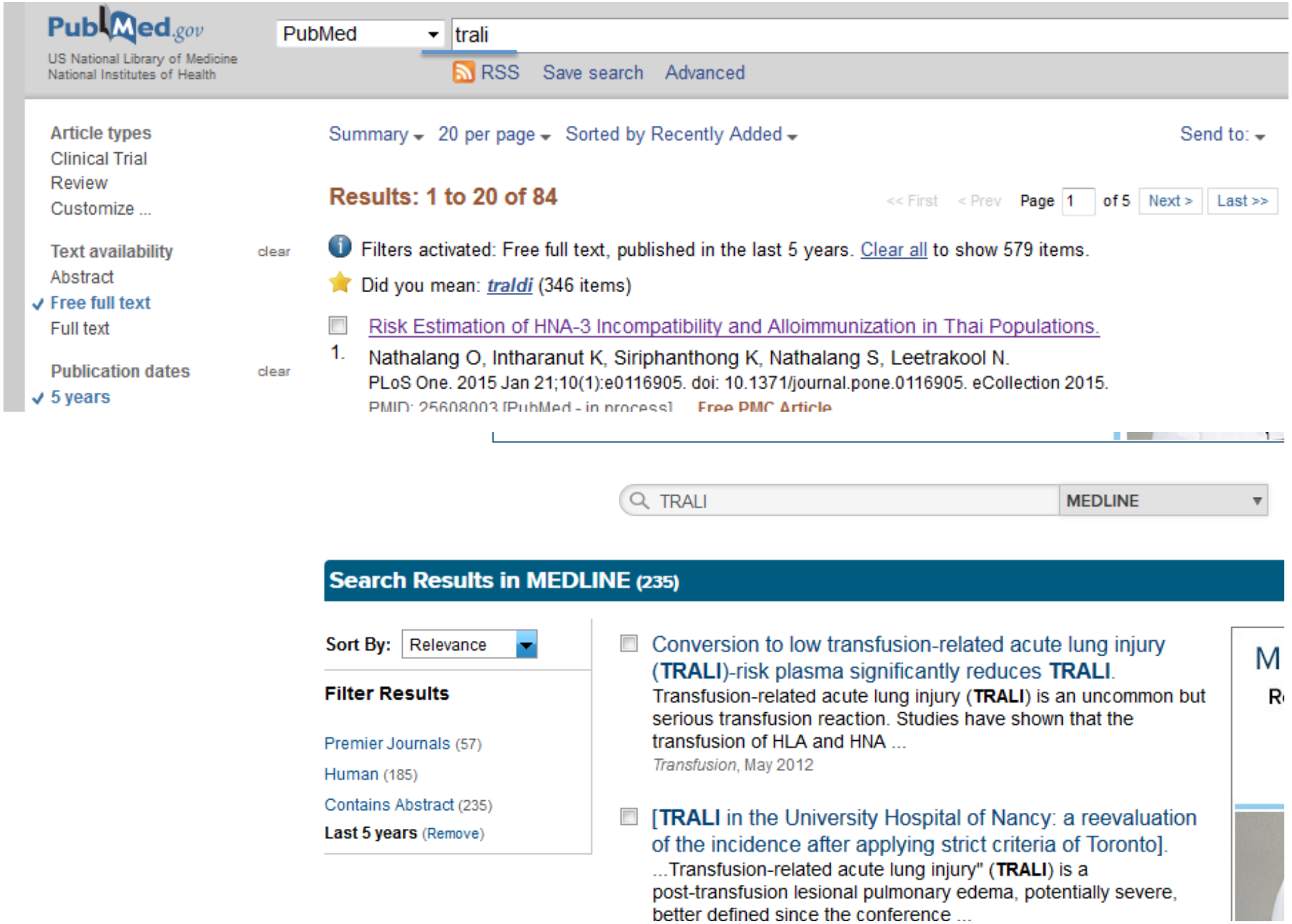
TRALI-синдром: состояние проблемы

Грицан Алексей Иванович

Красноярский государственный медицинский университет им.проф. В.Ф. Войно-Ясенецкого

Краевая клиническая больница

2017 год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PLoS One. 2015 Jan 21;10(1):e0116905. doi: 10.1371/journal.pone.0116905. eCollection 2015.
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Conversion to low transfusion-related acute lung injury (TRALI)-risk plasma significantly reduces TRALI.
Transfusion-related acute lung injury (TRALI) is an uncommon but serious transfusion reaction. Studies have shown that the transfusion of HLA and HNA ...
Transfusion, May 2012
[TRALI in the University Hospital of Nancy: a reevaluation of the incidence after applying strict criteria of Toronto].
...Transfusion-related acute lung injury" (TRALI) is a post-transfusion lesional pulmonary edema, potentially severe, better defined since the conference ...

Дефиниции

- TRALI – острое повреждение легких (ОРДС), связанное с переливанием (трансфузией)
- Possible TRALI – вероятно TRALI
- TACO – перегрузка (недостаточность) кровообращения, ассоциированная с переливанием (трансфузией)

