

Review of Abdominal Damage Control and Open Abdomens: Focus on Gastrointestinal Complications

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Abstract

Massive trauma and abdominal catastrophes carry high morbidity and mortality. In addition to the primary pathologic process, a secondary systemic injury, characterized by inflammatory mediator release, contributes to subsequent cellular, end-organ, and systemic dysfunction. These processes, in conjunction with large-volume resuscitations and tissue hypoperfusion, lead to acidosis, coagulopathy, and hypothermia. This “lethal triad” synergistically contributes to further physiologic derangements and, if uncorrected, may result in patient death. One manifestation of the associated clinical syndrome is the development of intra-abdominal hypertension (IAH) and the abdominal compartment syndrome (ACS). The development of ACS is insidious. If not recognized and treated promptly, ACS leads to multi-system organ failure (MSOF) and mortality. Improved understanding of IAH and ACS led to the development of damage control (DC)/open abdomen (OA) as surgical decompressive strategy. The DC/OA approach consists of three basic management steps. During the initial step the abdomen is opened, hemorrhage/abdominal contamination are controlled, and temporary abdominal closure is performed (Stage I). The patient then enters Stage II – physiologic restoration with core rewarming, correction of coagulopathy and completion of acute resuscitation. After physiologic normalization, definitive management of injuries and eventual abdominal closure (Stage III) are achieved. The authors will provide an overview of the DC/OA approach, as well as the clinical diagnosis of ACS, followed by a discussion of DC/OA-associated complications, with focus on digestive system-specific complaints.

Key words

Damage control – open abdomen – intra-abdominal hypertension – abdominal compartment syndrome – gastrointestinal complications.

Abbreviations

ACS – Abdominal compartment syndrome; DC – Damage control; IAA - Intraabdominal abscess; AH – Intraabdominal hypertension; IAP – Intraabdominal pressure; ICU – Intensive Care Unit; MSOF – Multi system organ failure; NPWT – Negative pressure wound therapy; PVH – Planned ventral hernia.

Introduction

The term “damage control” reportedly originated in the Navy and referred to a strategy designed to stop fire and flooding in combat situations so that a ship at risk of sinking can be salvaged [1]. Damage control (DC)/open abdominal (OA) approach, although used since the 1940's, was popularized in the late 1980's as a method of salvaging critically ill patients with physiologic compromise due to massive hemorrhage [2, 3]. Although first described formally in civilian trauma population, DC/OA has been used in the military to facilitate prompt surgical control of bleeding/contamination and early evacuation of injured soldiers, with resultant improvement in survival rates [3-5]. The DC/OA approach has also been employed in critically ill non-trauma surgical patients [6, 7].

Massive trauma and abdominal catastrophes entail significant morbidity and mortality. In addition to the primary insult, a secondary injury associated with the release of inflammatory mediators often occurs during the acute resuscitation phase [8]. Subsequent cellular and end-organ dysfunction contributes to the development of acidosis, coagulopathy and hypothermia [8, 9]. This “lethal triad” exacerbates existing physiologic derangements and, if uncorrected, almost invariably results in mortality. One of the manifestations of this systemic syndrome is the development of intra-abdominal hypertension (IAH) and the emergence of abdominal compartment syndrome (ACS)

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[7, 8]. The clinical development of ACS is often insidious. If not recognized and treated promptly, ACS leads to multi-system organ failure (MSOF) and mortality. Improved understanding of pathophysiology associated with IAH/ACS supports therapeutic use of DC/OA to halt the progression of IAH, ACS, and MSOF [2, 6]. IAH/ACS contributes to systemic venous congestion due to elevated intra-abdominal pressure [10]. Although non-operative management of ACS has been described in highly selected patients, most experts agree that “mainstream” therapy for ACS involves prompt abdominal decompression [11].

The DC/OA approach consists of three principal management steps [12]. The first step is abbreviated surgery for rapid control of hemorrhage and/or abdominal contamination, with hemostatic packing and temporary abdominal coverage (Stage I). The patient then enters Stage II, which consists of physiologic restoration (i.e., core rewarming, correction of coagulopathy, completion of acute resuscitation). After physiologic normalization is achieved and all underlying causes of the ACS are identified/treated, definitive surgical restoration (i.e., re-establishing bowel continuity) and abdominal coverage (Stage III) are completed over variable amounts of time (typically, days to weeks). Due to severity of the underlying disease and unconventional character of the DC/OA approach, a number of complications can be seen in DC/OA patients. Many of these complications affect the digestive system.

