INVITED REVIEW

Open Abdomen in a Critically Ill Patient

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Abstract

One of the damage control strategies used to avoid or treat abdominal compartment syndrome is "open abdomen (OA)," where the facial edges and the skin is left open, exposing the abdominal viscera. Although it reduces the mortality both in trauma and non-trauma abdominal complications, it does create a significant challenge in an intensive care setting, as it has physiological consequences that need early recognition and prompt treatment both in the intensive care unit and in the operating room. The article aims to review literature on "open abdomen," describe the challenges in such cases, and proposes a guideline for the intensivist in managing a patient with an OA.

Keywords: Abdominal compartment syndrome, Enteroatmospheric fistula, Negative pressure wound therapy, Open abdomen, Peritoneal sepsis, Primary or temporary abdominal closure.

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Introduction

Damage control surgery (DCS) techniques like open abdomen (OA), where the facial edges and the skin are purposefully left open, thereby exposing the abdominal viscera, are used in 10–15% of trauma laparotomies[1] Pancreatitis, abdominal trauma, massive or extensive burns, ruptured aortic aneurysm, sepsis, and retroperitoneal hemorrhage are some scenarios, where pressure-related end-organ dysfunction occurs due to increase in the volume of the intra-abdominal content manifesting in the form of hemodynamic, renal, ventilatory, and central nervous system compromise. The general indications for OA are damage control 40%, facilitate early second look 25%, multiple reasons 20%, excessive contamination 10%, and decompression to prevent or treat abdominal compartment syndrome (ACS) 5%.[2,3]

Although using OA strategy can reduce mortality,[3] it creates multiple challenges for the intensivist, as these patients are highly catabolic, loose a lot of fluid and proteins, suffer from nutritional insufficiency, and hemodynamic instability. OA also creates a challenge for the surgeon due to the development of an enteroatmospheric fistula (EAF), where the gastrointestinal content leaks into the OA field that can be catastrophic. The other surgical issues are ileus (13%), anastomotic leak (7%), fascial dehiscence (11%), and surgical site infection (19%).[4] Subsequently, the fascia retracts if it is not closed early and a ventral hernia develops.[5]

Ogilvie was the first to use this technique more than 80 years ago with the intent to allow the intra-abdominal infection to drain (principal of source control) to treat intra-abdominal sepsis.[6] The first international consensus conference (ICC) on OA was held in Milan in December 2014 to develop evidence-based guidelines to correctly identify the indications for OA, to choose the right technique for temporary abdominal closure (TAC), treat enteric complications, and subsequently close the abdominal wall effectively.[7] According to published guidelines of The Eastern Association for the Surgery of Trauma (EAST) practice management committee, level III evidence exists to support the use of the OA technique in trauma in the presence of acidosis (pH < 7.2), hypothermia (temperature < 35°C), and coagulopathy, where >10 units of red blood cells have been transfused or >6 L of crystalloids have been administered in 24 hours.[2,8,9]

Björck modified his classification of OA in 2016 (Table 1).[10]

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Abdominal Compartment Syndrome

Intra-abdominal pressure (IAP) is defined as the steadystate pressure present within the abdomen and is approximately 5–7 mm Hg in adult ICU patients. Intra-abdominal hypertension (IAH) is defined as sustained and persistent pathological increase in IAP >12 mm Hg and is graded from I to IV. When IAH is caused due to injury or disease in the abdominopelvic area, it is called primary IAH. IAH caused by conditions not originating in the abdominopelvic cavity is secondary to IAH. Abdominal compartment syndrome is defined as a sustained and persistent IAP >20 mm Hg [with or without an abdominal perfusion pressure (APP) <60 mm Hg] associated with

Table 1: Björck amended classification of open abdomen 2016[10]

Grade	*Description*	
I	A	Clean OA without adherence between bowel and abdominal cavity
	B	Contaminated OA without adherence/fixity
	C	Enteric leak, no fixation
II	A	Clean OA developing adherence/fixity
	B	Contaminated OA developing adherence/ fixity
	C	Enteric leak, developing fixation
III	A	Clean, frozen abdomen
	B	Contaminated, frozen abdomen
IV		Established enteroatmospheric fistula, frozen abdomen

new organ dysfunction.[11] Level I evidence whether one should resuscitate using this as an end point is not clear.[12]

