

Acute abdominal compartment syndrome

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Acute abdominal compartment syndrome can be an elusive clinical diagnosis for which critical care physicians must exercise a high index of suspicion in the differential diagnosis of hemodynamic compromise and hemorrhagic shock. The purpose of these *Rounds* is to describe a case of abdominal compartment syndrome and to summarize the pathophysiology, differential diagnosis, etiology, and treatment of this entity. The Research Ethics Committee of the Montreal Heart Institute granted approval for use of the related images for publication purposes.

A middle-aged male was admitted to the intensive care unit (ICU) for management of lower gastro-intestinal bleeding and hemorrhagic shock. His medical history was remarkable for cirrhosis (Child-Pugh Stage C) secondary to hepatitis C, with previous ligation of esophageal varices. He also had undergone a colonoscopy with a cecal polyp

resection five days earlier. The initial blood work-up showed a 6.0 g decrease in hemoglobin. The patient was mechanically ventilated and transfused with six units of packed red blood cells. Despite blood transfusion and aggressive resuscitation with crystalloids, noradrenaline was required at an infusion rate of $15 \mu\text{g} \cdot \text{min}^{-1}$ to maintain a mean arterial pressure (MAP) >65 mmHg. Esophagogastroduodenoscopy showed no active bleeding. A colonoscopy was planned, and 4 L of an oral preparation (Golytely) were to be administered via a nasogastric tube (NGT). After 30 min and 1 L of the oral preparation had been administered, the patient's blood pressure decreased to 50 mmHg and the noradrenaline infusion was increased to $53 \mu\text{g} \cdot \text{min}^{-1}$ (Fig. 1a). The patient's abdomen became increasingly tense and he developed anuria. His intravesical pressure at this time was 28 mmHg (Fig. 1b). A diagnosis of acute compartment syndrome (ACS) was made, and ultrasound-guided paracentesis (Figs. 2 and 3a, b) and nasogastric aspiration were performed. Two and one-half litres of ascitic fluid and 1 L of the enteral solution were removed through aspiration. Shortly after this intervention, the noradrenaline requirements decreased to $10 \mu\text{g} \cdot \text{min}^{-1}$ to maintain the MAP >65 mmHg. On angiography, an intracecal bleeding site was identified and the cecal artery was embolized. The bleeding stopped and the patient later stabilized. A review of computed tomography scans of the abdomen performed one week prior to this acute event revealed signs of liver cirrhosis with portal hypertension and ascitis (Fig. 4 [videos #1 and #2 online at www.springerlink.com]). Note was made of the fact that the inferior vena cava was significantly narrowed in its intrahepatic portion.

Abdominal compartment syndrome is defined as a sustained abdominal pressure >20 mmHg with evidence of organ dysfunction relieved by abdominal decompression.¹

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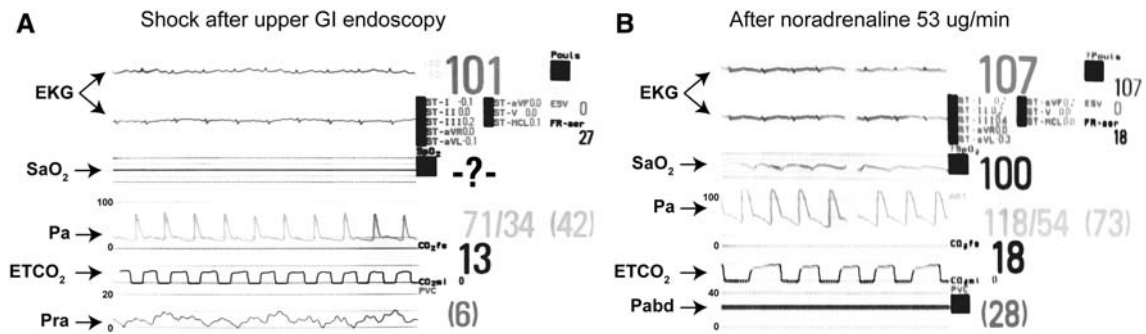


Fig. 1 **a** Hemodynamic response following upper endoscopy. The patient's blood pressure (BP) was 71/34 mmHg and the end-tidal CO₂ was 13 mmHg with right atrial pressure (RAP) = 6 mmHg. *EKG* electrocardiogram. **b** Hemodynamic response to an increase in

noradrenaline infusion. The patient's BP increased to 118/54 mmHg. The abdominal pressure (*Pabd*) of 28 mmHg confirmed the diagnosis of abdominal compartment syndrome