Intra-abdominal hypertension and the abdominal compartment syndrome

Intra-abdominal hypertension and the ACS manifest clinically with tense, distended abdomen, progressive hypotension, oliguria, and increased airway pressures [13]. Early recognition of IAH/ACS is essential (Table I) [1]. There are three main subtypes of ACS. Primary ACS is associated with clearly definable abdominal pathologies that result in elevated intra-abdominal pressures (IAP). Secondary ACS is associated with IAP elevations due to extra-abdominal factors (i.e., massive fluid resuscitation for non-abdominal causes such as large body surface area burns). Tertiary ACS can be defined as recurrent ACS in the setting of pre-existing laparostomy or initial non-operative therapy performed for either primary or secondary ACS. The diagnosis of IAH/ACS requires high index of clinical suspicion and may be confirmed with bladder pressure assessments (Table II). Therapy must be instituted promptly. While surgery remains the mainstay of therapy for IAH/ACS, non-surgical treatment has been described (Table III). [11] Diagnostic/therapeutic approaches to IAH/ACS are shown in Fig. 1.

The surgeon should consider DC/OA in scenarios complicated by the presence of the “lethal triad”. The identification of coagulopathy (non-surgical bleeding from abdominal surfaces), hypothermia (body temperature $\leq 34^{\circ}\text{C}$), and acidosis (pH <7.2) should trigger the decision to abbreviate the surgical procedure (i.e., expedited hemostasis/

Table I. Summary of definitions, signs, symptoms, diagnostic approaches, and therapeutic interventions associated with intraabdominal hypertension and abdominal compartment syndrome. Adapted from World Society for Abdominal Compartment Syndrome (WSACS) Algorithms (<http://www.wsacs.org/algorithms.php>) and Consensus Guidelines (Malbrain et al. Intensive Care Med 2006;32:1722 and Malbrain et al. Acta Clin Belg Suppl 2007:44).

	Intraabdominal Hypertension (IAH)	Abdominal Compartment Syndrome (ACS)
Intraabdominal pressure measurements	Sustained or repeated elevation in intraabdominal pressure of >12 mmHg Grade I: IAP 12-15 mmHg Grade II: IAP 16-20 mmHg	Sustained IAP >20 mmHg associated with new organ dysfunction or failure Grade III: IAP 21- 25 mmHg Grade IV: IAP > 25 mmHg
Clinical signs	Overt signs may be absent Abdominal distention or tense abdomen may be present	Hypotension Increased Peak Inspiratory Pressure Decreased urine output
Diagnostic methods	IAP pressure measurements - High index of clinical suspicion - Bladder pressure (most common) - Gastric pressure - Rectal pressure	IAP pressure measurements with new-onset end-organ dysfunction or failure
Prevention	Limit resuscitation volumes Limit crystalloid vs colloid	Limit resuscitation volumes Limit crystalloid vs colloid Early recognition and treatment of IAH Avoid fascial closure with severe IAH or suspected ACS
Therapy	Maintain abdominal perfusion pressure (APP) >50 -60 mmHg Consider increased sedation Brief trial of chemical paralytics Supine position Consider bladder/gastric decompression Abdominal percutaneous catheter decompression	Surgical decompression for sustained, refractory IAH progressing to organ failure

Table II. Optimal technique for measuring bladder pressure as a surrogate of intra-abdominal pressure. Compiled from De Waele et al. *Intensive Care Med* 2006;32:455 and Fusco et al. *J Trauma* 2001;50:297.

• Supine patient positioning
• Zero transducer to iliac crest at midaxillary line
• Ensure relaxed abdominal musculature
• Instill 25 mL sterile saline into aspiration port of urinary catheter
• Measure via aspiration port, at end-expiration, 30-60 seconds after infusion

control of contamination) and plans for reoperation and definitive repairs after achieving physiologic stabilization [2, 5, 14, 15]. Whenever IAH/ACS is suspected intraoperatively, leaving the abdomen open may prevent IAH from progressing to ACS. In cases of secondary/tertiary ACS, the decision-making process is more difficult, especially when abdominal source of clinical deterioration is not immediately suspected.

Implicit to the DC/OA approach is the planned surgical re-exploration, typically 12-72 hours after the index operation. While some surgeons advocate temporary skin-only closure in the interim period, others cover the abdominal defect with various temporary dressings [1, 6, 16, 17]. Of note, “tertiary” ACS can occur among patients already undergoing DC/OA management whose temporary abdominal closure/dressing is too constrictive in relation to the underlying abdominal viscera.

