Transfusion-Related Acute Lung Injury

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Abstract Transfusion-related acute lung injury (TRALI) is related to the transfusion of blood components. Typically, it is a clinical syndrome, characterized by the sudden onset of dyspnea, hypoxemia and bilateral non-cardiogenic pulmonary edema. A 83-year-old female patient with a history of AML developed TRALI after receiving 6 units of platelets. TRALI symptoms was started 10 min later the transfusion. AML is a risky group for TRALI. While giving transfusion to the risky groups of TRALI one must be more careful. The mortality rate caused by TRALI will decrease if the patient who is thought to have TRALI or who has bilateral pulmonary edema without any other reason showing the existence of TRALI is given ventilatory support at the right time.

Keywords TRALI · Syndrome · Ventilator

Introduction

Transfusion-related acute lung injury (TRALI) is related to the transfusion of blood components. Typically, it is a clinical syndrome, characterized by the sudden onset of dyspnea, hypoxemia and bilateral non-cardiogenic pulmonary edema [1]. TRALI, is the most common reason of deaths that is related to transfusion. However, incidence of deaths is not known exactly [2]. The TRALI incidence is 1/2,000–1/5,000 for all blood components and 1/432 for platelets and occurs within 6 h of transfusion [1, 3]. Plasma

and platelets are the most common blood products which cause TRALI [2].

Case

An 83-year-old female patient. In the patient's laboratory tests, who was admitted to hematology polyclinic with fatigue and ecchymose complaints, thrombocytopenia and neutropenia were found out. After the observation of peripheral blood smear; since, blast and dysplastic neutrophils were observed; bone marrow aspiration, biopsy and flowcytometric tests were held considering the existence of myelodysplastic syndrome or acute leukemia. After the test results, the patient was diagnosed by acute myeloid leukemia. Because the patient was too old and did not accept to use chemotherapy, the patient was observed with supportive treatment. The patient was given platelet concentrate or platelet concentrate by apheresis when her platelet counts were under $10 \times 10^3/\text{UL}$. When the patient's hemoglobin count was under 8 g/dL she was given erythrocyte concentrate. The patient who had headache during the observation in January 2012 and whose thrombocyte level was under $5 \times 10^3/\text{UL}$, had a cerebral CT scan and bleeding was not observed. The patient was given 6 units platelet concentrate which was randomly pooled, irradiated and depleted leukocytes. The patient, who had sudden onset of dyspnea 10 min after the transfusion, was thought to have TRALI. Auscultation of lung fields which was carried out early during the complaints was normal. Pulse oximetry showed that O_2 saturation was 60 %. Her blood pressure was 140/100 mmHg, pulsation was 110/min. The patient was given O_2 via the oxygen mask and was injected antipyretics, corticosteroids, diphenhydramine and IV diuretics. The breathing problem of the patient increased within 10 min.

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Fig. 1 Bilateral pulmonary infiltration

Fig. 2 X-ray, after the ventilatory support treatment

Pulse oximetry showed that O_2 saturation decreased as 40 % even during the O_2 treatment. When the patient's, who developed cyanosis, lung was examined; crepitant rale was diagnosed around bilateral upper zones. Afterwards, the patient was intubated and was given ventilatory support.

X-ray results showed the existence bilateral pulmonary edema (Fig. 1). ECG results showed nothing other than sinus tachycardia. Echocardiography results showed that systolic functions were normal. pH 7.08, pO_2 114.5 mmHg, pCO_2 50.8 mmHg, HCO_3^- 14.3 mmol/L were found in arterial blood gas which was taken just after the patient was given mechanical ventilation. The patient, who was observed for 18 h via mechanical ventilation, was given $NaHCO_3$ infusion, IV diuretics, corticosteroid and eptantoin (because of convulsion). It was observed that pulmonary edema decreased within hours when the patient was supported by mechanical ventilation (Fig. 2). The patient, who was observed for nearly 18 h via mechanical ventilation, was extubated at the 18th h. The laboratory results are shown with the following Table 1.

