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CLINICAL INFORMATION

Hemostatic Resuscitation in Traumatic Hemorrhagic Shock: Case Report

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Abstract

Background and objectives: The aim of this paper is to report a case in which the damage control resuscitation (DCR) approach was successfully used to promote hemostatic resuscitation in a polytraumatized patient with severe hemorrhagic shock.

Case report: Female patient, 32 years of age, with severe hemorrhagic shock due to polytrauma with hip fracture, who developed acidosis, coagulopathy, and hypothermia. During fluid resuscitation, the patient received blood products transfusion of fresh frozen plasma/packed red blood cells/platelet concentrate at a ratio of 1:1:1 and evolved intraoperatively with improvement in perfusion parameters without requiring vasoactive drugs. At the end of the operation, the patient was taken to the intensive care unit and discharged on the seventh postoperative day.

Conclusion: The ideal management of traumatic hemorrhagic shock is not yet established, but the rapid control of bleeding and perfusion recovery and well-defined therapeutic protocols are fundamental to prevent progression of coagulopathy and refractory shock.

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Introduction

Uncontrolled hemorrhage accounts for 30-40% of early mortality in trauma and more than 80% mortality in the operating room, reported as the leading cause of potentially preventable death [1]. These patients often progress to varying degrees of coagulopathy, hypothermia, and metabolic acidosis, considered the lethal triad of trauma and major predictive of morbidity and mortality in polytraumatized patients.

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Fluid resuscitation in traumatic hemorrhagic shock is still a matter of debate. The classical approach (infusion of large volumes of fluid), type of fluid, and the goals to be achieved have been controversial, as there is evidence that conventional strategies may exacerbate coagulopathy, bleeding, and mortality [2,3].

The concept of hemostatic resuscitation was developed from the experience of emergency medicine in the military, especially during wars in Vietnam, Iraq, and Afghanistan. Hemostatic resuscitation involves early administration of blood products in order to restore both perfusion and coagulation and minimize the use of large volumes of crystalloid and their dilutive effect on coagulation [4,5].

In this context, resuscitation is the early use of whole blood or packed red blood cells (PRBC), fresh frozen plasma (FFP), and platelet concentrate (PC) high administration with fixed ratio between products [5-7].

The objective of this case report is to discuss a case in which the hemostatic resuscitation has been used successfully in a patient victim of polytrauma with severe hemorrhagic shock.

Case report

Female patient, aged 32 years, with polytrauma resulting from car crash against truck, who was brought to the emergency department of the Hospital das Clínicas of the Faculdade de Medicina da Universidade de São Paulo by the Emergency Care Rescue Time (Grupo de Resgate e Atendimento a Urgências – GRAU), air medical service, in the city of São Paulo.

Upon admission, the patient was unconscious, using a cervical immobilization collar, intubated, and on ventilatory support. During clinical examination, she presented with tachycardia; hypotension; anisocoria; Glasgow coma scale 3; and pelvic, humerus, and left jaw fractures; in addition to active bleeding in the right frontoparietal region and large hematoma in the pelvic and hypogastric region.

Additional tests, such as ultrasound, showed no free fluid in the abdominal cavity, chest X-ray without changes, and brain computed tomography with evidence of bleeding into the right ventricle, but without signs of intracranial hypertension.

The initial fluid resuscitation was performed with two liters of Ringer's Lactate and three PRBCs. After closed external immobilization of the pelvis, the patient was immediately taken to the operating room (OR) for hip fracture surgical treatment. The time interval between the patient's arrival to hospital and OR admission was approximately 60 minutes.

In the OR, blood pressure was not identified at the initial noninvasive hemodynamic monitoring, and the patient had only palpable carotid pulse (140 ppm) (Table 1). After obtaining large-bore venous access (venoclysis with a 14G catheter), fluid resuscitation and invasive vascular monitoring with mean arterial pressure (MAP) and central venous pressure (CVP) was initiated.

Table 1 Initial Data Monitoring in the Operating Room.

Mean blood pressure	40 mm Hg
Heart rate	144 ppm
Temperature	34.6ºC
Central venous pressure	3 mm Hg
$ETCO_2$*	25 mm Hg

*$ETCO_2$: end tidal CO_2.

Initial phase of resuscitation was made with the rapid infusion system of warm fluids (Level 1 infuser®) for transfusion and adjusted to maintain MAP > 70 mm Hg, obtained with adrenergic pharmacological support (noradrenaline).