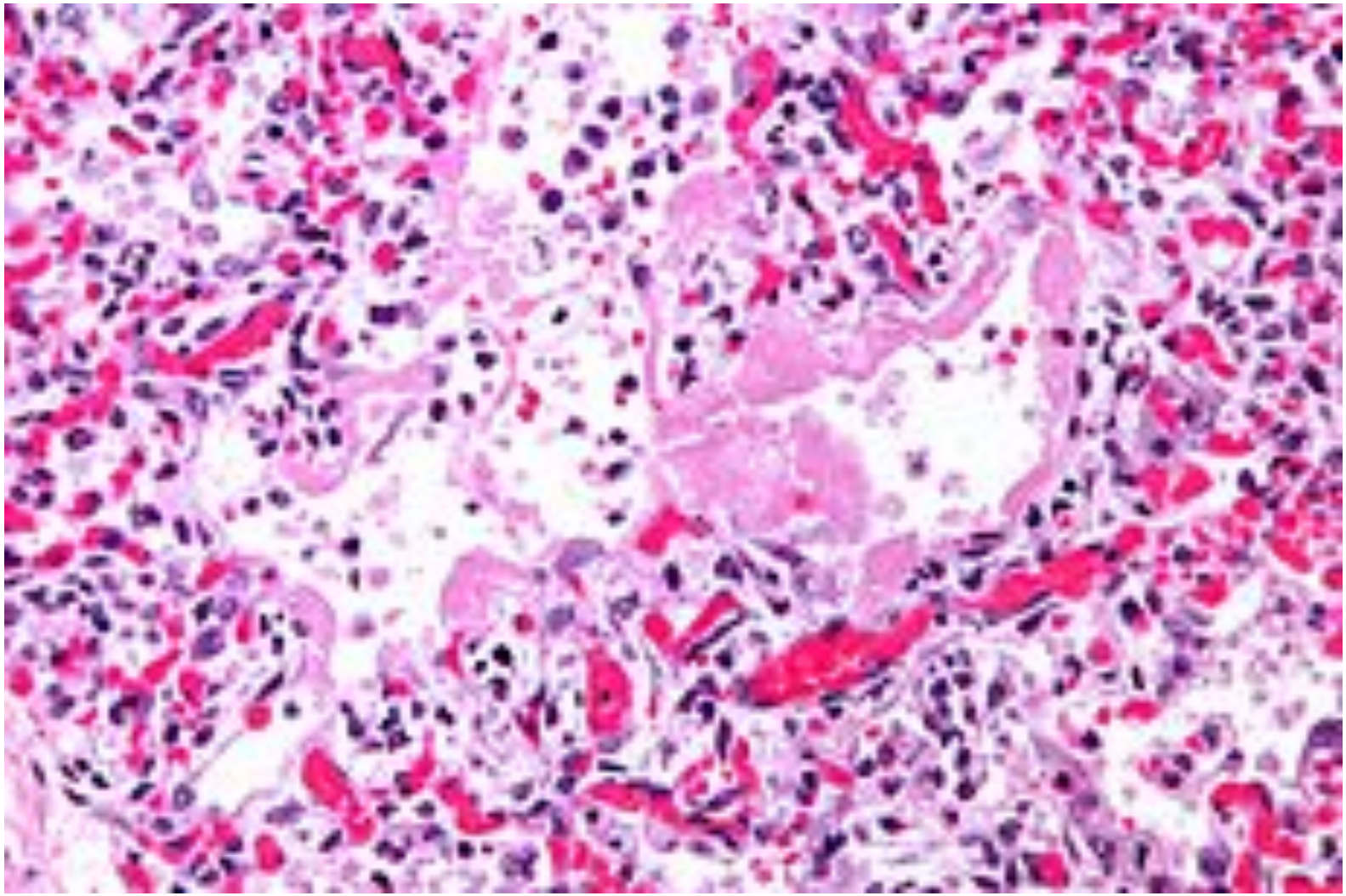
- TRALI – некардиогенный отек легких, в основе которого лежит повышение проницаемости эндотелия легочных капилляров, резвившееся вследствие переливания крови и ее компонентов (Popovsky V.F., 1983)

Основные «причины»

- Цельная кровь
- СЗП
- Эритроцитарная масса
- Тромбоцитарная масса
- Криопреципитат

- Иммуноглобулины (описаны единичные случаи)

Распространённость и патофизиология



Частота развития

- В среднем – 1: 1323 введенные дозы различных препаратов (2008 год)
- 1:4410 доз эритроцитарной массы (Siliman C.C., 2003)
- 1:7900 доз СЗП (Wallis S., 2003)
- 1:200 доз СЗП от доноров женщин, имевших более 2-х беременностей (Palfi D., 2001)
- 1:3 дозы СЗП от доноров, препараты крови которых ранее стали причинами развития TRALI (Kopko P.M., 2002)
- 1:317 доз тромбоцитарной массы (Clarke S., 1994)

Transfusion-related acute lung injury: from bedside to bench and back

Beth H. Shaz,[1,2] Sean R. Stowell,[2] and Christopher D. Hillyer[1,3]

[1]New York Blood Center, New York, NY; [2]Center for Transfusion and Cellular Therapies, Department of Pathology and Laboratory Medicine, Emory University, Atlanta, GA; and [3]Division of Hematology/Medical Oncology, Department of Medicine, Weill Cornell Medical College, New York, NY

BLOOD, 3 FEBRUARY 2011 • VOLUME 117, NUMBER 5

Transfusion-related acute lung injury (TRALI) is the leading cause of transfusion-related mortality. To determine TRALI incidence by prospective, active surveillance and to identify risk factors by a case-control study, 2 academic medical centers enrolled 89 cases and 164 transfused controls. Recipient risk factors identified by multivariate analysis were higher IL-8 levels, liver surgery, chronic alcohol abuse, shock, higher peak airway pressure while being mechanically ventilated, current smoking, and positive fluid balance. Transfusion risk factors were receipt of plasma or whole blood from female donors (odds ratio = 4.5, 95% confidence interval [CI], 1.85-11.2, *P* = .001), volume of HLA class II antibody with normalized background ratio more than 27.5 (OR = 1.92/100 mL, 95% CI, 1.08-3.4, *P* = .03), and volume of anti–human neutrophil antigen positive by granulocyte immunofluoresence test (OR = 1.71/100 mL, 95% CI, 1.18-2.5, *P* = .004). Little or no risk was associated with older red blood cell units, noncognate or weak cognate class II antibody, or class I antibody. Reduced transfusion of plasma from female donors was concurrent with reduced TRALI incidence: 2.57 (95% CI, 1.72-3.86) in 2006 versus 0.81 (95% CI, 0.44-1.49) in 2009 per 10 000 transfused units (*P* = .002). The identified risk factors provide potential targets for reducing residual TRALI. (*Blood*. 2012;119(7): 1757-1767)

- TRALI возникает в пределах 6 часов после трансфузии.
- Высокая частота при использовании плазмы и тромбоцитов, чем при трансфузии эритроцитов (со смертельным исходом случаев TRALI для плазмы 1: 2-300 000; тромбоцитов, 1: 3-400 000; эритроцитов, 1:25 002 000), а также наличие HLA - антител более 80% случаев).
- Минимизация использования плазмы и тромбоцитов доноров с HLA – антителами привели к снижению частоты TRALI

