Dealing with Massive Hemorrhage and Disseminated Intravascular Coagulopathy

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Massive Hemorrhage

• Loss of a total blood volume within a 24 h period (Mollison et al, 1997)

• a 50% blood volume loss within 3 hrs or a rate of loss of $\geq 150$ ml/min (Fakhry & Sheldon, 1994).
# Clinical Classification of Blood Loss

(based on est. blood vol of 85cc/kg –70kg pt.)

<table>
<thead>
<tr>
<th>Class</th>
<th>Acute Blood Loss</th>
<th>% Lost</th>
<th>Clinical Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1000cc</td>
<td>15</td>
<td>Palpitations, dizziness</td>
</tr>
<tr>
<td>2</td>
<td>1,500cc</td>
<td>25</td>
<td>Tachy (100-120), Orthostatic BP changes ↓ pulse pressure</td>
</tr>
<tr>
<td>3</td>
<td>2000cc</td>
<td>35</td>
<td>Tachy (120-140), Hypotension (Systolic 80-100 mg/Hg) Cool extremities</td>
</tr>
<tr>
<td>4</td>
<td>2500cc or &gt;</td>
<td>40</td>
<td>Tachy (≥140), Shock (Systolic &lt;60) Oliguria, air hunger</td>
</tr>
</tbody>
</table>
Expected Fall in Pt’s HgB

- Bedside Pearl:
- For every 500 cc of blood loss expect the patient’s HgB to fall by ~1 gram in the acute scenario
“What happens normally with clotting”

Endothelia Disruption ("injury")

Platelet Activation ("plugs hole")

Intrinsic Clotting ("help arrives")

Cascade

Fibrin Meshwork ("layers of screen doors" cover hole)

Plasmin (Clipper trims meshwork to fit)

FSP ("Fragments of the screen door" filtered out)
A “Practical” Clotting Cascade

PET
Calcium, Thromboplastin, 7
(CAT - 7)
(Thromboplastin is “GUNK” from IUFD, AFE, Abruptio, tissue injury, endotoxins, etc.)

PiTT
8, 9, 11, 12, PLTS

Common Pathway
Calcium, 5, 10
“Calcium Five and Dime”

Prothrombin

Thrombin

Fibrinogen → FIBRIN
Plasminogen → Plasmin (Clipper)

FSP
DIC

Disseminated (systemic)

Intravascular

Coagulopathy

“Systemic rather than local generation of Thrombin and Plasmin”
Obstetric Causes of DIC

Extrinsic “GUNK” in the circulation that activates the cascade

- Abruptio Placentae
- AFE
- IUFD
- Septicemia
- Massive Hemorrhage
- Preeclampsia
- Transfusion RXN
- Fatty Liver
“What Problems Arise from DIC?”

FSP
“Screen Door Fragments”

Leads to PLTS, Dysfunction, & Prevents Fibrin Polymers

Bleeding

Damage endothelia lining of pulm capillary bed (acute Lung Injury)

Damage Surface of RBC’s (Hemolysis)

Plugs the Microcirculation

Tissue Necrosis (Organ Injury)
Kidney, Liver, etc.
“Diagnosis of DIC”

Clinical Exam - “Most Important”

- Bleeding from unrelated site
  (Venipuncture oozing, epistaxis, hematuria, gingival/mucosal bleeding, purpura, or petechiae)
- Shock out of proportion to Blood Loss (Bradykinin)
- Renal Failure, lung injury and other end organ injury
“Laboratory Diagnosis of DIC”

“Basics” of Abnormal Labs:
- Low Fibrinogen (< 300 mg)
- Prolonged PT (Then PTT)
- Low Platelets (< 150 K)
- Elevated FSP (or D-Dimer)
- Hemolysis on peripheral smear
- Prolonged Thrombin Time
  (Time Fibrinogen → Fibrin)
“General Points” Laboratory

- PT/PTT become prolonged when Fib < 100 mg % or Factors depleted by ≥ 50%.
- 15% of Pts with DIC have NL FSP (over degradation)
- FSP > 330 ug/ml - uterine inertia, PP bleeding
- Clot observation (< 6 min:Fib > 150mg%) if no clot after 30 min:Fib < 100 mg%
  if clot forms and lyses w/in 30 min: ↑ Plasmin
Treatment of DIC

Basic Tenets:

• Treat underlying disorder
• Aggressively support blood volume, blood pressure and tissue oxygenation.
• Component therapy
• Push for NSVD
• Avoid conduction anesthesia
Component Therapy: Indicated for abn bleeding or subnormal values prior to surgery or vaginal delivery.