According to the ICC on OA, empiric use of OA in trauma patients is indicated in the case of DCS for bleeding injuries requiring packing and planned re-exploration within a day or two (Grade II, LoE II) and if there is extreme visceral or retroperitoneal swelling or elevated bladder pressure after surgery (Grade II, LoE II), as these are the risk factors for ACS.[7] A pH <7.2, core temperature <34°C, estimated blood loss >4 L, blood transfusion >10 U of packed red blood cells, systolic blood pressure <70 mm Hg, lactate levels >5 mmol/L, base deficit >−6 in patients older than 55 years or >−15 in patients younger than 55 years, and/or prothrombin time >1.6 are indications of a patient who is physiologically fatigued and likely to develop ACS.[9,13] As these risk factors are also associated with increased mortality and morbidity, the surgeon should therefore perform an abridged procedure and leave the abdomen open.[13]

ACS develops in acute pancreatitis due to peripancreatic inflammation, visceral edema secondary to resuscitation, and ileus. Percutaneous drainage has less morbidity and mortality than decompressive laparotomy and should be performed only if there is persistent organ dysfunction and IAH, despite catheter drainage.[13,14] Although early relief in ACS reduces hospital length of stay and mortality, it is still controversial regarding which method to use.

Initial Non-surgical Management of Abdominal Compartment Syndrome

The WCACS recommends that a baseline IAP should be recorded if two or more risk factors for ACS are present. Daily serial monitoring of IAP should be done at least once in an 8-hour shift; however, the optimal frequency has not been recommended.[15] The gold standard for IAP measurement has been the intravesical technique using a Foley manometer U tube by instilling 25 mL of physiological saline in the bladder.

Hypertonic saline and colloids are preferred over isotonic fluids, and restrictive fluid strategies should be used for resuscitation to prevent the development of ACS.[16] Nonsurgical techniques that should be initiated are use of nasogastric and/or rectal tubes to remove the intraluminal contents, administering agents or enema that increases gut motility, use of neuromuscular blocking agents in ventilated patients, reducing the enteral intake, and colonic decompression. All these techniques have only theoretical benefit in decreasing IAP.

Thoracic epidural after initial resuscitation has been evaluated in a blinded, nonrandomized, prospective study by Hakobyan in which they found that it significantly reduced the mean IAP (16.8 mm Hg to 6.3 mm Hg), thereby increasing the APP without any hemodynamic compromise.[17] Percutaneous catheter drainage of any space-occupying fluid collection or resuscitation induced ascites can potentially improve APP and organ dysfunction, thereby avoiding a laparotomy. If the decrease in IAP is <9 mm Hg, and the amount of fluid drained is <1 L, it should be considered as a failure. If fulminant ACS develops with evidence of organ failure, laparotomy should be considered.[11,18]

Effects of Intra-abdominal Hypertension and Clinical Manifestations of Abdominal Compartment Syndrome

Rise in IAP impedes the venous return causing pressure changes that have the following multisystem effects and hemodynamic changes.[14,19,20]

Respiratory System

Raised diaphragm, poor compliance, and decreased functional residual capacity, and basal atelectasis, presenting as low-tidal volume, hypoxia, hypercarbia, and raised airway pressures.

Gut and Liver

Reduced splanchnic, hepatic, and portal flow presenting as edema, ischemia, and necrosis of the gut, metabolic acidosis, and higher incidence of hepatic artery thrombosis and portal vein thrombosis

Renal System

Decreased blood flow to the kidneys, renal venous congestion causing cortical arteriolar compression presenting as decreased urine output, rising creatinine, and renal failure.

Circulatory System

Compression of the inferior vena cava resulting in reduced preload and increased afterload presenting as hypotension, reduced cardiac output, and false elevation in central venous pressure (CVP) and wedge pressure.

Central Nervous System

Increased intrathoracic pressure with decreased cerebral venous outflow presenting as elevated intracranial pressure.

Important changes that occur at the time of open decompression are:[14]

- Immediate improvement in lung compliance and tidal volume; hence, ventilatory parameters should be carefully monitored and readjusted.
- Increase in the cardiac output: if the patient is on inotropes, they should be titrated as per the recommended mean arterial pressure.
- A significant drop in central venous pressure may be observed; hence, resuscitate accordingly.
- Reduction in intracranial pressures by as much as 10 mm Hg-extremely important in patients of concomitant traumatic brain injury and position of the patient should be optimized accordingly.

