Using TEG to Guide Treatment of Hemorrhage... TIC ... Coagulopathy of TBI...

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Disclosure Statement
- Bader
  - Board of Directors: Secretary
  - Neurocritical Care Society
  - Mentor
  - Bard
  - Haemonetics
  - Medical Advisory Board
  - Brain Trauma Foundation and Neuoptics
  - Scientific Advisory Board
  - Cerebrotech
  - Stock options
  - Neuoptics and Cerebrotech

Learning Objectives
- Identify specific trauma patient populations at great risk for hemorrhagic shock and pathophysiology of hemorrhage/Trauma Induced coagulopathy
- Describe the normal/abnormal dynamic clotting parameters of Thromboelastography (TEG) and propose treatment using an algorithm
- Strategize treatment options involving actual cases of hemorrhage

Coagulation Continuum
- Thrombosis
- Bleeding
- Balance

Hemostatic Process
- Endothelium damaged
- Platelet plug formed (white clot)
- Thrombin generated on platelet surface
- Platelet-fibrin plug formed (red clot)
- Clot lysis

Hemorrhage States
- Trauma
  - Trauma Induced Coagulopathy
  - Traumatic Brain Injury
- Intracranial Hemorrhage
  - ICH
  - SAH
- GI Bleeding
- Liver disease/disorders
- OB Hemorrhage
- Ruptured vessels
Trauma Injury, Hemorrhage, & TBI

- Trauma/Injury is the 2nd leading cause of death globally
  - 40% of mortality associated with injury due to uncontrollable hemorrhage
- 1/3 of severely injured trauma patients sustain Trauma Induced coagulopathy (TIC)
  - Poorly understood mechanisms
  - Several theories
- Coagulopathy of TBI (CTBI) is a component of TIC
  - Multiple theories contribute to early platelet dysfunction
  - Correlation between severity of TBI and platelet dysfunction

The Coagulopathy of Trauma

Coagulopathy of TBI (CTBI)

- Presence of CTBI ranges 10-97% in ROL due to many factors
  - Heterogeneity of patients, types of lab tests, timing of tests, and lack of clear defined consensus to define CTBI
  - Associated with poor outcomes
  - Blunt TBI: coagulopathy increases mortality (50% vs 17.3%) compared to no coagulopathy
  - Factors increase risk include GCS<8, ISS>16, hypotension on admit, cerebral edema, SAH, shift

Coagulopathy of TBI (CTBI)

- Multiple Factors – Multiple Theories
  - TF release by the brain
  - Tissue/platelet degeneration
  - Disseminated intravascular coagulation (DIC)
  - Activation of Protein C

Coagulopathy of TBI (CTBI)

- Platelets & Platelet Activating Factor Theories
  - TBI may result in platelet hyperactivity
  - Platelet activating factor (PAF) induces aggregation and contributes to hypoxia-induced breakdown of the BBB
  - Tissue Factor normally not exposed to circulating blood volume...in TBI brain tissue (rich in TF) & platelets (breakdown) release TF in response to the injury and other cellular dynamics

Exhausted Platelet Dysfunction

BBB disruption releasing TF (Castellino et al 2014)

- Qualitatively different form that found in most tissues (unexposed to soluble clotting factors –unsaturated by factor VII)
- Liberation of free TF into circulation, provokes TF binding to VIIa on a massive scale
  - Results in stimulation of thrombin production in the initiation phase
  - Flood of TF generated thrombin results in platelet exhaustion syndrome
  - Large numbers of circulating platelets gain in a refractory state
    - Leads to Platelet inhibition at the ADP receptor site (Davis et al 2013)
    - Platelets incapable of stimulation and cannot form a stable thrombus through usual pathways
  - Platelet count usually normal (Davis et al 2013)
  - No evidence of fibrinolysis (Davis et al 2013)
Platelet Dysfunction as an Early Marker for CTBI

Platelet Dysfunction is an Early Marker for Traumatic Brain Injury-Induced Coagulopathy

Platelet Dysfunction

Platelet count (Platelet count) within 15 min

Human: ADP inhibition 93% in TBI

Massive Transfusion Protocol

Massive Transfusion Protocol

Fluid resuscitation

– Crystalline resuscitation done judiciously to avoid dilutional coagulopathy and tissue edema

