Damage Control Philosophy in Polytrauma

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Abstract

Severe traumatic injury is a public health care problem; with injuries accounting for 12% of the global mortality. Continued improvement in the survival of severely injured trauma patients is a paramount goal. Bypass/damage control surgery following trauma has developed as a major advance in surgical practice in the last twenty years. The principles of damage control surgery defied the traditional surgical teaching of definitive operative intervention and were slow to be adopted. Currently, damage control surgery has been successfully utilized to manage traumatic thoracic, abdominal, extremity, and peripheral vascular injuries. In addition, damage control surgery has been extrapolated for use in general, vascular, cardiac, urologic, and orthopaedic surgery. Stone et al were the first to describe the "bypass" approach of staged surgical procedures for severely injured patients. This approach emerged after their observation that early death following trauma was associated with severe metabolic and physiologic derangements following severe exsanguinating injuries. Profound shock along with major blood loss initiates the cycle of hypothermia, acidosis, and coagulopathy. During the 1980s, hypothermia, acidosis, and coagulopathy were described as the "trauma triangle of death" which makes the prolonged and definitive operative management of trauma patients dangerous. The management technique, now described as "damage control" by Rotondo et al, involves a multiphase approach, in which reoperation occurs after correction of physiologic abnormalities.

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Introduction

The surgical treatment of haemodynamically unstable patients with penetrating abdominal injuries during war continues to improve. These wounds were considered fatal during the early 20th century. Liver packing for abdominal trauma was first described in 1908, but it was discontinued due to high incidence of infection [1]. During World wars I and II the military surgeons recognised the importance of speedy abdominal surgery. With improvements in weapon technology, the use of high velocity bullets resulted in greater energy transfer. The magnitude of the intra-abdominal cavitation also caused severe tissue destruction, shockwave rupture of gas filled intestine at sites remote from the wound track and multiple solid organ damage [2]. These injuries required prolonged laparotomies. It was during these procedures that the lethal triad of acidosis, hypothermia and coagulopathy were identified. It would often manifest as nonsurgical bleeding after three to six hours of surgery.

In 1983, Harlan Stone re-introduced the concept of rapid packing and termination of the laparotomy when intra-operative coagulopathy became clinically apparent [3]. In this study, 17 patients were managed by an initial laparotomy, followed by packing in patients with an

observed clinical coagulopathy, then completion of the surgical procedure once the coagulopathy was improved. This resulted in 11 survivors, with a mortality rate of 35%. Ten years later Rotondo coined the phrase Damage Control Surgery (DCS) [4]. Damage Control was a naval term. It described the techniques like stuffing mattresses into gaping holes, extinguishing local fires, dogging down watertight doors to limit flooding and damage spread. These principles kept the ship afloat and maintained mission integrity [5]. This allowed the ship to continue to fight, until a feasible plan could be formulated for later definitive repair.

Pathogenesis of 'Lethal triad' and its Consequences (Fig. 1)

Hypothermia is a consequence of severe exsanguinating injury and subsequent resuscitative efforts. Severe haemorrhage leads to tissue hypoperfusion and diminished oxygen delivery, which leads to reduced heat generation. Clinically significant hypothermia is important if the body temperature drops to <36°C for more than 4 hours. Hypothermia can lead to cardiac arrhythmias, decreased cardiac output, increased systemic vascular resistance, and left shift of the oxygen-hemoglobin dissociation curve. It can also induce coagulopathy by inhibition of the coagulation cascade [4]. Low temperature also impairs the host's

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Fig. 1: Lethal Triad.

immunologic function.

Hypothermia is aggravated by heat loss from either environmental factors or surgical interventions. The multidisciplinary team caring for trauma patients must make every effort to prevent heat loss and help to correct hypothermia.

