Josh's Corner

Articles that shaped my practice

Subject: Tranexamic Acid

Article 2 –

Moore HB, Moore EE, Gonzalez E, Chapman MP, Chin TL, Silliman CC, Banerjee A, Sauaia A. Hyperfibrinolysis, physiologic fibrinolysis, and fibrinolysis shutdown: The spectrum of postinjury fibrinolysis and relevance to antifibrinolytic therapy. J Trauma Acute Care Surg. 2014 Dec;77(6):811-7. doi: 10.1097/TA.0000000000000341.

Synopsis –

Acutely injured patients admitted to Denver Health Medical Center from 2010 to 2013 who had TEGs within 12 hours of their injury were retrospectively reviewed. Patients were broken into three groups: hyperfibrinolytic, physiologic, and "shutdown." The "shutdown" group comprised 115 patients, physiologic comprised 32, and hyperfibrinolysis comprised 33. The hyperfibrinolytic group was more likely to receive blood products and have a massive transfusion (defined as more than 10 units of RBCs inside 6 hours). An increase in mortality was seen in both the hyperfibrinolytic and "shutdown" groups. Exsanguination was the leading cause of death in the hyperfibrinolytic cohort (66% of all deaths), whereas the primary cause in the "shutdown" cohort was traumatic brain injury (45%), followed by multi-organ failure (40%).

Details –

The study was supported by three NIH grants, the Colorado Clinical and Translational Science Award Grant, and Haemonetics. Patients were excluded if they had an ISS less than 15 or if they had taken an anticoagulant before their injury. The cut-off for hyperfibrinolysis was set at a Ly30 of 3% based on studies by the groups in Houston and Denver. The threshold for the "shutdown" group was made using the receiver operating characteristic curve on mortality in the study population. Based on the optimal specificity and sensitivity for mortality using the Youden index, the Ly30 of 0.8% was chosen as the dividing point between the physiologic and "shutdown" groups. Upon review of the variables, the only two that came out as statistically significant between the three groups was systolic blood pressure and the need for massive transfusion. The hyperfibrinolytic group was much more likely to have a lower blood pressure. The study also found that the amount of fibrinolysis was not normally distributed.

Questions raised –

Is the study population representative of trauma patients arriving in the trauma bay at your institution? What determines if a patient arrives hyperfibrinolytic versus in "shutdown?" Is the level of fibrinolysis causative, contributive, or just related

to the final cause of mortality? Does the level of fibrinolysis change over time, particularly over the course of 3 hours from the time of injury? Morrison et al. has stressed following the CRASH-2 protocol for dosing tranexamic acid following trauma because of fibrinolytic activation not apparent by viscoelastic measures.[3] Is a more cautious approach appropriate? Could the dose of tranexamic acid in the CRASH-2 study push a patient from the physiologic cohort into the "shutdown" cohort, potentially affecting mortality? What is the best strategy for dosing antifibrinolytics in patients with significant hemorrhage? Should patients in the "shutdown" group receive some sort of fibrinolytic?

References –

1.) Cotton BA, Harvin JA, Kostousouv V, Minei KM, Radwan ZA, Schöchl H, Wade CE, Holcomb JB, Matijevic N. Hyperfibrinolysis at admission is an uncommon but highly lethal event associated with shock and prehospital fluid administration. J Trauma Acute Care Surg. 2012;73(2):365Y370; discussion 370.

2.) Chapman MP, Moore EE, Ramos CR, Ghasabyan A, Harr JN, Chin TL, Stringham JR, Sauaia A, Silliman CC, Banerjee A. Fibrinolysis greater than 3% is the critical value for initiation of antifibrinolytic therapy. J Trauma Acute Care Surg. 2013;75(6):961Y967; discussion 967.

3.) Morrison JJ, Ross JD. Is viscoelastic evidence of hyperfibrinolysis the ideal indicator for tranexamic acid administration in trauma? J Trauma Acute Care Surg. 2013 Oct;75(4):743. doi: 10.1097/TA.0b013e3182a53873.