• Normalthermia Maintenance
  – Warm fluids and Bair Hugger

• Transfusion Related Medications
  – TXA
  – Calcium Chloride
  – Reversal agents for anticoagulant, antiplatelet therapy

• Ongoing Assessment of Lab values
  – Point-of-care testing (Chem 8, PT/INR, Lactate)
  – ABG, CBC, PT/INR/PTT/INR

• Endpoints of Resuscitation
  – Rota-driven massive transfusion
  – Goal-directed transfusion
  – TEG
Measuring TIC and CTBI
Value of Viscoelastic Analysis
- Viscoelastical Hemostatic Assays (VHAs) tests that reflect the new understanding of hemostasis
  - Initiation → Amplification → Propagation
  - TEG and ROTEM
- VHAs assess properties of coagulation in whole blood
  - Can differentiate between low fibrinogen and reduced platelet function as the cause of impaired clot strength as well as systemic hyperfibrinolysis
- Clinical value of VHA is corroborated by > 30 clinical studies on patients with massive hemorrhage—
  - Demonstrates Superiority over conventional coagulation tests

Assessing Coagulopathy after TBI

Hemostasis Monitoring:
TEG Hemostasis System
- Whole blood test
- Measures hemostasis
  - Clot initiation through clot lysis
  - Net effect of components
- TEG system
  - Laboratory-based
  - Point of care
  - Remote, can be networked
  - Flexible to institution needs

TEG Technology:
How It Works
- Cup oscillates
  - Pin is attached to a torsion wire
  - Degree of pin movement is a function of clot kinetics
- System generates a hemostasis profile
  - From initial formation to lysis

Thrombin Formation (Clotting Time)
The R Parameter: Identified
- Reaction time
  - Time from the start of the test, when the pin is stationary, to the time of initial fibrin formation when fibrin creates a connection between the surface of the cup and the surface of the pin
- Expression of enzymatic reaction—the ability to generate thrombin and fibrin
- Normal range 5-10 minutes

Thrombin Formation Abnormalities
The R Parameter: Elongated R
- Possible causes of imbalance:
  - Slow enzymatic reaction
  - Possible etiologies:
    - Factor deficiency/dysfunction
    - Residual heparin
    - Anticoagulants
    - Warfarin
    - Novel AC
- Common treatments:
  - FFP
  - Protamine
  - PCC
Fibrinogen

The α (Angle) Parameter: Identified

- Rate of increase in pin oscillation amplitude as fibrin is generated and cross-links are formed
  - Conversion of Fibrinogen → fibrin
  - Interactions among fibrinogen, fibrin, and platelets
  - The faster the rate of fibrin generation, the greater the increase in pin oscillation amplitude, and the larger the angle.

Normal α (Angle) is 53-72 degrees

Fibrinogen Abnormalities

The α (Angle) Parameter: Low α

A low angle suggests a slow rate of fibrin formation, which could lead to bleeding.

- Possible causes of imbalance:
  - Slow rate of fibrin formation
  - Possible etiologies:
    - Low fibrinogen levels or function
    - Insufficient relative amount of thrombin generation
    - Platelet deficiency/function

Common treatments:
- FFP
- Cryoprecipitate

Fibrinogen Abnormalities

The α (Angle) Parameter: High α

- Possible causes of imbalance:
  - Fast rate of fibrin formation
  - Possible etiologies:
    - Platelet hypercoagulability
    - Fast rate of thrombin generation

Since a high angle is the result of an imbalance in other phases of the hemostatic process, there is no specific common treatment for it. Possibilities for reducing the angle are anticoagulation and platelet inhibition.

Fibrinogen Abnormalities

The α (Angle) Parameter: Low alpha Angle

Pt admit with history of liver, liver failure, septic shock...

