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Damage control strategy in bleeding trauma patients

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Hemorrhagic shock is a main cause of death in severe trauma patients. Bleeding trauma patients have coagulopathy on admission, which may even be aggravated by incorrectly directed resuscitation. The damage control strategy is a very urgent and essential aspect of management considering the acute coagulopathy of trauma and the physiological status of bleeding trauma patients. This strategy has gained popularity over the past several years. Patients in extremis cannot withstand prolonged definitive surgical repair. Therefore, an abbreviated operation, referred to as damage control surgery (DCS), is needed. In addition to DCS, the likelihood of survival should be maximized for patients in extremis by providing appropriate critical care, including permissive hypotension, hemostatic resuscitation, minimization of crystalloid use, early use of tranexamic acid, and avoidance of hypothermia and hypocalcemia. This review presents an overview of the evolving strategy of damage control in bleeding trauma patients.

Key Words: blood transfusion; critical care; hemorrhage; injury; resuscitation

Review Article

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INTRODUCTION

In South Korea, injury is a major cause of death, especially for younger age groups, and according to a 2015 nationwide survey, approximately 30% of deaths due to trauma would have been preventable if adequate treatment had been administered [1]. Furthermore, the top cause of death in trauma is uncontrollable bleeding [2]. "Damage control" is a term used in the maritime industry to refer to the emergency management of situations that may cause a vessel to sink. At the scene of an accident, only temporary, limited, urgent repairs are conducted to prevent sinking and then the damaged ship is brought to the dock for definitive repairs. In a similar sense, damage control surgery (DCS) refers to operations performed in patients whose condition is in extremis due to bleeding. In DCS, many procedures are omitted in order to focus on preserving vital aspects of physiology, based on the concept that severely injured patients cannot withstand the prolonged procedures and physiological insults associated with definitive repair [3-6]. In other words, definitive care of trauma patients in extremis in a single operation is like fully repairing a damaged ship at the scene without adequate equipment.

DAMAGE CONTROL RESUSCITATION

Damage control resuscitation (DCR) is an extension of the concept of damage control in severe trauma patients and accepted as complementary to DCS. Conceptually, DCR encom-

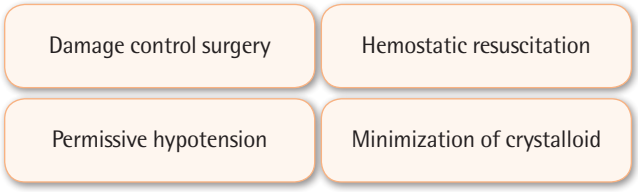

Figure 1. Four main components of damage control resuscitation.

passes not only DCS, but all critical care approaches that correct trauma-induced coagulopathy and provide optimal resuscitation [7-11]. DCR incorporates permissive hypotension to prevent clots from dislodging, minimization of crystalloid use, hemostatic resuscitation, body rewarming, and early hemorrhage control (Figure 1).

PERMISSIVE HYPOTENSION AND MINIMIZATION OF CRYSTALLOID USE

Permissive hypotension is also known as hypotensive resuscitation. The main concept is restricting the amount of resuscitation fluid and vasopressors to maintain blood pressure in the lower-than-normal range until the bleeding is controlled. Achieving a balance between organ perfusion and hemostasis is crucial for bleeding trauma patients, and clinicians must walk the tightrope between bleeding and hypotensive shock. If the blood pressure is higher, it is more likely that any clots that have formed will be dislodged by the high pressure of the blood and the amount of bleeding will increase. Maintaining normal blood pressure in an uncontrolled bleeding situation may therefore worsen survival, which provides a rationale for maintaining lower-than-normal blood pressure. The target systolic blood pressure of permissive hypotension is 80–90 mm Hg; however, permissive hypotension is only a temporary tool to be used until the source of bleeding is controlled because prolonged hypotension can cause ischemic damage to end-organs, including the brain and kidney, and worsen lactic acidosis [12]. Therefore, in patients with severe traumatic brain injury (TBI), this rule does not apply, and the new target for TBI patients with glasgow coma scale ≤8 is a mean arterial pressure >80 mm Hg [12].

Target of Permissive Hypotension

Another issue is the minimization of crystalloid use. The Advanced Trauma Life Support (ATLS) guidelines traditionally recommended the infusion of 2 L of crystalloid in patients with suspected hemorrhagic shock [13]. However, in the current 10th edition of the ATLS guidelines, only 1 L of crystalloid

KEY MESSAGES

- Damage control surgery (DCS) focuses on rapid control of hemorrhage and contamination for patients in extremis.
- DCS is usually conducted within a very limited time, and non-urgent procedures are omitted or postponed to a subsequent operation.
- Damage control resuscitation incorporates a broad range of approaches to save the lives of bleeding trauma patients, including DCS, permissive hypotension, hemostatic resuscitation, minimization of crystalloid use, early use of tranexamic acid, and avoidance of hypothermia and hypocalcemia.

