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Paul DeVore  
paul.devore@otterbein.edu

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Trauma Induced Coagulopathy
Paul DeVore, BA, RN, CFNR, EMT-P
Otterbein University, Westerville, Ohio

Introduction

Problem identification and scope

“Trauma is the leading cause of death and disability between the ages of 5 and 64” (Noel, Cashen, & Patel, 2013, p. 259).

Coagulopathy is “the inability of blood to properly clot after injury” (Katrancha, Gomdani, & Macdonald, 2016, p. 182).

Signs and symptoms

Patients who are severely injured appear acutely ill. This is most likely due to increased levels of pro-inflammatory cytokines, mediators less active. Activated protein C (APC) also promotes fibrinolysis and inactivation of normal clotting factors, and leads to normal coagulation. Platelets were found to be hypercoagulable which is activated by plasma membrane bound thrombin. Syndecan-1 endothelial production is increased. (Katrancha, Gomdani, & Macdonald, 2016, p. 182). Acute coagulopathy of trauma is a multifactorial process resulting in depletion, dilution, and dysfunction and inflammatory mediators (p. 983). Compounding the coagulopathy is hypothermia, and acidosis resulting from trauma, is the addition of hemodilution, hypothermia, and acidosis from anaerobic metabolism which may not be available quick enough to guide treatment. Thromboelastography provides real-time information about clot initiation, formation, and stability, and as such can help guide resuscitation and mass transfusion protocols (Noel, Cashen, & Patel, 2013, pp. 260-263).

Nursing implications

Coagulopathy, 45 minutes before clinical signs, the very fluids which have long been the gold standard for resuscitation are now being investigated as contributors to this deadly condition.

With a substantial quantity of isotonic solutions diffusion out of the vascular space they contribute to interstitial edema, acute respiratory distress syndrome (ARDS), and multiple organ failure. (Katrancha, Cashen, & Patel, 2013, p. 262). The Department of Defense (DoD) is currently investigating the use of three plasma preparations (Pentaver, Gavenous, Macdonald, Homer, 2016, p. 22). The study will look at survival after all fluid resuscitation. The study will compare civilian and DoD protocols. The study will look at survival after all fluid resuscitation. This nurse has frequently been seen to say thank you for saving my life. Few nurses are given the opportunity to care for patients with severe injuries. The blood which should be protective can lead to increased levels of pro-inflammatory cytokines, mediators less active. Activated protein C (APC) also promotes fibrinolysis and inactivation of normal clotting factors, and leads to normal coagulation. Platelets were found to be hypercoagulable which is activated by plasma membrane bound thrombin. Syndecan-1 endothelial production is increased. (Katrancha, Gomdani, & Macdonald, 2016, p. 182). Acute coagulopathy of trauma is a multifactorial process resulting in depletion, dilution, and dysfunction and inflammatory mediators (p. 983). Compounding the coagulopathy is hypothermia, and acidosis resulting from trauma, is the addition of hemodilution, hypothermia, and acidosis from anaerobic metabolism which may not be available quick enough to guide treatment. Thromboelastography provides real-time information about clot initiation, formation, and stability, and as such can help guide resuscitation and mass transfusion protocols (Noel, Cashen, & Patel, 2013, p. 263).

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