Fibrinogen Abnormalities

The α (Angle) Parameter: High α

Patient is given way to much fibrinogen in the OR

Platelet Function

The MA Parameter: Defined

- Maximum amplitude
- Clot strength = 80% platelets + 20% fibrinogen
- The stronger the clot, the greater the amplitude of pin oscillation.
- Platelet function influences thrombin generation and fibrin formation

Normal MA=50-70 mm
Platelet Function Abnormalities

The MA Parameter: Low MA

- Abnormalities in the MA value represent an imbalance in the hemostatic system, and are typically associated with platelet function because of the 80% contribution by platelets to clot strength
- Possible causes:
  - Insufficient platelet-fibrin clot formation
  - Poor platelet function
  - Low platelet count
  - Low fibrinogen levels or function

Common treatments:
- Platelet transfusion

Platelet Function Abnormalities

The MA Parameter: High MA

- Possible causes:
  - Excessive platelet activity due to platelet hypercoagulability
  - Patients with an abnormally high MA are at higher risk of a thrombotic event

Common treatments:
- Antiplatelet agents
  - Note: Should be monitored for efficacy and/or resistance (See Module 6: Platelet Mapping)

Fibrinolytic Abnormalities

LY30 Parameter: Primary Fibrinolysis

- When fibrinolysis is greater than the rate of clot formation, or when it causes the breakdown of new clots, bleeding typically occurs. This condition is primary fibrinolysis and is identified with the TEG analyzer by an LY30 value of greater than 7.5% (or EPL > 15%), combined with a CI value of less than or equal to 1.0
- Possible causes:
  - Excessive rate of fibrinolysis
- Possible etiologies:
  - High levels of tPA
- Common treatments:
  - Anti-fibrinolytic agent

Fibrinolytic Abnormalities

LY30 Parameter: Primary Fibrinolysis

Postinjury Fibrinolysis: S/P MVC

Mortality rate by percent fibrinolysis

- Mortality rate by percent fibrinolysis

- Mortality rate by percent fibrinolysis

- Mortality rate by percent fibrinolysis

- Mortality rate by percent fibrinolysis

- Mortality rate by percent fibrinolysis
What about Rapid TEG?

Rapid TEG

- Jeger and colleagues evaluated Rapid TEG
- r-TEG utilizes tissue factor in addition to kaolin for activation of the clotting cascade
- 20 trauma patients: r-TEG results available < 20 min. vs. > 30 min. for TEG, PT/PTT

Jeger V et al J Trauma 2009

Normal TEG vs r-TEG

Rapid TEG predicting coagulopathy

<table>
<thead>
<tr>
<th>TABLE 4. Multivariate Logistic Regression Model Predicting MT (≥10 Units PRBC) in the First 6 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictor</td>
</tr>
<tr>
<td>--------------------</td>
</tr>
<tr>
<td>ACT &gt; 128 s</td>
</tr>
<tr>
<td>Age (yr)</td>
</tr>
<tr>
<td>Male gender</td>
</tr>
<tr>
<td>Blunt mechanism of injury</td>
</tr>
<tr>
<td>White race</td>
</tr>
<tr>
<td>ED systolic blood pressure</td>
</tr>
<tr>
<td>ED heart rate</td>
</tr>
<tr>
<td>Positive FAST exam</td>
</tr>
</tbody>
</table>
r-TEG predicting NO blood

Table 5: Multivariate Logistic Regression Model Predicting No PRBC Transfusions in the First 6 h

<table>
<thead>
<tr>
<th>Factor</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACT &lt; 105 s</td>
<td>1.85</td>
<td>1.060-3.185</td>
<td>0.028</td>
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<tr>
<td>Age (yr)</td>
<td>0.99</td>
<td>0.978-1.007</td>
<td>0.340</td>
</tr>
<tr>
<td>Male gender</td>
<td>0.88</td>
<td>0.486-1.606</td>
<td>0.866</td>
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<tr>
<td>Blunt mechanism of injury</td>
<td>1.56</td>
<td>0.868-2.833</td>
<td>0.136</td>
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<tr>
<td>White race</td>
<td>0.60</td>
<td>0.353-1.037</td>
<td>0.068</td>
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<tr>
<td>ED systolic blood pressure</td>
<td>1.00</td>
<td>0.985-1.027</td>
<td>0.558</td>
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<tr>
<td>ED heart rate</td>
<td>0.98</td>
<td>0.976-0.998</td>
<td>0.020</td>
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<tr>
<td>Positive FAST examination</td>
<td>0.62</td>
<td>0.313-1.238</td>
<td>0.117</td>
</tr>
</tbody>
</table>