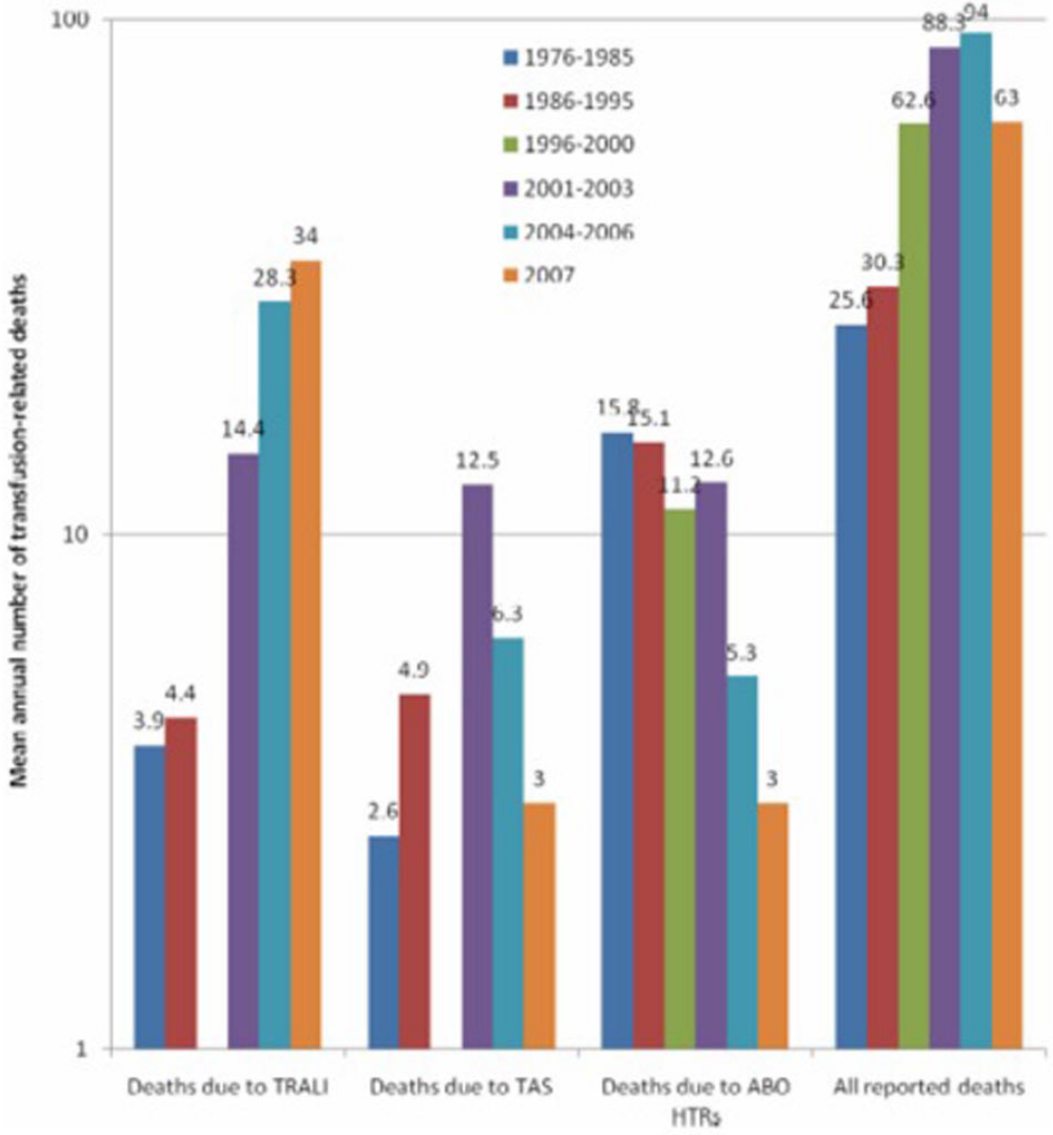


Figure 1. Transfusion-related fatalities reported to the FDA.[14] The 3 leading causes of known and reported allogeneic blood transfusion-related deaths, based on data reported passively to the US FDA over 32 years (1976-2007). For each of the 5 periods for which data have been made available, the figure shows the mean annual number of deaths deemed to be due to TRALI, transfusion associated sepsis (TAS), or ABO hemolytic transfusion reactions (HTRs), along with the mean total number of deaths reported to the FDA plotted on a logarithmic scale. Deaths reported to the FDA include donor fatalities, recipient fatalities in which allogeneic blood transfusion (ABT) was not deemed to be the probable or major cause of death, and recipient fatalities due to TRALI, TAS, ABO HTRs, and other transfusion complications. Data on TRALI and TAS are not available for the period 1996 to 2000. (Reprinted with permission.)

Патогенез(1)

- Патогенез TRALI – переливание компонентов крови, которые содержат анти-HLA антиген или анти-нейтрофилов (HNA) антиген.
- Несмотря на существенные доказательства причастности трансфузии антител в возникновении TRALI, остается неопределенность. Одним из важных наблюдений является то, что родственные антитела не обнаружены во всех клинически диагностированных случаев моделей TRALI.
- Не у всех реципиентов при трансфузиях, содержащих анти-HLA или анти-HNA антитела развивается TRALI.
- Вероятно, что, кроме трансфузии имеются ко-факторы, связанные препаратом или реципиентом, которые важную роль в патогенезе TRALI.
- **Гипотеза – у реципиента должно быть основное заболевание (заболевания), скорее всего иммунное, которое, приводит к «восприимчивости» к TRALI, и который затем запускается трансфузией.**

Патогенез(2)

- Иммунное TRALI - развитие лейкоагглютинации при переливании компонентов крови, содержащих антитела против лейкоцитов реципиента

- Не иммунное TRALI – патологические эффекты липидов, которые накапливаются в компонентах крови при ее хранении

Transfusion. 2010 November ; 50(11): 2447–2454. doi:10.1111/j.1537-2995.2010.02715.x.

Female donors and transfusion-related acute lung injury: A case-referent study from the International TRALI Unisex Research Group

Rutger A. Middelburg, **Daniëlle van Stein**, **Barbara Zupanska**, **Małgorzata Uhrynowska**, **Ognjen Gajic**, **Eduardo Muñiz-Diaz**, **Nuria Nogués Galvez**, **Christopher C. Silliman**, **Tom Krusius**, **Jonathan P. Wallis**, **Jan P. Vandenbroucke**, **Ernest Briët**, and **Johanna G. van der Bom**

Abstract

BACKGROUND—Although quantitative evidence is lacking, it is generally believed that the majority of cases of transfusion-related acute lung injury (TRALI) are caused by female blood donors. We aimed to examine the relation between female donors and the occurrence of TRALI.