Clinical coagulopathy occurs because of hypothermia, platelet and coagulation factor dysfunction, which occurs at low temperatures, activation of the fibrinolytic system, and hemodilution following massive resuscitation. Platelet dysfunction is secondary to the imbalance between thromboxane and prostacyclin that occurs in a hypothermic state. Hypothermia and hemodilution produce an additive effect on coagulopathy. After replacement of one blood volume (5,000 mL or 15 units of packed red blood cells (RBCs) only 30-40% of platelets remain in circulation [6]. The prothrombin time (PT), partial prothrombin time (PTT), fibrinogen levels, and lactate levels are not predictive of the severe coagulopathic state.

Anaerobic metabolism starts when the shock stage of hypoperfusion is prolonged, leading to metabolic acidosis caused by the production of lactate. Acidosis decreases myocardial contractility and cardiac output [7]. Acidosis also worsens from multiple transfusions, the use of vasopressors, aortic cross-clamping and impaired myocardial performance. It is clear that a complex relationship exists between acidosis, hypothermia, and coagulopathy and that each factor compounds the other, leading to a high mortality rate.

Principles of Damage Control

The aim of damage control surgery is to stop haemorrhage and minimize contamination. Haemorrhage is controlled by ligation, temporary clamping, shunting or packing. Contamination due to hollow viscus injury is minimized by closure or resection without anastomosis.

Table 1

Physiologic guidelines that predict the need for damage control

S. No.	Physiologic parameter
1.	Hypothermia $\leq 34^{\circ}\text{C}$
2.	Acidosis pH ≤ 7.2
3.	Serum bicarbonate ≤ 15 mEq/L
4.	Transfusion of $\geq 4,000$ mL blood
5.	Transfusion of $\geq 5,000$ mL blood and blood products
6.	Intraoperative volume replacement $\geq 12,000$ mL
7.	Clinical evidence of intraoperative coagulopathy

Then abdomen is temporarily closed so as to prevent intra-abdominal hypertension. Planned reoperation to restore anatomy is performed after achieving normal physiology.

Not all trauma patients require damage control measures. The guidelines for initiating damage control are given in Table 1. Certain conditions and complexes of injuries assessed both preoperatively and intraoperatively require damage control.

Damage control, according to Asensio et al [8], implies immediate control of life-threatening haemorrhage, hepatic packing, pancreatic drainage, temporary hollow viscus closures, rapid stapled resections, splenectomy, nephrectomy, vascular pedicle clamping in situ, and the use of intraabdominal vascular shunts. Frequently, these patients experience abdominal compartment syndrome. Therefore, the posttraumatic open abdomen with temporary abdominal wall closure is used as an extension of damage control. Specifically, for chest injuries one should repair cardiovascular injuries, perform stapled pulmonary tractotomy, pack if needed, place chest tubes, and close the skin [8].

The second stage begins in the intensive care unit, (ICU) where the trauma surgery team tries to correct the metabolic disorders. Rewarming the patient is a high priority because coagulopathy and acidosis can be corrected and maintained only after the body temperature returns to normal. Further inspections are then made to identify injuries that may not have been detected in the initial survey. Twenty-four to 72 hours may be needed to correct metabolic derangements. The last stage of damage control involves the timing of reoperation when definitive procedures are performed. Reoperation is considered early if major blood losses continue. Usually, there is a window of 36-48 hours after the initial injury, between the correction of the metabolic disorder and the onset of the systemic inflammatory response syndrome and/or multiple organ failure. In this phase, definitive procedures are undertaken. Thorough reexploration is made for any additional injuries, and restoration of gastrointestinal continuity and vascular repair are done. Provisional feeding access may be

placed, which is followed by washout of the abdominal cavity and an attempt at definitive closure. The patient then returns to the ICU for further care.

Recent Developments

Warfare injuries have been a driving force for surgeons to innovate better methods of trauma care. Many time-tested methods of resuscitation and surgery were tested during Afghanistan and Iraq wars. New approaches to tackle the complex military trauma were developed. This has definitely resulted in reduced incidence of mortality due to war wounds. One of the new approaches is the Damage Control Resuscitation (DCR).

Damage control resuscitation: This an integrated approach where DCR and DCS are undertaken simultaneously. It involves permissive hypotension and haemostatic resuscitation along with damage control surgery [9].