Courtesy: Dr. Bryan Cotton

Table 1: Algorithm for TEG-Guided Blood Component Therapy

<table>
<thead>
<tr>
<th>TEG Abnormality</th>
<th>Blood Component Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Critical Value ≤ 95%</td>
<td>Aspirin, Thienopyridines, GP IIb/IIIa antagonists</td>
</tr>
<tr>
<td>Critical Value ≤ 90%</td>
<td>Aspirin, Thienopyridines, GP IIb/IIIa antagonists</td>
</tr>
<tr>
<td>Critical Value ≤ 85%</td>
<td>Aspirin, Thienopyridines, GP IIb/IIIa antagonists</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Activator Solution</th>
<th>Antithrombotic Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombin</td>
<td>-</td>
<td>Aspirin, Thienopyridines, GP IIb/IIIa antagonists</td>
</tr>
<tr>
<td>ADP</td>
<td>-</td>
<td>Aspirin, Thienopyridines, GP IIb/IIIa antagonists</td>
</tr>
<tr>
<td>AA</td>
<td>-</td>
<td>Aspirin, Thienopyridines, GP IIb/IIIa antagonists</td>
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Measuring Platelet Dysfunction

EG with Platelet Mapping

- Platelet function is analyzed using the TEG/PM assay.
  - Four individual samples of 360 µL of whole blood are placed into separate specialized cups from blue-capped collection tubes. Next, 10 µL of the prepared activator solution, comprised of reptilase, factor XIII, and phospholipids, is added to three of the cups.
  - The activator solution determines the MA of the fibrin clot (MA_fibrin).
  - Next, either 2 mM, final concentration of adenosine diphosphate (ADP), and/or 1 mM, final concentration of arachidonic acid (AA), are added to specialized cup(s) to determine the MA of the ADP (MA_ADPA) and AA (MA_AAA).
  - The maximum hemostatic activity (MA_haemostasis) is measured using a potassium Ca²⁺-activated whole blood sample collected in citrate, which is also gently inverted three to five times before TEG use.

Platelet Mapping Values

<table>
<thead>
<tr>
<th>MA_un</th>
<th>MA_ADPA</th>
<th>MA_AAA</th>
<th>MA_haemostasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>35</td>
<td>90</td>
<td>50</td>
</tr>
</tbody>
</table>

3/7/2018
Inhibited Platelets
Platelets are more inhibited:
TEG/PM tracing with platelet inhibition greater than normal.
A patient can have a normal Platelet count but the platelets are not functioning properly.

What can TRIP you up with TEG with Platelet Mapping

If the patient’s platelets are inhibited, how can we tell if the effect is coming from a bleed?

Standard TEG tracing in Kaolin activated. Kaolin promotes thrombin which is a potent activator of the platelet. Thrombin normally activates your platelets by creating a series of reactions that result in a rise in MACH or MAP. With Platelet Mapping you can tap into this thrombin generation. This is an active ADP receptor directly to show if the platelets are inhibited due to an issue with aggregation.

We must look at the TEG with Platelet Mapping to get the full picture...

Remember the Regular TEG (1st channel) has Citrated Kaolin in the sample. Kaolin turns on the Thrombin which is so powerful it attracts platelets and is able to SHUT DOWN any ADP or AA inhibition that might be present.

Table 1: Algorithm for TEG-Guided Blood Component Therapy

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<td>1. Platelet infusion</td>
</tr>
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<td>MA ADP &gt; AA ≥ 95</td>
<td>2. TEG/PM if going to OR still</td>
</tr>
<tr>
<td>% Inhibited Platelet Mapping: MA ADP &lt; AA ≤ 90</td>
<td>Treat platelet inhibition &gt; 50% if trending/bleeding</td>
</tr>
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</table>

58 year old TBI: 9/19

TEG R parameter, Alpha Angle, MA and U50 – all normal...BUT...We must look at the TEG with Platelet Mapping to get the full picture...

Remember the Regular TEG (1st channel) has Citrated Kaolin in the sample. Kaolin turns on the Thrombin which is so powerful it attracts platelets and is able to SHUT DOWN any ADP or AA inhibition that might be present.

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58 year old TBI:

TEG with Platelet Mapping there is heparin in the Green tube which restricts Thrombin and therefore allows the true ADP or AA inhibition on platelets to be revealed. This TEG with Platelet Mapping ADP is the 3rd sample (you have to click on 1,2 and 3 screens to view).

