

Abdominal compartment syndrome – the decision to operate is not so easy

I.T.A. Janssen* and J.G. van der Hoeven

Department of Intensive Care, Radboud University Medical Centre, Nijmegen, the Netherlands

Abstract

A 48-year-old male developed multi-organ failure due to abdominal compartment syndrome after trauma and abdominal surgery. He received surgical decompression after less invasive attempts to lower intra-abdominal pressure were not successful. We describe the diagnostic approach and measurements, which led to the decision to perform surgical decompression.

Introduction

Abdominal compartment syndrome (ACS) is a life-threatening condition defined as intra-abdominal hypertension (IAH) and subsequent organ dysfunction. ACS requires early recognition. If conservative measures fail to lower the intra-abdominal pressure (IAP), surgical decompression may be necessary. Measuring IAP is the first step in appropriate management, because either abdominal diameter or palpation of the abdomen is unreliable (accuracy of clinical abdominal examination for identifying elevated IAP is 77%) [1]. IAP can be measured either directly through needle puncture of the abdomen during peritoneal dialysis treatment or laparoscopy or indirectly using intravesicular pressure as measured through a bladder catheter or gastric pressure through a balloon catheter. Transvesicular measurement of IAP is the recommended method and commonly used [2]. IAH may affect all organ systems, but respiratory, cardiovascular and kidney function are affected most often. The decision to operate is not always easy. More specifically, increased IAP may be accompanied by other organ dysfunction due to various reasons, but not necessarily causally related. In the latter case, surgical decompression may be inappropriate. We describe the clinical decision making in a patient with a trauma related increase in IAP.

Case report

A 48-year-old male was admitted to our hospital after a traffic accident. He suffered a pelvic fracture, a comminute fracture of the distal tibia, a fracture of the second metacarpal bone of his right hand and cerebral contusion. CT-scan of the abdomen showed some paracolic fluid with fat stranding possibly due to mesenteric contusion. The patient was admitted to the intensive care unit (ICU) for monitoring and was hemodynamically and respiratory stable for more than 24 hours.

Two days after the accident the patient was transferred to the surgical ward. On the third day he underwent pelvic surgery with internal fixation and plate osteosynthesis of the tibia. During surgery, the patient bled from the iliac vein with a total bloodloss of 1300 ml and he was readmitted to the ICU.

The patient was tachycardic (130 bpm) with a mean arterial

pressure of 102 mmHg. His respiratory rate was 40/min although his oxygen saturation was normal with 3 litres of oxygen. He complained of severe abdominal pain. The abdomen was distended and tender on palpation, without palpable masses. The surgeon was consulted and decided to perform a laparotomy. During laparotomy, transection of the sigmoid was found with faecal spill, which resulted in a partial sigmoid resection and a temporary colostoma. When he returned from the operating room, he was ventilated with pressure support 8 cm H₂O and PEEP 10 cm H₂O with a FiO₂ of 0.4.

Over night, his clinical situation deteriorated with hypotension, oliguria and low central venous oxygen saturation of 55%. He developed acute renal failure. To evaluate the hemodynamic status a transpulmonary thermodilution catheter was inserted in the femoral artery. The cardiac index was 1.7 l/min/m² after insertion and goal-directed fluid resuscitation was initiated. Both global end-diastolic volume and extravascular lungwater were low.

The IAP was 31 mmHg and neuromuscular blockage was given which resulted in a lower IAP of 21 mmHg. A nasogastric tube was inserted and small volumes of gastric fluids were removed. Adequate sedation and analgesia was ensured with midazolam and sufentanil. After a few hours the ventilator pressure increased to a plateau >30 cm H₂O. To confirm the diagnosis of ACS several additional tests were performed. To exclude other causes of the high plateau pressure, a chest X-ray was made, which didn't show signs of ARDS or pneumonia. Pleural ultrasound excluded a pneumothorax or a large pleural effusion. Hemodynamic monitoring showed a persistent low cardiac output while echocardiography showed a hyperdynamic heart with a good left ventricular function.

The combination of increased IAP, high plateau pressure without another explanation, oliguria and normal cardiac function on ultrasound with low cardiac output confirmed the diagnosis of ACS

Correspondence to: I.T.A. Janssen, Department of Intensive Care, Radboud University Medical Centre, Nijmegen, the Netherlands, **E-mail:** Ingelante@hotmail.com

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and surgical decompression was performed. After decompression, ventilator pressures improved and diuresis already increased during surgery. All intra-abdominal organs at laparotomy were vital.