- Packed RBCs
- Fresh Frozen Plasma (FFP)
- Platelets
- Cryoprecipitate
- Massive transfusion protocols-1:1:1 or 6:4:1
## Blood Product Replacement

<table>
<thead>
<tr>
<th>Component</th>
<th>Contents</th>
<th>Volume</th>
<th>Anticipated Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Packed rbc</td>
<td>rbc, wbc, plasma</td>
<td>300cc</td>
<td>Increase Hgb 1g/dL per unit</td>
</tr>
<tr>
<td>Platelets</td>
<td>platelets, rbc, wbc, plasma</td>
<td>50cc</td>
<td>Increase platelet count 7000/mm³ per unit</td>
</tr>
<tr>
<td>FFP</td>
<td>plasma, clotting factors</td>
<td>250cc</td>
<td>Increase fibrinogen 5-10 mg/dL per unit</td>
</tr>
<tr>
<td>Cryoprecipitate</td>
<td>fibrinogen, factors V, VIII, XIII, vWF, fibronectin</td>
<td>15 cc</td>
<td>Increase fibrinogen 5-10 mg/dL per unit</td>
</tr>
</tbody>
</table>
Component Therapy

**Packed RBCs**

- “One unit of packed RBCs - increases total hemoglobin by 1 and total hematocrit by 3.

- Transfuse the hemorrhaging patient in shock to restore oxygen - carrying capacity and delivery
Component Therapy

Fibrinogen

Sources:
A. FFP (vol = 250cc): increases total Fibrinogen in Circulation by 5-10 mg% per unit
B. Cryoprecipitate (vol = 35-40cc): Also increases total Fibrinogen by 5-10 mg% per unit. (contains no AT III)

Replace to correct total Fibrinogen ≥ 100 mg %
Component Therapy

Platelets

- "Single Donor" Platelets - one unit increases total circulatory platelet count by 30-60,000 (less infectious risk)
- "Pooled" Platelets - One unit increases total circulatory platelet count by ~ 7000
  (6 pack - 42,000)

Replace plts to 60,000

(Prophylaxis if PLTS < 10-20,000 or in preoperative patient < 50K)
Recombinant Activated Factor VII: A New Weapon in the Fight Against Hemorrhage

- Vit K – dependent protein → approved (Hemophilia) Promotes clotting through extrinsic pathway (tissue factor) (Complexes with tissue factor → activates Factor IX and X, and generates thrombin)
- Dose 60-80 micrograms / Kg IV bolus
- Controls bleeding rapidly – 10 minutes!
- Initially, very few adverse effects reported – TED is a clear risk (7% in ICH tx study, Mayer 2005)
- Short ½ life (2 hours)
- High Cost – 1400.00/milligram

Bouwmeester FW. Obstet Gynecol 2003
Danilos J. Obstet Gynecol 2003
Additional Agents

• **Aprotonin**: Directly inhibits Plasmin
• **Tranexamic acid**: Like aminocaproic acid but 10x stronger (prevents plasmin binding to fibrin)

• Both may reduce Blood transfusion by 30%
• (primarily cardiac surgery)

• Cochrane review—Data from Henry, et al 2001
Summary

Reviewed/Discussed:

- Massive hemorrhage and the normal clotting process.
- The clotting cascade!
- The basic pathophysiology of DIC.
- The obstetric causes of DIC.
- Clinical/Laboratory Diagnosis.
- Management/Treatment of DIC and massive hemorrhage.