58 year old TBI:

TEG with Platelet AA is the 4th sample (you have to click on 1, 2 and 4 screens to get this to show). It reveals a MA AA of 20.9 and a 70% platelet inhibition of the AA receptors.

The Treatment is geared toward consideration of – is the patient oozing/bleeding? – Give platelets.

Does the patient have contusions that have the potential to blossom and become larger hemorrhagic contusions? Possibly give platelets.
Putting it all together...

- Applications
- Case Studies

21 year old Male - Ped vs Train

Red Trauma Alert...1035
- GCS 1-4-2
- VS HR 160 BP 92/50 R28
- O2 saturation 60%
- Hgb 11.9/Lactate 8.4
- Diagnostics
  - Left chest pulmonary contusion, fx clavicle, scapulae, left rib fractures (1-3, 5, 7, 8-9) hemopneumothorax
  - Pelvis: left side rami fractures/acetabular fx, right sacral fx
  - Facial fractures: R maxillary sinus, right zygoma/nasal fx, orbital emphysema, fx anterior right orbital floor
  - CT abdomen: active hemorrhage within the left flank, left gluteal region, near the left sacroiliac joint, near the left medial gluteal muscle, suspected mesenteric and retroperitoneal contusions, severe left hydronephrosis from uteropelvic junction obstruction, right adrenal contusion.

Other injuries
- CT brain: bilateral apical parasagittal parenchymal hemorrhages, SDH, contusions, and cerebral edema
  - ICP opening pressure 30s
- Interventional OR: embolization of internal iliac artery

What is this?
Platelet Mapping

Massive Transfusion Protocol

- TXA completed at 1500

Post TXA

Order:
2 U FFP
1 Cryo
2 Packs Platelets

Post TXA Platelet Mapping

Post op 1710

Post Op 1710
Liver Disease & Coagulopathy

- TEG: ETOH hx and cirrhosis (massively bleeding)

Liver Disease & Coagulopathy

- Treated with
  - PCC: K Centra
  - DDAVP
  - Multiple Blood Products

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Product Description</th>
<th>Unit#</th>
<th>Volume</th>
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<tbody>
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<td>2/22/16</td>
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<td>Leuk-reduced RBC</td>
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<td>Pheresis Platelets</td>
<td>W200616833767</td>
<td>201</td>
</tr>
</tbody>
</table>

Liver Disease & Coagulopathy

Patient required more platelet transfusions
Stopped bleeding

Case Study Trauma
Spinal Cord Injury
Trauma Case: Spinal Cord Injury

- EMS Report
  - 51 year old female riding bike with husband when hit from behind at high rate of speed and thrown in the air.
  - GCS 7 (E4, V2, M1) on medic arrival
  - BP 90/9, HR 120, RR 16, Sats 99%
  - Pt with snoring respirations, attempted OPA but unable due to clenched jaw
  - 18 g IV established and 200 cc NS given
  - Medics on scene for 10 mins with additional 11 mins transport to hospital
  - Patient designated Critical Trauma

Diagnositics

- CT
  - Acute traumatic midline shift at fracture sites, associated with the concomitant dissociation injury
  - Acute traumatic shefrant anterior paravertebral
  - Transverse fracture of the right vertebral artery
  - Unilateral fx of both L1 and L2 transverse
  - Left lateral T1 and T2 vertebral body with inferior endplate
  - Multiple anterior wedge compression fx of T12
  - Acute traumatic midline fracture at T12
  - C2 and L1 vertebral body fx, condylar avulsion fx of right inferior ramus at L1
- MRI
  - Findings consistent with concomitant dissociation, widening of the bony -nerve
  - Gerbner anterior posterior view according to ligamentous injury
  - Anterior epidural fluid noted extending from C2 to C6
- Course of Care
  - Neurosurgery states CT concerning for atlanto-occipital dislocation
  - Patient denied as incapable for NIMI
  - Patient is at 1036 to Trauma Bay, patient on the way to trauma bay