STUDY DESIGN AND METHODS—We performed an international, multicenter case-referent study. TRALI patients who were diagnosed clinically, independent of serology or donor sex, and had received transfusions either only from male donors or only from female donors (unisex cases) were selected. The observed sex distribution among the donors of these TRALI patients was compared to the expected sex distribution, based on the relevant donor populations.

RESULTS—Eighty-three clinical TRALI cases were included; 67 cases received only red blood cells (RBCs), 13 only plasma-rich products, and three both. Among RBC recipients the relative risk (RR) of TRALI after a transfusion from a female donor was 1.2 (95% confidence interval [CI], 0.69–2.1) and among plasma-rich product recipients the RR was 19 (95% CI, 1.9–191). The p value for the difference between RBCs and plasma was 0.023.

CONCLUSION—Our data support the notion that plasma from female donors is associated with an increased risk of TRALI, while RBCs from female donors are not.

Факторы риска

Transfusion-related acute lung injury: incidence and risk factors

Pearl Toy,[1] Ognjen Gajic,[2] Peter Bacchetti,[1] Mark R. Looney,[1] Michael A. Gropper,[1] Rolf Hubmayr,[2] Clifford A. Lowell,[1] Philip J. Norris,[1,3] Edward L. Murphy,[1,3] Richard B. Weiskopf,[1] Gregory Wilson,[2] Monique Koenigsberg,[1] Deanna Lee,[1] Randy Schuller,[4] Ping Wu[1], Barbara Grimes,[1] Manish J. Gandhi,[2] Jeffrey L. Winters,[2] David Mair,[4] Nora Hirschler,[1,5] Rosa Sanchez Rosen,[1,3] and Michael A. Matthay,[1] for the TRALI Study Group

Transfusion-related acute lung injury (TRALI) is the leading cause of transfusion-related mortality. To determine TRALI incidence by prospective, active surveillance and to identify risk factors by a case-control study, 2 academic medical centers enrolled 89 cases and 164 transfused controls. Recipient risk factors identified by multivariate analysis were higher IL-8 levels, liver surgery, chronic alcohol abuse, shock, higher peak airway pressure while being mechanically ventilated, current smoking, and positive fluid balance. Transfusion risk factors were receipt of plasma or whole blood from female donors (odds ratio = 4.5, 95% confidence interval [CI], 1.85-11.2, *P* = .001), volume of HLA class II antibody with normalized background ratio more than 27.5 (OR = 1.92/100 mL, 95% CI, 1.08-3.4, *P* = .03), and volume of anti–human neutrophil antigen positive by granulocyte immunofluoresence test (OR = 1.71/100 mL, 95% CI, 1.18-2.5, *P* = .004). Little or no risk was associated with older red blood cell units, noncognate or weak cognate class II antibody, or class I antibody. Reduced transfusion of plasma from female donors was concurrent with reduced TRALI incidence: 2.57 (95% CI, 1.72-3.86) in 2006 versus 0.81 (95% CI, 0.44-1.49) in 2009 per 10 000 transfused units (*P* = .002). The identified risk factors provide potential targets for reducing residual TRALI. (*Blood*. 2012;119(7): 1757-1767)

• **Факторы риска у реципиента :** IL-8, хирургия печени, хроническая алкогольная интоксикация, шок, высокое пиковое давление в дыхательных путях в процессе ИВЛ, курение, и положительный баланс жидкости.

• **Факторы переливания:** плазма или цельной кровь доноров женского пола, уровень антител HLA класса II, и уровень HNA с положительным результатом гранулоцитов в тесте иммунофлуорисценции

Table 7. All components received during or within 6 hours in 89 cases of TRALI

Components	No. of cases	% of cases
Only whole blood	1	1
Only plasma	13	15
Only platelets	5	6
Only RBCs	20	22
Mixed components, no plasma	4	4
Mixed components, with plasma	46	52
Total	89	100

Table 8. Univariate patient risk factors for TRALI

	No. TRALI/no. without risk factor (%)	No. TRALI/no. with risk factor (%)	OR	Lower 95% CI	Upper 95% CI	*P*
Medical factors						
≥ 10 units, any component (per unit > 9)	NA	NA	1.17	1.05	1.31	.006
Shock before transfusion	50/184(27.2)	36/69(56.5)	4.3	2.2	8.4	< .001
Fluid balance pre-TRALI (increment per liter)	NA	NA	1.17	1.08	1.28	< .001
Acute renal failure	66/213(31.0)	23/41(57.5)	2.5	1.22	5.2	.010
Smoking history (current vs other)	67/212(31.6)	18/36(50.0)	1.99	0.95	4.2	.07
Peak airway pressure > 30 cmH_2O if mechanically ventilated	73/230(31.7)	16/21(69.6)	5.6	2.1	14.9	< .001
Chronic alcohol abuse	75/229(32.8)	12/18(88.7)	3.0	1.07	8.7	.04
Severe liver disease	68/217(31.3)	21/36(58.3)	2.9	1.34	6.2	.007
Surgery type, reference group = no surgery			1			
Liver	74/233(31.8)	15/20(75.0)	12.1	3.4	43.1	< .001
Spine	78/230(33.9)	11/23(47.8)	3.9	1.32	11.3	.013
Cardiac	77/227(33.9)	12/26(46.2)	3.3	1.21	9.2	.02
Vascular	85/243(35.0)	4/10(40.0)	2.8	0.65	12.0	.17
Other	67/178(37.6)	22/75(29.3)	1.40	0.69	2.8	.35
Markers of inflammation before transfusion						
Log_{10} IL-6	NA	NA	1.65	1.04	2.6	.03
Log_{10} IL-8	NA	NA	1.79	1.02	3.1	.04
Biologically plausible protective factors						
Immunosuppression	66/166(39.8)	22/85(25.9)	0.52	0.28	0.95	.03
Leukemia or lymphoma	82/216(38.0)	6/36(16.7)	0.32	0.12	0.83	.02
Pre-TRALI platelet count < 50×10^9/L	72/176(40.9)	10/50(20.0)	0.36	0.17	0.79	.010
Pre-TRALI WBC count < 0.5×10^9/L	81/221(36.7)	2/15(13.3)	0.32	0.07	1.51	.15
Patient illness severity as indicated by location of the patient at the time of transfusion*						
Intensive care unit (reference group)			1			
Operating room or postoperative recovery unit	46/157(29.3)	43/96(44.8)	1.05	0.51	2.2	.90
Floor	75/185(40.5)	14/68(20.6)	0.22	0.09	0.54	< .001
Hematology-oncology floor	85/229(37.1)	4/24(16.7)	0.19	0.05	0.68	.010
Outpatient	88/244(36.1)	1/9(11.1)	0.11	0.012	1.01	.05

NA indicates not applicable because the factor is a continuous variable.
*May or may not include the first transfusion.

