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Pneumology

Acute Post-Transfusion Pneumonia: About a Case

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Abstract **Case Report**

Acute post-transfusion pneumopathy or TRALI (acronym for "transfusion-related acute lung injury"), is defined by an acute respiratory distress syndrome occurring within six hours of a transfusion. It is a classic complication of LBP (Labile Blood Products) transfusion. Recent data have contributed to a better understanding of its mechanisms, two of which are well defined: an immunological conflict on the one hand, and activation of neutrophils by lipid factors on the other. The treatment of TRALI is of lesional pulmonary edema: oxygen therapy and respiratory assistance. We report a case of acute post-transfusion pneumonia, which occurred in a 23-year-old young man following the transfusion of a platelet pellet.

Keywords: Acute transfusion pneumopathy, TRALI, pulmonary edema.

INTRODUCTION

Acute post-transfusion pneumopathy or TRALI is an acute respiratory distress syndrome that occurs no later than six hours after a blood product transfusion. It is a rare accidental complication whose frequency is estimated between 1/5000 and 1/500000 of injected blood products.

The pathophysiological mechanism still remains debated; TRALI would be the consequence of an immunological conflict between donor and recipient either incriminating granulocytic Ag or HLA Ag. Sometimes, when no immunological reaction is proven, the incrimination of the activating lipids which are released during the process of preservation of blood products is evoked. But regardless, acute post-transfusion pneumonitis remains a major pulmonary inflammatory reaction with lesions of the pulmonary capillaries. This lesion leads to an increase in the permeability of the pulmonary capillaries [1].

It is a picture of respiratory distress of variable severity associated with a radiological alveolar interstitial syndrome mimicking a acute pulmonary edema but not cardiogenic.

We report the case of a 23-year-old young patient, followed for bone marrow hypoplasia, who benefited from repeated transfusions of platelets and red blood cells, and who presented an acute respiratory distress syndrome following the transfusion of a pocket of platelet pellet.

CASE REPORT

This is a male patient, 23 years old, with no notable cardiac or pulmonary history, followed, for a year, for autoimmune hepatitis, with discovery of bone marrow hypoplasia; under corticosteroid and anti-androgen therapy, with regular transfusion support made up of repeated transfusion of packed red blood cells and platelets on a regular basis.

Following a platelet transfusion from a pocket of platelet pellet, the patient presented sudden acute dyspnea, a productive cough bringing up mucous sputum, with feverish sensation and chills. The picture appeared two hours after the transfusion, which prompted a consultation in the emergency room.

The examination found a conscious but polypneic patient at 24 cycles/min, tachycardia at 110 bpm, oxygen saturation at 93% AA with the presence on auscultation of bilateral crackles.

The frontal chest X-ray showed bilateral alveolar-interstitial snowstorm images without enlargement of the cardiothoracic index (Figure A).

A thoracic scannographic complement was performed to rule out a COVID infection given the current pandemic context and which objectified alveolar-interstitial damage with scattered ground glass images at the level of the two pulmonary fields (Figure B). A Covid PCR was performed which came back negative.

The patient was hospitalized in our pulmonology department, put on oxygen therapy alone with a good evolution in 24 hours, without recourse to any medical treatment: disappearance of the rales on auscultation, improvement in oxygen saturation to 97% in Ambient Air and become eupneic.

Control radiological imaging (Figure C) proved satisfactory: cleaning of pre-existing lesions.

Faced with the post-transfusion character of the clinical picture, mimicking a acute pulmonary edema picture, and the rapid spontaneous favorable evolution under oxygen therapy alone, clinical and radiological; a transthoracic echocardiogram was performed and then returned to normal; then most likely diagnosis was acute post-transfusion pneumonia or TRALI.