(including the amount in the pre-hospital setting) is suggested [14]. Excessive crystalloid use is associated with a risk of dilutional coagulopathy because crystalloids contain no blood products (e.g., red blood cells [RBCs] and coagulation factors) and pose a risk of hypothermia, acute lung injury, abdominal compartment syndrome, multi-organ failure, and death [15-17]. A multicenter trial evaluating the minimization of crystalloids for penetrating torso injuries was reported in 2015 [18]. Compared with the standard fluid resuscitation group, the restrictive fluid resuscitation group exhibited lower intraoperative mortality (9% vs. 32%; $P<0.001$) and a shorter hospital length of stay (13 vs. 18 days; $P=0.02$). In 2017, Harada et al. [19] reported, in an analysis of 10-year trends in crystalloid resuscitation, that the decrease in high-volume crystalloid resuscitation paralleled a reduction of mortality.

HEMOSTATIC RESUSCITATION

Hemostatic resuscitation is now widely accepted as a transfusion therapy for bleeding trauma patients in extremis. The traditional component therapy that only gave packed RBCs (pRBCs) and crystalloids could not replace the coagulation factors consumed by bleeding. The concept of hemostatic resuscitation is the transfusion of blood products (pRBCs, fresh frozen plasma [FFP], and platelets) that closely approximate whole blood. In 2007, Borgman et al. [20] reported a survival benefit for a high ratio of plasma to RBCs in patients who received massive transfusion. A high plasma-to-RBC ratio was independently associated with survival benefits due to reduced death from hemorrhage. Early and aggressive plasma transfusion can reduce mortality from bleeding [20-23].

A high ratio of platelets to RBCs is also very important. In 2008, Holcomb et al. [23] investigated the plasma-to-RBC and

platelet-to-RBC ratios in 466 civilian trauma patients who underwent massive transfusions. They divided the patients into four groups according to ratios of FFP to RBCs and platelets to RBCs of 1:2, as follows: (1) high FFP-to-RBC and high platelet-to-RBC ratios, (2) high FFP-to-RBC and low platelet-to-RBC ratios, (3) low FFP-to-RBC and high platelet-to-RBC ratios, and (4) low FFP-to-RBC and low platelet-to-RBC ratios. The patients with high FFP-to-RBC and high platelet-to-RBC ratios had significantly higher survival rates at 6 hours, 24 hours, and 30 days than the other groups.

However, the optimal FFP-to-RBC and platelet-to-RBC ratios remained unknown. In 2015, the Pragmatic, Randomized Optimal Platelet and Plasma Ratios (PROPPR) study was conducted, in which 680 patients were randomized to receive either a 1:1:1 or 1:1:2 ratio of plasma, platelets, and RBCs [24]. Overall mortality was not significantly different between both groups, although death from exsanguination was less common in the 1:1:1 group than in the 1:1:2 group. In conclusion, FFP-to-RBC and platelet-to-RBC ratios of at least 1:2 are recommended for DCR.

BODY REWARMING

Hypothermia is a common problem in the resuscitation of trauma patients because of cold exposure, cold resuscitation fluids, significant blood loss, and shock. The problem is that hypothermia can negatively affect coagulation function. One study showed that relative clotting factor function was sensitively decreased by temperatures ranging from 37°C to 25°C; even if there was no deficiency in clotting factors, their function was severely impaired to less than 10% of normal [25]. In 1994, Gubler et al. [26] demonstrated that dilutional coagulopathy had an additive effect on hypothermia. Hypothermia does not decrease the level of clotting factors or platelets, but directly affects their function. Therefore, treating hypothermia actively with heated fluids, heated blanket, and a warm environment is recommended.

TRANEXAMINIC ACID

Tranexamic acid (TXA) is an anti-fibrinolytic drug that interferes with the binding of plasminogen to fibrin. In theory, TXA can prevent clot breakdown and reduce blood loss in bleeding trauma patients. In 2011, the CRASH-2 trial was conducted to evaluate the effect of early administration of a short course of TXA on death from bleeding trauma [27]. The mortality of the group that received TXA within 1 hour of injury was 5.3%, compared to 7.7% in the placebo group, and TXA did not increase the risk of thrombosis. Patients who received TXA between 1 and 3 hours after the injury also had a survival benefit from bleeding to death. However, if TXA was given more than 3 hours after the injury, the risk of death due to bleeding significantly increased. Therefore, TXA should be first administered within 3 hours from injury. Specifically, 1 g of TXA is administered intravenously or intraosseously, mixed with 100 mL of saline for 10 minutes, followed by another 1 g steady drip over 8 hours.