Overview of abdominal closure and reconstructive techniques

Management of DC/OA is complex [6, 7, 18]. While DC/OA offers survival advantage in critically ill surgical/trauma patients, it may increase the risk of both early evisceration

and late hernia formation [19]. Open abdominal wounds can be temporized utilizing skin-only closure, sterile silastic membrane coverage, absorbable or non-absorbable mesh materials, negative pressure wound therapy (NPWT), and Velcro-like Wittmann patch [6, 20]. Definitive fascial closure should be pursued whenever possible [13]. Various wound care adjuncts may help facilitate fascial approximation/abdominal closure [17, 20, 21]. While some authors suggest that the Wittmann Patch and NPWT may be associated with improved rates of fascial closure [22], others utilize the “planned ventral hernia” (PVH) as the default pathway in cases where prompt primary fascial closure is not possible. Such PVHs are covered by split thickness skin grafts, with delayed fascial closure performed after the patient recovers from the acute illness [6, 23]. Occasionally, large hernia defects require extensive abdominal wall reconstructions utilizing abdominal component-separation techniques [23, 24]. Abdominal wall reconstruction is especially challenging in the presence of a fistula or stoma [7]. For this reason, ostomy creation should be avoided in DC/OA patients, and enteral anastomosis should be attempted during Stage II of DC/OA [1].

Complications related to the abdominal wall and abdominal cavity

Chronic ventral hernia

Chronic ventral hernia is very common in patients undergoing DC/OA, with a wide incidence range (13%-80%) depending on patient-specific factors and institutional patterns of practice (i.e., aggressive fascial repair versus PHV) [6, 19, 25]. The use of skin grafting for temporary coverage of the underlying bowel predictably results in ventral hernia [13]. Wound and intraabdominal infections/abscesses following closure of DC/OA also increase the risk of ventral hernia,

Table III. Strategies in non-operative approach to intraabdominal hypertension (IAH) and the abdominal compartment syndrome (ACS) [11]. Of note, non-surgical approach to IAH/ACS has to be viewed with caution and operative therapy should be considered at any time if non-response/failure of below-described techniques is noted. Adapted/modified from the World Society on Abdominal Compartment Syndrome (WSACS) at <http://www.wsacs.org/Images/medical%20management.pdf>.

Goal	Method	Comments	Strength of recommendation	Quality of evidence
Reduce IAP	Supine positioning	-	Weak	Low
	Increase sedation/analgesia	-	-	Insufficient evidence
	Chemical paralytics	Brief trial while utilizing other techniques	Weak	Low
Maintain organ perfusion	Target Abdominal Perfusion Pressure (APP) >50-60 mmHg by use of judicious resuscitation and/or vasoactive agents	APP = Mean arterial minus abdominal pressure ^a	High	Low
Decrease bowel wall edema/ hypervolemia	Hypertonic solution/ colloid resuscitation	-	Strong	Moderate
	Diuretics/hemodialysis	-	-	Insufficient evidence
Maximize available intraabdominal space	Bladder, gastric, and/or rectal decompression	-	-	Insufficient evidence
	Percutaneous catheter decompression of abdominal fluid collections	Commonly with US or CT guidance	Weak	Low

US-ultrasound; CT-computed tomography; ^aCheatham ML, et al. *J Trauma* 2000;49:621-626.

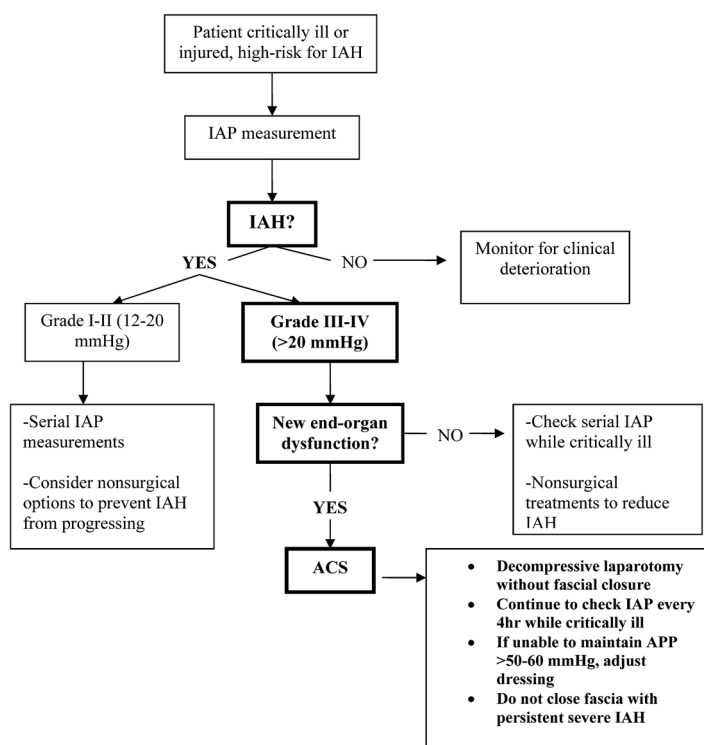


Fig 1. Algorithm outlining basic clinical approach to intraabdominal hypertension and the abdominal compartment syndrome. Adapted/modified from WSACS resuscitation algorithms at <http://www.wsacs.org/algorithms.php>. Legend: APP - Abdominal perfusion pressure; IAH-Intraabdominal hypertension; IAP-Intraabdominal pressure; ACS-Abdominal compartment syndrome.

