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Case Report

Transfusion-Related Acute Lung Injury (TRALI) or Transfusion-Related Lung Edema Occurring Postoperatively after the Removal of a Parieto-Occipital Tumor: A Case Report and Literature Review

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Abstract: TRALI is a post-transfusion acute respiratory distress syndrome that presents as acute non-cardiogenic pulmonary edema occurring within six hours after a transfusion. A 38-year-old female patient was hospitalized in intensive care for the management of postoperative complications following the resection of a parieto-occipital PEIC, complicated by intraoperative hemorrhagic shock requiring the transfusion of 2 units of red blood cells and 2 units of plasma. Preoperatively, the clinical and biological examination was unremarkable. Under general anesthesia, the estimated blood loss of 1000ml required transfusion of 2 units of packed red blood cells and 2 units of fresh frozen plasma. At 4 hours post-transfusion and 3 hours postoperatively, she developed respiratory distress associated with macroscopic hematuria. She exhibited diffuse bilateral crackles with fine wheezing. Oxygen saturation was 65% under mechanical ventilation. Blood gases showed hypoxemia with hypercapnia. Laboratory tests revealed anemia with Hb: 6.9 g/dl, thrombocytopenia at 59,000/mm³, AST 148.9, ALT 302.1. Transthoracic ultrasound was normal. Thoracic CT angiography showed findings consistent with acute lung injury edema. Furosemide 40 mg was administered every 6 hours for 3 days, in combination with nebulization sessions and corticosteroid therapy. After 24 hours of treatment, the progress was satisfactory with normal saturation. At 72 hours, the lung fields, CT scan, and laboratory results had returned to normal. TRALI is a syndrome whose clinical diagnosis involves identifying pulmonary edema without a hemodynamic or cardiac component.

Keywords: TRALI, Transfusion, Respiratory distress.

INTRODUCTION

Transfusion-Related Acute Lung Injury (TRALI) refers to acute pulmonary edema (APE) occurring after the administration of labile blood products (LBP) [1]. In 1951, post-transfusion hypersensitivity reactions manifesting as non-cardiogenic pulmonary edema were reported. However, it was in 1985 that this entity was identified by Popovsky [2] and designated TRALI (Transfusion-Related Acute Lung Injury).

Transfusion-related pulmonary edema presents as transfusion-related acute respiratory distress syndrome (ARDS) occurring within six hours of a transfusion, with non-cardiogenic acute pulmonary edema. Diagnosis is both clinical and radiological, the main finding being the development of pulmonary edema during or following a transfusion in the absence of any obvious cause.

All labile blood products containing plasma can cause TRALI. As for blood-derived products, intravenous immunoglobulins [3] have been implicated, as well as hematopoietic stem cell injections [4].

We report a case of TRALI following a transfusion of packed red blood cells (PRBCs) and fresh frozen plasma (FFP) in a 26-year-old woman.

OBSERVATION

A 38-year-old woman was hospitalized in intensive care for the postoperative course of an excision of a parieto-occipital intracranial expansile process (ICEP) (figure 1 and 2) complicated by intraoperative

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hemorrhagic shock requiring the transfusion of two RBCs and two FFPs.

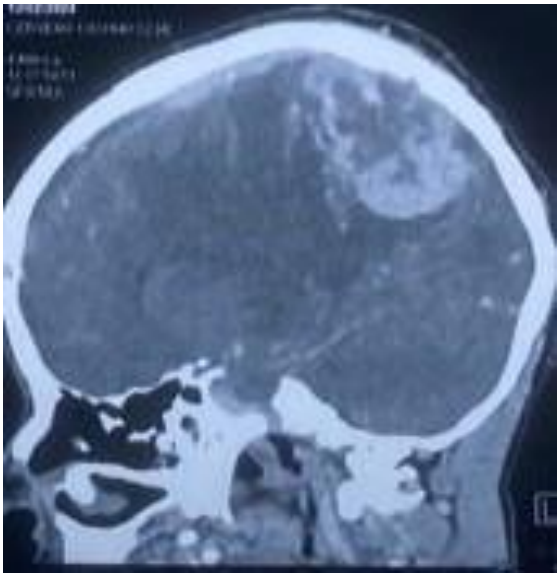

Figure 1: Parieto-occipital PEIC with a large perilesional edema exerting a mass effect on the midline structures with collapse of the ipsilateral ventricle and exclusion hydrocephalus

