7-31-2019

Trauma-Induced Coagulopathy

Zachary Wise
Otterbein University, wise3@otterbein.edu

Trauma-Induced Coagulopathy

Zachary S. Wise BSN, RN, CCRN, SRNA

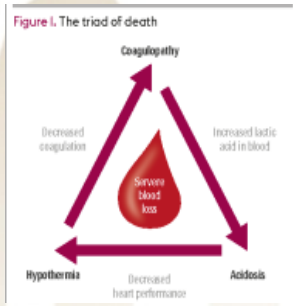
Otterbein University, Westerville, Ohio

Introduction

- "A trauma patient's greatest risk of death after the first 24 hours of injury stems from a combination of 3 conditions: hypothermia, acidosis, and coagulopathy" (Katrancha & Gonzalez, 2014).
- "Unless this cycle can be broken, the patient's death is unavoidable" (Mizobata, 2017).
- Viscoelastic tests such as thromboelastogram (TEG) provide real time assessment of blood properties to help guide correction of the trauma triad (Pigna, Lippi, Saronni, & Cervellin, 2018).

Working in the trauma population, a significant pathophysiological issue is the "lethal triad" stemming from various forms of injury (Mizobata, 2017, p. 2). The triad is comprised of the three variables that contribute the most to a patient's risk for mortality: hypothermia, acidosis, and coagulopathy. Critical care management of this triad is largely focused on resuscitation of the hemorrhagic and coagulopathic population to aide in restoring natural physiologic function (Katrancha & Gonzalez, 2014, p. 61). With that, recent studies have sought out the efficacy of utilizing TEG to guide resuscitation during this high acuity phase (Mohamed et al., 2017, pp. 2-3).

This complex pathophysiological process is often seen in critical care. In the trauma population, "uncontrolled hemorrhage" is the isolated cause of death in forty percent of patients (Mizobata, 2017, p. 1). Moving forward, effective treatment and reduction of this percentage will be based upon rapid reversal of the lethal triad. To accomplish this, an emphasis on restoration of clotting factors, optimal understanding of guided massive transfusion protocol, and surgical management must be understood (Mizobata, 2017, pp. 4-7).



(Keane, 2016)

Signs and Symptoms

TIC has numerous symptoms that manifest as a result of an initial injury. Many symptoms are compounding and intertwined as a direct result of the pathophysiology of the lethal triad. While numerous symptoms will vary based upon mechanism of injury, common manifestations of the severely compromised TIC patient include:

- Altered levels of consciousness
- Hypothermia – body core temperature of less than 36.5°C
- Metabolic acidosis – hypoperfusion may induce lactic acid accumulation
- Coagulopathy - inability to clot worsens with loss of factors
- Hypotension – due to acidosis, impaired cardiac performance, and volume loss
- Hemorrhagic hypovolemia

Laboratory Testing

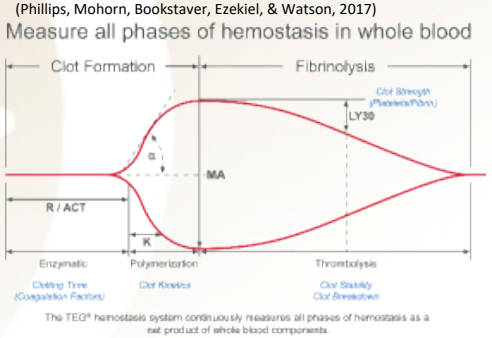
Primary labs for patient's experiencing TIC should be focused on TEG. This test provides a real time assessment of factors related to coagulopathy. With an understanding of TEG normal physiological values, appropriate resuscitation can be achieved with less blood products. Fewer, more effective, transfusions minimizes risks, complications, and cost of care (Mohamed et al., 2017).

Components of TEG include time to clot initiation (ACT or R-value), speed of clot formation (K-value and Alpha angle), strength of clot (MA), and how much clot lysis has occurred after 30 minutes (LY30).