However, there has been some criticism of the routine use of TXA. In 2016, Moore et al. [28] investigated 2,540 severe trauma patients with an Injury Severity Score >15 and showed that the fibrinolysis shutdown type was the most common, whereas the hyperfibrinolytic type that needed antifibrinolytic agents only accounted for 18% of cases. Therefore, the authors recommended individualization of TXA usage after confirmation of coagulation status through a viscoelastic assessment.

CALCIUM

Calcium is an important cofactor of the coagulation cascade. In bleeding trauma patients who need blood transfusion, hypocalcemia may be a problem because of citrate, a calcium chelating agent contained in many blood products. The effects of hypocalcemia cannot be evaluated through routine laboratory tests, and even 1 unit of citrate-containing blood products can further lower calcium ion levels to the point that they approach critical values of hypocalcemia associated with lower survival [29-33]. Calcium ion levels should be monitored and maintained within the normal range. To correct hypocalcemia, calcium chloride is preferable due to the amount of calcium that it contains, as 10% calcium chloride contains 270 mg of elemental calcium per 10 mL, whereas 10% calcium gluconate contains 90 mg of elemental calcium per 10 mL [12].

DAMAGE CONTROL SURGERY

In the 1980s, an important paradigm shift occurred in the treatment of bleeding trauma patients. DCS is a treatment strategy that focuses on rapid control of hemorrhage and contamination [3,4,34]. Time is very limited for DCS—usually within 90 minutes—because of the concept that severely injured patients cannot withstand the prolonged procedures and physiological insult that would be required for definitive repair. Therefore, the bowel is left in a discontinuous condition and

vessels may be kept patent with temporary shunts.

Three steps of DCS

First step is abbreviated operation, including bleeding and contamination control, as well as temporary abdominal closure. Second is critical care for physiological restoration in the intensive care unit. Third is definitive surgery. DCS is now accepted as a component of DCR and is concomitantly conducted with DCR in most cases.

CONCLUSION

The damage control strategy is a very urgent and essential aspect of management for bleeding trauma patients, considering the acute coagulopathy of trauma and these patients' physiological status. The strategy incorporates an abbreviated operation, referred to as DCS, as well as appropriate critical care including permissive hypotension, hemostatic resuscitation, minimization of crystalloid use, early use of TXA, and avoidance of hypothermia and hypocalcemia. These approaches collectively improve the survival of bleeding trauma patients in extremis.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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Conceptualization & Data curation: HC. Formaly analysis & Methodology: MK. Project administration: HC. VIsualization: MK. Writing–original draft: HC. Writing–review & editing: MK.

REFERENCES

1. Jung K, Kim I, Park SK, Cho H, Park CY, Yun JH, et al. Preventable trauma death rate after establishing a national trauma system in Korea. J Korean Med Sci 2019;34:e65.
2. Sauaia A, Moore FA, Moore EE, Moser KS, Brennan R, Read RA, et al. Epidemiology of trauma deaths: a reassessment. J Trauma 1995;38:185-93.
3. Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. Ann Surg 1983;197:532-5.
4. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR 3rd, Fruchterman TM, Kauder DR, et al. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma 1993;35:375-83.
5. Sutton E, Bochicchio GV, Bochicchio K, Rodriguez ED, Henry S, Joshi M, et al. Long term impact of damage control surgery: a preliminary prospective study. J Trauma 2006;61:831-6.
6. Johnson JW, Gracias VH, Schwab CW, Reilly PM, Kauder DR, Shapiro MB, et al. Evolution in damage control for exsanguinating penetrating abdominal injury. J Trauma 2001;51:261-71.
7. Khan S, Davenport R, Raza I, Glasgow S, De'Ath HD, Johansson PI, et al. Damage control resuscitation using blood component therapy in standard doses has a limited effect on coagulopathy during trauma hemorrhage. Intensive Care Med 2015;41:239-47.
8. Ball CG. Damage control resuscitation: history, theory and technique. Can J Surg 2014;57:55-60.
9. Midwinter MJ. Damage control surgery in the era of damage control resuscitation. J R Army Med Corps 2009;155:323-6.
10. Parr MJ, Alabdi T. Damage control surgery and intensive care. Injury 2004;35:713-22.
11. Mizobata Y. Damage control resuscitation: a practical approach for severely hemorrhagic patients and its effects on trauma surgery. J Intensive Care 2017;5:4.
12. Spahn DR, Bouillon B, Cerny V, Duranteau J, Filipescu D, Hunt BJ, et al. The European guideline on management of major bleeding and coagulopathy following trauma: fifth edition. Crit Care 2019;23:98.
13. American College of Surgeons. Advanced trauma life support program for doctors. 7th ed. Chicago: American College of Surgeons Committee on Trauma; 2004.
14. American College of Surgeons. ATLS advanced trauma life support. 10th ed. Chicago: American College of Surgeons; 2018.
15. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg 2003;138:637-43.
16. Duke MD, Guidry C, Guice J, Stuke L, Marr AB, Hunt JP, et al. Restrictive fluid resuscitation in combination with damage control resuscitation: time for adaptation. J Trauma Acute Care Surg 2012;73:674-8.
17. Duchesne JC, Guidry C, Hoffman JR, Park TS, Bock J, Lawson

