Chapter 10

Damage Control Surgery

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Abstract

Objective: The basis of damage control surgery rests on quick control of life-threatening bleeding, injuries, and septic sources in the appropriate patients before restoring their physiological reserves as a first step followed by ensuring of the physiological reserves and control of acidosis, coagulopathy, and hypothermia prior to complementary surgery.

Material and methods: Knowing when to perform damage control surgery will increase the likelihood of survival. There are three main criteria that are important in the selection of patients: (1) critical physiological factors, (2) complex injury causing the loss of physiological reserves, and (3) other conditions in trauma patients. Acidosis, acquired coagulopathy, and hypothermia (death triangle/the lethal triad) which are among critical physiological factors come to the fore in patient selection. In patients predicted to undergo damage control surgery, a replacement with crystalloids is applied after establishing a wide vascular access before reaching the hospital with the purpose of maintaining acceptable vital functions until reaching the hospital.

In the rapid/primary surgery stage, the purpose is controlling bleeding and contamination. With the purpose of controlling bleeding and hemostasis, packing, clamping, ligation, and shunting procedures are applied to the four quadrants or a balloon catheterization is done. Following hemorrhage control, the colon and intestines are examined. Primary suturation, simple resections, closed absorbent systems, and external drainage are preferred for controlling contamination. However, reconstructive surgeries, stoma forming, and nutrition ostomies are not applied in this quick laparotomy. Then, abdominal closure (temporary abdominal closures; TAC) is done. In the second stage of damage control surgery (resuscitation), patients are taken into an intensive care unit for a period of 24–48 h for the enabling of aggressive resuscitation and patient monitoring. The main objective here is the elimination of problems caused by the acidosis, coagulopathy, and hypothermia triangle. Following 24–48 h of resuscitation after primary surgery in intensive care, planned definitive surgery is

performed (the third stage of damage control surgery). The packing materials are carefully removed. After all injuries are detected and any hemorrhages are stopped, complementary gastrointestinal repair (such as resections and anastomoses) is done and if it is not necessary, then ostomy and the opening of enteric feeding tubes are avoided. Closed system drainages and a nasoenteric feeding tube are placed if necessary. If abdominal closure cannot be fully done, temporary abdominal closure is done in the fourth stage.

Results: After damage control surgery procedures, there was an improvement in survival rates.

Conclusion: Damage control surgery and damage control management of the patient are important for improved survival rates and success of treatment before the lethal triad occurs deeply.

Keywords: damage control surgery, trauma, abdominal injury, sepsis, death triangle

1. Introduction

Before damage control surgery was defined, classic surgical procedures applied regardless of the physiological condition of patients would produce high rates of mortality. Prolonged intervention for definitive surgical procedures would result in the depletion of the patient's reserves and result in a process deadlier for the patients [1]. Although damage control surgery was initially planned for use on patients with severe abdominal trauma, today severely septic patients who undergo surgery are also included in this group [2, 3]. Damage control surgery continues to develop during the quarter-century period in which it was defined.

For the first time in the 1980s when the treatment principles were defined, Stone et al. stated that they reduced the high mortality rates by one-third [4]. Burch et al. [5] in an ongoing process mentioned the packing procedure in liver injury. With the start of the process, Rotondo et al. [6] mentioned "Damage Control Surgery" for the first time in 1993, and in their publication in which they applied damage control procedures to patients with major abdominal visceral and vascular injury, they stated that there was an improvement from 11 to 77% in survival rates [6].

In fact, the basis of damage control surgery rests on quick control of life-threatening bleeding, injuries, and septic sources in the appropriate patient before restoring their physiological reserves as a first step followed by ensuring of the physiological reserves and control of acidosis, coagulopathy, and hypothermia prior to complementary surgery [7]. Patients are monitored in intensive care units with the purpose of ameliorating this deadly triad and afterwards, their complementary surgeries and abdominal closing procedures are performed [1, 7].