Intra-abdominal Sepsis

The primary goal in the management of any infectious process is source control; hence, in the presence of intra-abdominal sepsis, infection should be drained, necrotic material debrided, and intestinal injury repaired by performing a laparotomy. Mortality among acidotic patients who are hemodynamically unstable with significant systemic involvement due to intra-abdominal sepsis can be reduced by application of OA.[21]

The proposed benefit of OA is this subset of patients is that ACS can be prevented in those requiring high-volume resuscitation and repeated peritoneal toileting. It has been suggested that by removing cytokine-laden peritoneal fluid during peritoneal toileting, systemic inflammatory response can be reduced, and a prospective study analyzing the inflammatory component in the peritoneal fluid and serum of patients with temporary abdominal dressing after DCS is currently being conducted.[22] Reexploration or a "second-look" operation may also be needed in patients with complex liver injury, duodeno-pancreatic injuries, and penetrating injuries caused by a blast or high-velocity weapons that lead to loss of the abdominal wall.

Ruptured Abdominal Aortic Aneurysm

ACS develops in patients of ruptured AAA secondary to large volume resuscitation, reperfusion injury and formation of hematomas that are large space-occupying lesions causing pressure changes. Elective delayed fascial closure should be considered in patients who have the following risk factors for development of ACS as identified by Rasmussen:[23]

- Systolic blood pressure <90 mm Hg for >18 minutes (preoperative shock)
- Cardiac arrest (preoperatively)
- Hypothermia <33°C
- Severe metabolic acidosis base deficit >13
- Massive intraoperative resuscitation >3.5 L/hour
- EAST guidelines recommend using DCS for patients who are at high risk for developing visceral edema or IAH.[8]

Management of Open Abdomen and its Definitive Closure

A multidisciplinary approach with close interaction between the surgical and intensive care unit team is required to manage a critically ill patient with an OA which should be done in a specific staged process[13,14] (Flowchart 1). This plan aims at reducing the risk of developing both primary and secondary ACS and ideally should begin right at the time of retrieval, followed through in the emergency room and ICU, till the operating room where the surgeon decides to keep an OA.

ICU Management

Prolonged OA delays extubation, predisposes to repeated infections, and increases the risk for enteroatmospheric fistulae, therefore optimizing the physiology for the early closure of the abdomen is the primary challenge for the intensivist. The "lethal triad" of hypothermia, acidosis, and coagulopathy[24] that develops in patients of hemorrhage needs to be reversed by judicious resuscitation in the ICU.

Hypothermia

There is additional insensible heat loss with an OA in comparison to a closed abdomen although it has not been quantified. Even if one uses warm intravenous fluids, humidified warm gases, and heating blankets, approximately 4.6°C are lost per hour during laparotomy, and a drop in core temperature from 34°C to <32°C has been associated with 40–100% increase in mortality in trauma patients.[25,26] The harmful effects of hypothermia are:

- Cardiac dysrhythmias causing decreased cardiac output.
- Left shift of the oxygen dissociation curve hence poor oxygen delivery.
- Coagulopathy due to enzymatic dysregulation (raised prothrombin time and partial thromboplastin time) and platelet dysfunction (prolonged bleeding time).

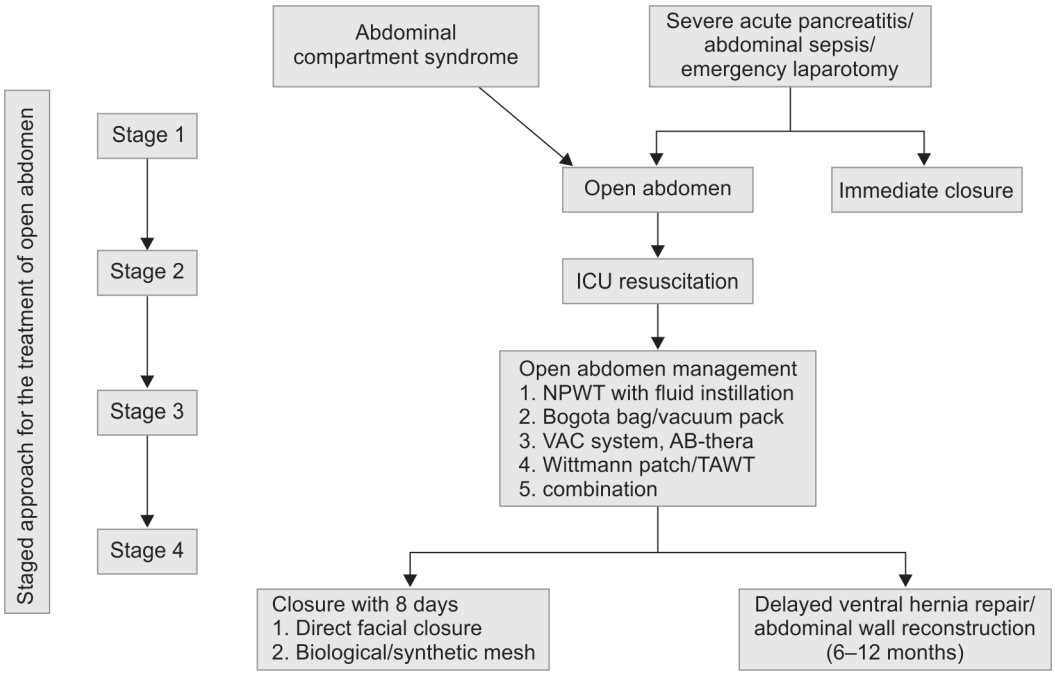
Management/prevention of hypothermia:

- Continuous core temperature monitoring
- Administer fluids via fluid warmers and use warm saline for irrigation
- Warm and humidify ventilator circuits
- Remove wet linen and keep minimum surface area exposed
- Increase ambient room temperature
- Convection blankets and heating mattress

Acidosis

Hypotension, hypothermia, and ongoing hemorrhage cause hypoperfusion of tissue and development of acidosis. Severe acidosis (pH < 7.1) further hampers tissue perfusion, worsening the hemodynamics, and coagulopathy. Look for compartment syndrome in an extremity or development of ACS, a missed injury or ongoing hemorrhage or occult sepsis, if lactate levels are increasing.[27]

Flowchart 1: Staged approach for the treatment of open abdomen (adapted from Coccolini et al.[14])



Management of acidosis:

- Monitoring of Arterial blood gases, electrolytes, and lactate clearance
- Avoid high-volume resuscitation to prevent increase in IAH which will further prolong OA
- Maintain hemodynamics
- Chase cultures and treat infection

Coagulopathy

Hypothermia, hypotension, acidosis, and ongoing hemorrhage requiring high-volume resuscitation in the form of intravenous fluids and blood products all-cause coagulopathy due to enzymatic dysregulation, hyperfibrinolysis, and platelet dysfunction.
Management of coagulopathy:[28,29]

- Increase core temperature to 37°C with 4 hours of arriving in the ICU
- Transfuse blood products to maintain PT < 15, platelet count >100,000 and fibrinogen level >100, in the ratio of 2:1:1 or 1:1:1 as per the PROPPR trial (exact ratio can still be debated).
- Point of care tests for coagulation like thromboelastography and ROTEM has been recommended to guide product administration during DCS.
- Requirement of more than 2 units of RBCs per hour for 3 hours is an indication for angiography or re-exploration laparotomy.

Ventilation

Patients with ACS have decreased pulmonary compliance, and high ventilatory pressures may be required to mechanically ventilate these patients. However, since the transpulmonary pressure is elevated in ACS, the high ventilatory pressures do not over distend the alveoli. Hence, if the tidal volume is decreased to lower the airway pressures, it will cause hypoxia and respiratory acidosis. This can be fatal when a tense abdomen in ACS is opened.

Therefore, once the abdomen is decompressed, the ventilatory settings must be changed to maintain appropriate tidal volume to prevent overexpansion of the alveoli. Maintaining high minute ventilation initially and administering bicarbonate and calcium can prevent a cardiovascular collapse that can be precipitated by the sudden increase in the acid load from the abdomen. Sudden increase in venous return when the abdomen is decompressed can cause right ventricular overload if there is preexisting pulmonary hypertension due to hypercarbia or myocardial dysfunction due to sepsis, which can be treated with dobutamine or milrinone. A concomitant large pleural effusion that is compromising ventilation, may also need to be drained.[27]

Risk factors for the development of acute respiratory distress syndrome (ARDS) are >10L fluid resuscitation in first 24 hours, aspiration, pneumonia, sepsis, intestinal ischemia-reperfusion syndrome, acute pancreatitis, alcohol, and drugs. Pre-emptive lung-protective ventilation strategies should be applied in the presence of any risk factor.[30]

Patients with OA are intubated and mechanically ventilated because of their underlying disease and there are several reports of patients of OA who have been extubated and made ambulatory with a low incidence of evisceration. The respiratory musculature usually compensates for the lack of negative subdiaphragmatic in the presence of an OA. A score of –2 to 0 on the Richmond Agitation-Sedation Scale/light sedation is usually sufficient when a temporary abdominal dressing is applied.

