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Review Article

Damage Control approach for managing severe maxillofacial trauma- A review

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ABSTRACT

Aim- severe maxillofacial trauma injuries expose patients to life threatening complications. These situations require rapid action in order to save the patient from "the lethal triad", hypothermia, acidosis and coagulopathy. Here we present an insight on protocols of Damage control surgery, to be followed in such situations. A comprehensive online database search was done and articles were critically reviewed, on DCS strategies and their outcomes.Damage control surgery is a rapid and effective approach to stabilize physiological and biochemical state in severely exsanguinating patients. Focus to save life, allows for considerations in slight morbidity. Further studies are advised to promote better understanding and its application.

Introduction

During the last two decades, advancement in pre hospital care and rapid mobilization, has ensured in expedient arrival of severe trauma patients in primary health centers. Secondly, advent of automatic weapons and ballistic missiles in modern warfare, have resulted in massive tissue destruction and multiple penetrating wounds, leading to severe exsanguination[1,2]. The traditional approach in these patients is not effective and increased incidence of mortality has been observed, despite anatomic repair. This occurs due to the combined effect of acidosis, hypothermia and coagulopathy ("the lethal triad")[3] and thus led to the emergence of "damage control"[4,5] strategy in the management of injured patients. Borrowed from the United States Navy it represents " the capacity of a ship to absorb damage and maintain mission integrity"[6]. In surgery[7], it refers to physiological and biochemical stabilization of patients by rapid control of hemorrhage, contamination, temporary wound closure and resuscitation to normal physiology in ICU, followed by subsequent re-exploration and definitive anatomical and functional repair. The principles of damage control surgery have been traditionally applied in abdominal trauma[8], as early as the American civil war. As surgical care evolved after the second world war, a shift towards single "definitive" surgery took place, showing good results in gunshot wounds from small caliber weapons but as more lethal and higher-velocity weapons became commonly used, the concept of surgery was revisited and DCS was adopted and better survival rate of patients were reported[9,10]. Recently the DCS concept has expanded to maxillofacial and neck trauma patients[11,12,]. The aim of

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this review is to systematically summarize the literature, and discuss the four stages of DCS, in the management of maxillofacial trauma.

Methods-

Search strategy

A literature search was performed using web-based on-line databases (PubMed, ISI Web of Science, Medscape, Google Scholar and Cochrane Library), and hand-searches of major journals, reference texts and published abstracts. For web-based on-line searches the following key words were used to identify relevant publications: "Damage Control Surgery" in maxillofacial trauma. The searches were confined to English language literature.

Article evaluation

Articles identified by on-line and hand searches were evaluated using a set of inclusion and exclusion criteria. The inclusion criteria's are as follows

Literature reporting on damage control surgery with combat-related maxillofacial trauma as part of a general discussion.

Literature reporting on damage control surgery with a specific focus on combat- related maxillofacial trauma.

Exclusion criteria

1. Literature reporting on damage control surgery not involving maxillofacial trauma.
2. Literature reporting on non-surgical management under the umbrella term of "damage control" procedures.

Discussion-

Severe maxillofacial trauma, can occur in various military and civilian environment. Civilian trauma is seen, mostly through road traffic accidents, falls, inter personal violence, sports and occupational injuries, which causes blunt trauma, resulting in closed or compound fractures, soft tissue lacerations and mild to moderate penetrating injuries. Trauma sustained in military combat or terrorist attacks, inflicts complex lacerations, comminuted fractures, high degree burns, profuse bleeding and contaminated wounds with extensive tissue loss[13]. These patients face several physiological challenges because of rapid hypovolemia, which leads to hypothermia, coagulopathies and metabolic acidosis. This lethal triad is extensively documented in literature and each component is discussed below.

Pathophysiology-

Hypothermia- is defined as a core temperature less than 35°C for more than four hours and occurs due to shock, prolonged exposure and dilution caused by large units of crystalloids and blood products infusion. Hypothermic patients are predisposed to arrhythmias, reduced cardiac output, increased systemic vascular resistance and left shift of the oxygen-hemoglobin saturation curve. Also it exacerbates coagulopathies by dysfunction of platelets and clotting cascades[14]. Hypothermia is often aggravated during initial hospitalization, by removing clothes of the patient and exposure, to evaluate systemic injury. Emergency room temperature is 15° C, which results in significant temperature loss[15]. This can be minimized by keeping the emergency operating room at 27° C.