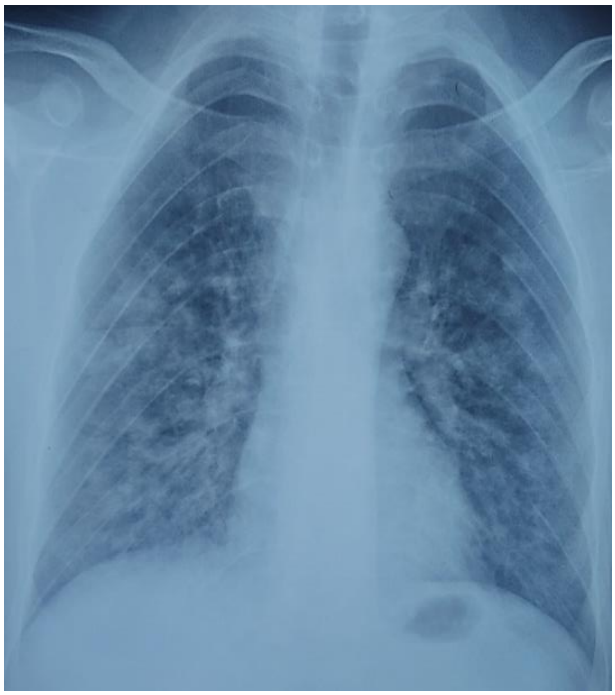

Figure A: Frontal chest X-ray: bilateral alveolar-interstitial syndrome

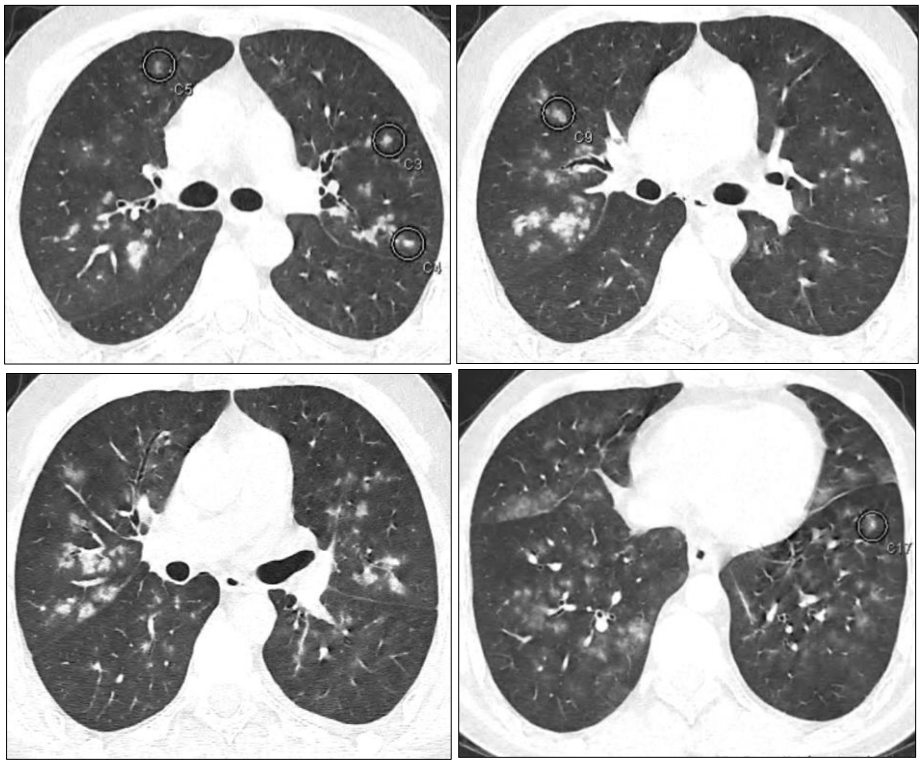

Figure B: Axial sections of a chest CT scan passing through the parenchymal window: an alveolar-interstitial lesion with scattered ground glass images at the level of the two pulmonary fields