Normal Values
- R: 4 to 8 minutes
- K: 1 to 4 minutes
- Alpha angle: 47-74 degrees
- MA: 55-73mm
- LY30: 0 to 8%

Treatment
- Prolonged R: Fresh Frozen Plasma or protamine
- Prolonged K/Reduced angle: cryoprecipitate
- Reduced MA: platelets
- Elevated LY30: Tranexamic acid

(Phillips, Mohorn, Bookstaver, Ezekiel, & Watson, 2017)



(Haemonetics, 2019)

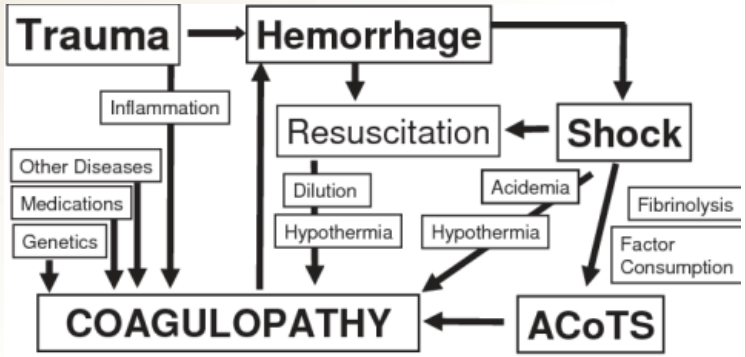
Pathophysiology

TIC is propagated by a cascade of physiological events, but the cycle is initiated by a severe injury. Regardless of mechanism such as blunt force or penetrating trauma, TIC can be manifested and will quadruple mortality compared to a trauma without TIC (Phillips, Mohorn, Bookstaver, Ezekiel, & Watson, 2017). After injury, coagulopathy issues manifest because of variables such as hypothermia, acidosis, hypoperfusion, and hemodilution.

Acute trauma promotes hemorrhage due to injury to organs or bone. As hemorrhage persists, several variables escalate. Loss of volume from hemorrhage results in hypotension and hypoperfusion; two lethal variables that are clinically addressed. Notably, hypoperfusion in the trauma patient is linked to the conversion of Protein C to Activated Protein C (APC). This is significant because APC inhibits cofactors that lead to the production of thrombin. Additionally, APC leads to an overactive tissue plasminogen activator in the body; rapid conversion of clotting factors ensues leading to excessive bleeding. Hypoperfusion also leads to the body's transition to anaerobic metabolism resulting in the accumulation of lactic acid. As lactic acid accumulates, metabolic acidosis worsens. Acidosis itself causes further disorders within the clotting cascade leading to prolonged clotting times and inferior clot performance. Furthermore, this hypotension and acidosis are frequently combatted with crystalloid resuscitation such as 0.9% sodium chloride while waiting for the delivery of blood products from the lab. The crystalloid resuscitation further antagonizes a hyperchloremic acidosis and leads to a dilution of blood and coinciding clotting factors.

Throughout the entire treatment of TIC, the patient is exposed to risk factors that contribute to worsening hypothermia. This issue begins when the patient is immediately affected by the trauma. At this point, the patient is exposed to whatever the environmental temperature may be. Additionally, when first responders arrive, clothing is often removed to visualize the full extent of trauma. Treatment includes room temperature or colder resuscitation with fluids such as chilled blood products. Depending on severity or mechanism of injury, a patient may also be exposed to the cold setting of the operating room to correct the insult that has occurred. This hypothermia is noteworthy because it further hinders the body's ability and drive to manufacture clotting factors.

(Katrancha & Gonzalez, 2014)



(Hess et al., 2008)

Effective utilization of TEG can optimize the ability to combat TIC. Data generated from TEG results helps to combat pathological issues worsened by non TEG management. Traditional management of TIC focuses resuscitation at a 1:1:1 ratio of PRBC, FFP, and platelets. TEG, with its in-depth and real time analysis of lacking clotting factors, provides the ability to generate effective clotting factors with less volume. This combination promotes coagulation while combatting excessive exposure to the negative properties of hypotension and hypothermia. Utilization of TEG also contributes to a reduction in critical care days and length of stay. Effective management of transfusions is patient specific. Algorithms outside of those generated by real time analysis will be based upon educated guesses rather than patient specific scenarios.