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2. When should damage control surgery be done?

Knowing when to perform damage control surgery will increase the likelihood of survival. There are four main topics that are important in the selection of patients: (1) critical physiological factors, (2) complex injury causing the loss of physiological reserves, and (3) other conditions in trauma patients (**Table 1**) [7].

1. Critical physiological factors
    - **a.** Hypothermia (<35°C)
    - **b.** Acidosis (pH < 7.2 or base deficit > 8)
    - **c.** Coagulopathy
        - **i.** Nonmechanical bleeding
        - **ii.** Prolonged PT
        - **iii.** Thrombocytopenia
        - **iv.** Hypofibrinemia
        - **v.** The need for massive transfusion (the need for more than >10 units of red blood or the need for body fluid replacement)
    - **d.** Prolonged time for definitive surgery (>90 minutes)
    - **e.** Hemodynamic instability or pre-existing hypoperfusion
2. Complex injury associated with the loss of physiological reserve
    - **a.** High-energy blunt trauma
    - **b.** Multiple penetrant injuries
    - **c.** Visceral injury combined with major vascular trauma
    - **d.** Injuries passing through body cavities (closed head trauma, major vascular injury, and pelvic trauma)
3. Other conditions in trauma patients
    - **a.** Injuries that are better treated by nonsurgical means such as hepatic or pelvic injury corrected with angiographic embolization
    - **b.** Variations in physiological reserve (the elderly, those with a large number of comorbidities, and athletes)

Table 1. Indications for patient selection for damage control surgery [7].

Acidosis, acquired coagulopathy, and hypothermia (death triangle/the lethal triad) which are among critical physiological factors come to the fore in patient selection. There is a multivariable relationship between these three basic conditions [1]. Also the hypotension that occurs emerges as an important parameter in patients on whom damage control surgery is being planned to be done.

High-energy blunt traumas that can lead to the depletion of physiological reserves, those with a large number of penetrating injuries, injuries where more than one compartment is affected, and injuries where visceral organ and vascular injuries have occurred together are indicators for damage control surgery [7].

In patients predicted to undergo damage control surgery, a replacement with crystalloids is applied after establishing a wide vascular access before reaching the hospital. The main goal of replacement therapy, especially applied to patients whose transportation to the hospital will be prolonged, is to maintain acceptable vital functions until reaching the hospital [8–10].

3. The death triangle (the lethal triad)

It consists of hypothermia, acquired coagulopathy, and acidosis and was defined for the first time by Burch et al [5]. This condition gives rise to the depletion of physiological reserves and to life-threatening consequences (**Figure 1**).

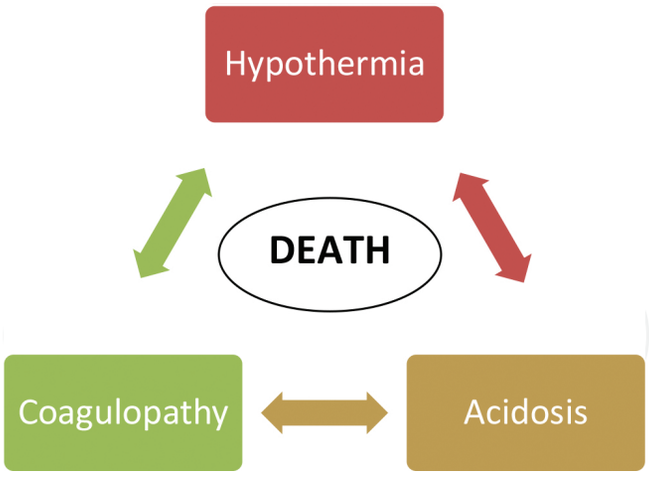

Figure 1. The death triangle (the lethal traid).