Table 12. Primary multivariate model of TRALI risk factors: antibodies transfused to the recipient controlled for recipient risk factors

	OR	Lower 95% CI	Upper 95% CI	*P*
Transfusion risk factors among all transfusions to each patient				
Total quantity of cognate anti-HLA-class II (MFI > 1500) per 10-fold increase	3.2	1.52	6.7	.002
Total volume of anti-HNA positive by GIFT among all units, per 100-mL increase	1.71	1.18	2.5	.004
Recipient risk factors				
Chronic alcohol abuse	5.9	1.22	28.3	.028
Fluid balance before transfusion (increment per liter)	1.15	1.02	1.29	.024
Peak airway pressure > 30 cmH_2O within 12 hours after intubation before transfusion	3.6	1.01	13.1	.048
Shock before transfusion	4.2	1.69	10.6	.002
Current smoker vs never or former smoker	3.4	1.22	9.7	.020
Liver surgery (transplantation)	6.7	1.25	35.7	.027
IL-8 concentration before transfusion, per 10-fold increase	3.0	1.30	6.8	.018

See Table 8 for numbers of patients for patient risk factors. Numbers of patients who received or did not receive this transfusion risk factor are not available because multiple imputation was used for units with missing data ("Statistical methods: risk factor analysis"). The patient's plasma IL-8 level measured before transfusion was also a predictor of risk in multivariate analysis. Because this variable was missing for 50 patients, reducing the set of analyzed patients to accommodate this variable disrupted the estimates of other risk factors, and so this was not included in the primary model but listed at the end of the table.

Mechanism of transfusion-related acute lung injury induced by HLA class II antibodies

Ulrich J. H. Sachs,[1] Wiebke Wasel,[1] Behnaz Bayat,[1] Rainer M. Bohle,[2] Katja Hattar,[3] Heike Berghöfer,[1] Angelika Reil,[4] Jürgen Bux,[4] Gregor Bein,[1] Sentot Santoso,[1] and Norbert Weissmann[5]

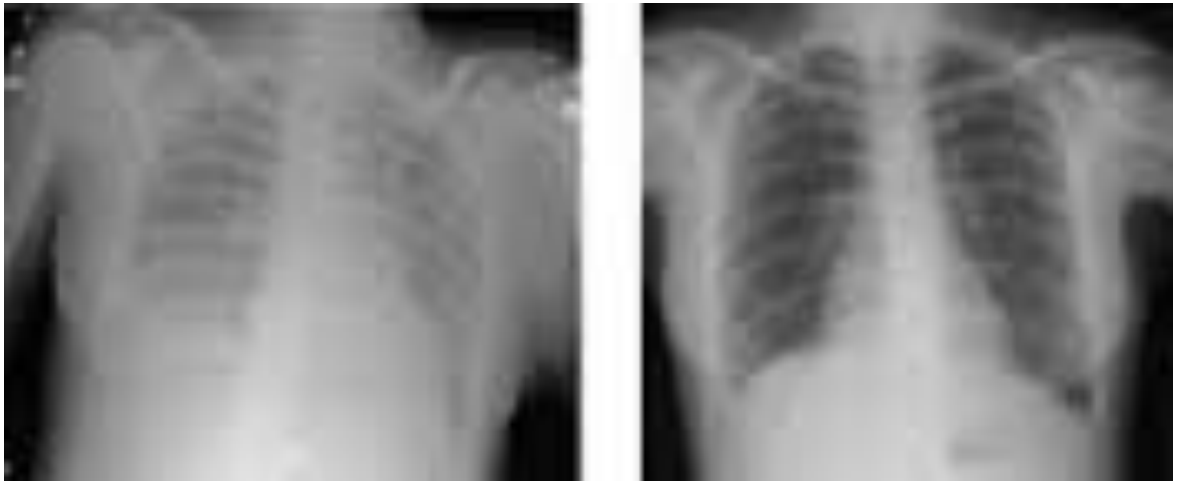
BLOOD, 13 JANUARY 2011 • VOLUME 117, NUMBER 2

Transfusion-related acute lung injury (TRALI) is the leading cause of transfusion-associated mortality in the United States and other countries. In most TRALI cases, human leukocyte antigen (HLA) class II antibodies are detected in implicated donors. However, the corresponding antigens are not present on the cellular key players in TRALI: neutrophils and endothelium. In this study, we identify monocytes as a primary target in HLA class II–induced TRALI. Monocytes become activated when incubated with matched HLA class II antibodies and are capable of activating neutrophils, which, in turn, can induce disturbance of an endothelial barrier. In an ex vivo rodent model, HLA class II antibody–dependent monocyte activation leads to severe pulmonary edema in a relevant period of time, whenever neutrophils are present and the endothelium is preactivated. Our data suggest that in most TRALI cases, monocytes are cellular key players, because HLA class II antibodies induce TRALI by a reaction cascade initiated by monocyte activation. Furthermore, our data support the previous assumption that TRALI pathogenesis follows a threshold model. Having identified the biologic mechanism of HLA class II antibody–induced TRALI, strategies to avoid plasma from immunized donors, such as women with a history of pregnancy, appear to be justified preventive measures. (*Blood*. 2011;117(2):669-677)

In conclusion, our study indicates that antibodies against HLA class II antigens are capable of inducing TRALI by monocyte activation and subsequent activation of neutrophils. Data obtained here support our previous assumption on a threshold model of TRALI and underline the importance of screening for HLA class II antibodies in suspected TRALI cases, supporting current strategies to avoid plasma from female blood donors with a history of pregnancy to reduce the number of TRALI cases.

В заключение, наше исследование показывает, что антитела против **HLA класса II** способны индуцировать TRALI путем активации моноцитов и последующей активации нейтрофилов. Данные, полученные здесь, поддерживает наше предыдущее предположение на модели TRALI и подчеркивают важность обследования на HLA антитела класса II при риске возникновения TRALI, не использовать плазму от доноров женского пола, имевших беременности, чтобы уменьшить количество случаев TRALI.