Gut Edema and Fluid Balance

Pathophysiology of gut edema in OA is multifactorial. During shock, the intravascular volume is preferentially shunted to the vital organs causing gut ischemia. Also, the mesenteric venous return is impaired when the IAP is elevated or the abdomen is packed, which causes congestion in an already ischemic gut. Post volume resuscitation, when an ischemic gut gets reperfused, there is free-radical mucosal damage and increased mucosal permeability leading to gut edema.[30] Inhibition of the lymphatic outflow via the cisterna chyli due to raised CVP has also been implicated in the formation of gut edema.

Hence, the primary goal in the ICU is to minimize volume overload and prevent gut edema by balanced resuscitative measures so that a primary fascial closure (i.e., fascia-to-fascia closure of the abdominal wall within the index hospitalization) is possible after laparotomy as fluid-related weight gain of >10% is a major risk factor for failure of primary closure.

Static indices like CVP and pulmonary artery occlusion pressure (PAOP) used to measure the intravascular volume may not be reliable in the presence of ACS or OA. Cheatham et al. in a study found right ventricular end-diastolic volume index (RVEDVI) to be a more accurate predictor of intravascular volume but could not give an optimal figure to target during resuscitation and recommended using markers like lactate to assess end-organ perfusion.[31] Ghneim et al. recommend using continuous stroke volume variation (SVV) as an end point, as it helps the intensivist to strictly titrate resuscitation to avoid hypoperfusion and volume overload, as it improved the time to primary fascial closure by 1 day.[32]

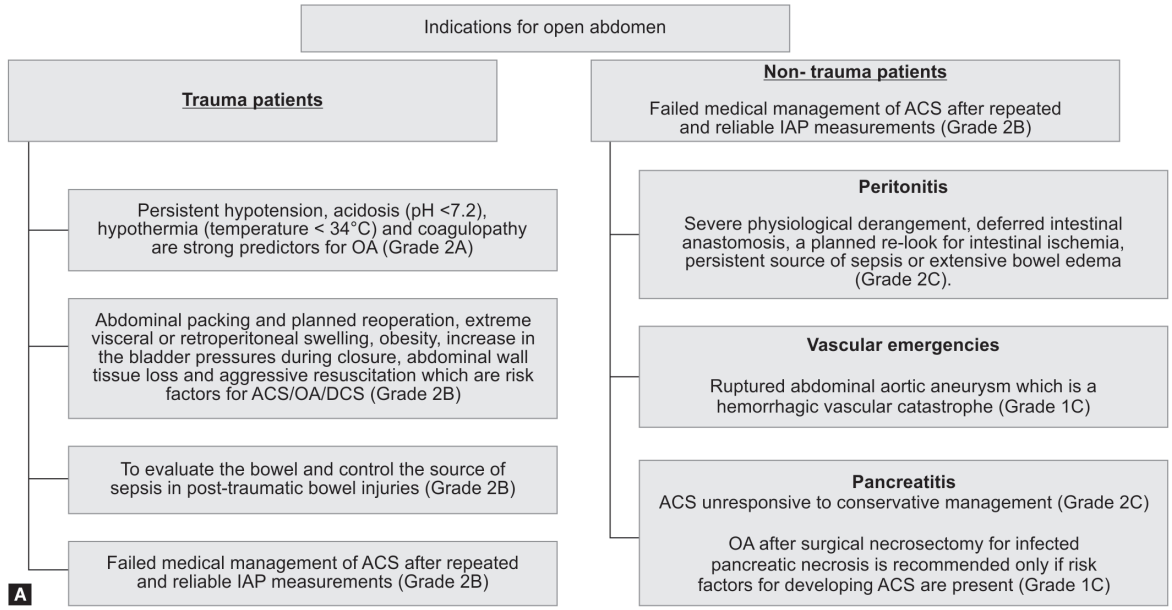
About 3% saline which is hypertonic causes shift of the fluid to the intravascular space and potentially can attenuate the inflammatory response; hence, in a small observational study higher rate of primary fascial closure was possible, but it was not statistically significant.[33] There is no randomized control trial about the use of albumin in OA, but since there is significant protein loss, there could be potential therapeutic benefit by using albumin as a resuscitative fluid.