1st TEG in Trauma Room 1040

- Received a Superpack of Platelets / DDAVP @ 1420

2nd TEG in OR @ 1719

- No Platelet Mapping

Post Op Report

- Transferred to SICU intubated with anesthesia
- 1 L NS, 1 L Albumin 5%
- EBL 500mL
- Urine output 850
- Given Rocuronium, Ancef, Propofol, dopamine, phenylephrine, norepinephrine during the procedure
- Post op labs: CBC, BMP, TEG
Post op Labs & TEG with platelet mapping

- PRBC, platelets, plasma, cryo transfused

4th TEG

- R is prolonged at 14.4 minutes
  - Requires FFP transfusion – 1 unit @ 0345

Platelet Mapping

MA ADP and MA AA are both normal

Patient given another Pack of Platelets at 0652 for Plts 121

5th TEG

- Normalized TEG and TEG with Platelet Mapping

Patient Course of Care

- 9/3 Patient emergently to OR for Occiput-T1 fusion, decompression/reduction of fx, and EVD. Patient on hypothermia protocol x 24 hrs
- 9/7 Patient to IR for IVC filter and DHT
- 9/8 Patient to OR for T4-T7 decompression/fusion
- 9/14 Patient extubated. Moving 2/5 UE and 4/5 LE
- 9/15 Patient transferred to Kaiser. On D/C Pt GCS 15 and moving 2/5 UE and 5/5 LE

CODE STROKE: ICH

EMS Pre hospital report

- 55 year old female
- Chief complaint: Seizure
  - Per family report
    - Nausea and vomiting x2 days
    - Family thought it was food poisoning
    - Pt had complained of HA for 2 days
- Presented with Right sided weakness facial droop and ALOC
Arrived at ED

- Physical exam
  - Eyes with forced deviation
  - Right facial droop
  - VS 160/71, HR 53 RR 16, RA 99, T 97.7
- Code Stroke called off to CT
- Current meds
  - Paxil, Norco 5/325, Gabapentin
  - Ibuprofen intermittently for her HA

CT – Left sided intracranial hemorrhage extending into the ventricles

Initial Labs

Clinical Course

- Patient further decompensates
  - Intubated
  - Mannitol, Levetiracetam given
- Neurosurgery consulted
- To OR for ICP and brain tissues monitors
- TEG drawn

Treatment of TEG

- DDAVP 0.3 mcg/kg IV for ADP/AA inhibition
- Super pack Platelets
- To the OR
OR 1215

- Decompressive craniotomy
- Intracranial pressure monitor placed in ventricle with frank blood
- Removal of Frontal Cavernous Malformation
- Tissue oxygenation monitor

Post OP Cat Scan

Arrives ICU

- Repeat TEG & Labs
- Temp 35.5 C (95.9 F)
- Multiple Drips

Post op TEGS

TEG treatment

- Super pack platelets given
- No obvious bleeding
  - Frank blood still draining for ICP monitor
  - ICP Pressure 12-16 mmHg
- TEG with platelet mapping and PFA ordered

Comparison of Labs and TEG 2230

Normal PFA
Abnormal TEG ADP
Clinical Course

- Hypothermia X 48 hours then rewarmed
- Day 7
  - Back to OR for Left craniectomy
    - Increased cerebral edema when rewarmed.
  - Hypothermia X 48 hours
- Day 15
  - Brain Hardware D/C

Clinical Course

- Day 18
  - Trach and G & J tubes
- Day 24
  - Transferred to Step down unit
- Day 30
  - Transferred to ARU
  - Independent at home at 6 months

Case Study POLY Trauma

- EMS Report
  - 58 y/o Male – Side swiped off Motorized Scooter
  - GCS (E4, V3, M5)
  - L Pupil 6mm/NR
  - BP 142/78, HR 126, RR 28, Sats 91% (NRB)
  - Left Lower Extremity Deformity
- Critical Trauma Designation
  - Anticipate Injuries
  - Room Prep
Trauma Resuscitation

- Primary Assessment
  - Airway Established / Disability
  - Breathing: Sats 80%
    - Needle Thoracostomy & LEFT CT (100cc output)
  - VS: CL Right Fem
    - NS 500cc
    - 3% NS 200
- Secondary Assessment
  - Chest X-ray
  - Tubes/Lines
  - Labs
  - Neuroprotective Interventions