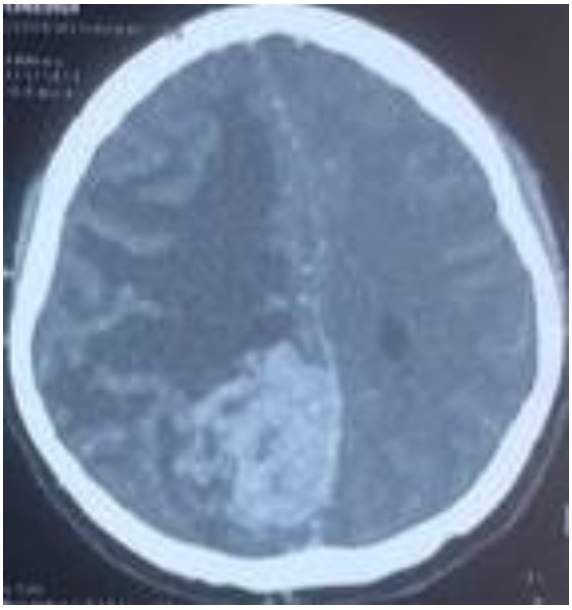

Figure 2: Axial section of a contrast-enhanced cerebral CT scan showing parieto-occipital PEIC with a large perilesional edema exerting a mass effect on the midline structures with collapse of the ipsilateral ventricle and exclusion hydrocephalus

The preoperative clinical and laboratory examinations were unremarkable. She was placed under general anesthesia with orotracheal intubation and experienced hemorrhagic shock during the procedure, with an estimated blood loss of 1000 ml requiring transfusion of 2 units of packed red blood cells and 2 units of fresh frozen plasma. During surgery, the tumor was resected en bloc. The duration of anesthesia and surgery was 4 hours 20 minutes and 4 hours 50 minutes, respectively. After surgery, the patient was transferred to the intensive care unit and extubated at 1 hour postoperatively, as her respiratory, neurological, and hemodynamic status was satisfactory.

Four hours after the transfusion, or three hours post-surgery, she presented with respiratory distress associated with gross hematuria. Physical examination revealed diffuse bilateral crackles with fine wheezes. Peripheral oxygen saturation (SpO2) was 65% on a high-concentration mask at 15 L/min. A chest CT scan showed pulmonary edema with a lesion. Figure 3

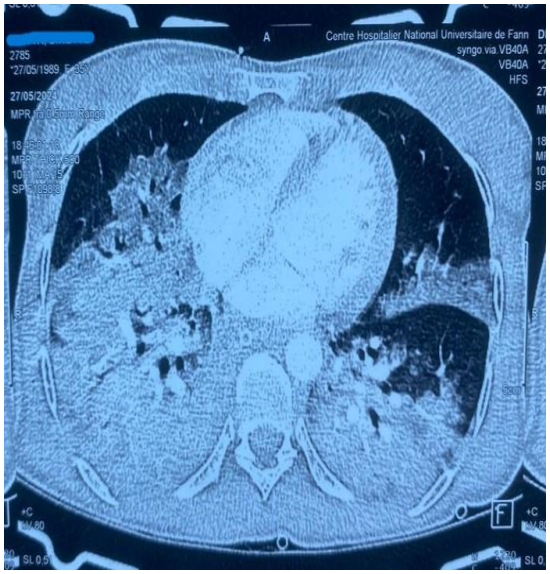

Figure 3: Axial CT scan of the chest with contrast enhancement in the parenchymal window showing bilateral alveolar consolidation in the middle and lower left lobes, associated with areas of ground-glass opacities in the perilesional region on the right and the lingual lobe. Normal appearance of the tracheobronchial tree. No pleuropericardial effusion. Conclusion: Appearance suggestive of pulmonary edema with a lesion

Elsewhere, the neurological examination was normal, with clear consciousness and no neurological deficits. The cardiovascular examination was normal, with normal blood pressure and adequate urine output. Macroscopic hematuria resolved within 10 hours. Table I details the results of the post-operative assessment.

Table I: Results of the post-operative assessment

Red Blood Cells	**2.33 Tera/L**	**Creatinemia**	**8.44 mg/l**
Hemoglobin	6.9g/dl	ALAT	302.1 (7.5 N)
Hematocrit	19.4	ASAT	148.9(4.2 N)
Leaflets	59000/mm³	PaO2	80mmHg
Sodium	153 mEq/L	PaCO2	50mmHg
Potassium	3.2 mEq/L	Chloremia	118 mEq/L

No abnormalities were detected on the electrocardiogram. Doppler echocardiography showed good biventricular systolic function, normal filling pressures, and no pulmonary arterial hypertension (PAH). A chest X-ray was not performed. A chest CT angiogram was performed. No residual tumor was detected on the follow-up brain CT scan (Figure 4).

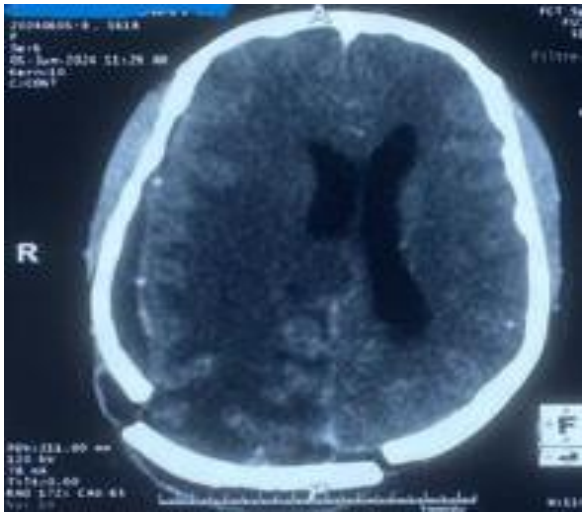

Figure 4: Axial section of a cerebral CT scan with contrast showing no residual tumor visualized with right fronto-parieto-occipital vasogenic edema associated with a 10mm thick ipsilateral subdural collection with mass effect on the midline structures with beginning of subfactorial herniation

Furosemide 40 mg was administered intravenously every 6 hours for 3 days, in combination with nebulization sessions. After 24 hours of treatment, the patient's condition was satisfactory and oxygen saturation was normal. At 72 hours, the lungs were clear and the chest CT scan was normal.

