



HOSTED BY



ELSEVIER

Contents lists available at ScienceDirect

Journal of Acute Disease

journal homepage: www.jadweb.orgCase report <http://dx.doi.org/10.1016/j.joad.2015.03.003>

Transfusion-related acute lung injury: A case report

Emmanouil Petrou^{1*}, Vasiliki Karali², Vasiliki Vartela¹¹Division of Cardiology, Onassis Cardiac Surgery Center, Athens, Greece²First Department of Propaedeutic and Internal Medicine, Athens University Medical School, Athens, Greece

ARTICLE INFO

Article history:

Received 19 Jan 2015

Received in revised form 2 Feb 2015

Accepted 15 Mar 2015

Available online 10 Jul 2015

Keywords:

Transfusion-related acute lung injury

INR

Transfusion

ABSTRACT

Transfusion-related acute lung injury is the most common cause of serious morbidity and mortality associated with the transfusion of plasma-containing blood components. The syndrome can be confused with other causes of acute respiratory failure. Herein, we describe a 71-year-old man who was transfused with fresh frozen plasma due to prolonged INR, and died of what was considered as transfusion-related acute lung injury, despite treatment.

1. Introduction

Transfusion-related acute lung injury (TRALI) is the most common cause of serious morbidity and mortality associated with the transfusion of plasma-containing blood components. The syndrome can be confused with other causes of acute respiratory failure. Herein, we describe a 71-year-old man who was transfused with fresh frozen plasma due to prolonged INR, and died of what was considered as TRALI, despite treatment.

2. Case report

A 71-year-old man was admitted to our hospital due to prolonged INR (13.5). The patient had a history of coronary artery bypass grafting for coronary artery disease and mitral valve replacement with a metal one 14 years earlier. Since then he was treated with acenocoumarol. On admission the patient was hemodynamically stable with normal vital signs. The 12-lead surface electrocardiogram revealed a known atrial fibrillation with an acceptable ventricular response. The treatment team

decided to administer fresh frozen plasma (FFP) in order to correct the prolonged INR. An uneventful transfusion of 2 FFP units took place and a repeat INR measurement was scheduled for a few hours later.

However, 4 h after FFP transfusion, the patient experienced a sudden onset of dyspnea, tachypnea (35 breaths/min), cyanosis, profuse diaphoresis and hypoxemia (oxygen saturation < 75%). The patient's blood pressure was 170/90 mmHg, and his temperature was normal. On physical examination, breath sounds were markedly decreased, and chest auscultation revealed bilateral diffuse crackles. There were no signs of volume overload, and his jugular venous pressure was normal. There were no electrocardiographic changes, and the test results for cardiac enzymes were within normal limits. A complete blood count revealed elevated white blood cells (30000), compared to 5500 on admission, and platelet reduction. A chest radiograph showed coarse alveolar infiltrates and a normal cardiac silhouette, findings that are indicative of noncardiogenic pulmonary edema. Echocardiography demonstrated a preserved left and right ventricular function with no other eminent pathology. On admission to the intensive care unit, the patient was stabilized with the administration of intravenous diuretics and corticosteroids.

Due to following marked hypotension, the attending team decided to initiate inotropic treatment with dopamine and dobutamine. During the following hours, the patient gradually became anuric and was set on hemofiltration. Transaminasemia was established, and 12 h after admission, selective intubation and mechanical ventilation due to mixed acidosis was decided.

*Corresponding author: Emmanouil Petrou, 29 Kioutacheias Street, GR-142 31 Nea Ionia, Greece.

Tel: +30 6945112509

Fax: +30 2102751028

E-mail: emmgpetrou@hotmail.com

Peer review under responsibility of Hainan Medical College.

Disseminated intravascular coagulopathy was excluded. A few hours later the patient died despite the full support described above.

3. Discussion

TRALI was first reported in 1951^[1], and 1957^[2], and findings from the initial case series were published in 1966^[3]. As distinguishing biomarkers are absent, TRALI is a clinical diagnosis. The lack of a standardized definition of TRALI has contributed to under-diagnosing of this syndrome, as well as has hindered its epidemiology and research investigation. In recognition of this problem, a case definition of TRALI based on clinical and radiological parameters was formulated during a consensus conference by the US National Heart, Lung and Blood Institute in 2004^[4]. The definition is derived from the widely used definition of acute lung injury (ALI) and its more severe form, acute respiratory distress syndrome (ARDS), as proposed by the North American-European Consensus Conference consensus^[5]. These criteria include the acute onset of hypoxia with bilateral pulmonary infiltrates, no evidence of left ventricular overload (transfusion-associated circulatory overload) and the presence of a risk factor for ALI/ARDS. TRALI is defined as the fulfillment of the definition of ALI within 6 h after transfusion in the absence of another risk factor for ALI. Although this definition appears straightforward, a complicating factor is that the characteristics of TRALI are indistinguishable from ALI due to other etiologies, such as pneumonia, sepsis, aspiration, lung contusion, multiple fractures, and pancreatitis. Using this definition would rule out the possibility of diagnosing TRALI in a patient with an underlying ALI risk factor who has also received a transfusion. To identify such cases, the terms “possible TRALI” or “transfused ALI” were developed, which allow for the presence of another risk factor for ALI^[6]. Given the uncertainty of the relationship of ALI to the transfusion in possible TRALI, this term facilitates separate categorization in surveillance systems to permit comparisons across systems.