Fig. 2 **a** Right lower quadrant echographic examination before peritoneal fluid removal using a 5 MHz phased-array ultrasound. **b** A 10° medial tilt resulted in an image in which a bowel loop appeared close to the abdominal wall. **c** A 10° lateral tilt resulted in a more appropriate site for paracentesis. In addition, in a patient with portal hypertension, varicose veins of the abdominal wall can be visualized and avoided during the procedure

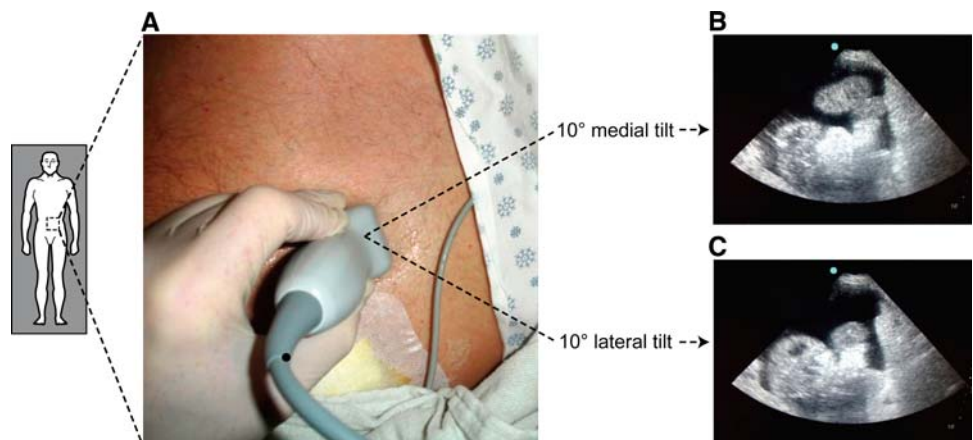
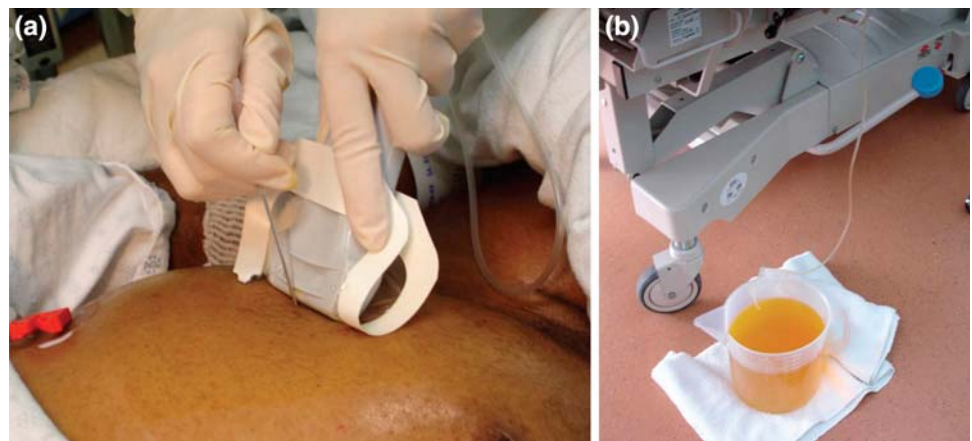


