Worldwide ten percent of death are due to trauma, greater than that of HIV, tuberculosis, and malaria combined. In contrast to other causes of trauma, death exsanguination occurs rapidly (median 2- 3 hours) after the presentation and accounts for roughly half of trauma deaths.

Over the last 10 years, the management of major hemorrhage in trauma patients has changed radically. This is mainly due to the recognition that many bleeding patients when they come to the emergency have an established coagulopathy before the dilutional effects of fluid resuscitation. Traumatic coagulopathy has been demonstrated in patients who received little or no IV fluid therapy, negating the long-held belief that iatrogenic hemodilution is the main causative factor in traumatic coagulopathy. This has led to the use of new terminology **ATC***(acute traumatic coagulopathy)*; trauma induced coagulopathy. The presence of this impairment early after trauma is an important predictor for increased organ dysfunction, infection and overall mortality.

Phases:

Cap and Hunt Classified trauma associated coagulopathies into 3 phases.

- **First phase** is immediate activation of multiple hemostatic pathways with increased fibrinolysis in association with tissue injury and /or tissue hypo-perfusion.
- **Second phase** involves therapy related factors during resuscitation.
- **Third phase**, post resuscitation phase is an acute phase response leading to pro thrombotic state predisposing to venous thromboembolism.

Of these three phases, first phase corresponds to ATC and clinical features of first phase along with the pathophysiologic factors of second phase provide the characteristics of trauma induced coagulopathy.

European management guidelines for trauma induced coagulopathy:

Initial assessment and management:

- Extent of traumatic hemorrhage assessed.
- Patient in shock with identified source of bleeding to be treated immediately.
- Patient in shock with unidentified source of bleeding send for further

investigation.
- Coagulation, hematocrit, serum lactate, base deficit assessed.
- Antifibrinolytic therapy (tranexamic acid within 3 hours after injury) initiated.
- Patient with history of anticoagulant therapy assessed.

Resuscitation:

- Systolic blood pressure of 80-90 mm Hg achieved in absence of TBI.
- Measures to achieve normothermia implemented.
- Target Hemoglobin Level 7-9 mg/dl achieved.

Surgical Intervention:

- Damage control surgery performed in hemodynamically unstable patient.

Coagulation management:

- Massive transfusion protocol with high plasma red blood cell ratio employed.
- Target fibrinogen level 1.5 -2g/l achieved.
- Target platelet level achieved.
- Prothrombin complex concentrate administered if indicated due to Vitamin K antagonist,oral anticoagulant or evidence from viscoelastic monitoring.

Conclusion:

Acute traumatic coagulopathy is caused by endogenous factors but can be worsened by improper medical management. Drivers of ATC are activation of protein C, disruption of endothelial glycocalyx, consumption of fibrinogen and exhaustion/ dysfunction of platelets. This result in reduced clot strength, auto-heparinization and hyper fibrinolysis. Patients presenting to emergency with an established coagulopathy are liable to poor outcomes and must be recognized as early as possible and managed directly and aggressively.

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