Abdominal Compartment Syndrome

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Injury/Disease Demographics

- Defined by sustained intra-abdominal pressure (IAP) greater than 20 mmHg in the presence of new organ dysfunction or evidence of splanchnic hypoperfusion.
- Intra-abdominal hypertension (IAH) typically precedes the abdominal compartment syndrome (ACS), and is defined by sustained pathologic elevation of IAP $\geq$ 12 mmHg.
- Normal IAP ranges:
    - In normal, non-obese adults: 5-7 mmHg.
    - Critically ill patients: 5-10 mmHg.
    - Morbidly obese patients: 5-14 mmHg
- Increased IAP leads to compression of intra-abdominal lymphatics and vasculature with progressive worsening of bowel perfusion leading to necrosis. Increased bowel wall permeability occurs with the intestinal ischemia, which results in increased extracellular volume and extravasation of fluid into the bowel, mesentery, and peritoneum and worsening of intra-abdominal pressure.
- Occurs in trauma, burn, emergency general surgery, and intensive care unit patients. Incidence has been reported from 1-36% in mixed population adult intensive care unit patients with significant mortality.
- Primary ACS occurs from a disease process or injury within the abdomen or pelvis.
- Secondary ACS occurs from a disease process or injury outside of the abdomen or pelvis.
- Recurrent ACS is defined by occurring after medical or surgical treatment of primary or secondary ACS.
- Risk factors include diminished abdominal wall compliance (abdominal surgery, burns, prone positioning, large ventral hernia repair), increased intra-abdominal or intra-luminal contents (ileus, acute pancreatitis, bowel edema, intra-abdominal infection, etc.), sepsis, large volume fluid resuscitation, massive blood transfusion, mechanical ventilation, and increased PEEP (> 10 mmHg) requirements.
- In patients undergoing an abdominal operation with multiple risk factors for the development of ACS, primary abdominal closure is contraindicated and therefore the development of ACS can often be prevented.
- Tips and tricks
    - It is important to note that even patients with an open abdomen and temporary abdominal wall dressings can develop recurrent ACS. Just because a patient has been surgically decompressed once does not preclude them from developing ACS a second time.

Clinical Presentation

- Physical exam alone is unreliable in diagnosing ACS, but a distended abdomen is the most common clinical feature. A distended abdomen in the presence of one or more risk factors mentioned above should prompt concern for ACS.

- Patients may have worsening of their respiratory status due to an increase in thoracic pressure from diaphragmatic elevation. Patients typically present with an increasing oxygen requirement and/or increased plateau pressures if mechanically ventilated.
- Cardiovascular changes likely to be seen include a reduction in cardiac output resulting from decreased preload via compression of the inferior vena cava as well as increased afterload. This leads to a decreased stroke volume which is often compensated by an increase in heart rate.
- In addition to a distended abdomen, signs and symptoms consistent with splanchnic hypoperfusion may be present. These signs include a worsening metabolic acidosis or elevation of serum lactate.
- Oliguria or anuria may also be present, secondary to decreased renal perfusion.
- Tips and tricks
    - Acute IAH leads to an increase in intracranial pressure (ICP) due to an increased thoracic pressure and impaired venous outflow from the brain. This association is important to remember in patients with presumed or known concurrent brain injury.

Evaluation & Diagnostics

- Physical exam alone is insufficient in diagnosing ACS.
- Suspicion for ACS should be followed by evaluation of intra-abdominal pressure. This evaluation is most commonly performed by measuring urinary bladder pressure.
- The urinary bladder pressure is measured by first draining the bladder, then instilling 25 mL of sterile saline into the bladder and clamping the drainage tube, distal to the instillation port. A needle is then inserted into the instillation port, and a pressure transducer attached.
- The patient should be supine at the time of bladder pressure measurement, and the transducer zeroed at the pubis symphysis or iliac crest in the midaxillary line.
- Intra-abdominal hypertension is present with a bladder pressure measurement of ≥ 12 mmHg, while ACS is defined as sustained pressures of > 20 mmHg with signs of new organ dysfunction or failure.
- Intra-abdominal hypertension may be present at baseline for patients with morbid obesity, large volume ascites, or who are pregnant. In patients with these conditions and a concern for ACS, bladder pressures may need to be measured serially in order to determine if an acute elevation exists.
- Tips and tricks for obtaining an accurate bladder pressure:
    - Ensure the bladder is adequately drained – this can be done with ultrasound.
    - If serial measurements are likely to be needed, mark the location of the transducer where it has been zeroed – this will help in reproducibility.
    - The patient must be completely supine without head of bed elevation as this can falsely elevate the measurement.
    - Be consistent with the saline instillation into the bladder.

