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Review Article

The challenge in management of hemorrhagic shock in trauma

Col Mathews Jacob [a,*], Praveen Kumar [b]

[a] Associate Professor, Department of Anaesthesiology and Critical Care, Armed Forces Medical College, Pune 411040, India

[b] Resident, Department of Anaesthesiology and Critical Care, Armed Forces Medical College, Pune 411040, India

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ABSTRACT

Transfusion and resuscitation practices in trauma have undergone a sea change over the past decade. New understanding of transfusion physiology and experiences in military trauma over the last decade has identified key factors taken as challenges in trauma. The most important challenge remains acute traumatic coagulopathy (ATC) which sets in early after a trauma and spirals the patient into shock and continued bleeding. World wide trauma is the leading cause of mortality. More than 6 million deaths occur due to trauma out of which 20% are due to uncontrollable bleeding. Out of the hospital admissions in trauma 20% develop coagulopathy. Mortality is three to four times higher in a patient with coagulopathy and thus prevention and correction of coagulopathy is the central goal of the management of hemorrhagic shock in trauma. Damage control resuscitation (DCR), a strategy combining the techniques of permissive hypotension, hemostatic resuscitation and damage control surgery has been widely adopted as the preferred method of resuscitation in patients with haemorrhagic shock. The over-riding goals of DCR are to mitigate metabolic acidosis, hypothermia and coagulopathy, This article looks at the importance of acute traumatic coagulopathy, its etiology, diagnosis, effects and resuscitation strategies to prevent it and to see the background behind this shift.

Introduction

Every year, around 5.8 million people die worldwide due to events related to trauma, which corresponds to about 9.7 per 100000 population making trauma one of the leading cause of death and disability in all age groups in both sexes. About 40% of trauma related deaths are due to hemorrhage or its consequences.[1]

Outcomes from severe hemorrhage remains poor, with mortality rates approaching 50% for patients requiring blood transfusion, or for those who develop a significant coagulopathy.[2] On an average one in every four patient who is severely injured has acute trauma related coagulopathy at the time of admission to the emergency room.[2]

Researchers over the years have proved that coagulopathy relating to trauma plays a major role in determining the morbidity and mortality in trauma patients.[3] This high degree

* *Corresponding author*. Tel.: +91 9049498131 (mobile).
E-mail address: drmathewsjacob@yahoo.com (M. Jacob).
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of morbidity and mortality can be reduced by early detection of acute traumatic coagulopathy followed by early damage control resuscitation.

Acute traumatic coagulopathy

Coagulopathy following trauma is multifactorial, involving various components of the coagulation system. Endothelium, platelets, fibrin activation and dysfunction, fibrinolytic pathways; all play a crucial role. Which of these mechanisms predominates, dependents on the severity of trauma. There appear to be six main initiators of acute traumatic coagulopathy: 1. Tissue injury 2. Shock – hypoperfusion 3. Hemodilution 4. Hypothermia 5. Acidosis and 6. Inflammation.

Tissue injury

All traumatic injuries invariably lead to tissue damage although the severity of tissue injury varies widely. Crush injuries are associated with maximum tissue damage. The severity of the injury closely correlates with the degree of coagulopathy.[2] Tissue injury leads to activation of both clotting and fibrinolytic systems. Endothelial tear and damage which occurs due to tissue trauma leads to activation of coagulation system as the injury leads to exposure of tissue factor and subendothelial collagen type III, which activates coagulation proteases leading to thrombin and fibrin formation at the site of exposure.

Polytraumatic injury results in tissue factor (TF) thromboplastin release from damaged cells. Acute coagulopathy of trauma (ACT) occurs early and results from tissue hypoperfusion and the tissue injury. Coagulopathy associated with traumatic brain injury (TBI) results from the interplay of various factors. Because of the high concentration of tissue factor in the brain tissue, TBI has believed to be associated with a greater degree of coagulopathy compared with injury in other body systems.[4]

Hypoperfusion

A central role in the emergence of acute traumatic coagulopathy is thought to be played by hypoperfusion resulting due to shock and hypotension.[5] There is a direct correlation between the degree of hypotension and the laboratory derangements in the coagulation profile.

Hypoperfusion in trauma patients is associated with a moderate, dose-dependent reduction in the activity of coagulation factors II, VII, IX, X, and XI, and a more pronounced reduction in factor V activity, which is relatively independent of the severity of shock. The mechanisms underlying decreased factor V activity may be due to activated protein C mediated cleavage.[5]

Markers of hypoperfusion, such as base deficit, might be better and more readily available predictors of who all require coagulation support than international normalized ratio or activated partial thromboplastin time.

Hemodilution

Hemodilution leads to dilution of coagulation factors leading to significant amount of coagulopathy post trauma. During trauma and shock, there is a fall in intravascular hydrostatic pressure which leads to shift of fluid from interstitial and intracellular spaces into the intravascular compartment leading to significant reduction in coagulation factors.[3] This resultant hemodilution is compounded by the administration of crystalloids and colloids.

Hypothermia

Hypothermia is usually observed in trauma patients due to environmental exposure, reduced heat production from underperfused tissues, excessive losses, administration of cold fluids and blood products.[6] Clinically significant levels of reductions in platelet function, aggregation and enzyme activity occur at core body temperatures of 33 °C and below.[7] Drop in temperature by 1 °C is associated with a 10% drop in platelet function.

The activity of tissue factor or FVIIa complex reduces linearly with temperature. The activity is reduced to about 50% at temperatures of 28 °C. At low temperatures, the attraction between von Willebrand factor is reduced causing defective platelet adhesions. However, within the temperature range of 33–36 °C which is usually seen in trauma patients, hypothermia in isolation rarely affects the coagulation system.[8]

Acidosis

Because of hypotension and hypoperfusion which are seen in trauma, there is a significant acidemia in the patient which is further compounded by excessive administration of chloride containing fluids. Fall in pH by itself impairs the function of plasma proteases.

A decrease in pH from 7.4 to 7.0 reduced the activity of FVIIa by over 90% and that of FVIIa/Tissue factor by over 60%.[9] Administration of buffer solutions to correct acidosis have not been shown to correct the coagulopathy that has already set in, indicating that acidosis does not merely reduce the coagulation protease activity.

Inflammation

Massive injury leads to activation of the immune system and the initial inflammatory immune response and has been defined as systemic inflammatory response syndrome (SIRS). This initial response is usually compounded by secondary insults like infections, ischemia/reperfusion or surgeries.[10] Inflammation shifts the hemostatic mechanisms in favor of thrombosis. Multiple mechanisms are at play including upregulation of tissue factor leading to the initiation of clotting, amplification of the clotting process by augmenting exposure of cellular coagulant phospholipids, inhibition of fibrinolysis by elevating plasminogen activator inhibitor1 and decreases in natural anticoagulant pathways, leading to downregulation of the protein C anticoagulant pathway. The decreased function of the natural anticoagulant pathways may be particularly problematic because