wound dehiscence, and premature absorbable mesh failure [13, 25, 26]. Mayberry et al reported an 80% rate of ventral hernia with the use of absorbable mesh for bridging fascial defect [25]. In addition, chronic ventral herniation may lead to erosion of the overlying skin, which can contribute to both soft tissue (i.e., cellulitis) and prosthetic mesh infections. Large ventral hernias may be associated with prolonged recovery due to physical discomfort/loss of function. If mesh is to be utilized for bridging a fascial defect, many surgeons use absorbable or biologic materials in the presence of contamination/OA wound [27, 28]. In the presence of concomitant infection or fistula, the use of synthetic meshes is associated with high rates of infection, fistulization, and hernia formation. Definitive abdominal wall closure is associated with recurrent herniation in 5-10% of cases, depending on the reconstructive method and patient factors [13, 21, 24, 29, 30].

Infectious complications in the open abdomen

Surgical site infections and intraabdominal abscesses associated with DC/OA occur in as many as 83% of cases [1, 13, 31, 32]. The incidence of abdominal infection/abscess depends on the extent of traumatic injuries and/or bowel pathology (i.e., contamination, perforation, ischemic bowel) at the time of initiation of DC/OA, as well as the presence of any subsequent iatrogenic/non-iatrogenic complications. Major factors to consider include bile leak (incidence of 8-33%) [1] and enterocutaneous fistula (incidence of 2-25%) [1]. Surgical site infections and abdominal abscesses may also contribute to postoperative fascial dehiscence (reported in up to 25% of DC/OA patients) [1].

Wound infection management is particularly difficult in the presence of underlying mesh material. Few data are available to guide clinical management in patients with complex abdominal wall reconstructions. However, examining the experience with synthetic mesh infections after non-DC/OA ventral hernia repairs, the traditional approach consists of mesh removal, with prosthetic salvage attempted only in exceedingly difficult highly selected cases [33]. While there are reports of mesh salvage facilitated by debridement with/without partial mesh excision, drainage of any associated abscess, and antibiotic therapy, it is clear that extensive mesh infection without removal is almost invariably associated with therapeutic failure and long-term complications [34]. Because of the potentially devastating consequences of prosthetic infections, especially in clinical scenarios of fistula resection, ostomy reversal, or enterotomy, biologic “meshes” are recommended if native tissue component repair (the preferred option) is not possible [26, 34]. Human- and porcine-based fascial bioprotheses can provide satisfactory bowel coverage but have shortcomings [28]. Problems associated with the use of these biologic materials include acute infection, mechanical failure, and long-term formation of diastasis-like bulge in the area of the original fascial defect [28].

While the development of isolated intra-abdominal abscesses (IAA) may complicate DC/OA management, the diagnostic/treatment approach should be individualized. While some IAA are not accessible percutaneously and may require operative drainage due to their anatomic location, most IAA and fluid collections are amenable to percutaneous

drainage [35]. In one large series, partial success or cure was achieved in 90.8% of IAA managed percutaneously [35]. However, percutaneous drainage procedures did incur a relatively high complication rate (10.4% overall; 2.8% major complications) [35].

Sterile abdominal collections in open abdomens

Delineation between infected and non-infected fluid collections in DC/OA patients is difficult. It is important, however, to accurately determine if intra-abdominal fluid present on diagnostic imaging requires drainage or whether it can be safely observed. In general, all fluid collections with appearance suspicious for an abscess should be treated promptly with a combination of targeted antibiotic coverage, percutaneous drainage, and/or operative therapy. Another challenging aspect of managing postoperative fluid collections is their relatively high incidence in the surgical patient. In a sonographic survey of abdominal surgical patients, the incidence of localized abdominal fluid collections was 19% on postoperative day 4, 6% on day 8, and 2.5% on day 12 [36]. A study of abdominal computed tomography in 66 liver transplantation patients demonstrated loculated non-infected fluid collections in 20% of cases [37]. The incidence of such benign abdominal fluid collections may be even higher in DC/OA patients. For symptomatic non-infected collections, percutaneous therapy produces favorable outcomes in over two-thirds of cases [35]. However, percutaneous drainage of non-infected fluid collections may result in secondary infection and institutional antibiotic prophylaxis and routine aseptic technique should be followed during percutaneous drainage procedures.

