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Diagnosis and Treatment of Trauma Induced Coagulopathy (TIC) Using Thromboelastography (TEG)

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Abstract

Trauma induced coagulopathy (TIC) has been recognized as a distinct entity associated with increased mortality, morbidity and transfusion requirements. Uncontrolled bleeding is the most frequent preventable cause of death in trauma patients reaching hospital alive. TIC has been long thought to develop as a result of hemodilution, acidosis and hypothermia often related to resuscitation practices. The lack of well defined diagnosis criteria for TIC impedes early identification and treatment. Most authors established the presence of TIC if Prothombin time (PT) and activated thromboplastin time (APTT) were 1.5 times over the normal value. Mechanisms contributing to TIC include anticoagulation, consumption, platelet dysfunction and hyperfibrinolysis. Thromboelastography (TEG) is a portable bedside device that gives qualitative results for coagulation function, based on clotting, kinetics, strength and lysis. The result is available within 3 - 10 minutes, in the form of curve. Depending on the results of TEG, early administration of tranexamic acid (TXA), recombinant factor VIIa and aggressive blood product transfusional management for TIC with a red blood cell:plasma:platelets ratio close to 1:1:1 could result in decreased mortality from uncontrolled bleeding. This article reviews the pathophysiology and management of TIC.

Keywords

Trauma Induced Coagulopathy (TIC), Thromboelastography (TEG), Fibrinogen, Tranexamic Acid (TXA), Recombinant Factor VII (rFVIIa)

1. Introduction

Trauma remains a leading cause of death and disability in adults in spite of advances in resuscitation, surgical management, and critical care [1]. From 25% to

35% of injured civilian trauma patients develop a biochemically evident coagulopathy when they arrive at the emergency department, though there is improved efficiency of trauma system and reducing the time interval between acute injury and treatment [2]. Trauma-induced coagulopathy (TIC) is a global inflammatory state accompanied by coagulation derangements, acidemia, and hypothermia, which occurs after traumatic injury. It occurs in approximately 25% of severely injured patients, and its incidence is directly related to injury severity. The mechanism of TIC is multifaceted; proposed contributing factors include dysregulation of activated protein C, increased tissue plasminogen activator (tPA), systemic endothelial activation, decreased fibrinogen, clotting factor consumption, and platelet dysfunction.

This article reviews the pathophysiology and management of TIC.

2. Diagnosis

Prolongation of prothrombin time (PT) and activated thromboplastin time (APTT) have been used by most authors to diagnose TIC. Bronhi *et al.* established the presence of TIC if PT and APTT were 1.5 times over the normal values [2]. The prevalence of prolonged PT is higher, but prolongation of the PTT is more specific. While these tests are simple and widely available, they have several limitations. PT and APTT reflect hemostasis in plasma during the first 60 seconds of clotting. Moreover, these tests have a turnaround time of 35 - 45 minutes, are carried out at 37˚C and pH7.5 and do not consider the presence of hypothermia, acidosis, hypocalcium and anemia. Therefore, the use of device which we can measure the value of PT and APTT for a short time has been proposed. Decreased platelet count and decreased platelet function also contribute to coagulopathy and poor outcomes following trauma, although little information about platelet function is evident from the platelet count alone.

{Thromboelastography (TEG—Heamonetics Corp, Braintree, MA USA)}

This is a portable bedside device that gives qualitative results for coagulation function, based on clotting, kinetics, strength and lysis. It accepts one specimen at a time and processes it immediately at room temperature, before the natural temperature of specimen changes. The result is available within 3 - 10 minutes, in the form of curve (**Figure 1**). A number of parameters can be measured.

It is to decide whether the bleeding is surgical or pathological, which clotting factors are missing the function of platelets and whether fibrinolysis is evolving normally. Transfusion of blood components, coagulation factors and additional medication can be administered rationally, based on the results.

3. Treatment

3.1. Tranexamic Acid (TXA)

The most studied antifibrinolytic has been tranexamic acid (TXA). TXA inhibits plasminogen activation, as well as plasmin activity, preventing fibrin clot lysis. The CRASH-2 study [3] evaluated the use of TXA verus placebo in trauma within

3 hours of injury. This was a multicenter RCT that recruited more than 20,000 trauma patients with hemodynamic compromise or at risk of significant bleeding. The study showed that the use of TXA was associated with a decrease in mortality and deaths resulting from bleeding, without an increase in thrombotic complications. Based on the key role of hyperfibrinolysis in TIC pathogenesis and the results of CRASH-2, several authors have proposed that the use of TXA should be a standard in trauma management [4].