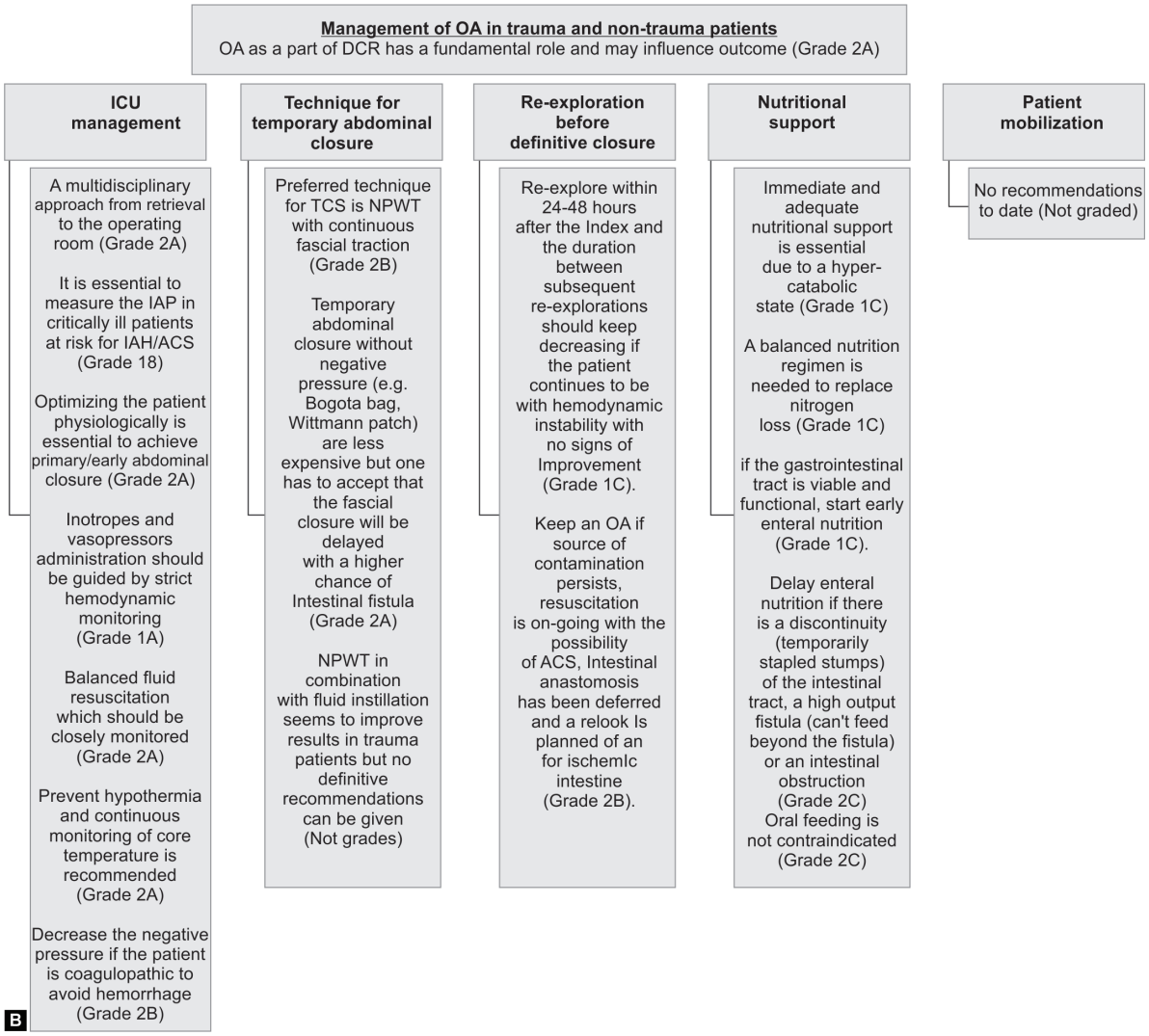
Nutrition

The only major contraindication to enteral feeding is intestinal discontinuity. It has been well refuted in the literature that exposing the viscera does not cause paralytic ileus and feeding in OA does not cause gut edema; hence, early full enteral feeding should be initiated when feasible to maintain gut integrity, modulate the systemic inflammatory response, decrease the rate of infection, decrease the rate of ventilator-associated pneumonia, achieve early primary abdominal closure and decrease the rate of fistulas.[34]

