Hypocalcemia and the Lethal Triad

SGT Ditzel Ricky M Jr
CCP-C, FP-C, TP-C, SO-ATP
AGENDA

- Case Review
- Definition
- Hypocalcemia
- Physiology
- SOF Initiative
- Evidence
- Protocols
- Synopsis
- Reference List
- Questions
Calcium(Ca++) is a major cation for multiple physiologic functions of the body. *** WHAT DOES Ca++ do?***

Ca++ is measured in two forms:

- Total Serum: 8.2-10.5mg/dL
- Serum Ionized: 4.5-5.2mg/dL
  - 1.3-1.5mmol/L
Case Review

- Location: Cooper University Hospital, Camden NJ
- Approximately 1600 EST A 21 y/o Asian female is brought to the ED via ground transport.
- G: PT is A&Ox1, supine, cool, pale and clammy, with bimanual vaginal pressure from the Resident OBGYN.
- O: PT has a spiral tear from her vagina to her uterus.
- Tx: Methergine, hemabate, mass transfusion, and Pitocin.
- Outcome: Surgical reconstruction of vaginal cavity and uterus, PT positively diagnosed with disseminated intravascular coagulopathy(DIC). Extubated 4 days post op.

- What could have gone better?
Serum Ionized Calcium <4.5mg/dL

<table>
<thead>
<tr>
<th>Acute Signs/Symptoms</th>
<th>Chronic Signs/Symptoms</th>
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<tr>
<td>Trousseau's Sign</td>
<td>Dementia</td>
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<tr>
<td>Chvostek's Sign</td>
<td>Dry Skin</td>
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<tr>
<td>Perioral Paresthesia</td>
<td>Abnormal dentition</td>
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<td>Fatigue</td>
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<td>Prolonged QT interval</td>
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<td>Seizures</td>
<td></td>
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</table>
Traumatic Hypocalcemia

\[ \text{DO2} = \text{CaO2} \times \text{CO} \]
Traumatic Hypocalcemia

\[ \text{DO2} = \text{CaO2} \times \text{HR} \times \text{SV} \]

\[ (\text{Spo2} \times 1.34 \times [\text{HGB}]) + (0.0003 \times \text{PaO2}) \]

\[ \text{CO} \]
Traumatic Hypocalcemia

\[ \text{Do2} = \text{CO} \times \text{SAO2} \times \text{HGB} \times 1.34 \]
## Traumatic Hypocalcemia

<table>
<thead>
<tr>
<th>100Kg M</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
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</thead>
<tbody>
<tr>
<td>Blood Loss (mL)</td>
<td>Up to 750</td>
<td>750-1500</td>
<td>1500-2000</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>Blood Loss (dL)</td>
<td>Up to 7.5</td>
<td>7.5-15</td>
<td>15-20</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Ca++ Loss (mg)</td>
<td>0-75</td>
<td>75-150</td>
<td>150-200</td>
<td>&gt;200</td>
</tr>
</tbody>
</table>

Coagulopathy
Traumatic Hypocalcemia

Protein Bound Ca++ mg/dL

Free Ionized Ca++ mmo/L

Acidosis

K. Ho 2016
• Citrate is metabolized in the liver
• Citrate in blood bags insignificant in a healthy liver
• Hemorrhage leads to hypothermia and decreased iC++
Traumatic Hypocalcemia

“You can’t punk physiology”

Anonymous
So what happens when there is an insult to the endothelium?
What was the role of calcium in this process?

PLA2

Arachidonic Acid

LOX  COX

TXa2
Physiology-Clotting Cascade

10

7

Fibrinogen

1

2

8

5

11

9

12

>0.56mmol/L Ca++

Fibrin
“There hasn’t been a new idea since 1776.”

LTC Theodore Redman
WB Questions?

Hypotensive resuscitation = hemostatic procedure, not optimal treatment
→ How long can you go hypotensive with WB (or components)?
→ How high can you bring BP without popping clot?

Hemostatic performance limits of stored WB
→ CPD to 21d vs. CPDA to 35d vs. components
→ WFWB added to stored: optimal ratio (3:1, 4:1, components?)
→ Leukoreduction, pathogen reduction, etc. effects?
→ New storage solution?
→ Better warmers?

LTOWB
→ Best titer methodology vs. antibody removal?

Prolonged Field Care
→ Can you “stretch” WB with albumin, fibrinogen, PCC?
“Ionized calcium levels in major trauma patients who received blood en-route to a military medical treatment facility”

Who: Royal British Military provided a retrospective study.
What: Compare the evidence of hypocalcemia in patients receiving blood transfusions.
When: Jan 2010-Dec 2014
How: 297 SM requiring blood transfusion were divided into a treatment group and non treatment group.
Results: Non-treatment group(166) 70% were hypocalcemic compared to the treatment group 28.3% were hypocalcemic.
Suggestions: 1 unit drops iCa++ to ~1.12mmo/L
2 units drops iCa++ to <1.0mmo/L
5 units drops iCa++ to <.8mmo/L

Kyle et al., 2017
“Concentration–dependent effect of hypocalcemia on mortality of patients with critical bleeding requiring massive transfusion: a cohort study”

Who: Western Australia University
What: Compare the sensitivity of concentrations to mortality.
How: 352 patients requiring mass blood transfusions from traumatic hemorrhage.
Results: Hypocalcemia was the most critical variable in determining mortality than fibrinogen, or acidosis levels. Determined that there is a linear concentration dependent relationship to mortality.

K. Ho 2016
Research

Conclusions
Proposal

- Hypocalcemia
- Acidosis
- Hypothermia
- Coagulopathy
Transfusion Criteria

- Two or more distal amputations or,
- One proximal amputation, or,
- Non-Compressible hemorrhage with signs of shock (SBP <100mmHg, and/or HR>100bpm).
- Controlled hemorrhage with signs of shock.
- Traumatic arrest within 5 minutes of loss of vital signs.
Mild Toxicity - Slow or stop transfusion until symptoms subside. Ensure proper mixture and concentration of citrate.

Severe Toxicity - Give 0.45 mEq elemental calcium or approximately 1ml of a 10% Calcium Gluconate (100mg/ml) for each 100mL citrated blood infused. Infuse over 10-20min for each 1 to 2gm of calcium gluconate. Diluted prior to administration (D5w or NS 100-250mL)

Administer appropriate IV Fluid bolus (500cc NS/LR) and re-assess casualty. Repeat bolus once after 30 minutes if still in shock.

If Blood products are available, consider resuscitation with plasma (FFP) and packed red blood cells (PRBCs) in a 1:1 ratio.
Proposed Protocol-DCR

Best

1. Obtain IV/IO access x2
2. Start infusion of LTOWB/FDP/pRBC through one line w/ fluid warmer attached.
3. Infuse 1G calcium chloride/gluconate in 100mL bag of NS bolus.
4. After infusion of calcium, flush line, infuse 1G TXA over 1-2min IV push.
Proposed Protocol-DCR

No blood/products

1. Obtain IV/IO access x2
2. Infuse 1G calcium Chloride/Gluconate in 100mL bag of NS bolus w/ fluid warmer.
3. Infuse 1G TXA IV push over 1-2min
Ionized calcium is a critical electrolyte for multiple physiologic functions throughout the body.

Hypocalcemia is directly related to the patients outcome.

Early treatment of hypocalcemia independent from citrate toxicity can decrease mortality rates.

Identification and treatment should take place in the platinum minutes.

Further research is needed to be conducted in this field to determine the perfect treatment plan.
Synopsis

School of Medicine & Health Sciences

THE GEORGE WASHINGTON UNIVERSITY


