Utilization of TEG and Platelet Mapping to Guide Resuscitation, in the Setting of Massive Transfusion

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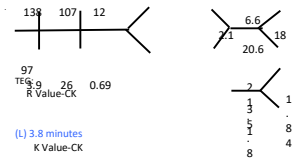
Introduction

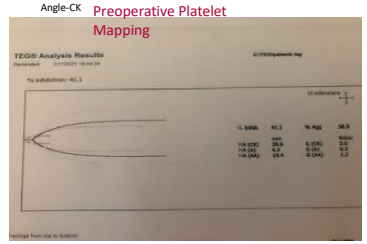
A 48-year-old male, with a history of cryptogenic cirrhosis c/b mesenteric venous thrombosis and subsequent intestinal failure, presents to the operating room for complete multivisceral transplantation (MTP; Stomach + Liver + Pancreas + Small Intestine)

Preoperatively

Preoperative Labs:



Preoperative Platelet Mapping



Operative Course

Upon presentation to the OR, patient underwent uneventful induction and intubation. Access included: bilateral radial arterial lines, right IJ 12 Fr triple lumen CVL, right arm 14G PIV and left arm 8.5 Fr RIC line. TEE probe was placed to assess hemodynamics throughout case. 1 U PRBC and 1 U of Platelets administered just after induction based off preoperative lab values.

Patient received a bolus of Amicar, followed by a continuous infusion at the direction of surgical team.

Given patient's extensive and complicated past surgical history, there was substantial blood loss upon abdominal dissection and Massive Transfusion Protocol (MTP) was initiated within the first hour of incision. The Level 1 ® Rapid Infuser was utilized for prompt resuscitation.

Thromboelastography (TEG) was used to help guide resuscitation

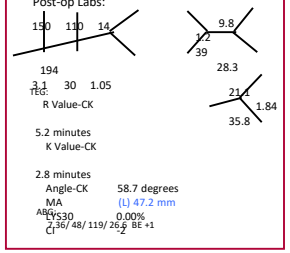
At the end of an almost 16-hour case, it was estimated that the patient lost ~60L of blood, and received 102 units PRBCs, 100 units FFP, 10 units cryoprecipitate, and 9 units platelets.

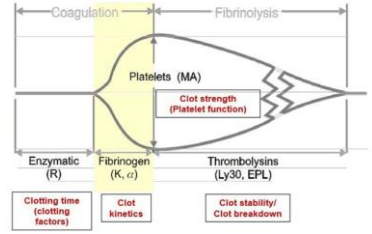
Surgical procedure included resection of the patient's native liver, stomach, pancreas, small intestine, subtotal colectomy, and splenectomy. Along with successful transplantation of stomach, liver, pancreas and small intestine.

Postoperatively

Postoperatively, the patient remained intubated and on high dose vasopressors. He was taken to the TICU for ongoing management.

Post-op Labs:





TEG	ROTEM	Description	Normal	Abnormality: Cause	Treatment
Reaction Time (R-Value)	Clotting Time	Time till initiation of fibrin clot formation	5 - 10 min	> 10 min: decreased clotting factors	FFP, protamine
K Value	Clot Formation time	Time to achieve 20mm clot, representing thrombin-platelet interaction	1 – 5 min	> 5 min: decreased fibrinogen	Cryoprecipitate, Fibrinogen
α - angle	α - angle	Rate at which fibrin cross-linking occurs	45 – 75°	< 45°: decreased fibrinogen	Cryoprecipitate, Fibrinogen
Maximum Amplitude (MA)	Maximum clot firmness	Maximum clot strength	50 – 75mm	< 50mm: decreased platelet count/function	Platelets, DDAVP
LYS-30	Clot Lysis	Degradation of clot 30 minutes after MA	0 – 10%	> 10%: increased clot breakdown	TXA, Amicar

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Discussion

TEG was developed in 1948, in Germany, to detect clotting factor deficiencies, in an effort to identify the specific cause of coagulopathies. Its availability was limited in the U.S. until the 1980's.

Conventionally, TEG is activated by Kaolin, which initiates the intrinsic pathway, resulting in R-time. As the clot begins to form, the TEG records the viscoroelastic changes that occur in the sample. A pin suspended in the sample is monitored for motion, as the fibrin-platelet bonding occurs.

TEG has gained acceptance in resuscitation of transplant, cardiac, trauma, GI bleeds, and obstetric patients

TEG can differentiate between low fibrinogen and reduced platelet function as the cause of impaired clot strength

A study (Speiss et al, 1995) involving TEG guided algorithms in CABG patients, indicated a decrease in overall transfusion requirements and number of re-explorations for bleeding from 5.7-1.5%.

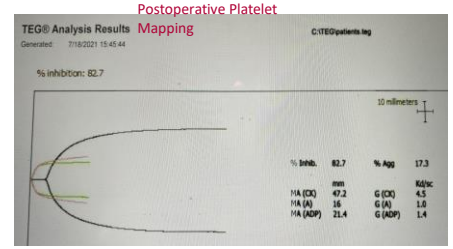
Recent movement to TEG-guided resuscitation over standardized MTP, indicating 1:1:1 strategy, may help guide each patient's individual resuscitation needs. This can include platelet mapping which helps to further understand qualitative function (Important with use of Antiplatelet medications and in states of profound coagulopathies). This may provide cost savings, if the clinician is able to limit amount of blood products given.

Often a limitation of TEG is time. Use of rapid TEG should result an R and K value in approx. 5 minutes and an alpha angle and MA in approx. 15 minutes, compared to 45 – 60 minutes with traditional TEG.

Additional adjuncts during resuscitation can include tranexamic acid, aminocaproic acid, cryoprecipitate, recombinant factor VII and prothrombin complex concentrates.

Important complications to consider during MTP include, acidosis, hypothermia, coagulopathy, exhausted platelet dysfunction, lung injury (TRALI), volume overload (TACO), infection, electrolyte abnormalities, and citrate toxicity.

Postoperative Platelet Mapping



Conclusion

At the time of this MVT, blood products were in short supply (specifically platelets), because of this our efforts to resuscitate in accordance with our TEG and platelet mapping results was unable to be achieved

Total number of blood products administered intraoperatively were ~221 units. TEG helped to guide our resuscitation. Post-operative ABG, Coags, CBC and TEG appeared relatively normal.

Interestingly, the patient's post-op platelet count was higher than his pre-op platelet count, but the patient's platelet mapping indicated that there was profound platelet dysfunction (approx. 82.7% inhibition)

Increasing prevalence necessitates understanding and utilization of TEG and Platelet mapping for intraoperative and postoperative resuscitation guidance.

Patient Update: The patient was weaned from vasopressors and extubated on POD 4. Unfortunately, around POD 11, patient had worsening renal function requiring CVVH, this was further complicated by pancytopenia, bowel obstruction and sepsis of unknown etiology and subsequent cardiac arrest. Patient was made DNR/DNI and care was withdrawn.