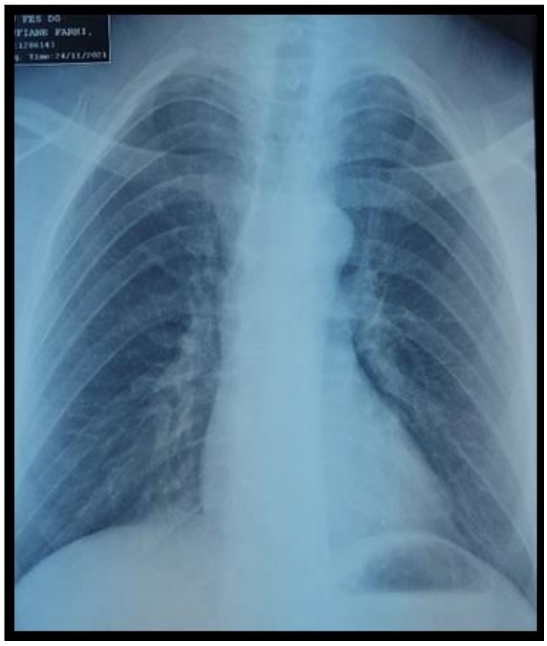

Figure C: Frontal chest X-ray: cleaning of the alveolo-interstitial syndrome

DISCUSSION

For 20 years, many actions have been taken to reduce the complications of blood transfusion. The risk of infection, especially viral, has decreased. Today, accidents of the immunological type seem to predominate. They include allergic reactions, alloimmunization accidents and TRALI (acronym for "Transfusion-Related Acute Lung Injury") [2, 3].

The acronym TRALI for "Transfusion-related acute lung injury" is a post-transfusion accident whose first description dates back to 1951 [4]. It is the first cause of post-transfusion death in the United States and the United Kingdom [5, 6] and the second cause of transfusion mortality in France [7].

For the pathophysiological side, and despite scientific progress, many questions are still being asked. But, classically, TRALI is linked to the aggression of microvascular endothelia and alveolar basement membranes by polymorphonuclear neutrophils activated by the injected blood product. Nevertheless, the pathogenetic, etiological and physiopathological mechanisms underlying this accident are not unequivocal and are the subject of discussions illustrating a need to better standardize the criteria and the diagnostic approach. These considerations led to the meeting of an international consensus conference which took place in 2004 [8] and which resulted in a number of criteria for defining a case of TRALI [10].

The physiopathology of TRALI is based on the theory of the two hits model [9]. This hypothesis, currently considered the most probable, is based on the existence of a recipient-dependent mechanism and a concomitant donor- dependent mechanism [9].

The pathophysiology of TRALI is therefore complex and assumes the coincidence of at least two factors:

- The first corresponds to a particular susceptibility of the recipient responsible for intra-pulmonary leucostasis
- The second corresponds to the blood transfusion, and which is represented by l activation of these accumulated leukocytes and leads to the release of the contents of their granules which would be directly responsible for endothelial damage. In this series of events, various pro-inflammatory factors are involved, including C5a, TNFα, IL1 and IL8 [10].

More details: This cascade of mechanisms results in leukocyte adhesion and rigidity. Then, the activation and degranulation of sequestered polymorphonuclear lead to endothelial and alveolar membrane damage [9].

This second stage may be of: immunological origin, by antigen-granulocyte antibody conflict (antiHLA I antibody, antineutrophil antibody, antiHLA II antibody); these antibodies can be sought in the donor and are more rarely identified in the recipient; or non-immunological: by accumulation of bioactive lipid molecules (lysophosphatidylcholines), during the consumption of cellular blood products, by a decrease in antioxidant activity linked to hypoxia or by the release of sCD40L, a pro-inflammatory mediator present in products labile blood; mediated by vascular endothelial growth factors (VEGF) particularly in neutropenic patients. This second probability is the most plausible for our patient.

Returning to the American-European consensus conference of TORANTO in 2004 which had resulted in a certain number of criteria to define a case of TRALI [10], with in particular the absence of clinical, radiological and ultrasound acute heart failure with elimination of the others causes of Acute Lung Injury (ALI), particularly infectious [11-15].

Recognize first of all an acute pneumopathy "ALI":

- ❖ Acute trigger.
- ❖ Hypoxemia.
- ❖ Bilateral infiltrate on X-ray.
- ❖ No evidence of circulatory overload.

Retain the post-transfusion link: TRALI

- ❖ Absence of pneumonia before transfusion.
- ❖ Its occurrence during or within 6 hours after transfusion.
- ❖ Absence of relationship with another ALI factor.

