

Trauma Crânio-Encefálico Isolado como Causa de Coagulopatia Precoce: Caso Clínico*Isolated Traumatic Brain Injury as a Cause of Early Coagulopathy: Case Report*Rafael Marques Pires¹, Ana Pinto Carneiro¹, Ana Margarida Ferreira¹, José Luís Ferreira²**Autor Correspondente:**

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A coagulopatia associada ao trauma está descrita no trauma crânio-encefálico isolado. Apresentamos um caso onde a tromboelastometria (ROTEM®) foi o único sinal de alerta para coagulopatia precoce.

Paciente do sexo feminino, 77 anos, vítima de queda de escadas. Assistida no local, foi intubada orotraquealmente e transportada ao Serviço de Urgência. Realizou tomografia computadorizada de crânio que evidenciou hematoma subdural agudo. Não apresentava outras lesões traumáticas ou sinais de hemorragia ativa na admissão, mas foi feita colheita de sangue para ROTEM®. Durante a craniotomia, apresentou instabilidade hemodinâmica e sinais de coagulopatia. Os resultados do primeiro ROTEM® evidenciaram ausência de formação de coágulo, e o suporte transfusional foi iniciado. Houve melhoria clínica, e o ROTEM® pós-transfusão mostrou melhoria na formação de coágulo. Admitida na Unidade de Cuidados Intensivos, faleceu 24 horas após o trauma inicial, por lesão neurológica irreversível.

A identificação precoce da coagulopatia associada ao trauma é essencial, evitando a deterioração na abordagem inicial, contribuindo para um melhor prognóstico.

Palavras-chave: Alterações da Coagulação Sanguínea; Lesões Encefálicas; Tromboelastometria

Abstract

Trauma induced coagulopathy has been reported in cases of isolated traumatic brain injury. We report a case where thromboelastometry (ROTEM®) was the only warning sign of the ensuing coagulopathy.

A 77-years-old female, fell down multiple stairs. Assisted on scene, she was intubated and transported to the emergency department. Head computed tomography showed an acute subdural hematoma. No other injury or signs of active bleeding were identified on admission, but a blood sample for ROTEM® was collected. During craniotomy she developed hemodynamic instability, as well as signs of coagulopathy. First ROTEM® showed absence of clot formation and transfusion therapy was started. There was clinical improvement, and a post-transfusion ROTEM® showed improved clot formation. Admitted to the Intensive Care Unit, the patient died 24 hours after the initial neurologic injury, of irreversible brain damage.

Early identification of coagulopathy associated with trauma is crucial, avoiding deterioration during initial management, and contributing to an improved outcome.

Keywords: Blood Coagulation Disorders; Brain Injuries; Thrombelastography

INTRODUCTION

Traumatic brain injury is the leading cause of death and disability in the trauma population and the leading cause of death in all patients younger than the age of 40.¹ Trauma induced coagulopathy has been reported in cases of isolated traumatic brain injury (ITBI) and is associated with prolonged intensive care unit (ICU) stays and detrimental outcomes, with high mortality and morbidity.¹⁻³ Approximately one third of patients with ITBI have associated coagulopathy.^{3,4} Early detection of coagulopathy is important in order to counteract the hemostatic disturbances. Standard tests such as prothrombin time (PT), activated partial thromboplastin

time (aPTT), fibrinogen concentration and platelet count are widely used during initial resuscitation in trauma patients.⁵ However, the conventional coagulation assays focus on selected aspects of coagulation, which may not be appropriate for acute traumatic coagulopathy. Full blood viscoelastic hemostatic assays (VHA), such as rotational thromboelastometry (ROTEM®) and thromboelastography (TEG®), provide a more complete assessment of hemostasis and as point-of-care devices should be able to provide results in a clinically useful time frame for targeted therapy.⁶ Liberal screening and early intervention in patients with clotting disorder associated with severe head injury is very important, even when

there are no signs of coagulopathy.

We describe a case of ITBI where the thromboelastometry (ROTEM®) was the only warning sign of the ensuing coagulopathy.