Диагностика и клиника



Pathology Consultation on Transfusion-Related Acute Lung Injury (TRALI)

Amy E. Schmidt, MD, PhD, and Jill Adamski, MD, PhD, for the Education Committee of the Academy of Clinical Laboratory Physicians and Scientists

Table 1
TRALI Diagnostic Criteria

Берлинские дефиниции

	Consensus Definitions
ALI	Acute onset; hypoxia (PaO_2/FiO_2 ≤300 mm Hg or oxygen saturation by pulse oximetry <90% on room air); bilateral pulmonary infiltrates by chest radiograph; no evidence of left atrial hypertension (ie, circulatory overload)
NHLBI	ALI developing ≤6 hours after transfusion; preexisting risk factors for ALI permissible
CCC	ALI developing ≤6 hours after transfusion; no alternative ALI risk factors permissible; *possible* TRALI: for patients with preexisting risk factors for ALI

ALI, acute lung injury; CCC, Canadian Consensus Conference; FiO_2, fraction of inspired oxygen; PaO_2, partial pressure of arterial oxygen; NHLBI, National Heart Lung and Blood Institute.

Table 2
TRALI vs TACO

Condition	Signs and Symptoms	Supporting Data	Pathogenesis
TRALI	Respiratory distress; tachypnea; hypoxemia; hypotension; noncardiogenic pulmonary edema; fever; onset within 6 hours of transfusion	Decrease in WBC count; bilateral pulmonary infiltrates on chest radiograph	HLA class I or II antibodies; human neutrophil antibodies; biological response modifiers
TACO	Respiratory distress; tachypnea; hypoxemia; cardiogenic pulmonary edema; hypertension; tachycardia; rapid improvement with diuretics	Bilateral pulmonary infiltrates on chest radiograph; increased heart size, vascular congestion, and/or pleural effusions; pulmonary artery occlusion pressure >18 mm Hg; elevated BNP	Transfusion of large volume or rapid infusion of blood products

BNP, B-natriuretic peptide; HLA, human leukocyte antigens; TACO, transfusion-associated circulatory overload; TRALI, transfusion-related acute lung injury; WBC, white blood cell.

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ISSN 1897–5593

REVIEW ARTICLE

Transfusion-related acute lung injury: A dangerous and underdiagnosed noncardiogenic pulmonary edema

Krzysztof Jaworski[1], Krystyna Maślanka[2], Dariusz A. Kosior[1, 3]

Table 1. Diagnostic criteria for transfusion-related acute lung injury (TRALI) [3, in our modification].

TRALI	Possible TRALI
1. Acute lung injury:	1. Acute lung injury:
A. Acute onset of symptoms	A. Acute onset of symptoms
B. Hypoxemia: $PaO_2/FiO_2 < 300$ or $SpO_2 < 90\%$ (no oxygen) or other clinical signs of hypoxemia	B. Hypoxemia: $PaO_2/FiO_2 < 300$ or $SpO_2 < 90\%$ (no oxygen) or other clinical signs of hypoxemia
C. Bilateral infiltrates on frontal chest radiograph	C. Bilateral infiltrates on frontal chest radiograph
D. No evidence of left atrial hypertension	
2. No preexisting acute lung injury before transfusion	2. No preexisting acute lung injury before transfusion
3. Development of symptoms during or within 6 h of transfusion	3. Development of symptoms during or within 6 h of transfusion
4. No temporal association with alternative risk factors for acute lung injury (foreign body aspiration, pneumonia, inhalation of toxins, burns, drowning, polytrauma, drug overdose, acute pancreatitis, cardiopulmonary bypass)	4. Possible temporal association with alternative risk factors for acute lung injury (foreign body aspiration, pneumonia, inhalation of toxins, burns, drowning, polytrauma, drug overdose, acute pancreatitis, cardiopulmonary bypass)