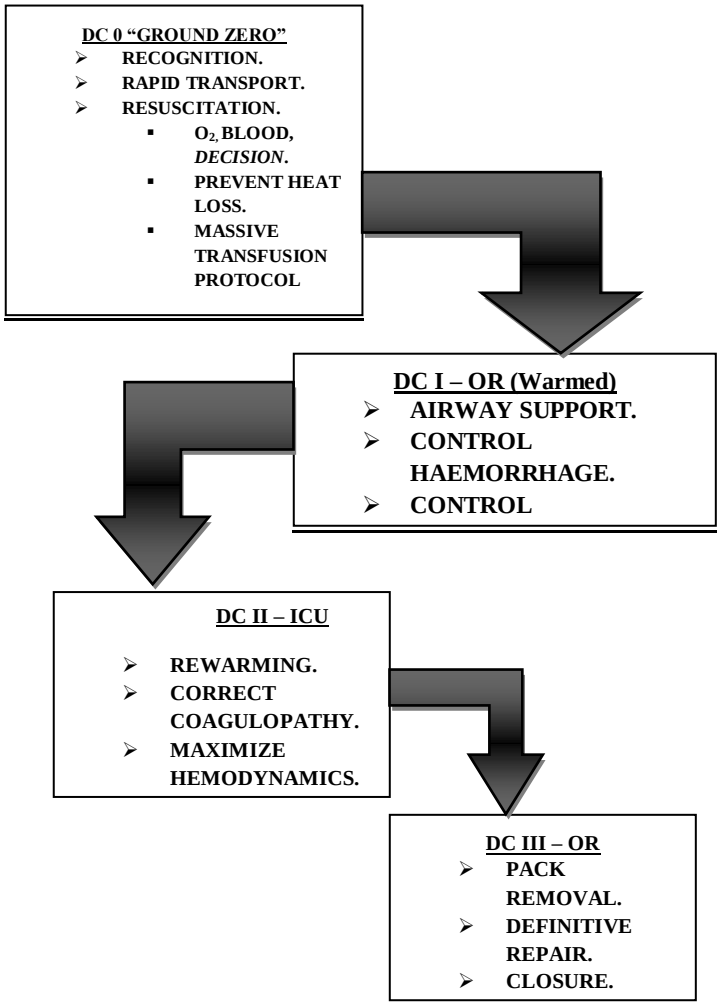

Fig. 1. The four phases of Damage control, from Johnson, Graclas, Schwab et al. J Trauma, 2001

Acidosis- hypoperfusion of tissues is associated with a shift of aerobic to anaerobic metabolism and production of lactic acid. Immediate measures should be taken, to limit the degree of acidosis by, controlling hemorrhage, adequate oxygen delivery through resuscitation with blood products. Urine output and lactate clearance[16] should be monitored and oxygen saturation as well as gastric pH should be regularly measured. The main goal is to restore patient's hemodynamics.

Coagulopathy- is multifactorial in origin, hypothermia causes platelet dysfunction and thereby affecting the initiation and propagation of platelet aggregation, as well as thromboxane B_2 production. Clotting factors are diluted by excessive resuscitation, causing prolonged coagulation. Hypothermia also leads to increased PT and PTT[17,18].

Rotondo and Schwab[8] coined the term "damage control" and outlined the parameters of performing three staged approach in trauma patients. DC I, consists of assessment of airway patency and breathing support, control of bleeding and contamination and abbreviated wound closure. DC II consists of ICU resuscitation, hemodynamic stabilization and tertiary examination to access all injuries. DC III consists of re-exploration and definitive repair. Recently Johnson and Schwab[19], introduced a fourth part "Damage control Ground Zero", it represents early interventions in pre hospital setup. (Fig. 1) .

