Polytrauma with Massive Transfusion

Section 1: Case Summary

Scenario Title:	Polytrauma with Massive Transfusion
Keywords:	Trauma. Massive Transfusion.
	A 35 year old man crashes his motorcycle on the highway and is brought in by EHS. He is hemodynamically unstable. In the ED the patient has a primary survey and initial resuscitation with activation of trauma team, OR, General/Trauma Surgery. He requires activation of massive transfusion protocol. After intubation and placement of chest tube in the ED the patient is transferred to the OR for trauma laparotomy and continued transfusion / resuscitation.

Goals and Objectives	
Educational Goal:	Preparation of ED team for massive transfusion protocol in unstable trauma patient
Objectives: (Medical and CRM)	• Initial stabilization and management of severely injured trauma patient • Review of implementing a Major Hemorrhage Protocol, identifying barriers and successes from physician, nursing, laboratory, RT and technical/IT perspectives. • Clear handover and transfers of care between teams
EPAs Assessed:	

Learners, Setting and Personnel				
Target Learners:	☐ Junior Learners	☐ Senior Learners	☒ Staff	
	☒ Physicians	☒ Nurses	☒ RTs	☒ Inter-professional
	☐ Other Learners: IMIT, Porters, Lab staff.			
Location:	☐ Sim Lab	☒ In Situ – ER	☐ Other:	
Recommended Number of Facilitators:	Instructors: 1			
	Confederates: Paramedics PRN			
	Sim Techs: 1			

Scenario Development	
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Section 5 - Part 1: Scenario Progression - ER Setting

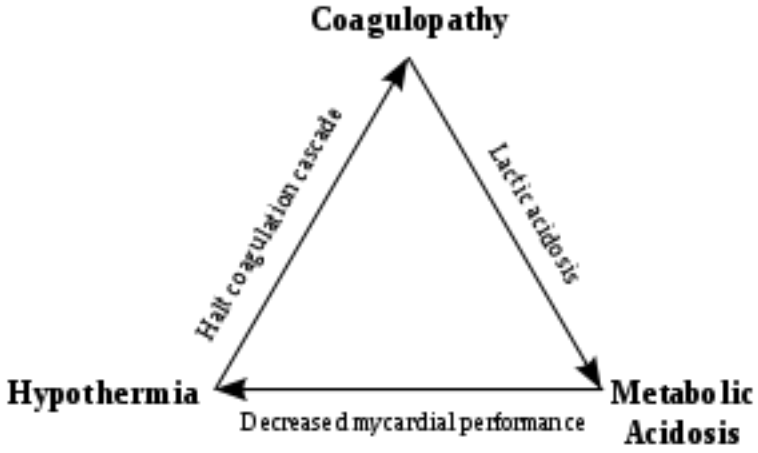
ER Trauma Room				
Patient State/Vitals	Patient Status	Learner Actions, Modifiers & Triggers to Move to Next State		Facilitator Notes
1. Baseline State Rhythm: Sinus tach HR: 128 BP: 85/55 RR: 30 O_2SAT: 91% NRB T: 35.2°C GCS: 8 (E2 V2 M4) Gluc 6.5	Moaning patient.	Expected Learner Actions ☐ Receive EHS Report ☐ Transfer safely to ER stretcher ☐ Remove clothing ☐ Place on monitors ☐ Obtain more IV access or IO ☐ Primary Survey ☐ EFAST – (*identify free fluid in abdomen, left hemo-pneumothorax)* ☐	Triggers *For progression to next state* -Should progress to next stage within 3 minutes	
2. Initial Management Rhythm: Sinus tach HR: 128 BP: 80/50 RR: 30 O_2SAT: 91% NRB GCS: 8 (E2 V2 M4)	Patient quiet after analgesia	Expected Learner Actions ☐ Call for blood STAT ☐ Initiate MHP ☐ Call TTL, OR, General Surgery ☐ Analgesia, TXA ☐ Portable CXR, PXR ☐ Analgesia, TXA ☐ Trauma labs ☐ Identify / communicate priorities to team.	Modifiers - If no blood called for STAT or no MHP requested within 5 minutes patients BP drops to 60/P and confederate identifies concerns	
3. Procedures Rhythm: Sinus tach HR: 120 BP: 75/40 RR: 30 O_2SAT: 91% NRB GCS: 8 (E2 V2 M4)		Expected Learner Actions ☐ MD to prepare / insert left chest tube ☐ MD to prepare to intubate patient ☐ 2 units of uncrossed blood arrives ☐ Post intubation sedation/analgesia ☐ Warm patient / Bair Hugger	Modifiers - If no chest tube or blood then patient has PEA arrest and will not have ROSC until this is done Triggers -Once intubated and left chest tube in-situ Trauma/GenSurg arrives	
4. Handover HR: 110 BP: 95/50 RR: 15 O_2SAT: 95% intubated GCS: 3 T		Expected Learner Actions ☐ Provide summary to Surgeon ☐ Discussion about stability for CT vs direct to OR ☐ Continue MHP ☐ Quickly reduce / splint wrist and ankle ☐ Initial Labs available ☐ Phone call with Hematopathology	Modifiers - Chest tube puts out 650cc blood, slowing Triggers - Surgery accepts care and will take to OR	

Appendix C: Facilitator Cheat Sheet & Debriefing Tips

The **trauma triad of death** is a medical term describing the combination of hypothermia, acidosis and coagulopathy.[1] This combination is commonly seen in patients who have sustained severe traumatic injuries and results in a significant rise in the mortality rate.[2] Commonly, when someone presents with these signs, damage control surgery is employed to reverse the effects.