Discussion

IAP is the steady state pressure concealed within the abdominal cavity. For most critically ill patients, an IAP of 5 to 7 mmHg is considered normal [3]. ACS is a serious condition, which results in multi-organ failure if unrecognized or inadequately treated. ACS is defined as a sustained IAP of more than 20 mmHg (with or without abdominal perfusion pressure less than 60 mmHg) that is associated with new organ dysfunction/failure [4]. In the ICU however, multi-organ failure with concomitant raised IAP can be due to several causes and it is important to make the right diagnosis. The pathophysiology of multi-organ failure with ACS is explained in table 1 [5].

A medical management algorithm was suggested by the World Society of the Abdominal Compartment Syndrome (Table 2)

Table 1. The pathophysiology of abdominal compartment syndrome.

Organ dysfunction	Pathophysiology
Respiratory	Increased IAP pushes the diaphragm into the thoracic cavity causing increased intrathoracic pressure, thoracic compliance decreases, airway pressure increases in mechanical ventilation, residual capacity is decreased and oxygenation is impaired due to ventilation/perfusion mismatch.
Cardiac	Increased IAP impairs venous return, increases left ventricular afterload because of increased systemic vascular resistance, which leads to impaired cardiac output.
Renal	Increased IAP causes direct compression of the renal parenchyma. Decreased perfusion of the kidneys due to decreased cardiac output, and the increased water and sodium retention due to activation of the renin-angiotensin system leads to kidney failure and oliguria or anuria.
Cerebral	High intrathoracic pressure causes increased jugular venous pressure and impairs venous return from the brain, causing increased intracranial pressure and decreased cerebral blood flow.
Gut	Decreased cardiac output and increased splanchnic vascular resistance impairs splanchnic perfusion and can lead to ischemia.
Extremity	Increased IAP increases femoral venous pressure, increases peripheral vascular resistance and reduces femoral artery blood flow by as much as 65%.

Table 2. Medical management algorithm by the World Society of the Abdominal Compartment Syndrome. The interventions should be applied in a stepwise fashion. If there is no response to these interventions consider surgical abdominal decompression.

Evacuate intraluminal contents.
1. Insert nasogastric and/or rectal tube. Initiate gastro-/coloprokinetic agents.
2. Minimize enteral nutrition. Administer enemas.
3. Consider colonoscopic decompression. Discontinue enteral nutrition.
Evacuate intra-abdominal space occupying lesions.
1. Abdominal ultrasound.
2. Abdominal CT. Percutaneous catheter drainage.
3. Consider surgical evacuation of lesions.
Improve abdominal wall compliance.
1. Adequate sedation and analgesia. Remove constrictive dressings, abdominal eschars.
2. Consider reverse Trendelenberg position.
3. Consider neuromuscular blockage.
Optimize fluid administration.
1. Avoid excessive fluid resuscitation. Aim for zero to negative fluid balance bij day 3.
2. Resuscitate using hypertonic fluids/colloids. Fluid removal through diuresis once stable.
3. Consider hemodialysis/ultrafiltration.
Optimize systemic/regional perfusion.
1. Goal-directed fluid resuscitation.
2. Hemodynamic monitoring to guide resuscitation.

[4]. Because surgical decompression is a treatment with frequent complications and serious consequences, it is important to lower the IAP with less invasive interventions first.

The algorithm clearly shows that the first step is to decrease IAP by inserting a nasogastric and/or rectal tube, avoiding excessive fluid resuscitation, drainage of intra-abdominal fluids and muscle relaxation.

In our patients, these steps led to a decrease in IAP, although still above 20 mmHg. With progressive organ failure we excluded other causes and performed surgical decompression, which resulted in immediate improvement of organ function.

The hemodynamic measurements with ACS deserve special attention. Because IAH increases intrathoracic pressures, both pulmonary artery occlusion (wedge) pressure and central venous pressure increase, despite a decrease in transmural pressure [6]. Volumetric parameters such as global end-diastolic volume or intrathoracic blood volume are a more appropriate indicators of a patient's volume status. Pulse pressure variation or stroke volume variation may be increased but not necessarily predict fluid responsiveness. Increased intrathoracic pressure results in increased pulse pressure variation or stroke volume variation. This means a cutoff of 10-12% pulse pressure variation or stroke volume variation to determine fluid responsiveness cannot be used. Instead a 20-25% pulse pressure variation or stroke volume variation cutoff has been proposed [7]. Left ventricular afterload is often increased which further decreases stroke volume and thereby cardiac output [8]. It is important to realize that overzealous fluid resuscitation results in even more elevated IAP by increasing ascites and intestinal edema.

Conclusion

We describe a case of multi-organ failure after abdominal surgery due to ACS. After exclusion of other explanations the patient received appropriate decompression with immediate recovery of organ function.

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