Abdominal Compartment Syndrome

A case base approach to Intra-Abdominal Hypertension & Abdominal Compartment Syndrome

Brandon Braddock, MPAS, PA-C
Pulmonary/Critical Care
Objectives

- Identify etiologies of Intra-abdominal hypertension and Abdominal compartment syndrome
- Identify clinical signs and symptoms of Intra-abdominal hypertension and Abdominal compartment syndrome
- Recognize and implement the use of bladder pressures for the diagnosis of Intra-abdominal hypertension and Abdominal compartment syndrome
- Recognize and understand treatment modalities for Intra-abdominal hypertension and Abdominal compartment syndrome
Patient History

- Patient S: 35 year old obese Caucasian female

- PMHx
  - Chronic anemia
  - Poly-substance abuse w/ IVDA
  - Alcohol abuse
  - Alcohol induced Cirrhosis
  - R hip abscess
  - Chronic pancreatitis
Surgical Hx

- Bilateral total hip arthroplasty – 2009
  - Recurrent R hip infections and chronic dislocations
Chief Complaint:
- Altered mental status
  - Last seen in her usual state of health by patient’s significant other one night prior when she went to bed.

HPI:
- Reportedly, upon waking, the patient was confused and was repeatedly saying “I’m scared.” Patient’s significant other denied patient’s active IVDA however did report patient takes morphine & dilaudid which she obtained “on the streets”
Vital Signs
- Temperature: 95.9°F
- Heart rate: 120bpm
- Respiration rate: 26 breaths/min
- SpO2: 95%
- Blood pressure: 79/48mmHg

Relevant physical Exam:
- Pale, jaundiced appearing – moderate distress
- Lungs clear
- Normal BS, soft, non-tender, non-distended abdomen
- + Edema/erythema of R hip w/ blanching & purulent drainage
- Awake, alert – not following commands
Relevant Lab Results
- WBC 3.1
- Hgb 5.8
- Platelets: 127
- aPTT 68.4
- PT 34.1/INR 3.44
- Glucose 149
- BUN 10/Cr 2.9
- Sodium 126
- Potassium 2.7
- Arterial lactate 4.55

Relevant Imaging Studies
- CT brain – neg
- CT abdomen/pelvis - pancolitis, chronic pancreatitis, bibasilar consolidation
- CT R. lower extremity – Chronic hip dislocation, surrounding edema
Severe sepsis with septic shock and multi-organ failure secondary to:
- Pancolitis
- PNA
- UTI
- Cx (+) MSSA Bacteremia

Acute hypoxemic respiratory failure defined as hypoxemia w/ PaO2 < 60 on ABG
- Required intubation & full MVS

Septic shock induced hypotension defined as: a systolic BP less than 90mmHg for a period greater than 15 minutes despite adequate fluid resuscitation or Lactate > 4 (previous guidelines)
- Refractory to IVF (30ml/kg) & required vasopressor support
Acute stage 3 renal failure defined as a rise in sCr 3x baseline or UO < 0.3ml/kg/hr for > 24 hours or anuria for > 12 hours despite adequate fluid resuscitation secondary to septic shock
- Metabolic acidosis secondary to renal failure, type A hyper-lactatemia & decreased strong ion difference
- Electrolyte abnormalities secondary to renal failure & septic shock

Disseminated Intravascular Coagulopathy (DIC) secondary to septic shock:
- Thrombocytopenia
- Coagulopathy

Acute encephalopathy secondary to septic shock
- Altered mental status & no intracranial abnormality identified on CT brain
Clinical Course

- Large volume fluid resuscitation: 25L +
  - IVF – isotonic solution (NS 0.9%) re: hypotension
  - PRBC & FFP re: anemia & coagulopathy
  - IV Abx re: infectious etiologies
  - IV sedation (Fentanyl/Propofol) re: vent management targeting a RASS -1
  - Significant third-spacing (interstitial edema)

- Mechanical ventilatory support

- Multi-disciplinary team
  - Intensivist, Ortho, Nephro, ID, PCP, Heme/Onc, General Surgery, PT/OT/ST, RN, Respiratory, Dietary, Social Services
Repeat Physical Exam

- Hypoactive bowel sounds
  - Misleading – majority of critically ill patients develop hypoactive bowel sounds
- Distended abdomen
- Firm/rigid abdomen
- Generalized anasarca – third spacing
- Decreasing urinary output
- Increasing vasopressor requirement
- Worsening hypoxemia
  - Increased peak pressures – indicative of poor chest wall compliance or increased abdominal pressures
  - Increased FiO2 requirement
    - Resultant atelectasis
Diagnostics

Intra-vesicular pressure (bladder pressure): greater than 25

Repeat CT abdomen/pelvis:
Diffuse severe colonic wall thickening (Colitis)
Assessment 2.0

- Severe sepsis with septic shock and multi-organ failure – refractory to aggressive treatment
  - Required multiple vasopressors