DATA & Diagnostic Studies

- Off to CT ...
- Diagnosis:
  - Skull Fx’s
  - Facial Fx’s
  - R/L SDH & Cerebral Contusions
  - L 1-9 Rib Fx’s:
  - PTX & Pulmonary Contusions
  - LEFT Scapula & Clavicle Fx
  - OPEN Fibula Fx

DATA & Diagnostic Studies

- Labs
  - Hemogram:
    - H/H: 12.0/37.6
    - Plts: 201,000
  - Chemistry: WNL
  - Lactate: 3.11
  - TEG & Coag’s
  - Type & Cross
  - ABG

Treatment Options

<table>
<thead>
<tr>
<th>Normal MA: 80-70 mm</th>
<th>Low: MA (~ 50 mm)</th>
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<tbody>
<tr>
<td></td>
<td><img src="TEG_MA_41.7" alt="" /></td>
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<tr>
<td><img src="TEG_MA_3.8" alt="" /></td>
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<tr>
<td><img src="TEG_MA_2.8" alt="" /></td>
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</tbody>
</table>

- MA ADP 3.8 / 100% Inhibited
- MA AA 2.8 / 100% Inhibited

2 Super Packs of PLT’s Given & Repeat TEG performed
Disposition

- Operating Room
  - Evacuation of SDH
  - Placement of ICP & PbtO2 Monitors
  - A-Line
- Resuscitation in OR
  - Crystalloid
  - Blood Products
    - PLT’s

Admit to SICU

- 1700 Arrival to SICU
  - VSS
  - CT output 200cc over 2 hours
  - Labs Collected
- Clinical Picture
  - Sedation
  - Vasopressors
  - FloTrac Initiated
  - Lab Results ...

Look at the Patient

- Clinical Picture
  - Is the Patient Bleeding?
  - What are the Vitals?
  - FloTrac and Hemodynamics

Clinical Course

- 0600 ...
- Vitals & FloTrac
- CT output
- Labs
  - H/H 6.2/17.9
  - Plts 89,000
- Vasopressor to augment BP
**Blood & Platelet Transfusion**
- **Blood Products**
  - 2 U PRBC’s given
  - 1 Super Pk Platelets
- **Outcomes**
  - FloTrac
  - Vasopressor Titration
- **Repeat TEG**

**Outcome**
- **LOS 16 D Acute Care**
  - SICU 2 weeks
    - Extubation failed day #6
  - PCSU day #14
- **Discharged to Neuro Rehab Center**
  - Rancho IV-V: impulsive
  - Ortho Stabilized & WBAT (PT/OT)
  - Resolving Rib fx’s & Pulmonary Contusions

**Heat Stroke Case**
- 27 year old Marine found down unknown amount of time with bruise on head
  - Initial Temperature 109 degrees F or 42.6 degrees Centigrade
  - GCS 1-1-1
  - Packed in ICE and transported to Mission Trauma
  - Arrived at Mission with GCS 3, Temp 102.7 degrees F
  - Iced saline 2 liters
Admit to SICU 90 minutes

• ICU Admit
  – Intubated with sedation (propofol)
  – BP 133/66 HR 122
  – Orders to hold sedation at 2pm to see if pt can be extubated
  – NO urine output
  – Oral T 101.7 Temp on Foley cath 38.4 showing 4 arrows up indicating anticipated increase in temperature of 2 degrees/hour – No urine output.
• Call to Trauma MD regarding fluid status and no urine output

Admit to SICU 90 minutes

• Labs 1230: 16.4/45 WBC 6.8 Na 143 K 5.9 Cl 109 CO2 20
  – BG 59 (amp D50 given) BUN 36 Cr 2.3 LA 8.21 CK 1199 CK MB 10
  – Amylase 254 PT 17.8 INR 1.6 PTT 36
• TS ordered 3rd liter of iced saline

  • 1309 BG 45 (D50 given)
  • 1329 BG 66 1400
  • BG 48 (D50 given)
  • 1420 85
  • 1450 45 (D50 given)

6 hours later

• 1550: NA 149 K 4.3 Cl 117 Co2 18 BG 35 (D50 given)
• 1632: 1 pack Platelets given
• 1647: BG 31 (D50 given)
• Pt has explosive diarrhea with development of massive GI hemorrhage
  – Intensivist consulted TEG ordered.
• 1642
  – TEG1: R = 83 minutes. No Alpha Angle, No MA
  – FLAT LINE R parameter…….