Damage Control – Ground Zero

It refers to early recognition of injury severity, in prehospital or casualty setup. Protocols to be followed are notification to anesthetist, operating room and blood bank, control of airway and immediate resuscitation to restore red cells and plasma volumes. Emphasis should be on minimizing the emergency room stay and shift the patient to operating room. Throughout this period warming of the patient is advised to control hypothermia.

Damage Control – I

Initial assessment should be done to determine whether the airway is patent and breathing is adequate. The oral cavity is inspected under proper

Table 1 Scores for massive transfusion

TASH score	**1. SBP: < 100 mmHg = 4 pts, < 120 mmHg = 1 pt**
(0–28 pts, Increasing TASH-scores were	**2. Hb: < 7 g/dl = 8 pts, < 9 g/dl = 6 pts, < 10 g/dl = 4 pts, < 11 g/dl**
= 3 pts,	
associated with an increasing probability	**< 12 g/dl = 2 pts.**
for MT requirement)	**3. intra-abdominal fluid : 3 pts**
	4. complex long bone and/or pelvic fractures: AIS 3 or 4 = 3 pts, AIS
5 = 6 pts	
	5. HR: > 120 bpm = 2 pts
	6. BE: < -10 mmol/l = 4 pts, < -6 mmol/l = 3 pts, < -2 mmol/l = 1 pt
	7. gender: male = 1 pt
ABC score	**1. penetrating mechanism: no = 0 pt, yes = 1 pt**
(0–4 pts, a score of 2 or greater was	**2. SBP < 90 mmHg: no = 0 pt, yes = 1 pt**
used to predict the need for MT)	**3. HR > 120 bpm: no = 0 pt, yes = 1 pt**
	4. positive FAST: no = 0 pt, yes = 1 pt

TASH trauma associated severe hemorrhage, SBP systolic blood pressure, pts points, pt point, Hb hemoglobin, AIS abbreviated injury score, HR heart rate, bpm beats per minute, BE base excess, FAST focused assessment with sonography for trauma, MT massive transfusion

lighting and any blood clots, loose or fractured teeth, foreign bodies, dirt and mucus are removed. Crepitations on palpation of neck are suggestive of laryngeal trauma or pneumomediastinum. Diminished breath sounds on auscultation may occur due to pneumothorax, hemothorax or flail chest. Wheezing and dyspnea imply lower airway obstruction, agitation and cyanosis represents hypoxia. Chin-lift and head tilt maneuvers, open the airway, but should be avoided in patients with cervical spine injury. Jaw thrust, while maintaining cervical spine allignment is advised in such

Tranexamic Acid	within 3 h of injury loading dose 1 g over 10 min, followed by 1 g over 8 h
Ionized Calcium Levels	maintain in normal ranges during MT
Plasma : RBC	at least 1:2 (preferably 1:1) maintain Hb levels: 7–9 g/dl maintain coagulation parameters (repeated monitoring of PT, aPTT, fibrinogen levels, platelets count, viscoelastic testing) in normal ranges during MT
Fibrinogen	3–4 g administer in case of thromboelastometric signs of a functional fibrinogen deficit or a plasma fibrinogen level of less than 1.5 to 2 g/l
Platelet Count	50 × 109/l if ongoing bleeding and/or TBI: 100 × 109/l initial dose 4–8 single platelet units or 1 aphaeresis pack
Blood Pressure	SBP: 80 to 90 mmHg until hemorrhage control (no TBI) if severe TBI (GCS ≤ 8) MAP ≥ 80 mmHg consider rFVIIa if major bleeding and traumatic coagulopathy persist despite maximal attempts to stop bleeding in case of pre-trauma therapeutic

Table 2 Hemostatic/Hemodynamic resuscitation following major trauma

MT massive transfusion, Hb hemoglobin, PT prothrombin time, aPTT activated partial thromboplastin time, TBI traumatic brain injury, SBP systolic blood pressure, MAP mean arterial pressure, GCS Glasgow Coma Scale, rFVIIa recombinant activated coagulation factor VII, PCC prothrombin complex concentrate.