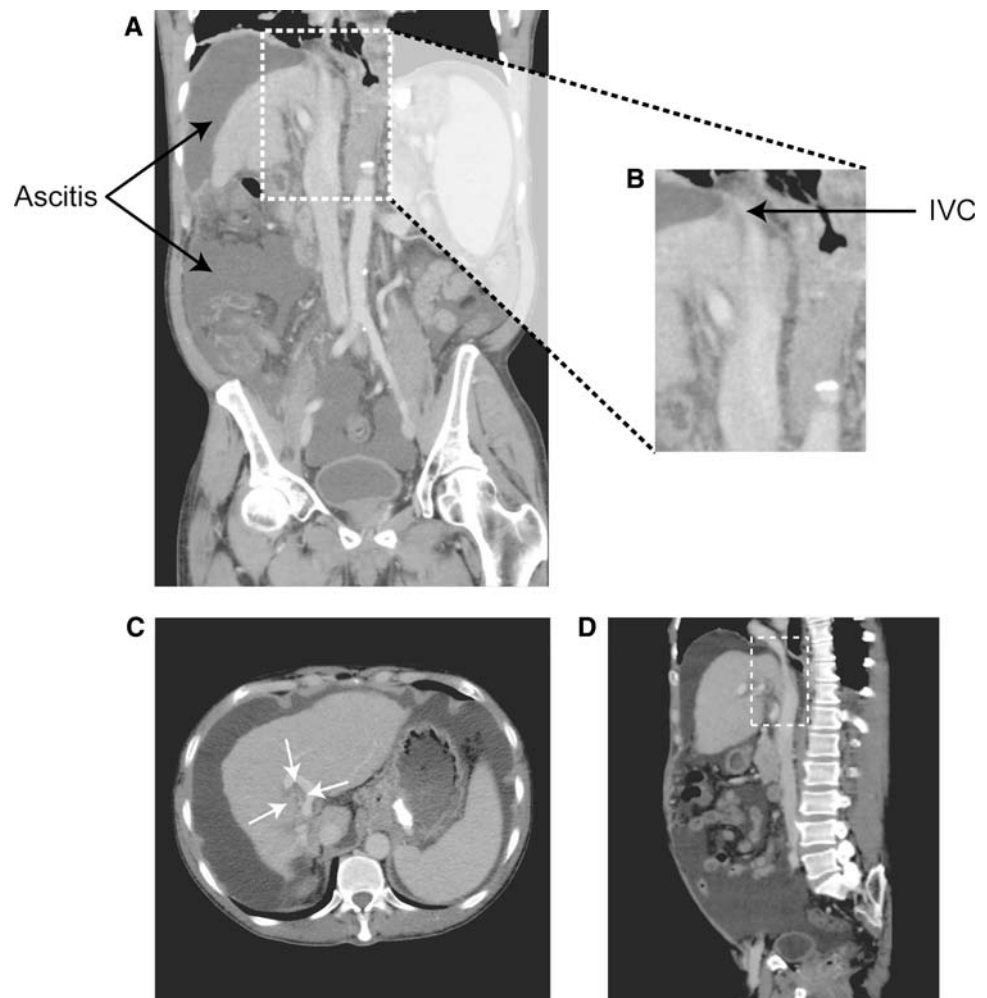
Fig. 3 **a** The paracentesis can be safely performed under direct ultrasound guidance using a sterile technique and adhesive (Opsite™ Flexigrid™, Smith & Nephew, UK). **b** This sterile technique is done using a #14 G catheter. The catheter is left in situ for slow drainage



The term intra-abdominal hypertension (IAH) is used in the presence of abdominal pressures from 12 to 20 mmHg. Increased pressure in a nonexpendable compartment reduces capillary bed perfusion and promotes bacterial translocation followed by the activation of inflammatory cytokines. The latter promotes leakage through vascular

walls and edema, which further contributes to the rise in intra-abdominal pressure (IAP). The reduction in the abdominal perfusion pressure (APP), defined as the difference between MAP and IAP, leads to organ ischemia. The associated rise in abdominal pressure increases the resistance to venous return (Fig. 5). This will reduce

Fig. 4 **a** Computed tomography image showing a small cirrhotic liver with ascites. **b** Narrowing of the intrahepatic vena cava (IVC) on the coronal view. This is a common finding in advanced cirrhosis. **c** Axial cut of the upper abdomen showing a small dysmorphic liver with partial portal vein thrombosis. **d** Sagittal view showing the narrowing of the inferior vena cava. (See videos #1 and #2 at www.springerlink.com)



venous return and lead to low cardiac output and shock.² Furthermore, as the IAP increases, the diaphragm is pushed cephalad, which reduces the thoracic or the extra-pulmonary compliance. The consequences of this condition include reduced renal glomerular filtration, an oligoanuric state, hepatic dysfunction, and intestinal ischemia. Acute compartment syndrome has been shown to be an independent risk factor for mortality in the ICU.³