3.1. Hypothermia

Extensive injury as well as the resuscitation wherein we performed massive transfusion also contributes to its formation. Tissue hypoperfusion due to serious bleeding occurs and deterioration of oxygenation occurs as a result. Consequently, hypothermia occurs [1].

In particular, the hypothermic condition continuing below 36°C for a long time (longer than 4 h) becomes clinically significant. Mortality rates rise up to 100% in patients with multiple injuries and whose body temperature is below 32°C. The decrease in oxygenation and in tissue perfusion due to bleeding in particular plays a role in its formation [11].

If hypothermia persists, it leads to cardiac arrhythmia, decrease in cardiac output, increase in systemic vascular resistance, and a leftward shift in the oxygen dissociation curve in the long term. It also leads to the impairment of the immune system and its suppression as a result of this [12].

It also deepens acquired coagulopathy, which is another important issue (with a decrease in the activation of coagulation factors, platelet dysfunction, impairment of endothelial permeability, and stimulation of the fibrinolytic system). This results in uncontrolled bleeding.

3.2. Coagulopathy

The balance between bleeding and bleeding control mechanism is disrupted due to trauma. Although there are many underlying factors, massive transfusion and hypothermia are important.

Especially in hypothermic patients, it leads to platelet dysfunction by disrupting the interaction between von Willebrand and the platelet glycoprotein 1b-IX-V complex. A decrease in the metabolic rate of coagulation factors occurs below 35°C [13].

Massive blood transfusions lead to hemodilution and the aggravation of coagulopathy and acidosis due to this.

Although prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen levels help, clinical suspicion is essential for diagnosis. In particular, extensive hemorrhages not due to surgical causes (such as from injuries, serosal surfaces, and the skin) help in making a clinical diagnosis [1].

3.3. Metabolic acidosis

In trauma patients, anaerobic respiration increases and lactic acidosis arises due to prolonged hypoperfusion. It gets aggravated with multiple blood transfusions, aortic clamping, and insufficient myocardial functioning. As it gets aggravated, it increases coagulopathy and due to this also hypothermia [1].

4. Stages of damage control surgery

4.1. Stage I (rapid/primary surgery)

Rapid surgery is applied with the purpose of controlling bleeding and contamination. The abdomen is entered with a midline incision extending from the xiphoid to the symphysis pubis. With the purpose of controlling bleeding and hemostasis, packing, clamping, ligation, and

shunting procedures are performed to the four quadrants or a balloon catheterization is done [14].

Following hemorrhage control, the colon and intestines are examined with the aim to prevent contamination. If perforation is observed, contamination is tried to be prevented by primary suturing and connecting or with a stapler. If the injured small intestine loop is below 50%, a simple single resection can be applied. Ensuring continuity of the bowel is not in the foreground. In the case of a biliary or pancreatic injury, closed absorbent systems and external drainage procedures are preferred. However, reconstructive surgeries, stoma forming, and nutrition ostomies are not applied in this quick laparotomy [1, 15].

Before the abdomen is closed, the inside of the abdomen is washed with warm solutions.

Then, abdominal closure (temporary abdominal closures; TAC) is done with the Baker technique, which today has taken the place of methods like the Bogota bag and clamping of the skin. Plastic-coated abdominal covers are laid in such a way as to protect the visceral tissues beneath and closed absorbent systems are laid on this plastic cover at the level of subcutaneous tissue. Meanwhile, the skin is protected. Ready kits are available for this technique (KCI V.A.C. and ABThera, Kinetic Concepts, Inc., San Antonio, TX; Renasys systems, Smith & Nephew, Inc., St. Petersburg, FL). In this way, the tension that causes abdominal compartment syndrome is reduced [7, 15].

In a septic abdomen, primary surgical treatment mainly focuses on controlling the contamination. To this end, resections and drainages are carried out. According to the source (hollow organ injury, pancreatic injury, or hepatobiliary injury), a wide source control can be ensured with a vacuum-aided TAC as in a trauma [7].