The three conditions share a complex relationship; each factor can compound the others, resulting in high mortality if this positive feedback loop continues uninterrupted.

Severe bleeding in trauma diminishes oxygen delivery, and may lead to hypothermia. This in turn can halt the coagulation cascade, preventing blood from clotting. In the absence of blood-bound oxygen and nutrients (hypoperfusion), the body's cells burn glucose anaerobically for energy, causing the release of lactic acid, ketone bodies, and other acidic compounds into the blood stream, which lower the blood's pH, leading to metabolic acidosis. Such an increase in acidity damages the tissues and organs of the body and can reduce myocardial performance, further reducing the oxygen delivery.



Damage control surgery (**DCS**) is a technique of surgery used to care for critically ill patients. The leading cause of death among trauma patients remains uncontrolled hemorrhage and accounts for approximately 30–40% of trauma-related deaths.[1] This technique places emphasis on preventing the "lethal triad", rather than correcting the anatomy.[2][3] Damage control surgery is meant to save lives. A multi-disciplinary group of individuals is required: nurses, respiratory therapist, surgical-medicine intensivists, blood bank personnel and others. While this lifesaving method has significantly decreased the morbidity and mortality of critically ill patients, complications can result. This procedure is generally indicated when a person sustains a severe injury that impairs the ability to maintain homeostasis due to severe hemorrhage leading to metabolic acidosis, hypothermia, and increased coagulopathy.[4] The approach would provide a limited surgical intervention to control hemorrhage and contamination. This subsequently lets clinicians focus on reversing the physiologic insult prior to completing a definitive repair. While the temptation to perform a definitive operation exists, surgeons should avoid this practice because of the deleterious effects on patients can result them succumbing to the physiologic effects of the injury, despite the anatomical correction.

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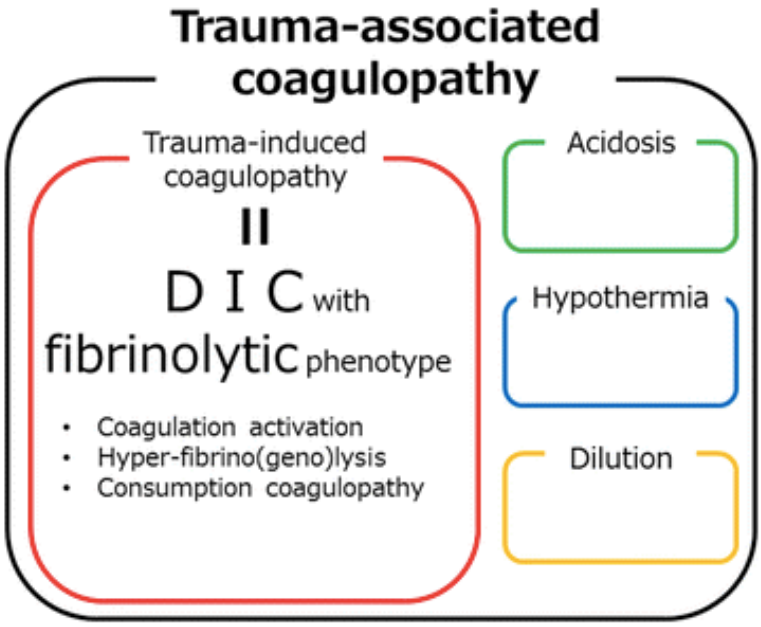
Disseminated intravascular coagulation (DIC) is characterized by systemic activation of blood coagulation, which results in generation and deposition of fibrin, leading to microvascular thrombi in various organs and contributing to multiple organ dysfunction syndrome (MODS). [1, 2] Consumption of clotting factors and platelets in DIC can result in life-threatening hemorrhage. [3]
Derangement of the fibrinolytic system further contributes to intravascular clot formation, but in some cases, accelerated fibrinolysis may cause severe bleeding. Hence, a patient with DIC can present with a simultaneously occurring thrombotic and bleeding problem, which obviously complicates the proper treatment.
The subcommittee on DIC of the International Society on Thrombosis and Haemostasis has suggested the following definition for DIC: "An acquired syndrome characterized by the intravascular activation of coagulation with loss of localization arising from different causes. It can originate from and cause damage to the microvasculature, which if sufficiently severe, can produce organ dysfunction." [4]
DIC is estimated to be present in as many as 1% of hospitalized patients.[5] DIC is not itself a specific illness; rather, it is a complication or an effect of the progression of other illnesses. It is always secondary to an underlying disorder and is associated with a number of clinical conditions, generally involving activation of systemic inflammation.

Fig. 1



Trauma-associated coagulopathy and trauma-induced coagulopathy. Trauma-associated coagulopathy is caused by multiple factors and includes trauma-induced coagulopathy, which is caused by trauma itself.

References

1. Pathophysiology of trauma-induced coagulopathy: disseminated intravascular coagulation with the fibrinolytic phenotype Mineji Hayakawa *Journal of Intensive Care* volume 5**, Article number: 14 (2017)**