situations[13]. Oral and nasopharyngeal airway devices can be placed in spontaneously breathing patients. Nasal airway should be avoided in patients with midface or craniofacial fractures[20]. To maintain a definitive airway, placement of endotracheal tube is mandatory, this can be done by rapid sequence intubation, as most of the patients may have full stomach, bag-valve-mask ventilation may lead to gastric distension, regurgitation and risk of aspiration. To avoid this patient should be preoxygenated followed by administration of induction agents and neuromuscular blocking agents. An assistant should apply cricoid pressure to prevent regurgitation. In patients suffering from respiratory arrest, agonal respirations or deep unresponsiveness, immediate endotracheal intubation is indicated, termed as crash endotracheal intubation, its advantages are technical ease and fast placement but disadvantages are potential of increased intracranial pressure, emesis and aspiration. In conditions where intubation is not possible, airway is secured surgically by cricothyroidotomy or tracheotomy. In emergency cricothyroidotomy is preferred as it can be done rapidly and the risk of damaging the thyroid isthmus is lower[21]. Tracheotomy is an elective procedure, which has to be planned and performed in sterilized surgical setting.

Once the airway is established, bleeding should be controlled by anterior or posterior nasal packs, oropharyngeal packs and pressure dressings. Mandibular fractures are temporarily stabilized with bandages in maxilla-mandibular occlusion. Partially avulsed facial structures e.g ear, nose and lips, should be manipulated in proper position and fixed with dressings, for later attachement. Injuries to the parotid gland, duct and facial nerves should be cleaned cautiously, and planned for repair afterwards. Facial lacerations should be cleaned thoroughly with hydrogen peroxide and antibacterial agents, foreign body inside the wound should be

removed and packed. Severe cases of hemorrhage, should be controlled by vascular ligation, bipolar electrocoagulation or angiographic embolization[22,23]. External carotid injuries should be ligated and transposition of external to internal carotid artery is advised when the latter cannot be repaired[24]. Bleeding vessels in the neck should be controlled by direct pressure with gauze.

Damage Control – II

Restoration of blood volume in severely exsanguinating patients is done by massive transfusion, it is defined as transfusion of ≥ 10 RBC units within 24 hrs or transfusion of > 4 RBC units over 1 hr and replacement of > 50% of total blood volume (70 ml/kg in normal adult male, 60 ml/kg in females) with blood products within 3 hrs[25]. For early recognition and haemodynamic management of such patients, Trauma associated severe hemorrhage (TASH)[26] and ABC[27] scores are advised (Table 1).

Generally, in post trauma patients, acute traumatic coagulopathy[28,29] occurs, which is characterized by increased regulation of endothelial thrombodulin, which forms complexes with thrombin and promotes anticoagulation[30], as well as endothelial dysfunction-induced hyperfibrinolysis[31]. In such situations hemostatic resuscitation, should be considered, by utilization of blood and blood products in early treatment phase[32] (Table 2). Repeated monitoring of prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen levels and platelet count should be done. Viscoelastic testing e.g thrombelastography, helps in early detection of coagulopathy, but decision is mainly based on clinical condition of the patient.

Recently, studies conducted by Prospective Observational Multicenter Major Trauma Transfusion (PROMMT)[33], hypothesized that administration of RBC:plasma:platelets at 1:1:1 ratio was associated with decreased mortality rates in bleeding patients.

Damage Control – III

Timing for planned reoperation is critical , there is usually a window period of 36 to 48 hours after the trauma. In this phase definitive procedures are done, packs are removed, minor bleeding vessels are controlled, A complete exploration of other injuries is done and facial fractures are reduced and fixed with plates. Haemostasis is achieved and patient is shifted to ICU, vitals are monitored continuously.

Conclusion

Damage control surgery represents an important landmark in life threatening trauma situations and gives a chance to survive in otherwise hopeless conditions. The DCS emphasizes on patient survival, thus potential for morbidity and prolonged complicated course of life, should be accepted. But to truly evaluate the effectiveness of the concept, further research through randomized control trials, are advised and also to separate patients with isolated maxillofacial injuries from those of multisystem trauma when managed under DCS principles. A better understanding and cross-disciplinary training on this concept is recommended, “The persons who sweat more in peace, bleed less in war”.

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