Complications related to bowel and feeding access

Fistulae and the open abdomen

Enterocutaneous fistulae (ECF) are the second most common type of abdominal complications associated with DC/OA. The incidence of ECF in DC/OA patients varies between 5-19% depending on the presenting diagnosis/indication for DC/OA [6, 38]. A special problem unique to DC/OA is the entero-atmospheric fistula (Fig. 2) [6]. Management of this type of fistula is challenging due to lack of vascularized tissue coverage over the exposed bowel, which virtually precludes spontaneous healing. Continuous efflux of enteric contents, combined with chronic exposure of the viscera to air, contribute to elevated catabolic activity, protein loss, infection/sepsis, and high mortality. Although usually unsuccessful, sporadic cases of definitive fistula closure using fibrin glue and acellular dermal matrix have been reported [39]. It is important to limit the care of the OA to providers who are both familiar with wound topography and are experts in OA wound care. Adequate nutrition is crucial to wound healing and fistula closure.

Once entero-atmospheric fistulae form, NPWT may be helpful in “isolating” fistula contents from the rest of the wound [33]. The key to clinical approach in this scenario is adequate control of fistula effluent. In addition, NPWT may also increase eventual fistula closure rate [40, 41]. Surgical exteriorization/proximal diversion, though beneficial in highly selected cases, may be difficult in face of mesenteric

foreshortening due to associated soft tissue/bowel edema. A “floating stoma” has been described wherein the surgeon sutures the edges of the fistula to a plastic silo over which a stoma appliance is then placed [33]. Specialized wound drainage devices offer an alternative, but require extensive nursing support. Intubation of the fistula in setting of DC/OA is not recommended as it may make the fistula opening larger. Coverage of fistula with well vascularized soft tissue represents the most effective strategy for control and eventual healing, but does not guarantee fistula closure [42]. Resection of a chronic fistula after patient is stabilized and fistula-associated infection is controlled constitutes another option. Surgical or percutaneously placed feeding tubes should be avoided in the setting of active DC/OA therapy due to the

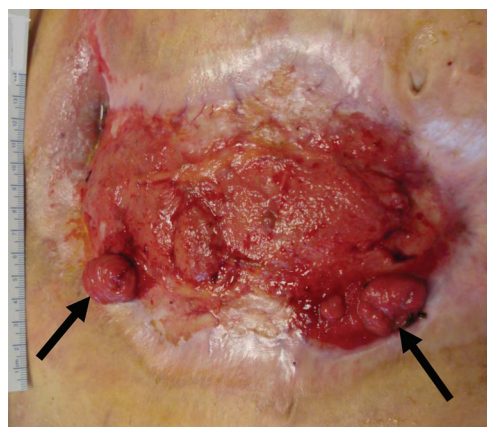


Fig 2. An example of a complex open abdominal wound featuring entero-atmospheric fistulae (arrows).

risk of fistula development at the site of the tube entry into the bowel [43]. It is prudent to wait until abdominal closure/bowel coverage has been accomplished before placing percutaneous or surgical feeding access [44].

Enterocutaneous fistula management is based on the overall patient status, fistula output quality/quantity, as well as the anatomic location (proximal/distal) and other fistula characteristics (i.e., presence of foreign body or length of the fistula tract) [45]. Much like the approach to DC/OA itself, the clinical care of fistulae consists of three phases: (a) patient stabilization – treatment of acute metabolic, hemodynamic, and infectious complications; (b) fistula investigation – delineating the anatomic characteristics including fistula location and tract length; and (c) treatment phase [45]. In the authors’ experience, proximal high-output fistulae rarely close without surgery while distal low-output colonic fistulae may close spontaneously with little therapy. It is important to document bowel patency distal to the fistula prior to surgical fistula takedown as the presence of untreated distal bowel obstruction will preclude favorable outcome of any such procedure.

Ileus and bowel obstruction

Ileus is a well-known gastrointestinal complication with multiple operative and non-operative etiologies (i.e., inflammatory and sympathetic responses, anesthetic

administration, mechanical disruption of normal peristalsis/anatomy, severe electrolyte abnormalities, and others) [46, 47]. Ileus in DC/OA scenarios likely has multifactorial etiology, with certain elements specific to the circumstances leading to the DC/OA approach. In particular, massive resuscitation, whether administered in the treatment of hemorrhage, sepsis, or other etiology of shock, may lead to profound bowel edema with subsequent gut motility dysfunction [48]. Avoiding excessive fluid resuscitation, up to and including the use of advanced hemodynamic monitoring devices and vasoactive agents, may help reduce iatrogenic tissue/bowel edema [49]. One interesting candidate therapy to help prevent ileus is the use of hypertonic saline during acute resuscitations (presumably via ameliorating resuscitation-induced intestinal edema) [50]. However, this therapy requires further investigation to identify appropriate dosage, timing, and suitable patient subsets.