After the fifth PRBC transfusion, the fluid transfusion protocol was triggered and the transfusion strategy followed the FFP/PRBC/PC ratio of 1:1:1, along with Ringer's Lactate infusion of 15 $mL.kg^{-1}.h^{-1}$ and correction of metabolic disorders. Table 2 presents data regarding the transfusion of blood products.

At the end stage of initial resuscitation, the patient had received PRBC (10U), FFB (9U), and PC (10U); the pelvic fracture had been surgically fixed, with surgical bleeding under control; there was evolution with improved perfusion rates, progressive reduction of the pharmacological hemodynamic support, and temperature recovery.

Laboratory tests confirmed the clinical improvement and successful recovery of perfusion, hemodynamics, and hemostasis. The massive transfusion protocol and infusion of norepinephrine were then discontinued (Table 3).

Surgical time was approximately 120 minutes; 30 minutes after the procedure the patient developed a skin rash and mild angioedema, which resolved spontaneously 2 hours later. Figure 1 shows the evolution of coagulopathy during patient's care.

The patient was taken to the ICU where she received packed red blood cells, without requiring other blood products. Postoperatively, she was hemodynamically stable without requiring norepinephrine and was kept under sedation and serial neurological evaluation due to the blood presence in the right ventricle, which evolved without surgical indication.

On the third postoperative day, sedation was suspended, resulting in extubation on the seventh day after surgery and discharged from the ICU to a secondary support unit on the eighth day. The patient was discharged from the hospital on the 63rd postoperative day.

Table 2 Blood Products transfused within 24 hours.

	EU	1st hour	2nd hour	3rd hour	Admission (ICU)**	12 hours	24 hours
PRBCs	2	3	3	2	1	0	0
FFP	0	4	3	2	0	0	0
CP	0	0	10	0	0	0	0

EU: emergency unit; ICU: intensive care unit; PRBCs: packed red blood cells, FFP: fresh frozen plasma, CP: platelet concentrate.

Table 3 First 24 Hours Laboratory Evolution.

	1st hour	2nd hour	3rd hour	Admission (ICU)**	12 hours	24 hours
INR	2			1.6	1.2	1.1
APTT-R	2.5			1.3	1.1	1
Temperature (ºC)	34.6	34.8	34.7	35	35.8	35.8
pH	6.8	7.2	7.2	7.2	7.3	7.4
Base excess (mEq.L^{-1})	-12	-3.1	-5.1	-7.3	-4.3	-1.8
Lactate (mg.dL^{-1})	58	39	35			
Hemoglobin (g.dL^{-1})	3.3	3.7	9.9	6.7	8.6	9.3
Hematocrit (%)	10.1	11.6	28.7	18.2	25.8	27.9

INR: institutional normalized ratio; R-APTT: ratio of activated partial thromboplastin time.

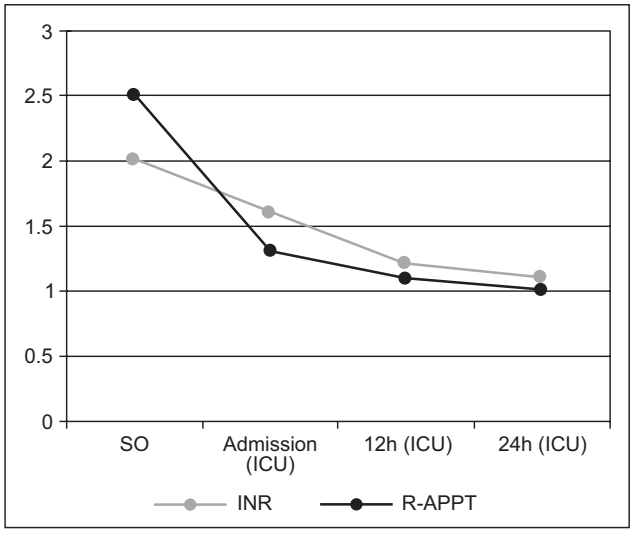

Figure 1 Behavior of INR and R-APTT over 24 Hours.

Discussion

This case report shows that an early, aggressive approach for hemorrhagic shock management, using the damage control resuscitation (DCR) strategy that focuses on hypotensive resuscitation and hemostatic transfusion to control surgical bleeding, plays an important role towards a successful treatment. Current approaches prioritize the rapid identification and correction of circulatory collapse, the major event responsible for mortality in these patients.