- Acute hypoxemic respiratory failure – declining status with increased V/Q mismatch + hypervolemia induced pulmonary edema requiring increased PEEP & FiO2

- Acute stage 3 renal failure – progressive to anuria despite optimal fluid resuscitation & improved sCr

- DIC – refractory w/ worsening coagulopathy – INR rising
Acute cholestasis secondary to septic shock as evident by rising aminotransferases & bilirubin

Critical-illness related adrenal insufficiency – required stress dose steroids

Worsening metabolic acidosis w/ pH 7.0 by ABG & worsening hyper-lactatemia despite initial improvement

DDx now includes
- Intra-abdominal hypertension & abdominal compartment syndrome as a result of
  - Diffuse colitis & generalized ileus
  - Cholestasis
  - Septic shock
  - Profound interstitial edema secondary to large volume resuscitation and septic shock induced capillary leak
Defining IAH & ACS

- **Intra-Abdominal Hypertension (IAH)**
  - A steady state pressure of greater than 12mmHg concealed within the abdominal cavity

- **Abdominal Perfusion Pressure (APP)**
  - Defined as Mean Arterial Pressure (MAP) – Intra-Abdominal Pressure (IAP)

- **Abdominal Compartment Syndrome (ACS)**
  - A sustained IAP > 20mmHg (with or without an APP < 60) that is associated with new organ dysfunction/failure – research purposes
Defining IAH & ACS

- Primary Intra-Abdominal Hypertension or ACS
  - Condition associated with injury or disease originating from the abdomino-pelvic cavity

- Secondary Intra-Abdominal Hypertension or ACS
  - Condition associated with injury or disease not originating from the abdomino-pelvic cavity
Intra-abdominal Hypertension

- Grade I - defined as intra-abdominal pressure 12-15 mmHg
- Grade II - defined as intra-abdominal pressure 16-20 mmHg
- Grade III - defined as intra-abdominal pressure 21-25 mmHg
- Grade IV - defined as intra-abdominal pressure > 25 mmHg
IAH Acuity

- **Acute**
  - Elevated IAP developing over the course of several hours i.e. trauma, intra-abdominal hemorrhage – **concerning for progression to ACS**

- **Subacute**
  - Elevated IAP developing over days i.e. critically ill patients
  - Critically ill patients generally have an IAP 5-7mmHg

- **Chronic**
  - Elevated IAP developing over months/years i.e pregnancy, obesity – generally not progressive to ACS
Risk Factors

**Primary**
- Abdominal trauma
- Abdominal surgery
- Cirrhosis w/ascites
- Gastroparesis/ileus
- Ogilvie’s syndrome
- Volvulus
- Pancreatitis
- Abdominal abscess
- Retroperitoneal bleed

**Secondary**
- Large IVF resuscitation
- Mechanical ventilation
- Sepsis and/or septic shock
- Burns
- Metabolic acidosis
- Third spacing/interstitial edema
Physiologic Consequence
Cardiovascular

- Impaired cardiac function
  - Increased IAP leads to reduced ventricular compliance & contractility
    - Can occur at IAP as low as 10mmHg
    - Exacerbated in underlying heart failure

- Reduced venous return
  - Increased IAP leads to compressed IVC and decreased preload & decreased cardiac output – exacerbated by hypovolemia
  - Increased systemic vascular resistance & venous hydrostatic pressure
    - Increased third spacing & DVT risk
Physiologic Consequence
Pulmonary

- Increased risk of alveolar barotrauma
  - Increased peak & plateau pressures
  - Reduced lung & chest wall compliance
  - Increased V/Q mismatch – arterial hypoxemia & hypercapnia
  - Accentuated in patient’s requiring large IVF, bronchospasm (COPD exacerbation) & pt’s requiring increased PEEP

- Increased risk of pulmonary infection/ventilator acquired pneumonia
  - Increased duration of vent support – poor ventilation
  - Increased atelectasis, edema, pleural effusions
  - May require NIPPV and/or MVS if not already
Physiologic Consequence
Renal

- Renal vein compression
  - Increases venous resistance and impaired venous drainage; studies suggest the primary cause of renal impairment

- Renal artery vasoconstriction & sympathetic response
  - Induced by sympathetic nervous system & RAAS system due to decreased cardiac output
    - Oliguria typically noted when IAP > 15mmHg
    - Anuria occurring when IAP > 30mmHg
Physiologic Consequence
Gastrointestinal

- Mesenteric ischemia
  - Decreased mesenteric vein flow due to increased intestinal edema leading to worsening IAP and possibly ACS as well as bowel necrosis and lactic acidosis – vicious cycle

- Studies suggested mesenteric blood flow is reduced with an IAP as low as 10mmHg
- Intestinal mucosal perfusion decreased with IAP > 20mmHg
Bowel hypo-perfusion
- Targeting abdominal perfusion pressure – 60mmHg (MAP – IAP)
- Increased bacterial translocation
  - Can occur at IAP of only 10mmHg – more prominent in cases of hemorrhage

