulmonary compromises	During the first 6 hours of the transfusion
Anaphylaxis	Generalized rash, flushing, wheezing, angioedema.	Usually immediately to the transfusion.
Acute hemolytic transfusion reaction	Costal pain, disseminated vascular coagulation, hypotension, fever.	Generally during the first 15 minutes after the transfusion has started.
Bacterial sepsis	Fever, hypotension	Generally during the first 15 minutes after the transfusion has started.

It includes tachypnea of acute onset, foamy lung secretion, hypotension, hypertension, fever, tachycardia and cyanosis. On auscultation, disseminated pulmonary rales are revealed but no third heart sound or gallop or other data of heart failure suggestive of hemodynamic overload are detected; there is also no engorgement of the jugular veins. The central venous pressure and the "wedge" pressure of the pulmonary capillaries are normal when they are determined.[12] The last two mentioned findings are fundamental to distinguish transfusion-related volume overload (TACO), as well as heart failure related to volumetric overload of any other origin; Differential diagnosis will be more difficult when both elements coexist (TRALI + volume overload or TACO).

Diagnosis will be based fundamentally on integrating clinical, radiological and haemogasometric elements, once the rest of the possible causes of ALI have been ruled out (Table 3).

Differential diagnosis

The differential diagnosis of a patient who develops a sudden pattern of respiratory failure after transfusion of blood products should include hemodynamic overload, anaphylactic reaction, bacterial contamination of transfused blood products and hemolytic transfusion reaction. High pulmonary pressure, hypertension, pulmonary edema (crepitating rales, gallop S3) Generalized rash, flushing, wheezing, angioedema, usually immediately to the transfusion.

Treatment

Support treatment based on the needs, and does not differ from the treatment of acute lung injury secondary to other etiologies. Severe cases require endotracheal intubation and mechanical ventilation while non-severe cases can be managed with oxygen therapy Non-invasive respiratory support with continuous positive airway pressure (CPAP) or positive airway pressure (BiPAP) may be sufficient in cases less severe, but endotracheal intubation with invasive mechanical ventilation is often required.[19]

The initial goal of hemodynamic treatment is to ensure adequate perfusion of the end organs. This can be achieved with fluid resuscitation and vasoactive support. Caution should be exercised with the early empirical administration of diuretic therapy, as it may cause hypotension, however, for patients with sustained hypoxemia and demonstrated haemodynamic stability, the administration of diuretic therapy may be an acceptable intervention.[14] In cases resistant to fluid therapy is forced to start inotropic. The use of corticosteroids remains empirical since there is no scientific basis to justify their use.

If a case of TRALI is suspected, the transfusion must be stopped and the hemoderivate sent to the blood bank to support the diagnosis by determining the leukocyte antibodies in the donated package, recurrences are not common in subsequent transfusions but other persons do not receive blood products that contain plasma from the donor[14] (Table 4).

Table 4: Donor management.

State	Criteria
No exclusion	• Donors with HLA antibodies that are not concordant with the receptors but assign them, in PULSE • Donors who are negative for leukocyte antibodies
Exclusion	• Positive donors for leukocyte antigens in concordance with the receptor antigen or having a positive cross-match with the recipient • Donors with HNA antibodies with identified HNA specificity.
Detention of transfusion	• If the Hb is> 10g / dL. • Prothrombin time (PT) <18 sec. • Partially activated thromboplastin time (aPTT) is <35 sec. • Platelet count is> 150,000 • Critical level of precipitates or fibrinogen if a level> 180g / L is reached.

Rule out other causes of pulmonary edema, especially volume overload or cardiac dysfunction.