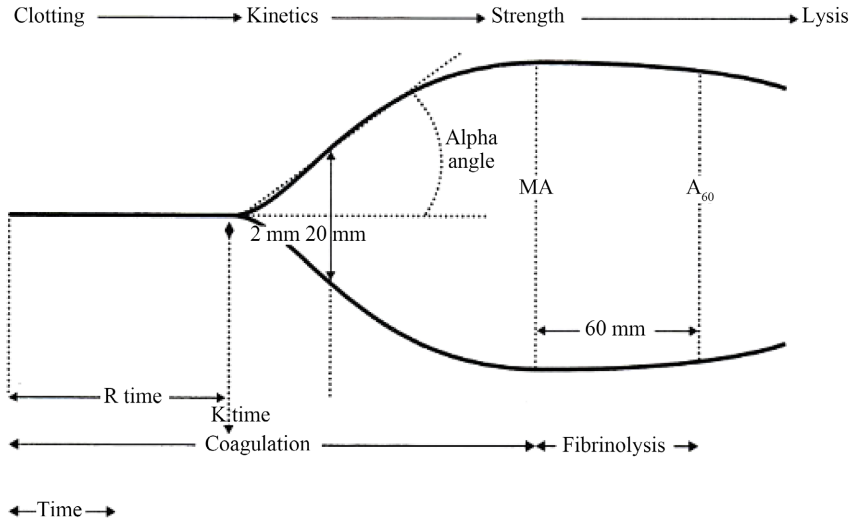

Figure 1. Thromboelastogram (From Edited by Kenneth D Boffard Manual of Definitive Surgical Trauma Care THIRD EDITION P44 Figure 3.1).

3.2. Fibrinogen

Hayakawa *et al.* [5] followed up hemostatic function in severe trauma patients for 24 hours.

When measuring hemostatic function over time in severe trauma patients, fibrinogen levels were <150 mg/dl in about 30% of patients when they arrived at the hospital. These levels continued to decline over time, but PT, APTT, and platelet counts rarely fell below dangerous levels. Therefore, if fibrinogen is 100 - 180 mg/dl, 2 g of fibrinogen was administered. If fibrinogen is below 100 mg/dl, 3 g of fibrinogen was administered.

3.3. Recombinant Factor VIIa

Two RCTs have evaluated the use of activated factor vii (rFVIIa) in trauma. This agent was developed for the treatment of hemophilia A or B. Recently, the control study [6], was performed to compare rFVIIa to placebo. This trial found a decrease in transfusion requirement but no mortality difference. Given these results and its elevated cost, rFVIIa is currently recommended only as a final option in controlling massive bleeding in blunt trauma [7].

3.4. Red Blood Cell:Plasma:Platelets Ratio

Determining an appropriate fluid resuscitation technique during trauma induced coagulopathy (TIC) is challenging. The addition of crystalloid or nonblood colloids further exacerbates a tenuous situation [8]. We advocated a balanced administration of RBC, plasma and platelets (1:1:1) for massive resuscitation. This is the closest representation of whole blood administration and provides maximal resuscitation while maintaining the ability for clot formation. US military data from Operation Iraqi Freedom tend to support this review.

4. Discussion

Effects of TIC include uncontrolled bleeding, massive transfusion, and multi-organ failure. Further, TIC corresponds to having a longer intensive care unit (ICU) and hospital length of stay (LOS), as well as a fourfold increase in mortality. Conventional coagulation tests (CCTs) offer valuable information in monitoring the different aspects of coagulation. However, CCTs have several limitations: (1) they fail to delineate the complex nature of TIC, (2) they are time consuming, and (3) they have questionable value in guiding transfusion requirements [9] [10]. A systematic review in 2011 suggested that CCTs are insufficient to diagnose TIC [11] [12]. Thrombelastography (TEG—Heamonetics Corp, Braintree, MA USA) provides an alternative diagnostic modality for TIC. And TEG are visco-elastic tests offering a prompt global overview of all dynamic sequential aspects of the TIC process by providing data on the speed of coagulation initiation, kinetics of clot growth, clot strength, and its breakdown. Clinicians have developed massive transfusion protocols (MTP) to prioritize the correction of coagulopathy by delivering blood products in a systematic and coordinated fashion. Moreover, by using TEG tests, physicians can obtain a more detailed assessment of all stages of hemostasis that allows for developing a patient specific goal directed therapy [13]. In trauma patients with TIC, evidence exists that TEG tests supersede CCTs in delineating the process of the clotting cascade and therefore are useful in guiding blood product transfusion [7]. Recent guidelines on the management of TIC encourage a treatment strategy focused on goal directed resuscitation with more restriction in the use of intravenous fluids during initial resuscitation. Indiscriminate administration of crystalloids, albumin, or even packed red blood cells (PRBCs) may cause cardiac and pulmonary complications, gastrointestinal dysmotility, and coagulation disturbances. Hence, TEG tests can provide detailed information on which blood products would prove most efficacious to the hemorrhaging trauma patient and accordingly help guide transfusion therapy in appropriate quantities.

If reaction time prolongation is observed based on TEG data, first consider administering FFP and then rFVIIa. If kinetic time prolongation is present, consider administering cryoprecipitate first, followed by rFVIIa. If the alpha angle is <65˚, first consider administering cryoprecipitate, then FFP or rFVIIa. If the maximum amplitude is <50 min, consider administering platelets first, then Desmoressin. If

Lysis at 30 minutes > 7.5%, consider administering tranexamic acid first.

5. Conclusion

Coagulopathy is frequent in trauma. Trauma induced coagulopathy (TIC) has been recognized as a distinct entity associated with increased mortality, morbidity and transfusion requirements. We would like to accurately diagnose the pathology using TEG data, administer blood products appropriately, and correct TIC coagulation and fibrinolysis abnormalities. These procedures could result in decreased mortality from uncontrolled bleeding.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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