

Editorial

Are PT and aPTT Efficient to Predict the Blood Transfusion in Burned Injury?

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Burn-related coagulopathy was first described in 1970s. Previous studies have revealed that if burned total body surface area (TBSA) exceeds 40% in burned patients, replacement of 20 units of blood products may be required [1]. Hence, critical blood and blood products are transfused with extensive burns, independent from the mechanism of the disease. Although the pathophysiological basis has not been precisely understood yet, transfusion-related complications have been clearly demonstrated including infection, acute respiratory distress, organ failure and prolonged stay in intensive care unit. Therefore, survival and patient safety will be enhanced by the use of targeted blood and blood product strategies to prevent possible coagulopathies.

In burned patients, lysis-induced losses occur and on the other hand tissue factor is released due to endothelial injury and coagulation activation occurs related to inflammatory cytokine storm. This process encourages the thrombin generation but also leads to consumption-coagulopathy by the triggering of fibrinolysis. Perioperative blood loss during escharotomy, debridement and graft-flap procedures exacerbate coagulopathy [2].

Today, the uses of viscoelastic tests which allow detailed monitoring of coagulation have made it possible to understand the underlying reason of disorders more precisely and to manage the treatment more effectively. Giving information about the part of hemostasis up to only 5% of thrombin generation, having differences arising from reagents between laboratories, the inability of monitoring in vivo fibrinolysis and prothrombotic state, incapability of setting the temperature suitable for the body temperature of the patient, inability to determine the blood levels of anticoagulant agents and indeed the most important of above all the prolonged turnaround time limit the use of conventional plasma-based tests, PT and PTT analysis [3].

The most common viscoelastic tests are TEG (Thromboelastogram) and ROTEM (Rotational Thromboelastometry). A viscoelastic test, thromboelastogram was first developed by Hartert in 1948 and took place in the clinical practice in 1980 when used in liver transplantation by Kang et al. Portable devices that al-

low bedside use without required a permanent and special space can evaluate all thrombin-mediated processes with viscoelastic measurements of 1-1.5 mL blood. Basically ROTEM is produced with TEG logic. Unlike TEG, in ROTEM, the movement is started by pin instead of receptacle and there is no torsion cable and the receptacle is not moved but the pin is rotated. Sample of the complete blood is placed in a cuvette and immersed in a cylindrical pin. There is a 1 mm aperture which is bridged by blood, between the pin and cuvette. As the blood begins to coagulate and the stability of the coagulum increases progressively, the coagulum restricts the rotation of the pin. This kinetic is detected mechanically and calculated by an integrated computer to the typical curves and numerical parameters. In fact, since the coagulum formation in complete blood is between the development of viscoelastic strength and the pin immersed in the receptacle, viscoelastic signal gives an idea about the formation of endogenous thrombin, fibrin polymerization and interaction between fibrin and platelets.

Experience of the practitioner and calibration can affect the results of these tests, therefore training is required. Although these tests are considered as expensive, it can be more economical when the unnecessary blood transfusions and subsequent treatment costs of the complications are considered. A study using ROTEM reported that the cost of blood and blood products were reduced 32% monthly [4].

In several publications, inadequacies of conventional coagulation tests have been demonstrated. 64% of the military patients injured with major trauma at war had abnormal TEG at the first application, whereas only 10% of them had abnormal PTT values [5]. In another study, perioperative allogeneic blood transfusion requirements have been proven to be reduced by viscoelastic tests [6]. Another recent study has shown that in burned patients, hypofibrinogenesis and fibrinolysis in the early stages are not the main cause of coagulopathy developed in the absence of massive bleeding and viscoelastic tests have been found to be successful in preventing unnecessary transfusions [6].

When we look more closely at the pathophysiology of burn-related coagulopathies, we can see that how complex

the phenomenon is. Although there are many terminologies for coagulopathy in burned patients, nowadays “acute / early-onset burn-induced coagulopathy” is used in literature. The primary causes of early phase coagulopathy in these patients are dilutional coagulopathy developed due to high volume fluid resuscitation, hypothermia due to aggressive heat loss and malfunction of proteins and enzymes in coagulation pathway due to tissue hypoperfusion. It is known that natural anticoagulants such as protein C, protein S, anti-thrombin III, and tissue factor pathway inhibitor [TFPI] decrease, on the other hand activated-factor VII [FVIIa], plasminogen-activator inhibitor type 1 [PAI1], and fibrin degradation products increase, hence ultimate balance is in favor of procoagulants. Although the increase in tissue-type plasminogen-activator (t-PA), which is released from damaged tissues after 24 hours following burn injury, appears to advocate a hyperfibrinolytic condition, there occur no hyperfibrinolysis since this is balanced by increase in PAI-1. It should be remembered that the increase in the acute phase reactivity of fibrinogen levels in these patients will also increase thrombotic susceptibility [7].

Although it is detected in a small proportion of burned patients, prothrombotic susceptibility in patients with large burned area triggers disseminated intravascular coagulation and impairment of organ perfusion due to microcirculatory obstruction, accelerating the progression of multiple organ failure. Therefore, it should be known that prophylaxis is rather needed to prevent venous thromboembolism in these patients.

Studies in massive hemorrhagic trauma patients have shown that survival has been improved by a massive transfusion protocol based on fixed ratio practice including early blood and blood product transfusion therapy. However, a treatment management method based on the assessment of conventional

laboratory results such as PT, PTT and INR obtained from burned patient with a complex coagulation disorder, may contribute to the development of thromboembolic complications in patients who are already in prothrombotic state. On the other hand considering the transfusion-related complications, we conclude that transfusion procedures planned with point-of-care tests for individual needs of burn patients would increase success rate in hemostatic management.

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