Although the administration of crystalloids is an established practice in cases of class I-II hemorrhage, the fluid resuscitation strategy in trauma with hemorrhagic shock (class III-IV) is up for debate [8,9]. In this scenario, the DCR strategy has received greater attention.

In traumatic hemorrhagic shock, the DCR strategy is one of the major therapeutic advances and its application is based on three pillars: abbreviated surgery, reversal of coagulopathy (hemostatic resuscitation), and hypoperfusion (permissive hypotension). This therapy is indicated in class III-IV hemorrhage and hemorrhagic shock and its goal is focused on fighting the lethal triad to abort the vicious cycle of bleeding and avoid the picture irreversibility.

The identification of viable candidates to therapy is the critical point of DCR. The triggers accepted for instituting the hemostatic resuscitation therapy include coagulopathy; blood transfusion > 10 U or > 4 U.h^{-1}; metabolic acidosis with base deficit > 5; temperature < 35ºC; and hemodynamic instability with insufficient response to resuscitation [3,10-13].

Reversing coagulopathy is the main goal for treating severe bleeding. The predominant mechanism of acute trauma coagulopathy (ATC) depends on the degree of microperfusion dysfunction, nature and severity of trauma, and deleterious effects of subsequent medical therapies. ATC has complex and multifactorial pathophysiology, which compromises hemostasis in its entire cascade. In addition, tissue hypoperfusion, metabolic acidosis, hypothermia, and hemodilution are other factors that trigger and maintain this disorder [14,15].

The metabolic markers of hypoperfusion in acute trauma have strong correlation with the incidence of ATC. In 20% of cases, frequently admitted patients with base deficit over six showed an incidence of coagulopathy, whereas patients without base deficit showed no change in laboratory markers of coagulation [14,16].

On the other hand, in a rapid attempt to restore tissue perfusion and oxygen delivery, polytrauma patients usually receive crystalloids and packed red cells that have no clotting factors, which causes strong dilutional effect on coagulation factors. Moreover, the clinical condition of these patients is worsened with the usual finding of metabolic acidosis (pH < 7.1) and hypothermia (temperature < 34°C) – factors that solely affect hemostasis.

The causes of hypothermia are multifactorial and interdependent, including changed central thermoregulation; reduced endogenous heat production, due to tissue hypoperfusion in hemorrhagic shock; exposure to low temperatures in the operating room; and infusion of crystalloid solution and blood products inadequately warmed [17].

Because the hemostasis process consists of an enzyme cascade dependent on a body temperature of about 37°C to occur normally, in case of hypothermia there is impairment of thrombin generation, platelet aggregation, and fibrin thrombi, which occurs simultaneously with hyperfibrinolysis [17-20].

Confirming the deleterious effects of hypothermia on coagulation, acidosis appears as a complicating factor, as it substantially reduces the rate of thrombus formation, assessed by thromboelastography, and platelet aggregation [5]. The high level of lactate (lactate > 90 mg.dL^{-1}) alone is proven to contribute to impaired hemostasis via *in vitro* thromboelastography [21].

The acidosis effect on coagulation is not reversed by simple pharmacological correction of acidosis with bicarbonate. Several investigators agree that pH must be over 7.2 before implementing circumstantial therapies for coagulation disorders, which reinforces the mandatory recovery of perfusion in coagulopathy treatment [19,20].

In the case reported, the patient was admitted with severe anemia (Hb = 3.3 $g.dL^{-1}$), acidosis (pH = 6.8^{-1}), hyperlactatemia (lactate = 58 $mg.dL^{-1}$), and moderate hypothermia. Given the gravity of the case, transfusion was initiated during surgery to maintain a ratio of 1:1:1 between blood products, with the intention of correcting anemia without increasing hemostatic disorder.

In a cohort study, Sperry et al. [6] found that when massive transfusion is performed, maintaining a high ratio of fresh frozen plasma and packed red blood cells, there is an overall reduction in transfusion requirements in the first 24 hours, despite the greater amount of plasma transfused. In our case, in the first 12 hours the patient received 11 units of PRBCs, 9 units of fresh frozen plasma, and 10 units of platelet concentrate.

There were some limitations in this case, such as the time to perform laboratory tests and the lack of platelet count. Thus, therapy during intraoperative period was given empirically based on surgical bleeding, clot formation in the field, and presence of microvascular bleeding.

Although the optimal therapeutic approach to traumatic hemorrhagic shock is not yet fully established, the rapid control of bleeding and recovery of perfusion, in addition to well-defined therapeutic protocols are fundamental to prevent progression of coagulopathy and refractory shock.

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