4.2. Stage II (resuscitation)

Following primary surgery, patients are taken into an intensive care unit for a period of 24–48 h for the enabling of aggressive resuscitation and patient monitoring. The main objective here is the elimination of problems caused by the acidosis, coagulopathy, and hypothermia triangle [1, 7, 16].

First, it is planned for the patient to be brought close to the euvolemic state to ensure end-organ perfusion. For this purpose, the patient is given blood products (such as erythrocytes and fresh frozen plasma [FFP]). The shock of the patient gets tried to be ameliorated with fluid resuscitation. Following these, techniques such as artery catheterization and pulse artery catheterization are applied [1, 17].

The hypothermic condition of the patients is important because hypothermia can cause acidosis and coagulopathy to deepen. The control of hypothermia begins with the quick termination of the initial operation. The quick removal of wet covers from the patient, raising of the room temperature in the operation room, the use of warm resuscitation fluids and ventilator air and heat regulating covers help warm up the patient in the initial surgery room. The patient should be exposed to heat for about 4 h before being taken into the intensive care unit. Pleural lavage can be applied to patients whose body temperature does not rise despite

the methods applied. If the body temperature continues to be low, continuous arteriovenous heating can be applied [6, 16].

Coagulopathy is the goal as a secondary objective. For this purpose, blood products and resuscitation are planned for the patient. In the first 24 h, replacement is applied to the patient according to the rule of 10s (10 units for erythrocyte suspension, fresh frozen plasma, and platelets each) [1]. Replacement is continued until 1 PT period is 15 s and the platelet number is 100,000/mm^3. If fibrinogen levels are low, cryoprecipitate can be applied every 4 h. In life-threatening nonsurgical hemorrhages, recombinant factor VIIa can be applied [1].

If sufficient resuscitation is ensured and the patient is exposed to heat and oxygenation is ensured, then oxidative respiration increases and the acidosis is corrected by itself [17].

4.3. Stage III (definitive/complementary surgery)

Following 24–48 h of resuscitation after primary surgery in intensive care, planned definitive surgery is performed [7]. First, the packing materials of the patient are carefully removed. After which all injuries are detected and any hemorrhages are stopped. Complementary gastrointestinal repair (such as resections and anastomoses) is done and if it is not necessary, then ostomy and the opening of enteric feeding tubes are avoided. After the inside of the abdomen is cleaned, closed system drainages are placed if necessary. A nasoenteric feeding tube is placed if necessary, and if abdominal closure cannot be fully done, temporary abdominal closure is done [7]. Rapid closures, moderately rapid closures, and long-term closures are among temporary abdominal closure techniques (**Table 2**).

1.	Rapid closure
	a. Only skin closure
	b. Placing a protective element such as a Bogota bag
	c. Vacuum-aided abdominal covers
2.	Moderate closure
	a. Successive skin or fascial closure
	b. Placing of interpositional mesh
	c. Vacuum-aided abdominal covers
3.	Long-term closure (planned ventral hernia)

Table 2. Closure options for abdominal injuries [7].

In patients with a septic abdomen, the septic source is debrided and drainage is applied. However, in order to avoid problems like abdominal compartment syndrome, relaparatomies or a planned relaparotomy can be done [7].

4.4. Stage IV (abdominal wall closure)

It is applied on patients whose abdomens could not be closed in definitive surgery and on whom temporary abdominal closure techniques are applied [15] (**Figure 2**).