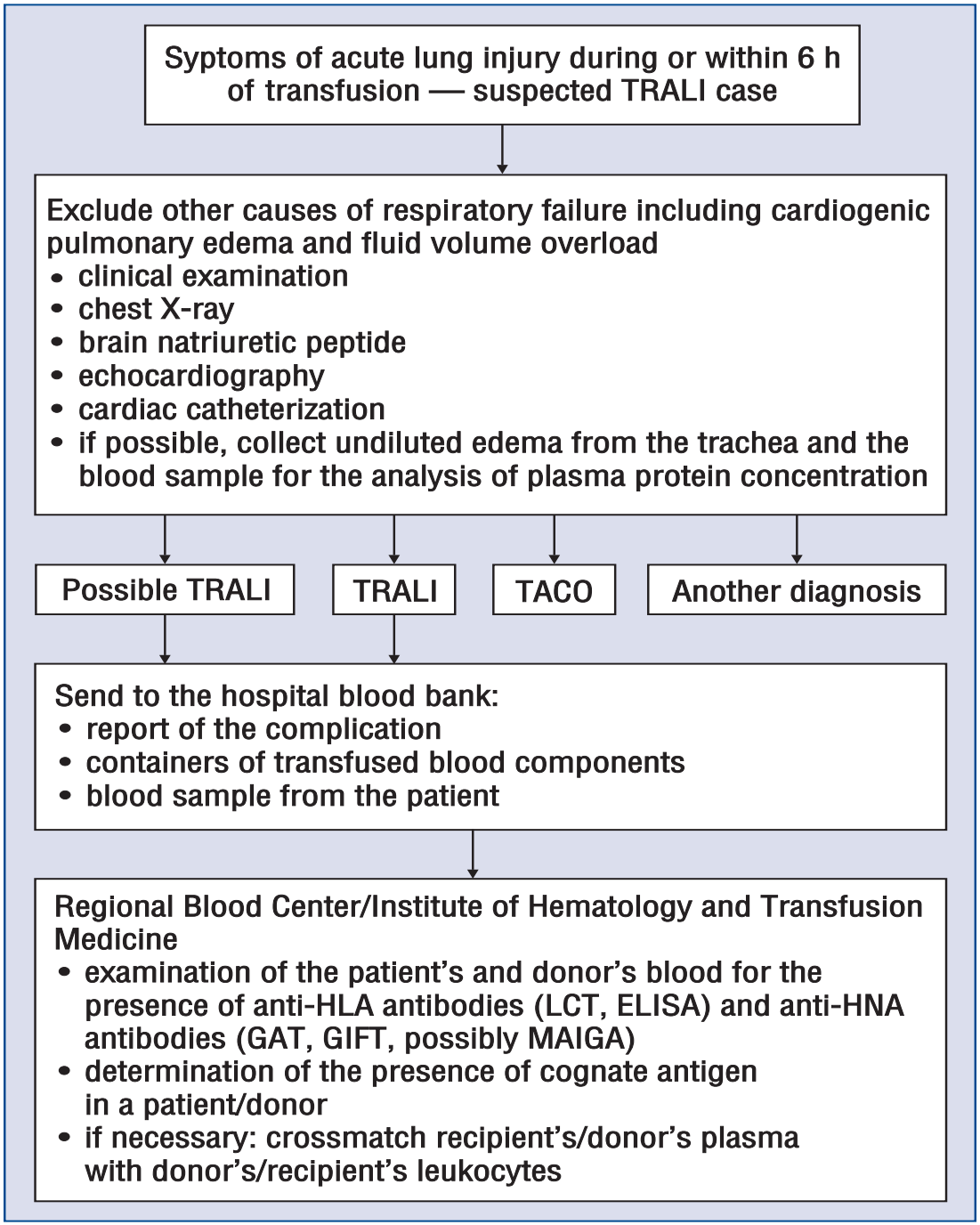


Figure 1. Transfusion-related acute lung injury (TRALI) diagnostic algorithm; TACO — transfusion-associated circulatory overload; HLA — human leukocyte antigen; HNA — human neutrophil antigen; LCT — lymphocytotoxicity test; ELISA — enzyme-linked immunosorbent assay; GAT — granulocyte agglutination test, GIFT — granulocyte immunofluorescence test; MAIGA — monoclonal antibody immobilization of granulocyte antigens.

- **Диагностика включает определение HNA или HLA антител в плазме донора и реципиента**

(Looney M.R., et al. // Chest., 2004. – V.126 (1). – P. 249-258)

В целом о клинике!!!

- **Развитие в первые 6 часов после переливания препаратов крови (но могут быть во время трансфузии!!!)**
- Одышка
- Кашель
- Озноб
- Интерстициальный, альвеолярный отек легких
- Лихорадка
- Артериальная гипотензия
- Острое начало
- Нарушение газообмена в легких
- Повышение внесосудистой воды в легких
- Двухсторонние инфильтраты на фронтальной РГОГК
- Отсутствие признаков острой левожелудочковой недостаточности
- **Отсутствие ОДН и ОРДС до трансфузии**

Интенсивная терапия

Crit Care Clin. 2012 July ; 28(3): 363–372. doi:10.1016/j.ccc.2012.04.001.

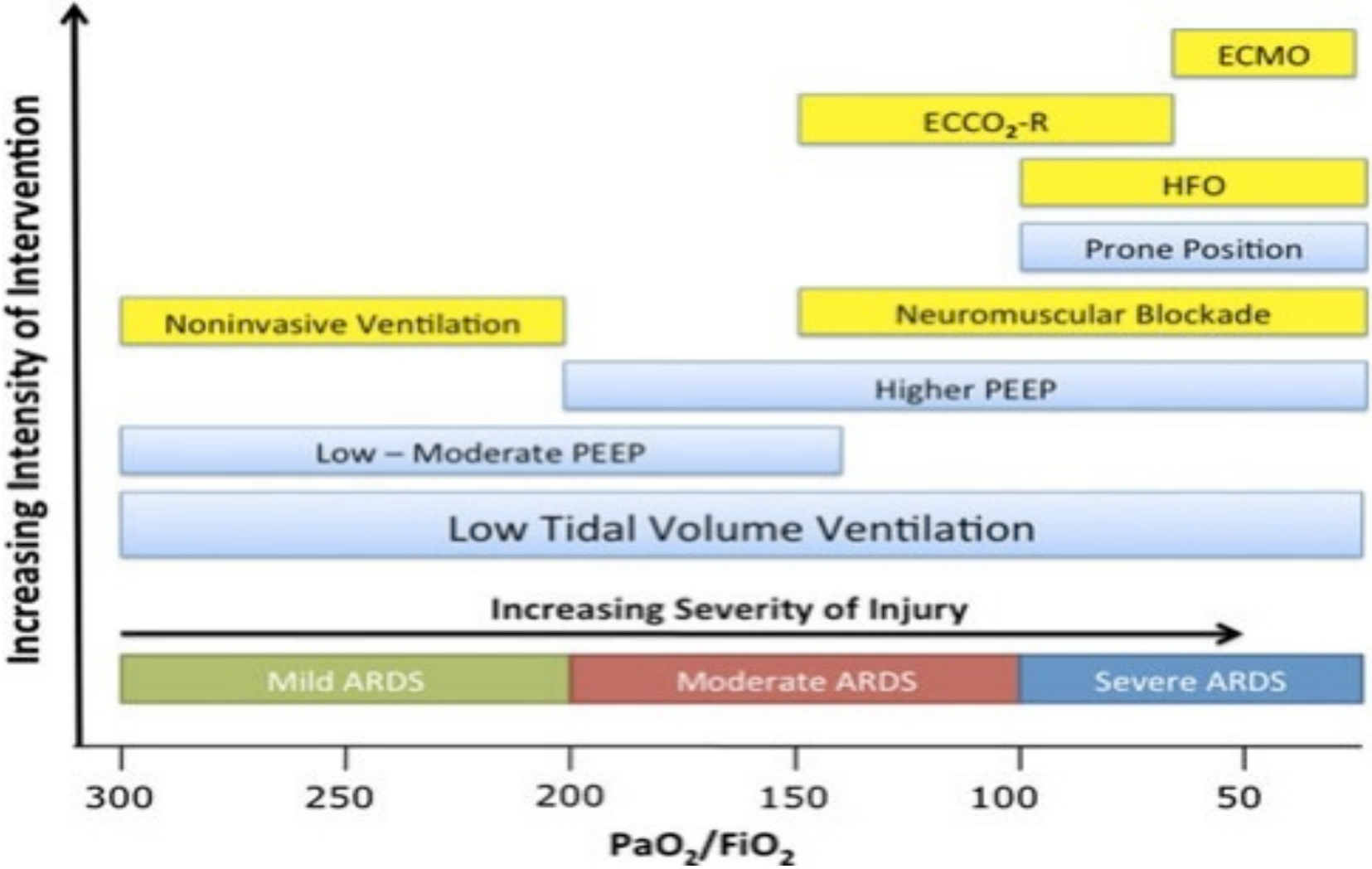
Transfusion Reactions: Newer Concepts on the Pathophysiology, Incidence, Treatment and Prevention of Transfusion Related Acute Lung Injury (TRALI)