        - Over instillation of saline and inconsistent amounts of saline can lead to inaccurate measurements.
        - Waiting 30-60 seconds after the instillation of saline into the bladder will allow the detrusor muscles accommodate for the instilled volume and provide a more accurate measurement.
    - Spontaneous breathing and abdominal contractions can falsely elevate bladder pressure measurements. Ensure the patient is comfortable if awake adequate sedation if the patient is intubated, as active abdominal muscle contractions during the measurement will falsely elevate pressure readings. This is particularly true for patients with COPD who may have increased abdominal muscle tension and forced expirations.
    - PEEP levels less than 15 mmHg have been shown to minimally impact bladder pressures. In patients requiring levels of peep greater than 15mmHg, serial measurements may be needed to determine acuity.

Imaging

- Although X-rays and computed tomography can be used to diagnose intra-abdominal sources of ACS, these modalities do not have a role in the diagnosis if ACS itself.
- Trans-abdominal ultrasound may be used to demonstrate compression of the inferior vena cava, which is not in of itself diagnostic, but it can be a marker of ACS
- Echocardiogram may show signs of increased intrathoracic pressure secondary to elevation of the diaphragm, decreased preload and increased afterload. These signs include decreased ventricular end diastolic volumes, reduced stroke volume, and reduced cardiac contractility. The ejection fraction can remain relatively normal to even slightly increased.

Role of Conservative Management and Associated Conditions

- If intra-abdominal hypertension exists without signs of organ dysfunction, serial bladder pressure measurements should be performed. In addition, the clinician should monitor for signs of organ dysfunction with hourly measurements of urine output and serial laboratory evaluations for acidosis.
- The World Society of the Abdominal Compartment Syndrome (WSACS) has published a medical management algorithm for patients with intra-abdominal hypertension prior to the development of ACS.[1] Techniques in this algorithm include:
    - Percutaneous drainage catheters for excessive intraabdominal fluid
    - Nasogastric decompression,
    - Goal directed fluid resuscitation and
    - Resuscitation with hypertonic fluids and/or colloids to avoid excessive fluid and crystalloid administration.

    - Correction of a positive fluid balance can be attempted with the use of diuretics and/or renal replacement techniques if appropriate.
    - Increasing abdominal wall compliance with adequate sedation and neuromuscular blocking agents should also be considered.
- However, once ACS has been confirmed with bladder pressure measurement of > 20mmHg and organ dysfunction, treatment is surgical decompression.
- Tips and tricks for conservative management:
    - In some situations, ultrasound-guided paracentesis may be useful to transiently decompress the abdomen and relieve intraabdominal hypertension. In some very closely monitored patients, this may prevent decompressive laparotomy
    - Early and continued coordination of care if multiple ICU teams are involved
    - Consider neuromuscular blockade early, particularly to ensure accurate bladder pressure measurements

Indications for Operative Treatment

- A bladder pressure of > 20 mmHg with signs of acute or progressing organ dysfunction should prompt surgical decompression.
- Signs of organ dysfunction include:
    - Oliguria or anuria
    - Reduced cardiac output
    - Hypotension
    - Metabolic acidosis refractory to appropriate fluid resuscitation
    - Elevated liver enzymes
    - Hypoglycemia
    - Elevated serum lactate
    - Respiratory failure with hypoxia, hypercapnea and elevated airway pressures.

Pre-Operative Preparation & Impact of Associated Injuries

- ACS without decompression is uniformly fatal. Once the diagnosis of ACS has been made, intervention is an emergency and should be performed immediately, without delay.
- Some authors have advocated for an aggressive increase in fluid administration immediately prior to surgical decompression in efforts to prevent the acute hypotension that usually occurs immediately following decompression. This hypotension is due to the abrupt decrease in vascular resistance and is managed with fluid resuscitation and/or vasopressors. Often patients with true ACS will markedly improve soon after decompression, however.
- If ongoing hemorrhage is suspected, coagulation abnormalities should be investigated and corrected either prior to or during surgery.
- Tips and tricks

- Ensure adequate intravenous access for resuscitation as needed during and after surgery
- If possible and not already in place, an arterial line should be placed once the decision is made to proceed with surgery.
- If the patient is too unstable to be moved to the operating room, a bedside decompressive laparotomy may be performed in the intensive care unit under sterile conditions with appropriate coordination with operating room, anesthesia, and nursing staff.
- The morbidity and mortality associated with a decompressive laparotomy is high, with rates up to 50%. Thorough and careful consideration with the patient's family or surrogate decision maker and the patient's primary care team regarding goals of care and likely outcomes should be had prior to operative intervention.

