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Transfusion Related Acute Lung Injury (TRALI)

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Introduction

Transfusion related acute lung injury (TRALI), is clinically defined as the new onset of acute lung injury and hypoxia with non cardiogenic pulmonary edema, within 6hrs post blood transfusion (Hendrickson and Hillyer, 2013). Though rare, TRALI is the leading cause of transfusion related morbidity and mortality (Alam, Huang, Yi, Lin & Hannach, 2014). The pathophysiology is complex with multiple pathways being identified in a "two-hit" mechanism with immune and non-immune factors. TRALI is different from, but often confused with, transfusion associated circulatory overload (TACO) which is associated with poor cardiac function and volume overload requiring alternative treatments. Increased knowledge of this deadly transfusion reaction is needed to identify the incidence, prevention, and treatment of TRALI with more specificity . As a future provider that will be administering a large amount of blood products in the operating room as well as requesting blood products be infused prior to operations, it is important to understand and identify when this specific reaction may occur. This topic is vital for advanced practice nurses to understand, as they will be directly identifying and treating these complications, ultimately saving lives.

Signs and Symptoms

TRALI	vs	TACO
Fever		No fever
Hypotension		Hypertension
Normal ejection fraction		Decreased ejection fraction
Pulmonary edema- Exudate		Pulmonary edema-Transudate
No response to diuretics		Significant response to diuretics
BNP < 200		BNP > 1200
WBC –transient leukopenia		WBC- unchanged
Hypoxia		
Dyspnea		
Tachypnea		
Tachycardia		
Cyanosis		
Chest x-ray with bilateral infiltrates		

Pathophysiology

First hit

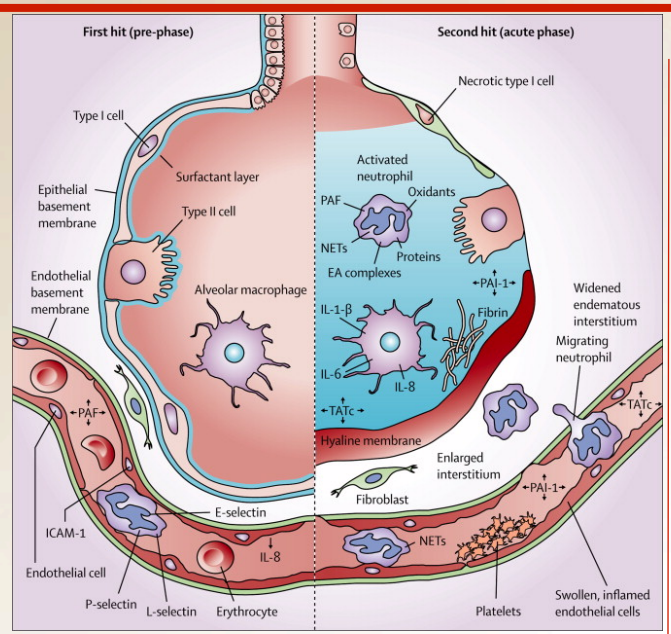
- Underlying disease such as sepsis or pneumonia causes sequestration of primed neutrophils to the activated pulmonary endothelium
- Neutrophils are attracted to the lung by release of cytokines and chemokines from upregulated lung endothelium.
- Loose binding by L-selectin takes place.
- Firm adhesion is mediated by E-selectin and platelet-derived P-selectin and intracellular adhesion molecules.

(Vlaar & Juffermans, 2013)

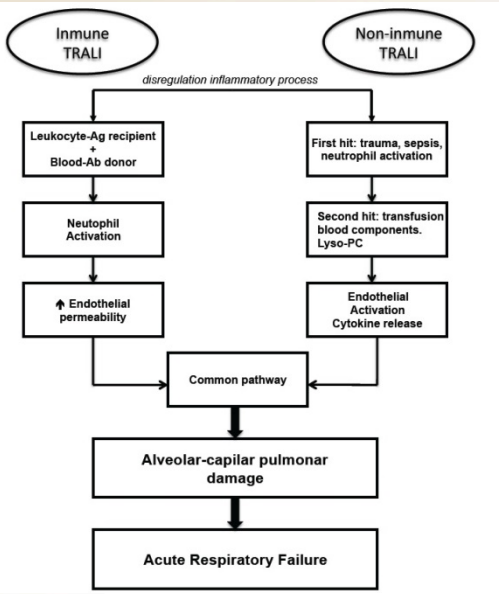
Second hit

- Initiated by transfusion of a blood component.
- Compounds induce neutrophil activation and release of cytotoxic factors causing endothelial damage and capillary injury.
- Reactive neutrophils secrete inflammatory mediators such as cytokines (IL-1, IL-6, IL-8, TNF-alpha), release proteolytic enzymes, and produce reactive oxygen species.
- Events initiate the cascade of immune or non-immune responses resulting in damage to the vascular endothelium and pulmonary capillary fluid leakage in to the alveolar space.

(Álvarez, Carrasco, Romero-Dapueto & Castillo, 2015)



(Vlaar & Juffermans, 2013)



(Álvarez, Carrasco, Romero-Dapueto & Castillo, 2015)

Immune Mechanism

- HLA or HNA antibodies in donor plasma react with antigens on recipient leukocytes leading to injury and fluid accumulation in the alveoli.
- Antibodies to HLA class I, HLA class II, or HNA antigens in blood donors have been reported in the majority of cases
- Multiparous female donors are most likely to have these antibodies.

(Hendrickson & Hillyer, 2013)

Non-Immune Mechanism

- Antibodies are not detected
- Caused by simultaneous transfusion of biologically active lipids or cytokines that accumulate during storage.

(Hendrickson & Hillyer, 2013)

Significance of Pathophysiology

It is important for providers to quickly recognize and differentiate TRALI from other pathologic processes. Knowing the origins and risk factors of TRALI based on the pathophysiology can help preemptively prevent the incidence. Male donors, female donors with no prior pregnancy, or donors negative for HLA-antibodies provide decreased risk of TRALI (Vlaar & Juffermans, 2013). The pathophysiology also has a direct effect on treatments that are effective and those that should be avoided. A classic example is that there is no benefit of diuretic or steroid therapy as in other respiratory failure events (Kleinman & Kor, 2017). Though both present similarly following blood product transfusion, a Taco will significantly respond to diuretics but a TRALI may be worsened by the same therapy due to the pathophysiology taking place.

Implications for Nursing Care

It is important that advanced practices nurses stay vigilant on the prevention of TRALI as a first resort by recognizing patients with predisposed risks. Once TRALI is diagnosed It is the advanced practice nurse's job to begin immediate supportive therapy. Oxygen therapy, Central access and Intubation may be a necessary intervention to perform if hypoxia and hypotension are severe. Providers must be aware of the pathophysiology and quickly stop any transfusions and provide fluid to treat hypotension, avoiding diuretics despite shortness of breath and bilateral chest infiltrates on x-ray film. Basic but quick supportive care is crucial along with follow up monitoring as more intervention is not always better in TRALI.

Conclusion

- Leading cause of transfusion related mortality.
- hypoxemic respiratory distress during or within six hours of transfusion.
- "Two-Hit" pathophysiology hypothesis.
- Therapy is supportive including fluid, oxygen, ventilator support and lung protective strategies.
- Avoid diuretics and steroids unproven to improve outcome
- Important to differentiate from TACO
- Patients who survive TRALI episodes generally recover completely.

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