The pathogenesis of TRALI has not been fully elucidated. In theory, any cell-containing blood product or plasma-rich blood product can cause TRALI. To the present, two hypotheses have been proposed. The first hypothesis suggests that TRALI is caused by donor antibodies against human neutrophil antigens or human leukocyte antigens (HLA) in the lungs of the recipient^[7]. However, the association between HLA antibodies in donor plasma and TRALI is not supported by hard data. A significant fraction of TRALI cases exhibit no detectable antibodies. Furthermore, many antibody-containing blood products fail to produce TRALI. An alternative hypothesis implicates a two-event model. The first event is an inflammatory condition of the patient (e.g. sepsis, recent surgery) causing sequestration and priming of neutrophils in the pulmonary compartment. The second event is the transfusion itself, containing either antibodies or bioactive lipids that have accumulated during blood storage, stimulating the primed neutrophils to release proteases^[8]. In both hypotheses the result is endothelial damage, capillary leak and extravasation of neutrophils.

The mainstay of treatment for TRALI remains supportive care. If the suspected blood product is still being transfused, it should be discontinued immediately^[9,10]. Furthermore, all

TRALI patients require additional oxygen, while mechanical ventilation is unavoidable in 70%–90% of the cases^[11]. In accordance with treatment of ALI patients, it could be speculated that restrictive tidal volume ventilation should be applied to avoid worsening of lung injury^[12]. Specific treatment strategies for TRALI, however, have not yet been developed. Therapeutic options for TRALI based on the investigation of G2A receptor have been reported, however results are not conclusive^[13]. Since the pathophysiology and etiology of TRALI have yet to be fully elucidated, and because there is no rapid diagnostic test, there are no clear recommendations for the prevention of new TRALI cases^[14].

Whereas mortality of ALI is 40%–60%, the majority of TRALI patients improve within 48–96 h after the insult, when appropriate respiratory support is supplied^[15]. However, a high level of suspicion is required for the prevention, and especially early treatment of the condition.

Conflict of interest statement

The authors report no conflict of interest.

References

- [1] Barnard RD. Indiscriminate transfusion: a critique of case reports illustrating hypersensitivity reactions. *N Y State J Med* 1951; **51**: 2399-402.
- [2] Brittingham TE, Chaplin H Jr. Febrile transfusion reactions caused by sensitivity to donor leukocytes and platelets. *J Am Med Assoc* 1957; **165**: 819-25.
- [3] Philipps E, Fleischner FG. Pulmonary edema in the course of a blood transfusion without overloading the circulation. *Dis Chest* 1966; **50**: 619-23.
- [4] Toy P, Popovsky MA, Abraham E, Ambruso DR, Holness LG, Kopko PM, et al. Transfusion-related acute lung injury: definition and review. *Crit Care Med* 2005; **33**: 721-6.
- [5] Kleinman S, Caulfield T, Chan P, Davenport R, McFarland J, McPhedran S, et al. Toward an understanding of transfusion-related acute lung injury: statement of a consensus panel. *Transfusion* 2004; **44**: 1774-89.
- [6] Triulzi DJ. Transfusion-related acute lung injury: an update. *Hematol Am Soc Hematol Educ Program* 2006: 497-501.
- [7] Lee JH, Kang ES, Kim DW. Two cases of transfusion-related acute lung injury triggered by HLA and anti-HLA antibody reaction. *J Korean Med Sci* 2010; **25**: 1398-403.
- [8] Silliman CC, Fung YL, Ball JB, Khan SY. Transfusion-related acute lung injury (TRALI): current concepts and misconceptions. *Blood Rev* 2009; **23**: 245-55.
- [9] Cherry T, Steciuk M, Reddy VV, Marques MB. Transfusion-related acute lung injury: past, present, and future. *Am J Clin Pathol* 2008; **129**: 287-97.
- [10] Popovsky MA. Transfusion-related acute lung injury. *Curr Opin Hematol* 2000; **7**: 402-7.
- [11] Wallis JP, Lubenko A, Wells AW, Chapman CE. Single hospital experience of TRALI. *Transfusion* 2003; **43**: 1053-9.
- [12] Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000; **342**: 1301-8.
- [13] Ellison MA, Ambruso DR, Silliman CC. Therapeutic options for transfusion related acute lung injury: the potential of the G2A receptor. *Curr Pharm Des* 2012; **18**: 3255-9.
- [14] Fabron A Jr, Lopes LB, Bordin JO. Transfusion-related acute lung injury. *J Bras Pneumol* 2007; **33**: 206-12.
- [15] Vlaar AP, Schultz MJ, Juffermans NP. Transfusion-related acute lung injury: a change of perspective. *Neth J Med* 2009; **67**: 320-6.