Hepatic dysfunction
- Impaired toxin clearance i.e Lactate
- Impaired production of coagulation factors
- Impaired protein production i.e Albumin
  - Leads to decreased hydrostatic pressure & development of ascites & increased IAP
  - Exacerbated in patients with underlying liver disease; our patient has a hx of EtOH cirrhosis
Physiologic Consequence
Central Nervous System

Cerebral Ischemia

- Increased intracranial pressure leading to decreased cerebral perfusion & ultimately cerebral ischemia
- Secondary to decreased venous return/decreased cardiac output
Symptoms

- Generalized complaints
  - Malaise/fatigue
  - Weakness
  - Dyspnea
  - Abdominal bloating
  - Abdominal pain

- Limitations in patient assessment/symptoms
  - Patient’s unable to communicate
    - Encephalopathic
    - Mechanical ventilatory support
Physical Signs

- Distended/firm abdomen
- Decreased bowel sounds - misleading
- Significant third spacing
- + Fluid wave (ascites)
- Increased JVD
- Worsening obtundation
- Cool/diaphoretic skin
- Progressive organ dysfunction despite treatment
  - Progressive oliguria/anuria
  - Increased vent support
    - Increased FiO2
    - Increased PEEP
    - Decreased tidal volumes
  - Refractory hypotension
Physical Signs

- Limitation in physical exam
  - Poor predictor of ACS; prospective cohort study (N=42) of blunt abdominal trauma demonstrated:
    - Sensitivity 56%
    - Specificity 87%
    - PPV 35%
    - NPV 94%
Diagnostics

- Imaging: not helpful in the diagnosis of ACS
  - Chest radiograph may show:
    - Low lung volumes, raised hemi-diaphragm, atelectasis
  - CT abdomen/pelvis may show:
    - Extrinsic compression of IVC
    - Abdominal distention
    - Renal compression
    - Bowel thickening
    - Inguinal herniation
Diagnostics

- Definite dx requires intra-abdominal pressure monitoring
  - Can be measured via multiple modalities including:
    - Intragastric
    - Intracolonic
    - Intravesicular (bladder)
    - IVC catheters

- Gold standard as defined by the WSACS for IAH/ACS screening:
  - Intravesicular (bladder) pressure
Bladder Pressure Technique

- Patient must be in supine position
- Clamp Foley catheter drainage tube
- Instill a maximum of 25ml sterile saline into the bladder via the aspiration port of the Foley
- 18g needle attached to a pressure transducer is inserted into the aspiration port (needle-less systems available)
- Pressure is measured at end-expiration once the transducer is zeroed at the level of the mid-axillary line
Noninvasive Bowel Decompression

- Nasogastric or orogastric tube placement
- Placement of bowel management systems. i.e Rectal tube placement
- Maintain supine position or head of bed no greater than 30 degrees of elevation if possible (risk assessment for VAP, aspiration, ICP)

Invasive Decompression

- Procedure dependent upon cause
  - Ascites $\rightarrow$ paracentesis
  - Abdominal abscess/hematoma/hemoperitoneum $\rightarrow$ percutaneous needle aspiration
  - Burns $\rightarrow$ escharotomy
Definitive therapy: treat underlying cause followed by:

- Abdominal decompression
  - Controversial in regards to timing of surgical decompression
    - All pt’s w/ IAP > 25mmHg
    - Some clinicians suggest decompression w/ IAP 15-25mmHg – felt there is improved APP & pt outcome
  - Some clinicians recommend following the APP
    - Retrospective study demonstrated APP <50mmHg more predictive of mortality w/ increased specificity/sensitivity than MAP or IAP alone
- Open abdomen with temporary closure
Therapeutic Intervention

Adapted from Intensive Care Med 2013;1109-1206
© 2014 World Society of the Abdominal Compartment Syndrome. All rights reserved.
Due to patient’s refractory and progressive multi-organ failure in conjunction with elevated intra-vesicular pressures she underwent abdominal decompression and was noted intra-operatively to have IAP in excess of 20mmHg

She was transferred back to SICU post-operatively in critical condition however tolerated the procedure adequately

Vasopressor requirement had improved

24hrs post-op she underwent closure of her open abdomen after resolution of the acute phase of her abdominal compartment syndrome
Unfortunately, due to patient S’s severe and refractory multi-organ failure triggered by severe sepsis and septic shock further treatment was deemed futile.

At the discretion of her significant other, as well as input from a multifaceted team, a terminal wean was requested.

Ultimately, the patient succumbed to her illness following withdrawal of life support.
Intra-abdominal Hypertension & Abdominal Compartment Syndrome can be caused by numerous etiologies both primary and secondary

Early recognition of developing signs/symptoms prompts urgent treatment

Although IAH alone is not a predictor of MOF, mortality for patients who progress to ACS ranges from 40-100 percent

Bladder pressures remain the gold standard for IAP monitoring

Definitive treatment is by surgical abdominal decompression
"All disease begins in the gut."
- Hippocrates
References