CASE REPORT

A 77-years-old female with history of hypertension and diabetes mellitus, fell down multiple stairs resulting in head trauma and loss of consciousness. Assisted on scene with Glasgow coma scale (GCS) 7, she was intubated and transported to the emergency department. At arrival the patient presented with a GCS 4 and anisocoria, and was hemodynamically stable. Head computed tomography (CT) showed acute subdural hematoma in fronto-temporo-parietal right side (Fig. 1), with acute compression and contralateral deviation of the right ventricle, which required emergency surgical decompression.

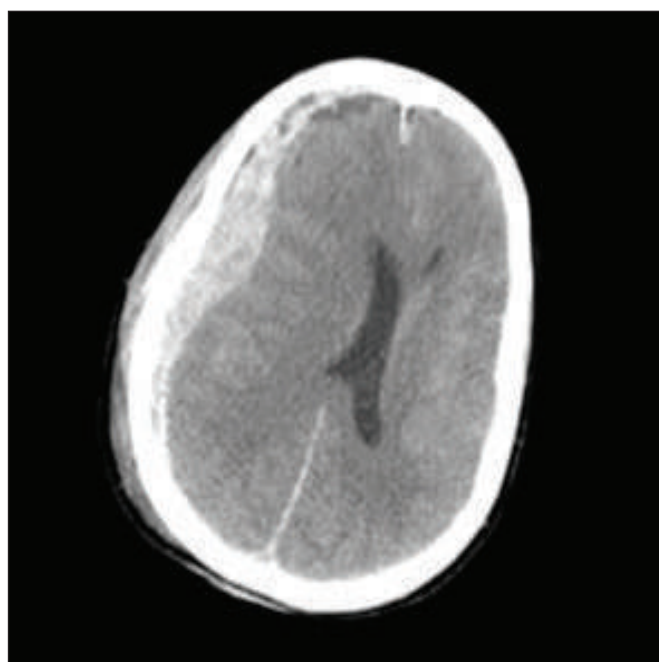


Figure 1. Preoperative brain computed tomography demonstrating acute subdural hematoma in right side.

No other trauma injury or signs of active bleeding were identified on admission. The first set of laboratory values was normal, without changes in standard tests such as PT, aPTT, fibrinogen concentration and platelet count but a blood sample for ROTEM® was collected before the patient went to the operating room (OR). During craniotomy she developed uncontrolled bleeding with hemodynamic instability requiring norepinephrine. The results of the first ROTEM® showed absence of clot formation and severe hyperfibrinolysis (Fig. 2).

Transfusion support was initiated with a total of 4 units of packed red blood cells (PRBC), 1 pool of platelets (PLT), 2 g of fibrinogen, 4 units of fresh frozen plasma (FFP) and 1 g of tranexamic acid. There was clinical improvement, allowing

for surgery completion with adequate hemostasis, and a post-transfusion ROTEM® showed improved clot formation (Fig. 3).

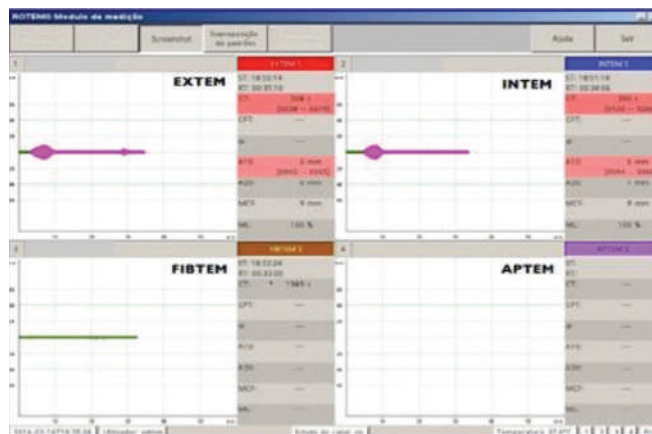


Figure 2. First ROTEM® analysis after trauma (hyperfibrinolysis).