S, et al. Low-volume resuscitation for severe intraoperative hemorrhage: a step in the right direction. Am Surg 2012;78:936-41.
18. Schreiber MA, Meier EN, Tisherman SA, Kerby JD, Newgard CD, Brasel K, et al. A controlled resuscitation strategy is feasible and safe in hypotensive trauma patients: results of a prospective randomized pilot trial. J Trauma Acute Care Surg 2015;78:687-97.
19. Harada MY, Ko A, Barmparas G, Smith EJ, Patel BK, Dhillon NK, et al. 10-Year trend in crystalloid resuscitation: reduced volume and lower mortality. Int J Surg 2017;38:78-82.
20. Borgman MA, Spinella PC, Perkins JG, Grathwohl KW, Repine T, Beekley AC, et al. The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. J Trauma 2007;63:805-13.
21. Savage SA, Zarzaur BL, Croce MA, Fabian TC. Time matters in 1: 1 resuscitations: concurrent administration of blood: plasma and risk of death. J Trauma Acute Care Surg 2014;77:833-8.
22. Shaz BH, Dente CJ, Nicholas J, MacLeod JB, Young AN, Easley K, et al. Increased number of coagulation products in relationship to red blood cell products transfused improves mortality in trauma patients. Transfusion 2010;50:493-500.
23. Holcomb JB, Wade CE, Michalek JE, Chisholm GB, Zarzabal LA, Schreiber MA, et al. Increased plasma and platelet to red blood cell ratios improves outcome in 466 massively transfused civilian trauma patients. Ann Surg 2008;248:447-58.
24. Holcomb JB, Tilley BC, Baraniuk S, Fox EE, Wade CE, Podbielski JM, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA 2015;313:471-82.
25. Johnston TD, Chen Y, Reed RL 2nd. Functional equivalence of hypothermia to specific clotting factor deficiencies. J Trauma 1994;37:413-7.
26. Gubler KD, Gentilello LM, Hassantash SA, Maier RV. The impact of hypothermia on dilutional coagulopathy. J Trauma 1994;36:847-51.
27. CRASH-2 collaborators, Roberts I, Shakur H, Afolabi A, Brohi K, Coats T, et al. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. Lancet 2011;377:1096-101.
28. Moore HB, Moore EE, Liras IN, Gonzalez E, Harvin JA, Holcomb JB, et al. Acute fibrinolysis shutdown after injury occurs frequently and increases mortality: a multicenter evaluation of 2,540 severely injured patients. J Am Coll Surg 2016;222:347-55.
29. Lier H, Böttiger BW, Hinkelbein J, Krep H, Bernhard M. Coagulation management in multiple trauma: a systematic review. Intensive Care Med 2011;37:572-82.
30. Perkins JG, Cap AP, Weiss BM, Reid TJ, Bolan CD. Massive transfusion and nonsurgical hemostatic agents. Crit Care Med 2008;36(7 Suppl):S325-39.
31. MacKay EJ, Stubna MD, Holena DN, Reilly PM, Seamon MJ, Smith BP, et al. Abnormal calcium levels during trauma resuscitation are associated with increased mortality, increased blood product use, and greater hospital resource consumption: a pilot investigation. Anesth Analg 2017;125:895-901.
32. Webster S, Todd S, Redhead J, Wright C. Ionised calcium levels in major trauma patients who received blood in the emergency department. Emerg Med J 2016;33:569-72.
33. Giancarelli A, Birrer KL, Alban RF, Hobbs BP, Liu-DeRyke X. Hypocalcemia in trauma patients receiving massive transfusion. J Surg Res 2016;202:182-7.
34. Schreiber MA. The beginning of the end for damage control surgery. Br J Surg 2012;99 Suppl 1:10-1.