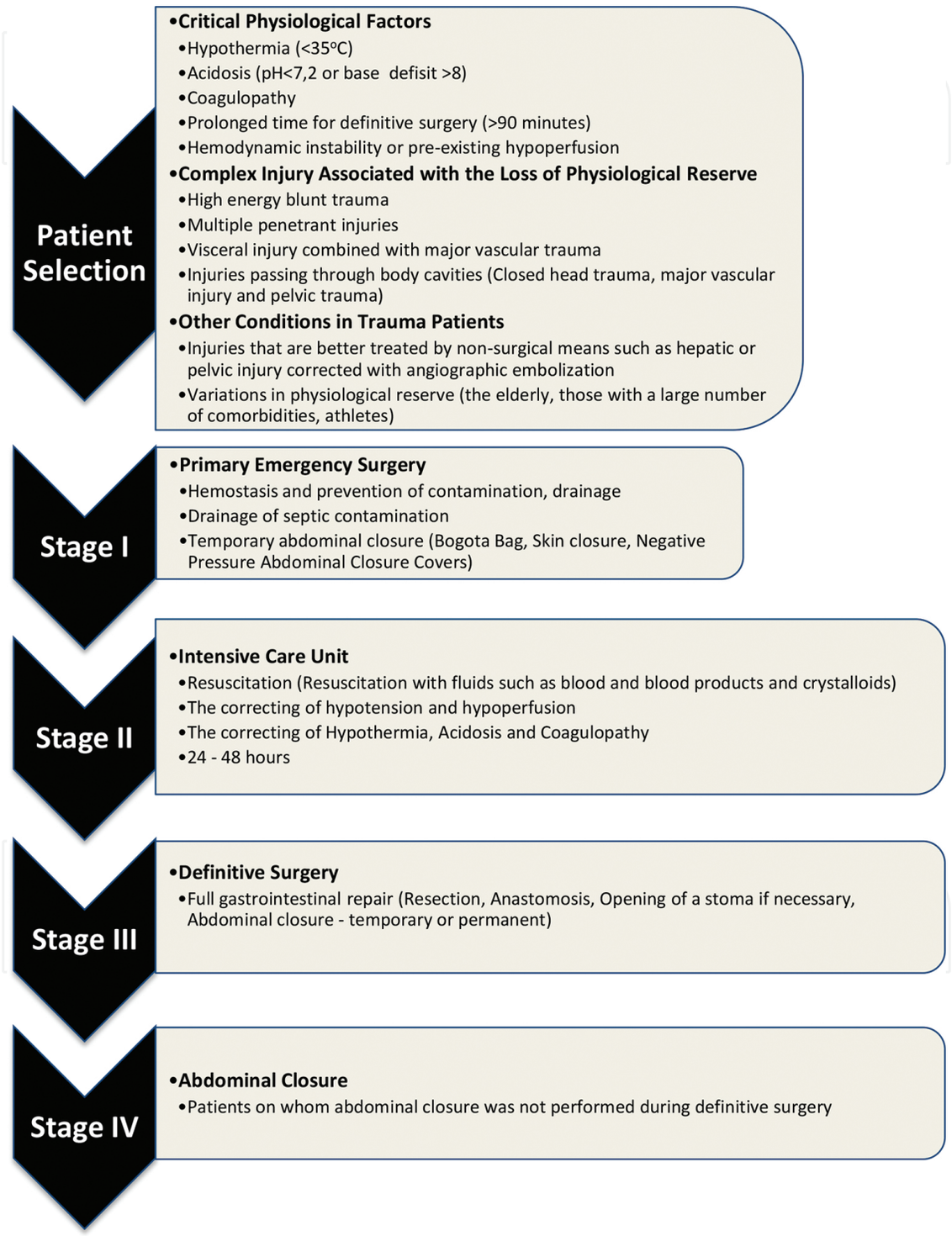

Figure 2. The stages of damage control surgery.

5. Complications

5.1. Abdominal compartment syndrome

In this condition, which arises especially after visceral swelling, hematoma, and packing procedures, a distended abdomen, increased peak airway pressure, insufficient ventilation, hypoxia, and oliguria or anuria are observed (**Table 3**) [18–20]. It can be measured with catheters placed in the bladder or catheters placed in the stomach. 35 cm H_2O is diagnostic for compartment syndrome. Rapid abdominal decompression is applied in the treatment [18].