The risk factors of ACS are summarized in Table 1 and can be divided into three categories: diminished wall compliance, increased intra-abdominal content, and capillary leak.^{3,4} Clinical manifestations are non-specific and include decreased urine output, high ventilatory pressures and a tense abdomen. Monitoring the intravesical pressure is essential to establish the diagnosis (Fig. 6). In patients with IAH and ACS, the APP should be maintained above 50–60 mmHg.⁵ Treatment should be directed towards management of the underlying cause. Specific goals should be to improve abdominal wall compliance, reduce abdominal fluid and/or air, and to correct the positive fluid balance. The most

definitive intervention is decompression laparotomy with temporary abdominal closure.⁶ However, this approach is not without risks and is not always curative.⁷ The use of diuretics, paracentesis, and dialysis can be very effective (Figs. 3 and 7).

In this particular case, the patient's rapid collapse can be explained by several factors for ACS. First, the narrowing of the intrahepatic vena cava is a typical finding in advanced cirrhosis (Fig. 4). Second, the esophagogastric endoscopy probably increased the IAP through air insufflation and the enteral preparation may also have contributed to the increased pressure. Third, aggressive fluid resuscitation with crystalloids could also have increased extravascular edema in the abdominal compartment with fluid redistribution. Finally, the APP was reduced by a hypotensive state associated with hemorrhagic shock. The hemodynamic instability was rapidly reversed via two mechanisms: reduction of the intraluminal and extraluminal content through the NGT drainage and bedside ultrasound-guided paracentesis. The hemorrhage was controlled through embolization of the cecal artery.

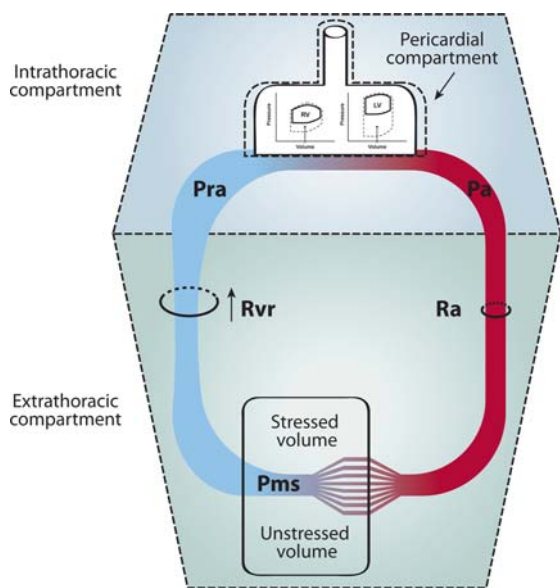


Fig. 5 Schematic representation of the circulatory system. The hemodynamic instability in abdominal compartment syndrome results primarily from an increase in resistance to venous return (R_{vr}) due to compression of the inferior vena cava. Since venous return depends on the gradient between the mean systemic venous pressure (P_{ms}) and the right atrial pressure (P_{ra}), when abdominal pressure exceeds the P_{ra} , the limiting pressure for venous return becomes the intra-abdominal pressure. The biventricular pressure volume curves of the right and left ventricle in the abdominal compartment syndrome are shown. The rise of abdominal pressure induced by abdominal compartment syndrome is transmitted to the thoracic compartment. Therefore, the absolute values of left and right ventricular filling will be elevated. However, the transmural pressure (pressure in – pressure out) will be reduced, and this may be associated with a normal or reduced ventricular volume. A discrepancy between high filling pressure and normal to reduced volume will then be observed. P_a arterial pressure, R_a arterial resistance

In conclusion, as illustrated by this case, ACS is a clinical entity that requires rapid diagnosis and treatment to prevent both short and long-term complications which can be fatal.

Table 1 Abdominal compartment syndrome

Diminished wall compliance
Abdominal surgery
Acute respiratory distress syndrome
Major burns/trauma
Mechanical ventilation
Prone position
Obesity (body mass index >30)
Intra-abdominal content
Liver dysfunction (ascitis)
Hemo/pneumoperitoneum
Increasing intraluminal fluid content (e.g., contrast enema)
Ileus/gastroparesis
Acute colonic pseudo-obstruction; colonic dilatation (Olgivie’s syndrome)
Tumour
Capillary leak/resuscitation
Massive resuscitation
Polytransfusion (>10 blood units/24 hr)
Acidosis (pH < 7.2)
Sepsis
Hypothermia (<33°C)
Hypotension
Coagulopathy
Major burns/trauma
Emergency laparotomy