Acute and subacute bowel obstruction in the setting of DC/OA (reported incidence, 2-21%) is most likely related to surgical adhesions [38, 51]. Long-term bowel obstruction is likely more common overall, but its incidence is poorly described. Regardless of the timing of post-DC/OA bowel obstruction, the initial therapy consists of bowel rest, fluid resuscitation with electrolyte replacement, and nasogastric suctioning [52, 53]. Due to the exceptional difficulty of operative approach in DC/OA patients with bowel obstruction (i.e., the presence of “frozen abdomen”) some have advocated prolonged nasogastric decompression with clinical observation in stable patients [54]. However, when signs of clinical deterioration (peritonitis, end-organ failure, hemodynamic instability) develop, operative intervention has to be undertaken regardless of the anticipated difficulty of adhesiolysis [53].

Enteral feeding and access in DC/OA patients

Despite significant/persistent bowel edema, resumption of gut function and safe feeding is possible in the setting of DC/OA [55]. Published studies report fewer infections, lower nutritional supplementation costs, and decreased fistulization rates when at least some enteral feeding is given during the DC/OA period [56, 57]. In the setting of a fistula, refeeding of the proximal effluent with/without administration of tube feeds into the efferent limb has been described and may be beneficial [58].

The presence of acute DC/OA constitutes a relative contraindication to placement of percutaneous/surgical feeding access [43, 44]. Some concerns exist regarding the relationship between early surgical feeding access placement in DC/OA patients and the risk of enterocutaneous fistula formation at the feeding access stoma site. While early enteral feeding has been shown to be of benefit to DC/OA patients, there is no compelling data to support early placement of surgical feeding access in these patients. Moreover, nasogastric and naso-duodenal tubes are suitable for providing adequate enteral feeding access in vast majority of patients with DC/OA. After abdominal closure, the risk of percutaneous endoscopic gastrostomy (PEG) placement should approximate that of other postoperative patients,

with overall success rates of 72-95% [44]. For patients able to undergo PEG placement but unable to tolerate gastric feeding, a conversion of PEG tube to a percutaneous jejunostomy (PEJ) device can be entertained, with evidence of decreased incidence of pulmonary aspiration [59]. With a dual-lumen PEG-PEJ (gastric/jejunal) tube, feedings can be administered via the jejunal port while the gastric port is placed to gravity drainage. Operative feeding access may be considered at the time of definitive abdominal closure in patients who are not candidates for or who failed percutaneous feeding access placement.

Hemorrhagic complications in damage control patients

Gastrointestinal bleeding associated with DC/OA therapy is usually encountered during the acute resuscitative (i.e., intensive care) stage of DC and can be broadly categorized as either post-surgical (i.e., anastomotic bleeding, mesenteric arterial hemorrhage) [60, 61] or as disease/treatment-related (i.e., “stress” gastric bleeding, pancreatic pseudoaneurysm bleeding) [62, 63]. Each of these primary groups can be further divided into intraluminal (i.e., “stress” gastric ulcer, anastomotic hemorrhage) [64, 65] or extraluminal bleeding (i.e., splenic vessel hemorrhage secondary to necrotizing pancreatitis) [66, 67]. Regardless of the source/cause of the bleeding, the initial clinical approach is similar. Early and adequate fluid resuscitation is the key, with focus on adequate vascular access, restoration of circulating blood volume and oxygen carrying capacity, correction of coagulopathy, and hemodynamic stabilization. Patient stabilization is then followed by a multi-modality approach – endoscopic control of bleeding [60, 68-70], percutaneous endovascular embolization [66, 71], and/or surgical therapy [72]. It is important to remember that late-stage OA are often characterized by the presence of severe adhesions and safe surgical exploration in search of the bleeding source may be effectively precluded in many cases. In such situations, interventional and/or endoscopic therapies may constitute the only viable options. For “stress” bleeding prevention, proton-pump inhibitors/H₂-receptor blockers are effective and should be utilized in critically ill patients [73]. Details of medical/endoscopic management of gastrointestinal bleeding are beyond the scope of this review, and the reader is referred to other sources for more information [60, 66, 68-71].