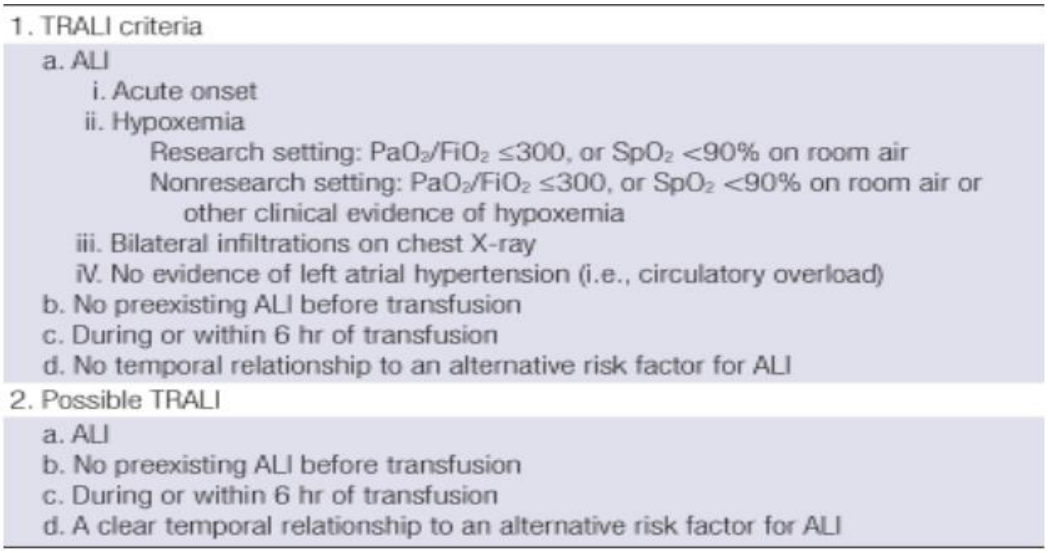

Figure D: Diagnostic criteria for TRALI and possible TRALI from the 2004 TORONTO Consensus Conference

The TRALI picture is nothing other than a lesional Acute Pulmonary Edema picture, but which occurs a few hours at most to 6 hours after the transfusion of a labile blood product. In its typical form, it is a picture of dyspnea with polypnea, tachycardia and bilateral crackles, with a slightly elevated temperature.

On the radiological level, the chest X-ray shows bilateral diffuse opacities, sometimes cottony, which can go as far as the image of a white lung. Thus mimicking a PAO but with a normal ICT (cardio-thoracic index).

Gasometry, if done, shows oxygen desaturation of arterial blood.

Several factors are likely to directly influence the occurrence of TRALI [18]:

Factors related to the blood product itself: use of several transfusion products that differ in their method of collection, preparation, leukodepletion and/or storage.

Factors related to the recipient: malignant hemopathy, HSC transplant, use of cytokines, Resuscitation, etc.

All LBPs, provided they contain plasma, have been implicated; and although the severity of TRALI is not correlated with the volume of injected plasma [19], apheresis platelet concentrates and therapeutic plasma are most often mentioned.

Finally, the shelf life of cellular PSL plays a role due to the release of lipid compounds of cellular origin during prolonged storage of PSL pouches. This factor has been particularly underlined by in vitro studies of experimental TRALI [20] and by the careful study of clinical cases comparing the average shelf life of the products in patients having a clinical reaction compared to transfused patients having no had no adverse reaction [21].

The treatment is symptomatic and is based on respiratory assistance by oxygen therapy with a mask started as soon as the first clinical signs appear, and mechanical ventilation if necessary. Recourse to vascular filling and vasoactive amines is sometimes essential. Corticosteroid therapy has no validated interest. Diuretics could be harmful due to the risk of hypovolaemia [22].

CONCLUSION

Despite the many advances made in terms of prevention of transfusion-related accidents, acute post-transfusion pneumonia, although rare, remains present. TRALI is still an incompletely elucidated transfusion accident, which makes knowledge of its etiologies insufficient to be able to implement effective preventive measures.

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