(Mohamed et al., 2017

Nursing Implications

Many differentials causing trauma induced coagulopathy require surgical intervention outside of the intensive care unit. However, preoperatively and postoperatively, nursing management of patient's with TIC revolves around identification and treatment of major symptoms of the pathological process (Mizobata, 2017). Nursing facilitated or inclusive implications of care include management of the following issues:

- Hypothermia must be combatted with active rewarming. Interventions include warmed intravenous fluids, heated blankets, and avoidance of exposure to ambient cooling.
- Preventing and identifying metabolic acidosis is integral in patient outcomes. Prevention of acidosis largely focuses on hemodynamic stability. Monitor for signs of inadequate end organ perfusion such as altered levels of consciousness and a decrease in urinary output.
- Appropriate and effective fluid resuscitation is imperative. Use of TEG accurately guides resuscitation with necessary blood products. TEG enhances the ability to avoid excessive crystalloid or colloid administration.
- Efficient administration of large amounts of blood products including packed red blood cells, fresh frozen plasma, platelets, cryoprecipitate, and more. Administration of these products facilitate the restoration of clotting factors lost during hemorrhage.
- Allowance of permissive hypotension in the absence of brain injury. Permissive hypotension promotes clot formation, but hypoperfusion of injured brain tissue can worsen injury. Without neurological damage, aim for a systolic blood pressure of 90mmHg.
- Consider pharmacological measures that may be in play. Many prescribed medications affect clotting capabilities and may require pharmacological reversal.
- Frequent assessment of vital signs, point of care labs, and serum labs to support clinical interventions advised per TEG guidelines.

(Katrancha & Gonzalez, 2014)

Conclusion

TIC is a medical emergency. With unmanaged hypothermia, acidosis, and coagulopathy, this pathological process advances without relent. Optimal outcomes are dependent upon rapid triage of injury and identifying appropriate treatment. Often, surgical measures are indicated. Preoperatively and postoperatively, care is focused on combatting the lethal triad. A major intervention for combatting coagulopathy is resuscitation with blood products.

Due to recent advancements in medical knowledge, the guidance of resuscitation can now be more effectively implemented with the utilization of TEG. The use of TEG has proven to be cost effective and incredibly beneficial in the reduction of mortality rates in the high acuity phase of TIC. Further studies are indicated to continually enhance the knowledge base that is available – especially as resuscitation with whole blood gains momentum in many clinical trials.

References

Haemonetics. (2019). https://teg.haemonetics.com/

Heier, H. E., Badloe, J., Bohonek, M., Cap, A., Doughty, H., Korsak, J., ... Soderstrom, T. (2015, August). Use of Tranexamic Acid in Bleeding Combat Casualties. *Military Medicine*, *180*(8), 844-846. https://doi.org/10.7205/MILMED-D-14-00592

Hess, J. R., Brohi, K., Dutton, R. P., Hauser, C. J., Holcomb, J. B., Kluger, Y., ... Bouillon, B. (2008, October). The coagulopathy of trauma: a review of mechanisms. *The Journal of Trauma Injury, Infection, and Critical Care*, *65*(4), 748-754. https://doi.org/10.1097/TA.0b013e3181877a9c

Katrancha, E. D., & Gonzalez, L. S. (2014, August). Trauma-Induced Coagulopathy. *Critical Care Nurse*, *34*(4), 54-63. https://doi.org/10.4037/ccn2014133

Keane, M. (2016, July). Triad of death: the importance of temperature monitoring in trauma patients. *Emergency Nurse*, *24*(5), 19-23. https://doi.org/10.7748/en.2016.e1569

Mizobata, Y. (2017, January 20). Damage control resuscitation: a practical approach for severely hemorrhagic patients and its effects on trauma surgery. *Journal of Intensive Care*, *5*(4), 1-9. https://doi.org/10.1186/s40560-016-0197-5

Mohamed, M., Majeske, K., Sachwani, G. R., Kennedy, K., Salib, M., & McCann, M. (2017). The impact of early thromboelastography directed therapy in trauma resuscitation. *Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine*, *25*(1), 1-11. https://doi.org/10.1186/s13049-017-0443-4

Phillips, J. B., Mohorn, P. L., Bookstaver, R. E., Ezekiel, T. O., & Watson, C. M. (2017, August). Hemostatic management of trauma-induced coagulopathy. *Critical Care Nurse*, *37*(4), 37-48. https://doi.org/10.4037/ccn2017476

Pigna, F., Lippi, G., Saronni, S., & Cervellin, G. (2018). Trauma-induced coagulopathy. A narrative review of goal-directed hemostatic resuscitation. *Emergency Care Journal*, *14*(3), 96-101. https://doi.org/10.4081/ecj.2018.7569

OTTERBEIN UNIVERSITY