Operative Technique

- A decompressive laparotomy is performed through a generous midline incision made through skin and carried down through the fascia. As the intra-abdominal contents typically push through the fascia once it has been even slightly opened, the surgeon should be extremely cautious not to cause an enterotomy while finishing the fascial incision.
- If the patient has had a recent midline laparotomy, the prior incision should be opened and the fascial incision extended in both directions is possible to ensure adequate decompression. Similarly, if a patient has a temporary abdominal closure in place, this should be removed and the fascial incision extended.
- After the intra-abdominal contents have been adequately decompressed, any excess intra-abdominal fluid should be removed and the abdomen copiously irrigated. Careful hemostasis should also be achieved.
- To prevent recurrence of elevated intraabdominal pressure, the abdominal incision is left open and a temporary abdominal closure placed.
- Temporary abdominal closure is achieved by the placement of a large silo or vacuum assisted dressing.
- Tips and tricks
    - This is not minimally invasive surgery. A large incision should be made. Abdominal compartment syndrome will not completely resolve if the incision is not large enough to allow for complete decompression.

Intraoperative Considerations

- Profound hypotension may occur immediately after abdominal decompression. Arterial monitoring should be present to aid in treatment, which may require rapid fluid or vasopressor administration

- Reperfusion syndrome after release of the abdominal wall may occur, resulting in temporary worsening of the patient's metabolic derangements and hemodynamic status. Treatment options include fluid resuscitation, sodium bicarbonate administration, and renal replacement therapy.
- The temporary abdominal closure used should provide both a non-adherent layer of protection for the intra-abdominal contents and allow for egress of fluid.
- Commercially available vacuum assisted dressings are most commonly used, and proponents cite better primary fascial closure rates with the use of this device. Concerns have been raised regarding the use of negative pressure wound therapies and the development of enteroatmospheric fistulae. Despite these concerns, the WSACS does recommend the use of negative pressure wound therapy as a strategy to obtain temporary abdominal closure.
- Tips and tricks
    - Abdominal compartment syndrome can recur. These patients are critically ill and may require aggressive fluid resuscitation and/or undergo significant fluid shifts. Both the incision and the temporary abdominal dressing (regardless of method) should be generous enough to allow for further distention and expansion of the intra-abdominal contents that may continue after surgery.

<u>Post-Operative Management</u>

- Postoperative management centers around the normalization of hemodynamic, metabolic, and coagulation parameters to allow for return to the operating room and primary fascial closure.
- Serial laboratory examinations should be performed to monitor the development and/or progress of reperfusion syndrome.
    - Worsening or refractory metabolic acidosis should prompt evaluation for new or worsening bowel ischemia.
    - Coagulation abnormalities should continue to be investigated and corrected postoperatively to prevent non-surgical bleeding.
- Goal directed resuscitation should be continued during the postoperative period to prevent excessive fluid administration while being mindful of the significant increase in insensible fluid loss with an open abdomen.
- Renal function should continue to be closely monitored as a diuretic phase also typically occurs within 72 hours of decompression. This physiologic diuresis may be augmented with the use of medications if renal function and hemodynamic status allows.
- Enteral nutrition is feasible in resuscitated patients and has been shown to be associated with increased rates of fascial closure for patients without bowel injuries.
- The patient should return to the operating room for attempted primary fascial closure ideally when all physiologic derangements have been corrected. This typically occurs between 24 and 72 hours.
- If fascial closure is not able to be performed secondary to fascial retraction, continued intra-abdominal edema, or unacceptable elevation of intraabdominal

pressure upon closure, successive fascial closure is attempted. Successive fascial closure can be achieved using negative pressure wound therapy, dynamic retention sutures, or commercially available devices such as the Wittmann patch (Starsurgical, Burlington, Wisconsin, USA). The fascia is then progressively tightened every 24-72 hours until it can be safely approximated.