Flat Line R....

1700 labs:

• PT 140 INR > 14 PTT > 180 Fibrinogen 52
• Given 2 FFP -4 Cyro - 2 Platelets & TXA 1000mcg @1837 with drip
• Na 147 K 4.1 Cl 115 CO2 20 BUN 33 Cr 3.43 AST 705 ALT 288 Alk Ph 58 CK 13105 CK MB 80.3

2ND TEG

2ND TEG w Platelet Mapping
3rd TEG

- MA ADP 4.4 MA AA 2.1 Platelet count 45,000 PT
  21.2 INR 2 PTT 36 Fibrinogen 130
- 1 FFP Platelets; 3 Cryo & 2 Plts

4th TEG

- MA ADP 12.1 MA AA 2.3 Fibrinogen 145 Platelet count 48,000
- Received cryo and platelets

LABS am:
- Na 143
- K 3.7
- Cl 107
- CO2 22
- BUN 40
- Cr 4.36
- Ca 7.3
- Mag 1.6
- CK 55,215
- CK MB 226
- WBC 1.8
- Platelet 28.1
- INR 2.6
- PTT 34
- Fibrinogen 156
- WBC 2.1
- Pt 6:1403
- Platelet mapping MA ADP 6.9 and MA AA 9.9

5th TEG

Pt 28.1 INR 2.6 PTT 34
Fibrinogen 156 WBC 2.1 11.3/31
Platelet 50,000 NA 143 K 3.6 Cl 106 CO2 24 Ca 7.4 Mag 1.4
AST 3034 ALT 1756

Tx: FFP 2 CRYO 1 PLT 1

ICU Phase and 6th TEG

- Quinton Cath placed...starting CRRT.
- I/O for 24 hours at 0600 16 liters In/5 Liters out
  - Urine 354 ml Stool 3100 OG 900
- Colonoscopy – No ischemic bowel seen
- TEG 6:1403
  - Platelet mapping MA ADP 6.9 and MA AA 9.9

ICU Care

- Multisystem Organ Failure
  - Neurologic – In coma
  - Rhabdomylosis and Acute Liver Failure
- On CRRT
- Call to Scripps Green for Transplant list
  - Transferred on Day 8
  - Received liver transplant on Day 9

ICU Care – Post Transplant

- Improved Neuro status
  - Woke up 3 days post transplant
- Rehab
  - Pt neurologically improved
  - Independent
- Post liver transplant
- Renal status
  - OFF Dialysis 8 weeks after incident
ABCs of TEG: Where to start?

- Become the Expert or Find a Nurse Colleague who wants to be content expert
  - Review/Read the literature on the use of TEG
  - Attend a lecture on how it is applied
  - Visit a center that has implemented a program
  - Become the expert!

- Find a Physician Champion
  - Get Physician BUY IN from the surgeons, intensivists, and Anesthesia
  - Find a champion from each area of expertise
    - Trauma, Neurosurgery, Neuro Critical Care, Intensivists, ED, Anesthesia

- Make friends with the Lab Manager or Perfusionist

ABCs of TEG: Where to start?

- Budget?
  - Donors are nice!
  - Build it into the budget cycle
- Once purchased: Form a MD team to build a hospital based protocol
  - Start with another hospital based protocol or develop one
  - Gain consensus: 2-3 meetings
- Educate staff
  - Physicians: Bring in physician content expert (national)
  - Nurses: Provide 1–1.5 hour lecture on TEG for key staff
- Get BUY IN from the rest of the hospital
  - Engage key nurses from ED/ICU, Laboratory personnel, OR personnel, and IT
  - Track Data on blood utilization
- Provide 24/7 support

Other uses….
Code Stroke: Vertebral-Basilar Aneurysm

- 41 yr old female with SAH: Disecting Basilar fusiform aneurysm
- Placement of pipeline x 3 in basilar into right vertebral artery
- Coil of right fenestration with 3x8 helical PC400 coil x 2
- Occlusion of distal left vertebral artery with 3x5 helical to close

Optimal Platelet Inhibition

- Given ASA 325 mg Plavix 300 mg
- Given additional Plavix 600 mg
- TEG repeated
- P2Y12 259

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