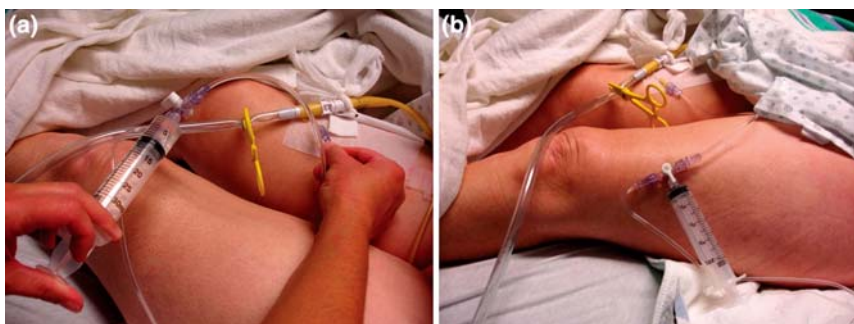
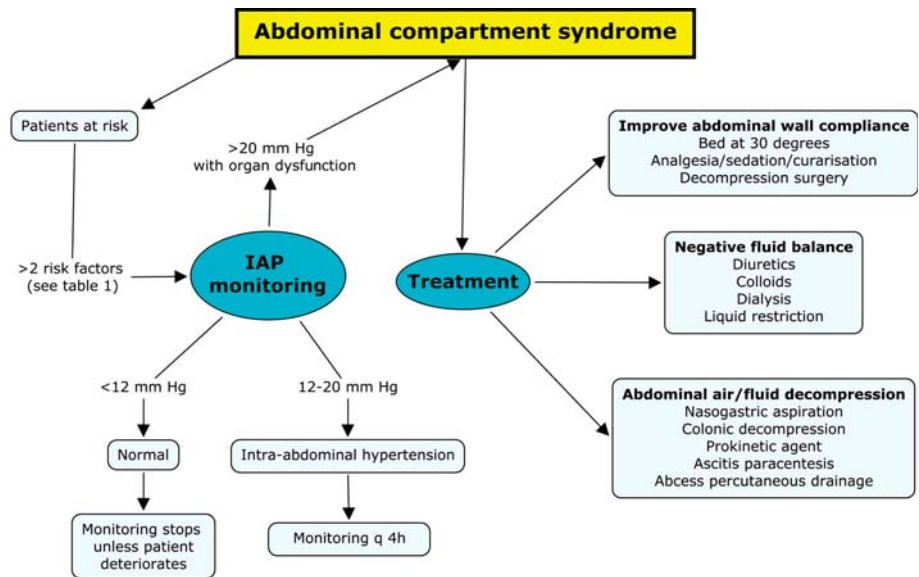


Fig. 6 Intra-abdominal pressure measurement: The system uses a normal saline solution (500 mL) connected to a standard pressure monitoring system, a 60 mL syringe, a Luer Lock, and 120 cm of rigid tubing (male–female) with a distal clamp. Steps: (1) The transducer should be zeroed at the phlebostatic axis (zeroing at the level of the mid-axillary line; the same level of the other transducers). (2) After placing the patient in a dorsal decubitus position (at 0°), verify that a Foley catheter is correctly positioned in the bladder (ultrasound scanning of the bladder can be performed for that purpose). (3) Connect the hemodynamic monitoring system to the 120 cm rigid tubing at the upper part of the 3-way stopcock. (4) Insert

a needle connected to the rigid tubing in the Foley and transiently clamp the Foley catheter. (5) Attach a 60 mL syringe to the monitoring system on the middle part of the 3-way stopcock and withdraw 25 mL of saline. (6) Turn down the 3-way stopcock and inject 25 mL of saline into the Foley catheter. (7) Open the 3-way stopcock to the monitoring system. Wait one minute to allow for stabilization of intravesical pressure. (8) Pressure measurement should be expressed in mmHg (Fig. 1b) and measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent. (9) Unclamp the Foley catheter and repeat the above steps if necessary

Fig. 7 An algorithmic approach to the diagnosis and management of abdominal compartment syndrome. IAP intra-abdominal pressure



Competing interests None declared.

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