DISCUSSION

The development of acute pulmonary edema (APE) following fluid resuscitation initially suggests a cardiac etiology. However, this clinical condition can also be a sign of post-transfusion injury, referred to as TRALI (transfusion-related pulmonary edema in English-speaking countries). This condition can occur immediately following a blood transfusion or after a transfusion-free interval of several hours.

Mortality related to TRALI is 20% in the general population and 58% in patients with serious underlying conditions [2]. TRALI accounts for 30–50% of transfusion-related deaths [5], and mortality during TRALI is estimated to be between 5% and 35% depending on the study [6]. Its incidence is estimated at approximately 8% in intensive care units [7] .

However, this data is still quite recent. Indeed, the exponential growth of the literature on TRALI dates back to the 2000s, with over 80% of published works. The vast majority of articles are case reports or review articles; very few case series have been published.

Previously, the prevalence of TRALI was estimated at approximately 1 case per 5,000 blood component transfusions. According to a prospective case identification study recently published in the United States, the risk would be slightly less than 1 case per 12,000 units transfused [8] .

As indicated in the Transfusion Incident Surveillance System (TISS) report for the period between 2011 and 2015, TRALI (confirmed and probable cases) is the leading cause of transfusion-related mortality in Canada (22%) after post-transfusion circulatory overload or TACO (42.6%). In the United States, it ranks second (24%) after TACO (34%) [9] .

The severity of TRALI is not correlated with the volume of plasma injected. Risk factors that have been implicated include: blood products that have increased after 4 days of storage [10] and products from immunized donors (multiparous women) [11-12].

The identification of the pathophysiological mechanisms relied on clinical (mainly hemovigilance), histopathological, and experimental data, both in vitro (primarily endothelial cell cultures) and ex vivo (isolated organs), as well as animal models. The few histopathological studies performed on patients who died rapidly from TRALI show interstitial and alveolar edema with accumulation of activated polymorphonuclear leukocytes in small vessels, capillaries, interstitial tissue, and pulmonary alveoli [13]. Electron microscopy also reveals large numbers of polymorphonuclear leukocytes that have lost their granular content in pulmonary microvessels, in direct contact with a damaged alveolocapillary membrane. These data suggest that TRALI results from the activation of neutrophils and endothelial cells, or both, through multiple mechanisms leading to the accumulation of neutrophils in the damaged endothelium of pulmonary capillaries, and subsequently to pulmonary edema and alveolar damage [7]. Two main pathogenic hypotheses are considered to explain TRALI:

- The single-strike hypothesis, where the occurrence of TRALI is solely dependent on a triggering factor contained in the blood product. This theory concerns immunological TRALI, where an antibody contained in the blood product can, by itself, cause TRALI.
- the double strike hypothesis where the occurrence of TRALI is conditioned by predisposing factors related to the recipient and leading to intrapulmonary leukostasis (first strike); on this ground occurs a second event related to the transfusion (second strike), immunological or not, which causes the activation of polymorphonuclear cells sequestered in the pulmonary circulation [14].

The diagnostic criteria are detailed in Table I.

Table I: Diagnostic criteria for TRALI (according to Kleinman *et al*. [15])

1. TRALI Criteria
a. Pulmonary edema due to injury
i. Quick installation
ii. Hypoxemia
- In the context of research work: PaO2/FiO2 ≤ 300 mmHg or SpO2 < 90% in ambient air
- Outside the context of research work: PaO2/FiO2 ≤ 300 mmHg or SpO2 < 90% in room air or other clinical evidence of hypoxemia
iii. Bilateral pulmonary infiltrates on chest radiograph
iv. No evidence of left atrial hypertension (i.e., volume overload)
b. Absence of pulmonary edema prior to transfusion
c. Appearance within six hours of the start of the transfusion
d. Absence of a temporal link with another risk factor for pulmonary edema injury

2. Possible TRALI criteria a. Pulmonary edema due to injury b. Absence of pulmonary edema prior to transfusion c. Appearance within six hours of the start of the transfusion d. Temporal link with another risk factor for pulmonary edema

Our patient meets all the criteria required to confirm TRALI. The outcome was favorable under treatment.

CONCLUSION

TRALI is a poorly understood syndrome whose clinical diagnosis relies on the clinical identification of pulmonary edema without hemodynamic or cardiac involvement. It is crucial to determine its impact in intensive care and to analyze the challenges associated with diagnosis and therapeutic management. To limit its incidence, blood products could be reserved for appropriate indications.

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