- a. Permissive hypotension – also known as “Hypotensive” or “Balanced” resuscitation – is a strategy of restricting fluid resuscitation until haemorrhage is controlled, while accepting a limited period of suboptimal organ perfusion. It is ensured by restricted fluid resuscitation to a volume sufficient to maintain a radial pulse. In polytrauma patients with head injuries, the importance of maintaining cerebral perfusion pressure is well recognized, consequently permissive hypotension is currently contraindicated in this setting.
- b. Haemostatic resuscitation is achieved by very early use of blood and blood products as primary resuscitation fluids with the aim of treating acute traumatic coagulopathy and at the same time prevent the occurrence of dilutional coagulopathy [10]. Although all three aspects of lethal triad are important, rapid treatment of coagulopathy is now recognized as central to improving outcome. It includes the administration of fresh frozen plasma, platelets, recombinant factor VIIa, cryoprecipitate, tranexamic acid and calcium. The high prevalence and severe impact of coagulopathy requires prompt treatment. Since the commonly available diagnostic tests are inappropriate in guiding treatment of trauma patients because of delay in getting their results which have poor sensitivity, the decision to initiate clotting factor replacement is clinical.

In patients expected to require massive transfusion, the US and British military practice is to give fresh frozen plasma and packed red blood cells in a 1:1 ratio. During the recent conflict in Iraq, it was found that there was a 46% reduction in mortality when FFP:PRBC ratio was 1:1 rather than the conventional 1:8 ratio [11].

Factor VIIa is an important component of coagulation cascade which is known to enhance local haemostasis at the trauma site. Randomized controlled trials of recombinant factor VIIa have shown a significant reduction in blood transfusion requirement in patients with blunt, but not penetrating trauma [12].

Conclusions

The exsanguinating trauma patient who requires massive transfusion incurs the greatest risk for the multifactorial interactions between acidosis, hypothermia, and coagulopathy. There continues to be an ongoing challenge to identify better predictors of outcome, improved means of resuscitation, greater understanding of physiologic derangements, and better timing to institute damage control.

Conflict of Interest

None identified

References

1. Feliciano DV, Burch JM, Spjut-Patrinely V et al. Abdominal gunshot wounds: an urban trauma center's experience with 300 consecutive patients. *Ann Surg* 1988; 208: 362-8.
2. Webster DW, Champion HR, Gainer PS. Epidemiologic changes in gunshot wounds in Washington DC 1983-90. *Arch Surg* 1992; 127: 694-7.
3. Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. *Ann Surg* 1983; 197: 532-6.
4. Rotondo MF, Schwab W, McGonigal MD et al. Damage control: an approach for improved survival in exsanguinating penetrating abdominal injury. *J Trauma* 1993; 35: 375-83.
5. Surface ship survivability: Navy war publications (NWP) 3-20.31 Washington DC: Department of Defense 1996.
6. Sugrue M, D'Amours SK, Joshipura M. Damage control surgery and the abdomen. *Injury* 2004; 35 : 642-8.
7. Burch JM, Ortiz VB, Richardson RJ et al. Abbreviated laparotomy and planned reoperation for critically injured patients. *Ann Surg* 1992; 215: 476-83.
8. Asensio JA, McDuffie L, Petrone P, Rhee JD et al. Reliable variables in the exsanguinated patient which indicate damage control and predict outcome. *Am J Surg* 2001; 182 : 743-51.
9. Holcomb JB, Watts S, Hodgets T et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma* 2007; 62 : 307-10.
10. Kirkman E et al. A proactive approach to the coagulopathy of trauma: the rational and guidelines for treatment. *JR Army medical corps* 2008; 153: 302-6.
11. Borgman MA, Spinella PC, Perkins JG 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.
12. Boffard KD, Riou B, Warren B et al. Recombinant factor VIIa as adjunctive therapy for bleeding control in severely injured trauma patients: two parallel randomised, placebo controlled, double blind clinical trials. *J Trauma* 2005; 59: 8-15.