David M. Sayah, MD, PhD, **Mark R. Looney, MD**, and **Pearl Toy, MD**
University of California San Francisco, San Francisco, CA

• Как и при других формах ОРДС, нет никакого специального лечения TRALI.
• Следует избегать введения диуретиков.
• При подозрении на TRALI трансфузию следует немедленно прекратить.
• В легких случаях достаточно кислородотерапии и рутинного ухода.
• В тяжелых случаях: ИВЛ, инфузионная терапия, мониторинг гемодинамики мониторинга и вазопрессоры (могут потребоваться).
• В редких случаях, гипоксемия в результате TRALI может быть настолько сильной, что может потребоваться ЭКМО.
• Есть данные об использовании при TRALI глюкокортикоидов, но четко их роль не определена.

Общие цели ИТ ОРДС

- 1) ликвидация заболевания, вызвавшего развитие ОРДС (проведение оперативного вмешательства, хирургическая санация очага инфекции, лечение шока и т.п.)
- 2) коррекция и поддержание приемлемого газообмена (подбор режимов и параметров респираторной поддержки, экстракорпоральные методы обеспечения газообмена)
- 3) улучшение легочного кровотока
- 4) гемодинамическая поддержка (инфузионная терапия, инотропные и вазоактивные препараты)
- 5) экстракорпоральные методы детоксикации
- 6) нутритивная поддержка
- 7) седация и анальгезия (атарактики, анестетики, наркотические анальгетики)
- 8) миорелаксанты только при тяжёлом ОРДС, на ранних этапах, кратковременно (до 48 часов)



Intensive Care Med (2012) 38:1573–1582
DOI 10.1007/s00134-012-2682-1

SPECIAL ARTICLE

The Berlin definition of ARDS: an expanded rationale, justification, and supplementary material

Niall D. Ferguson
Eddy Fan
Luigi Camporota
Massimo Antonelli
Antonio Anzueto
Richard Beale
Laurent Brochard
Roy Brower
Andrés Esteban
Luciano Gattinoni
Andrew Rhodes
Arthur S. Slutsky
Jean-Louis Vincent
Gordon D. Rubenfeld
B. Taylor Thompson
V. Marco Ranieri

Fig. 1 Aligning Therapeutic Options with The Berlin Definition (adapted from [48] with permission). This figure depicts potential therapeutic options according to the severity of ARDS. Boxes in yellow represent therapies that in the opinion of the panel still require confirmation in prospective clinical trials. This figure is just a model based on currently available information. In the coming years, various aspects of the figure will likely change; proposed cut-offs may move, and some therapies may be found to not be useful, while others may be added

Профилактика (1)

- Получение компонентов крови от доноров-женщин (в том числе цельной крови) является высоким фактором риска и уменьшение этого фактора риска в 2007-2008 годах уменьшила частоту TRALI - наблюдение в двух академических медицинских центрах с 2006 года по 2009 году с ~ 1: 4000 до ~ 1: 12000 .

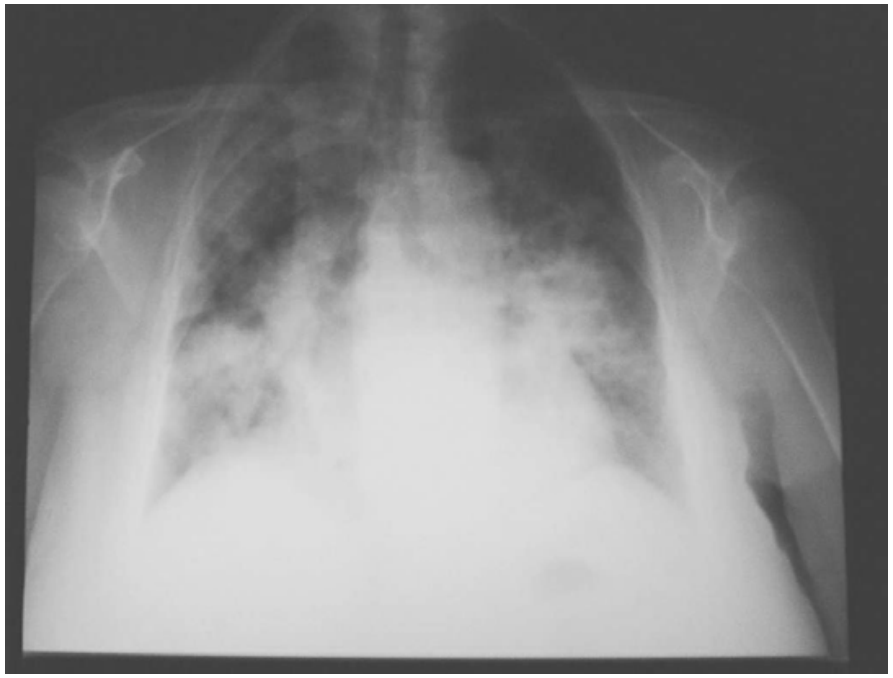
- Пациенты, имеющие факторы риска возникновения TRALI, такие септический шок, высокое PIP (> 30 см H2O) во время ИВЛ.

- **Рекомендуется уменьшить число трансфузий плазмы и тромбоцитов от доноров возможного высокого риска.**

Профилактика (2)

- Ограничение показаний к назначению трансфузии препаратов крови
- Проведение скрининга донорской крови на наличие антител против HLA и HNA
- Исключение из числа доноров лиц, трансфузия компонентов крови которых послужила причиной TRALI
- Заготовка СЗП только от доноров-мужчин
- Использование методов лейкоредукции
- Применение лейкофильтров
- Трансфузия отмытых эритроцитов, заготовленных по индивидуальному подбору

Благодарю за внимание!!!
Вопросы?



Адрес для переписки:
gritsan67@mail.ru

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