- If fascial reapproximation is unable to be achieved with progressive tightening techniques within 5-10 days, the decision is made towards a planned ventral hernia.
- The operative plan for a planned ventral hernia begins with the placement of one to two layers of absorbable polyglycolic mesh over the open abdomen. The mesh is then covered with a moist or negative pressure dressing. The wound is allowed to granulate through the mesh over a period of 1-3 weeks and a split thickness skin graft applied. The resultant ventral hernia can be repaired via an abdominal wall reconstruction 6-12 months later.
- Tips and tricks
    - Don't underestimate the value of nutrition in managing these patients postoperatively. Not infrequently, these patients have been critically ill prior to needing decompression and are malnourished. Starting enteral nutrition early is best. If enteral nutrition is not possible, parenteral nutrition should be considered and started if deemed appropriate.

Complications

- ACS is associated with significant morbidity and mortality, with pooled mortality rates in the literature between 39-54%.
- Although the open abdomen technique has been proven to be life saving and effective in reversing the vast majority of organ dysfunction, it is not without complications related to the operation itself.
    - Complication rates have been quoted from 17-54%, dependent upon the patient population and reason for the development of ACS.
- Complications of the open abdomen after a decompressive laparotomy for ACS include:
    - Wound infection
    - Intra-abdominal abscess
    - Enteroatmospheric fistulae.
- In patients with bowel injuries requiring resection and anastomosis, an increase in anastomotic leak rate is seen in patients with fascial closure that does not occur within 5 days of the first operation, and is a contributing factor to the development of enteroatmospheric fistulae.
- Overall there is an 8-14% incidence of enteroatmospheric fistulae associated with delayed or lack of primary closure of the abdomen.
- Specific risk factors that have been identified in the formation of enteroatmospheric fistulae include:
    - Large volume resuscitation
    - Increased number of surgical re-explorations
    - Sepsis

    - Mesenteric ischemia
    - Large bowel resection
    - Inability to achieve definitive fascial closure.
- Tips and tricks:
    - Overall, there is an association between increased number of complications and the longer an abdomen is left open. The plan for abdominal closure should start at the end of the decompressive operation.
    - Sequential closure techniques such as serial suturing of the fascia in conjunction with negative pressure dressings or devices such as the Wittman patch can and should be used to aid in primary fascial closure.
    - When fascial closure is obtained, the skin of the incision should be left open to prevent wound infections.

Considerations for Special Populations

- Obesity, and particularly morbid obesity continues to rise in the United States. There is evidence that intra-abdominal pressure is elevated at baseline in patients with significant truncal obesity. In addition, physical exam and detection of abdominal distention is difficult in this patient population. Patients with significant truncal obesity have been shown to have slightly elevated intra-abdominal pressure at baseline (8-10mmHg), but it is important to note these values do not meet the definition of intra-abdominal hypertension (> 12mmHg).
- Intra-abdominal hypertension has been shown to elevate intracranial pressure (ICP) and reduce cerebral perfusion pressure (CPP) independently from signs of pulmonary or cardiovascular dysfunction. This effect on ICP and CPP is due to increased intrathoracic pressures resulting in obstruction of cerebral venous outflow. In patients with TBI and elevations of ICP that are refractory to medical management, decompressive laparotomy should be considered at intra-abdominal pressures greater than 15 -20 mmHg.
- Patients with cirrhosis and ascites may develop intra-abdominal hypertension or abdominal compartment syndrome in critical illness. Abdominal decompression in these patients may be accomplished with paracentesis. Typically, rapid improvement in hemodynamics is seen. Patients requiring multiple paracentesis for recurrence may benefit from the placement of a temporary drain. In situations in which large volumes are drained, attention must be paid to volume and electrolyte status.

Recommended Readings

1. Kirkpatrick AW, Roberts DJ, De Waele J, et al. "Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome." *Intensive Care Med* 2013;39:1190-1206.
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3. Burlew CC. "The open abdomen: practical implications for the practicing surgeon." *Am J Surg.* 2012;204(6)826-835.
4. Roberts DJ, Ball CG, Kirkpatrick AW. "Increased pressure within the abdominal compartment: intra-abdominal hypertension and the abdominal compartment syndrome." *Curr Opin Crit Care.* 2016;22(2):174-185.
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6. Joshi D, Saha S, Bernal W, Wendon J, Auzinger G. "Advanced haemodynamic assessment of patients with liver disease and abdominal compartment syndrome." *Crit Care*2010;14(Suppl 1):P537.