Discussion

TRALI, can be defined as the onset of dyspnea which is related to the transfusion of blood products, usually occurring in the first 30 min of transfusion and non-cardiogenic pulmonary edema which is not caused by any other reason [1]. Patients with emergency cardiac surgery, hematologic malignancy, massive transfusion, sepsis, mechanical ventilation, multiparous female donors, human leukocyte antigen (HLA) class I and II antibodies in the platelet donor's plasma and plasma-rich components, are at the risk for development of TRALI [4, 5]. There is also a relationship between platelet age and TRALI. Antileukocyte antibodies, activation of the complement cascade, neutrophil (PMN)-mediated lung injury and pulmonary leukostasis are responsible for the pathogenesis of TRALI [6–8]. Furthermore, IL-6 and IL-8 are essential cytokines which stimulate PMN and play important role in pathogenesis of TRALI [9, 10]. As the platelet age increases, the level of IL-6 and IL-8 increase in the platelet concentrates. The infusion of these platelet concentrates increase the risk of TRALI [1, 11]. Being an AML patient she had hematologic disease therefore, she was among the risky group for TRALI. Moreover, since the

Table 1 The laboratory levels before TRALI and during the ventilation

	Pre-transfusion	After 10 min of transfusion	Start of Mechanical Ventilation	Sixth hour of mechanical ventilation	Twelfth hour of mechanical ventilation	Eighteenth hour of mechanical ventilation	After 1 h of extubation
st O_2 (%)	–	60	92	97	99	90	88
HR (minute)	76	110	109	89	87	95	109
BP (mmHg)	100/60	140/100	130/60	124/65	146/74	162/109	161/84
pH (mmHg)	–	–	7.068	7.320	7.431	7.464	7.480
pO_2 (mmHg)	–	–	114.5	111.1	110	111.4	59.5
pCO_2 (mmHg)	–	–	50.8	46.9	50.4	45.9	35

platelet concentrates that we used were for 5 days and included older thrombocyte, the patient was among the she was in high risk group for TRALI.

To diagnose TRALI; dyspnea with acute onset caused by acute lung injury, hypoxemia and bilateral infiltrates on frontal chest radiograph should be observed together with the absence of the evidence of left atrial hypertension and another reason to cause acute lung injury during or within 6 h of transfusion [5]. Since the echocardiography of the patient was normal, there was bilateral infiltrate on chest radiograph, which was observed within 10 min after the pooled platelet concentrate together with the acute onset dyspnea and hypoxemia and there was not other reason for ALI, the existence of TRALI was found out. Signs and symptoms include tachypnea, pulmonary secretions, hypotension (less commonly hypertension), fever, tachycardia and cyanosis. Auscultation of the lung fields reveals diffuse rales [5]. The patient had tachypnea, pulmonary secretion, tachycardia and cyanosis. However, the patient had hypertension which was not common. Although, auscultation of the lung fields was normal during the onset of dyspnea, diffuse rales were found out within 10 min. Laboratory tests which strongly support, but are not required for the clinical diagnosis of TRALI, include the demonstration of HLA class I or class II or neutrophil-specific antibodies in donor plasma. Because the patient was found to have TRALI as clinically, no other laboratory tests were required.

In most cases, mechanical ventilation and O₂ support are required for the treatment of TRALI. Different from acute respiratory distress syndrome which are caused by other reasons, patients with TRALI recover quickly with resolution of pulmonary infiltrates within 96 h of the transfusion. The mortality rate ranges from 5 to 10 % [6, 12]. It was observed that pulmonary infiltration disappeared just after the 18 h of ventilatory support and transfusion, and full recovery was observed after the patient was extubated. As soon as the TRALI is diagnosed, the necessity of ventilatory support should be checked. As in our case, if the patient is given ventilatory support without wasting much time, the mortality rate caused by TRALI will decrease.

As a result, in case of sudden onset of dyspnea, tachypnea and hypoxemia which developed within the first 6 h after the

transfusion, the first thing to consider is TRALI and it should be evaluated whether the patient needs ventilatory support or not. While giving transfusion to the risky groups of TRALI, one must be very careful. The mortality rate caused by TRALI will decrease, if the patient who is thought to have TRALI or who has bilateral pulmonary edema without any other reason showing the existence of TRALI is given ventilatory support at the right time.

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