- Ventilation protective of the lungs.
- Diuretics can be harmful in the hypovolemic patient.
- Corticosteroids have not shown any benefit.[20]

DISCUSSION

As already mentioned in the previous sections, it is secondary to the infusion of any blood product that contains plasma and has been described associated with the transfusion of cryoprecipitates, intravenous immunoglobulins and stem cell preparations. The incidence of this entity remains unknown, because it is underdiagnosed. Frequently the hypoxia that follows transfusion is managed empirically with diuretics, initially thinking that it is volume overload or hydrostatic pulmonary edema.[21]

The diagnosis of suspicion in the majority of cases is purely clinical, to confirm it, a chest x-ray and arterial blood gases are also required. The biggest challenge is to make an adequate differential diagnosis, mainly with volume overload, being the main factor that makes the differential diagnosis difficult, the massive transfusion of blood products and crystalloid solutions.

It is also important to consider the donor of the blood components and identify if there is a risk factor or history

that justifies or supports the diagnosis, for example, multiparous donors that have an increased risk of being carriers of antibodies linked to the development of TRALI.

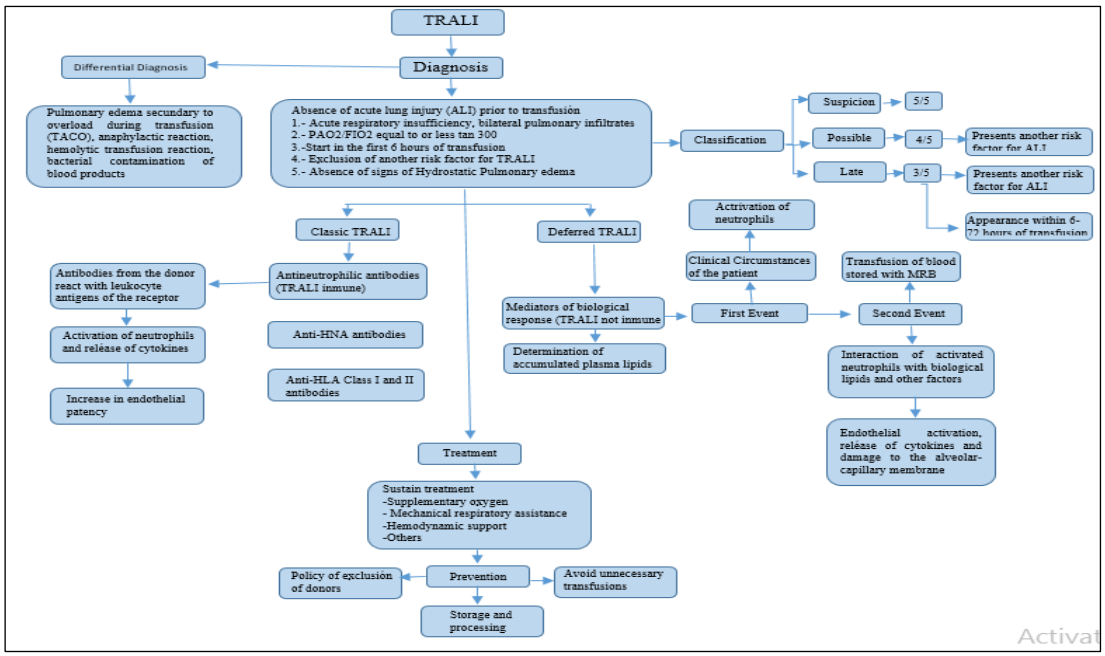

Figure 2: Diagnosis route.

CONCLUSION

TRALI is a rare pathological entity but with serious consequences in undiagnosed patients, due to its similarity with other post-transfusion complications such as hemodynamic overload, it is important to recognize the diagnostic criteria for this entity with special emphasis on identifying risk factors associated with other pathologies, since the premature initiation of medication as diuretics (ideal treatment for hemodynamic overload) may have consequences in patients with TRALI. Although this pathology usually does not exceed 6hours post transfusion, it can occur up to 72hours (delayed TRALI) and once the diagnosis is suspected, it will be based on clinical, radiological and haemogasometric elements as well as the necessary revision of the transfused packages looking for the presence of HNA and HLA antibodies and concluding with correct hospital management defining the severity of TRALI and if endotracheal intubation is necessary.

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