Serious abdominal injuries
The dissolution of intestinal contents
Pressure in primary fastia closure
Abdominal packing applications for coagulation
Massive transfusion that causes intestinal edema and distension
Failures in control of hemorrhage resulting in increased acidosis and coagulopathy

Table 3. Predisposing factors for increased intra-abdominal pressure in damage control surgery [21, 22].

5.2. Enterocutaneous fistulae and wound site problems

It can be observed up to 15% in abdominal trauma patients, especially patients with sepsis. Manipulations in internal organs in particular (especially open abdomen patients with pancreatic and colonic injuries) play a role [1]. When enteroatmospheric fistulae form, it is difficult for them to close and for the wound to be protected.

Hernias are one of the most frequently encountered complications of damage control surgery because of the nature of surgical interventions with open abdomen. Intra-abdominal abscesses, surgical incisional infections, sepsis, massive blood transfusions, malnutrition, and hypoproteinemia are factors that would negatively affect the recovery of wounds and cause incisional hernia [23].

5.3. Intra-abdominal abscesses

Abscesses whose incidence increases especially with abdominal packing are observed between 10 and 70% in abdominal trauma patients. Reasons for abdominal abscesses are colonic injuries, presence of a foreign object in the abdomen for more than 24 h, inadequate drainage of seepage blood and other body fluids, and foreign objects that enter the abdomen during a penetrating injury [23, 24].

Just as it can be corrected by radiological methods, surgical drainage can also be applied.

5.4. Organ-specific complications

5.4.1. Liver

Liver-related complications (e.g., hemorrhage, hemobilia, arteriovenous fistula, pseudoaneurysm, biloma, bile leak, and abscess formation) occur in approximately 20–45% of patients [23–25]. In diagnosis of postoperative complications such as hepatic or perihepatic abscesses or bilomas, abdominal CT and ultrasound (US) were used [23–25]. Postoperative prolonged hemorrhage can be associated with coagulopathy [25–27]. Up to 85% of majority of complications following liver trauma can be successfully managed with nonoperative techniques (such as endoscopic retrograde cholangiogram, percutaneous drainage, and angiography) [28].

5.4.2. Pancreas

Pancreatic injury is seen in 2–3% of severe abdominal injuries. Mortality due to pancreatic injury is generally caused by accompanying injuries. The most common specific complication following pancreatic injury is a pancreatic fistula (10–20%). Peripancreatic, subhepatic, and subphrenic fluid collections are commonly seen on US or CT after pancreatic trauma, and pseudocysts may occur as a result of undetected pancreatic duct disruption. Magnetic resonance cholangiopancreatography (MRCP) or endoscopic retrograde cholangiopancreatography (ERCP) provides accurate anatomical delineation of the duct injury [29].

Complications such as fistula, pseudocyst, and abscess can be treated with nonsurgical treatment by advanced radiological intervention and ERCP [29]. If this fails, operative interventions can be options for treatment of complications.

5.4.3. Colon, rectum, and small bowels

Anastomotic leakage is the most serious complication specific to intestinal surgery (2.9–15.3%) [30]. CT scan-guided percutaneous drainage should be performed in hemodynamically stable non-septic patients with a success rate of up to 80% [30].

5.5. Multiorgan failure(MOF) and acute respiratory distress syndrome (ARDS)

ARDS or MOF incidence ranges between 14 and 53%, depending on different series. In trauma patients, the risk factors of ARDS include the presence of sepsis, transfusion of more than 15 units of packed red blood cells in 24 h, pulmonary contusion, and long-bone fractures [4, 5, 31–33].

6. Conclusion

Trauma is a multisystemic and a multidisciplinary problem for physicians. Choosing the patient's appropriate treatment is the top goal of the physician. Damage control surgery and damage control management of the patient are important for improved survival rates and success of treatment before the lethal triad occurs deeply.

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