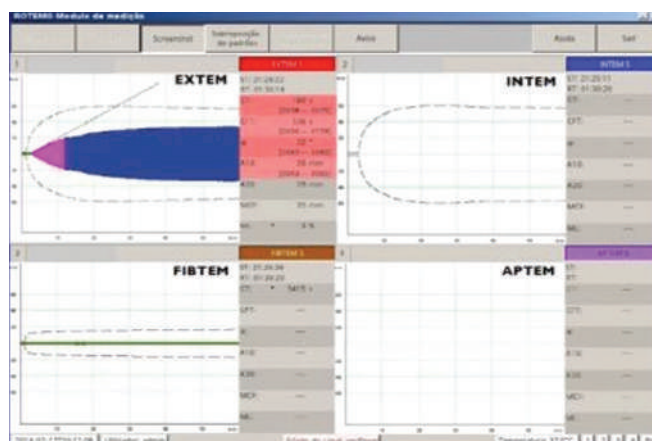


Figure 3. ROTEM® analysis after transfusion support.

Admitted to the ICU with a GCS 3, the patient was persistently hypotensive, despite volume and vasopressor support, accompanied by lactic acidemia. A decision to withhold further therapy was taken based on the irreversibility of the clinical condition, and she died 24 hours after the initial neurologic injury.

DISCUSSION

In our case report, thromboelastometry (ROTEM®) was the only warning sign of the ensuing coagulopathy. Despite the emergency surgical decompression, and transfusion approach, the patient had a poor prognosis and died 24 hours later of the neurologic injury. As described in literature, the presence of early coagulopathy was associated with a poor prognosis. In addition to the early coagulopathy, this patient had other unfavorable outcome risk factors, such as her age and severity of the head injury.

The management of patients with traumatic brain injury includes avoidance of hypoxemia, hypotension, and prevention of secondary brain damage and intracranial

hypertension. Coagulopathy associated with trauma may appear with ITBI and contributes to secondary brain damage.^{1,2}

The primary mechanisms of coagulation disorders in the setting of trauma are acute traumatic coagulopathy (ATC, also termed early trauma induced coagulopathy, early coagulopathy of trauma and acute coagulopathy of trauma-shock) and delayed coagulopathy secondary to haemodilution.⁷⁻¹⁰ Conventionally, the mechanism of ATC has been simplistically proposed as a result of depletion, dilution and dysfunction of procoagulant factors. Further, acidosis and hypothermia, which commonly follow traumatic injury, also add on to the hemostatic insult forming a vicious triad of coagulopathy, acidosis and hypothermia. To date, six key initiators of coagulopathy in trauma have been identified - tissue trauma, shock, hemodilution, hypothermia, acidemia, and inflammation.¹¹ There is significant evidence in the literature that the development of coagulopathy in patients with traumatic brain injury is an important indicator of poor prognosis.^{7,12} Poorer outcomes were associated with acute coagulopathy on admission or where coagulopathy developed in the first 24 hours post-trauma. Additional factors associated with unfavorable outcomes post ITBI were reported to be increasing age, male gender, pupillary reflex abnormalities, head injury severity and hypo-perfusion.^{1,12} Early identification of these patients is essential for an aggressive management of ATC, but also if there is a need for surgical decompression as was our case.

The true incidence of ATC in the setting of ITBI is largely unknown and has been estimated to be between 10% and 98%. This very wide range was attributed to inconsistent definitions of coagulopathy and criteria for ITBI. Other factors included differences in timing at which patients were tested and test sensitivity. Defining the true incidence may help clarify the rationale for empiric management directed at ATC and subsequent development of appropriate guidelines.¹³

Many investigators believe that ROTEM® is a valid predictor of coagulopathy and massive transfusion, and may be useful for diagnosis of early trauma coagulopathies and to direct blood-product transfusion.^{3,6}

Studies with adequate methodology and power are required to identify the role of repeated VHA measurements in haemostatic resuscitation guidance in trauma, and to establish the exact impact of early aggressive management of ATC in outcome.

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Confidentiality of data: The authors declare that they have followed the protocols of their work center on the publication of data from patients.

Proteção de Pessoas e Animais: Os autores declaram que os procedimentos seguidos estavam de acordo com os regulamentos estabelecidos pelos responsáveis da comissão de investigação clínica e ética e de acordo com a declaração de Helsinquia da associação médica mundial.

Protection of human and animal subjects: The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

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