Special topics related to DC/OA management

Mortality attributable to open abdomens and related complications

Mortality associated with DC/OA is largely related to the underlying (primary) diagnosis that necessitated DC/OA therapy. In early published experiences, DC for trauma patients who were in extremis was associated with mortality of 42% [2]. Since then, DC/OA has evolved to include other indications, up to and including temporizing intractable elevations of intracranial pressure following trauma [6, 7, 74, 75]. Recently reported DC/OA-associated mortality rates are 17-31% [29, 31, 32]. Excluding the primary etiology that led to the DC/OA approach, common

factors that cumulatively contribute to DC/OA-associated mortality include systemic inflammatory response, MSOF, severe infection/sepsis from a variety of sources, chronic protein loss, enterocutaneous/enteroatmospheric fistulae, and a plethora of operative factors complicating any subsequent high-risk surgical procedures [26, 31, 76]. Mortality may be further elevated in the presence of pre-existing malnutrition, chronic co-morbid conditions, obesity, and advanced age [30, 31, 77].

The major cause of mortality during the initial hospitalization of DC/OA patients is MSOF [8]. This may be associated with either the primary cause that led to DC/OA (i.e., the ACS, hemorrhagic shock, abdominal sepsis) or a number of secondary causes (i.e., subsequent infections or cardio-pulmonary complications). Sepsis related to intestinal leakage or fistulization is a prominent cause of mortality in DC/OA patients [31]. In one DC/OA series, mortality was 14% in patients with an enterocutaneous fistula versus 6% in patients without a fistula [30].

Following initial hospital discharge, bacteremia/sepsis associated with indwelling catheters may contribute to increased mortality in DC/OA patients (i.e., bloodstream infections associated with parenteral nutrition administration or urinary tract infections associated with indwelling urinary catheters). During the restorative phase of DC/OA, patients are at risk for venous thrombosis, pulmonary embolism, respiratory failure/pneumonia, pressure ulcers, and various infections following long/complex procedures involving hostile abdomen [26]. Despite the high overall mortality risk, it is well-established that DC/OA techniques, when indicated and used appropriately, confer significant survival benefit [6, 7]. Moreover, a number of potential risk factors associated with increased mortality in DC/OA patients may be mitigated with appropriate prevention and clinical optimization.

Post-DC/OA disability and loss of productivity

DC/OA therapy is often associated with prolonged ICU/hospital lengths of stay, increased ventilator days, and prolonged physical inactivity [29, 31, 76]. All of these factors are associated with infectious and respiratory complications, deep venous thrombosis/pulmonary embolism, as well as significant physical deconditioning [6]. In addition, the loss of abdominal domain may worsen over time, further complicating the overall patient recovery and limiting abdominal reconstructive options. If post-DC/OA ventral hernia is complicated by the presence of a fistula, an added level of complexity is introduced, with potential for worsening malnutrition, debilitation, and the need for advanced wound care [13, 30]. Based on limited evidence, functional status in DC/OA patients seems to be dependent on several factors, including the size of the hernia, the presence of skin and subcutaneous tissue overlying the midline defect, and the presence of a fistula [29, 78]. Cheatham et al reported that up to 55%-78% of patients eventually returned to work after abdominal closure or reconstruction [79, 80]. However, other studies of patients with large chronic ventral hernias show persistent significant impairment of activity, productivity, and quality of life [81].

Management of the pancreas during damage control

In trauma, the proximity of the pancreas to key other abdominal structures results in high incidence of other severe associated injuries, producing a heterogeneous mix of clinical injury patterns [82]. Commonly seen are splenic, gastric/duodenal, renal, hepatic, and vascular injuries (including the aorta, inferior vena cava, celiac, superior mesenteric, splenic and renal vessels) [82-84]. Moreover, the propensity of pancreatic injury to result in complications such as leaks, local tissue destruction, as well as pancreatitis-like inflammatory syndrome, make clinical approach to these injuries especially unforgiving [82, 84]. While the heterogeneity of associated injury patterns, combined with the relative infrequency of pancreatic injuries make comparisons of management strategies difficult, there is some evidence to suggest that adequate pancreatic drainage in patients with pancreatic injuries who require abbreviated laparotomy may limit mortality [82, 84, 85]. In non-trauma applications, pancreatic DC/OA may be even more challenging than DC performed for pancreatic trauma [6, 86-88]. Mortality associated with DC/OA performed for pancreatitis has been noted to be as high as 40% [87]. This mortality is higher than the 20-31% mortality associated with surgical management of pancreatitis without DC/OA, likely reflecting the true severity of illness among patients with pancreatitis who require formal DC/OA approach [89]. In addition, pancreatitis patients require more operations per patient during their DC/OA management and fascial closure is significantly less likely in cases of DC/OA involving pancreatitis [6, 87].