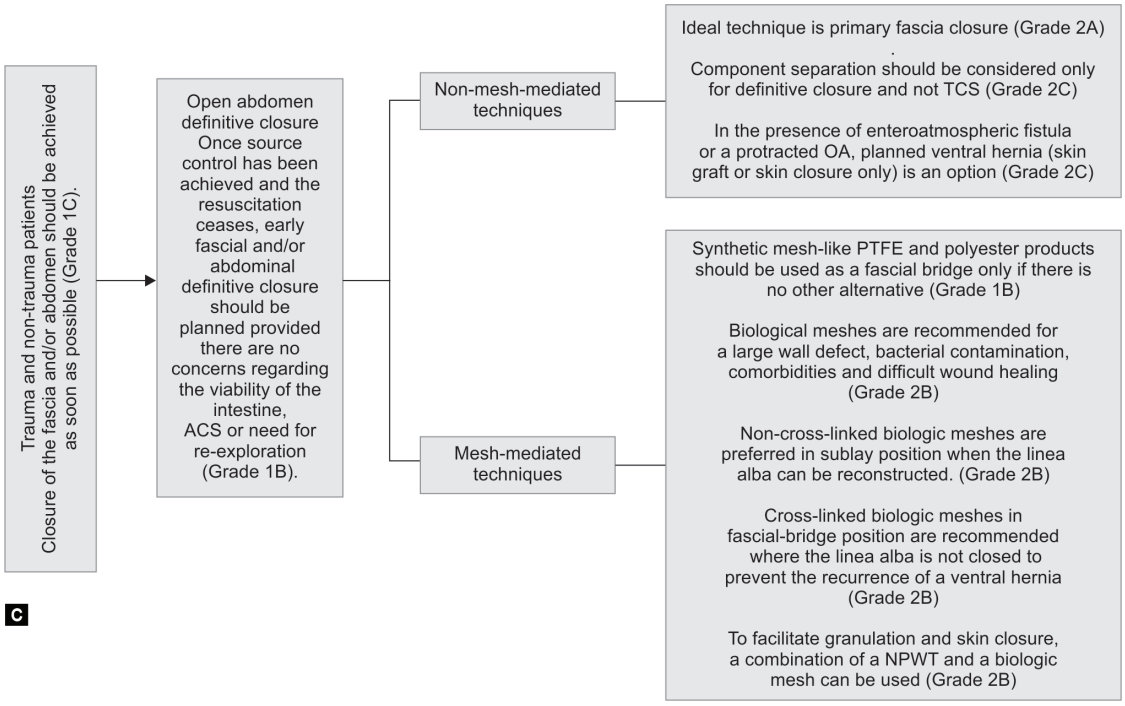
It is estimated that 2–4.6 g of nitrogen is lost per liter of abdominal fluid output. Nitrogen balance and caloric need should be carefully calculated, and adequate amount of fluid, electrolyte, and proteins should be replaced according to the amount of fluid lost through the abdominal wound, as this is a major cause for underfeeding. Significant amount of potassium, phosphorus, magnesium, and calcium is also lost into the peritoneal fluid and hence should be adequately replaced.[35]

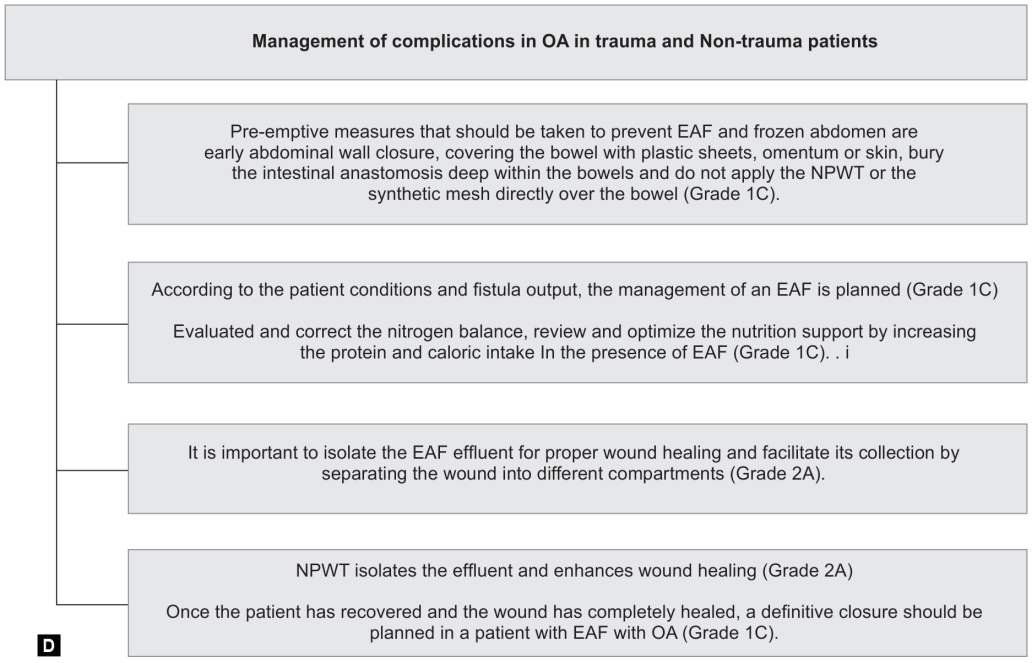
There are no guidelines regarding which site to feed (stomach vs jejunum), amount of enteral nutrition (trophic vs goal tube feeds), or the use of specialized formulas. Animal studies have shown that a high-fat enteral diet may decrease intestinal mucosal barrier when the peritoneal is exposed to air.





