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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 90 minutes post blood transfusion (Hendrickson and Hilayer, 2015). Though rare, TRALI is the leading cause of transfusion related morbidity and mortality (Ahmet, Huang, Yi, Li, & Hanchar, 2020). 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 your 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 who 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**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>TRALI</th>
<th>TACO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>Hypothermia</td>
<td>No Fever</td>
</tr>
<tr>
<td>No response to diuretics</td>
<td>Decreased ejection fraction</td>
<td>No response to diuretics</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Hypertension</td>
<td>No hypoxia</td>
</tr>
<tr>
<td>Anuria</td>
<td>Acute kidney injury</td>
<td>No anuria</td>
</tr>
</tbody>
</table>

**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.
- Firm adhesion is mediated by selectins and platelet-derived P-selectin and intracellular adhesion molecules.

#### Second hit

- Initiated by transfusion of a blood component.
- Compounds induce neutrophil activation and release of cytokotic factors causing endothelial damage and capillary injury.
- Reactive neutrophils secrete inflammatory mediators such as cytokines (IL-1, IL-6, IL-8, TNF-α), 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 the alveolar space.

(Álvarez, Carrasco, Romero-Daparto & Castillio, 2015)

### Immune Mechanism

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

(Hendrickson & Hilayer, 2015)

### Non-Immune Mechanism

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

(Hendrickson & Hilayer, 2015)

### Implications for Nursing Care

It is important that advanced practice 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. Oxygenation, 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 seen on a x-ray film. Real time support from a critical care is crucial along with follow up monitoring as more intervention is not always better in TRALI.

(Álvarez, Carrasco, Romero-Daparto & Castillio, 2015)

### Conclusion

- Leading cause of transfusion related mortality.
- Hypoxic 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.

### References Cited


### Additional Sources


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**Pathophysiology**

**First hit**

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- Neutrophils are attracted to the lung by release of cytokines and chemokines from upregulated lung endothelium.
- Firm adhesion is mediated by selectins and platelet-derived P-selectin and intracellular adhesion molecules.

(Álvarez, Juffermans, & Vlaar, 2015)

**Second hit**

- Initiated by transfusion of a blood component.
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