Special aspects of damage control for non-trauma

The DC/OA approach has been increasingly applied to critically ill non-trauma surgical patients [2, 6, 7]. This logical extension of the DC/OA paradigm is based on physiologic similarities between severely injured trauma patients and surgical patients with abdominal sepsis, bowel ischemia, intra-abdominal hemorrhage, severe pancreatitis, and other abdominal emergencies [6, 7, 88]. The acute care surgery model, wherein the trauma/surgical critical care specialist evaluates, performs surgery, and manages critically ill non-trauma emergency general surgery patients is gaining popularity. Although the initial cause behind the "lethal triad" may differ, final clinical effects are similar to those seen in trauma DC/OA patients [7]. At first, the concept of planned re-laparotomy involved a "second-look" surgery in cases of major abdominal catastrophe. Early studies did not find significant mortality differences between planned and unplanned re-laparotomy [90-92]. However, not all of the patients in those studies underwent what we now consider DC/OA (i.e., leaving fascia open). Even in this early experience, certain subgroups, especially cases where source control was not achieved during the index operation and those with diffuse fecal peritonitis, had lower mortality rates with planned relaparotomy [92, 93].

Modern non-trauma DC/OA approach is similar to trauma DC/OA [6]. It consists of three phases, beginning

with an abbreviated laparotomy (Stage I) conducted to stop hemorrhage and/or control peritoneal contamination. Temporary abdominal closure is utilized. This is followed by the “resuscitative phase” in the ICU setting (Stage II). Subsequent to that, a series of re-laparotomies facilitates definitive surgical repairs during the “restorative phase” (Stage III) [13, 87]. Lower-than-expected mortality has been reported when DC/OA was utilized in critically ill general surgical patients [7].

Damage control in pregnancy

Damage control in the pregnant patient is exceedingly rare [94]. This strategy may be indicated in both trauma and non-trauma obstetric patient [95]. Non-trauma indications during the third trimester of pregnancy include abdominal pregnancy, spontaneous hepatic rupture, or postpartum hemorrhage [95]. Based on the scant data, trauma DC with abdominal packing during pregnancy may be associated with anomalies in the uteroplacental flow and poor fetal outcomes despite surgical abdominal decompression [96]. Additional maternal and fetal monitoring may be required in DC/OA pregnant patients due to added physiologic and hemodynamic complexity [96].

Retained surgical foreign bodies and damage control operations

Retained surgical foreign bodies (RSFB) are preventable surgical errors that can cause significant harm to the patient and carry serious professional and medico-legal consequences [97, 98]. The body’s reaction to RSFB has been classified as exudative or fibrinous. Exudative reactions typically present early and manifest with sepsis, foreign body erosion/migration, abscess (30% of cases), and/or fistula formation (20%) [97-99]. Fibrinous reactions tend to present late, and can be characterized by granuloma formation, presence of soft tissue mass, abdominal pain, and/or bowel obstruction [97].

Abdominal RSFB account for >50% of all RSFB [97-99]. While most RSFB cases involve elective procedures, approximately 30% of cases are associated with emergency procedures [97, 99]. In fact, emergency surgery is among independent risk factors for RSFB, which also include unplanned changes in the procedure and elevated body mass index [97, 98, 100]. By extension, emergent DC/OA laparotomies may carry increased RSFB risk [97, 98, 100]. Little data exist concerning RSFB in trauma, with one study showing an incidence of approximately 1.2-1.4/1,000 among operative trauma cases [100].

An accurate count of surgical instruments and sponges during the surgical procedure is crucial [99]. If the initial and final counts are discordant then a recount should be performed along with re-inspection of the surgical wound and/or radiography of the surgical field [97]. Of note, a correct surgical count is no proof of RSFB absence, and as many as 60-80% of cases involving RSFB are associated with correct counts [97, 99]. Although routine use of surgical field roentgenograms is not recommended, this practice may be beneficial in cases considered to be “high risk” for RSFB [97]. Because of the risk of inflammatory/infectious

complications associated with surgical packs left in place for >4 days, patients undergoing DC/OA may benefit from periodic abdominal plain films to identify any potential RSFB, as well as from the routine use of roentgenograms immediately prior to abdominal closure. Finally, surgical sponges tagged with radio-frequency identification may help detect tagged sponges prior to closure [97].

Conclusions

Multiple trauma and abdominal catastrophes are associated with significant morbidity and mortality. Associated systemic inflammatory processes, combined with large-volume fluid resuscitation, may lead to the development of acidosis, coagulopathy, and hypothermia. This “lethal triad” synergistically contributes to further physiologic derangement and, if uncorrected, patient death. Among manifestations of the associated clinical syndrome are IAH/ACS. If not recognized and treated promptly, ACS leads to MSOF and mortality. Improved understanding of IAH/ACS led to the development of DC/OA as surgical decompressive strategy. The DC/OA approach halts the progression of ACS/MSOF but is associated with a number of complications. Knowledge of these complications and the awareness of preventive strategies may contribute to improved outcomes in critically ill surgical patients undergoing DC/OA management.

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