Figs 1A and B: (A) Indications for OA (summary statements WSES 2016) Coccolini F et al.;[9] (B) Management (summary statements WSES 2016) Coccolini F et al.[9]





Figs 1C and D: (C) Definitive closure (summary statements WSES 2016) Coccolini F et al.;[9] (D) Management of complications (summary statements WSES 2016) Coccolini F et al.[9]

Infection and Sepsis

Chances of an infectious complication increase after 8 days of OA. In all, 25% of patients with OA develop infectious complications in the form of wound infection, a deep abdominal abscess, or an intestinal fistula, which delays primary closure after an OA. The incidence of bloodstream infections is higher in such patients. Poor outcome is seen in almost 78% of the patients who develop gram-positive cocci and gram-negative bacilli intra-abdominal colonization. Antibiotics should be as per the disease process and cultures. There is limited role of prophylactic antibiotics, and a lot of emphases have been put to prevent catheter-associated bloodstream infections and VAP. Hence, whenever the patients are physiologically optimized, a primary fascial closure should be attempted.

Re-exploration

The first relook should be attempted within 24 hours and no later than 48 hours after the initial laparotomy. Any patient requiring more than 2 units of RBCs per hour for 3 hours should be posted for a re-exploration. Progressive closure should be attempted with each reoperation if definitive abdominal closure cannot be done.[36]

Surgical Management of Abdominal Compartment Syndrome/Open Abdomen

Temporary Abdominal Closure Techniques

The ideal technique should be easy to apply and remove so that the surgeon can rapidly access the surgical site, should drain secretions, make nursing care, and the primary closure easier, and be easily available and affordable.

Currently, the following are available—Bogota Bag, Wittmann Patch, Vacuum pack with an adjustable pump to set the negative pressure, AB-Thera, ABRA system.[9]

Definitive Closure

Definitive closure should be done by applying split-thickness skin graft with a synthetic mesh. Alternatively, a Biological prosthesis (collagen mesh) can be used which allows blood, growth, and anti-inflammatory factors to reach the surgical field. The non-crossed link meshes have a better tissue integration and local inflammatory reaction but have a faster re-absorption process.[9]

Conclusion

Abdominal compartment syndrome is the most common reason for leaving the abdomen open by reopening a laparotomy, not closing, or creating a fresh laparotomy. Routine monitoring of bladder pressures in high-risk patients should be a standard intensive care unit (ICU) protocol. The care of severely injured patients has improved with the application of OA. Surgery can be abridged in a physiologically depleted patient by doing a DCS as a part of DCM. OA can be managed by several TAC techniques; however, it is also associated with serious complications, such as severe fluid and protein loss, nutritional problems, EAFs, and development of massive incisional hernias. The most effective way to prevent or reduce these complications is to close the abdominal wall as soon as possible. Negative-pressure wound therapy may be associated with better outcomes than other temporary abdominal closure techniques. Definitive closure should be done only after the patients have recovered, depending on the duration of OA treatment and the size of the residual defect. The OA is associated with many early and late complications, including infections, gastrointestinal fistulas, and ventral hernias. Clinicians should be vigilant regarding the development of these complications.

Summary of statements from the World Society of Emergency Surgery guidelines (Fig. 1)[9]

Author Contribution

- Written the main script and complied the literature.
- Drafting the work or revising it critically for important intellectual content.
- Drafting the work or revising it critically for important intellectual content.

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