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Review Article

The gastro-renal effects of intra-abdominal hypertension: Implications for critical care nurses

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ABSTRACT

Intra-abdominal hypertension is classified as either primary or secondary – primary occurs due to intra-abdominal or retro-peritoneal pathophysiology, whereas secondary results in alterations in capillary fluid dynamics due to factors, such as massive fluid resuscitation and generalised inflammation. The renal and gastro-intestinal effects occur early in the progression of intra-abdominal hypertension, and may lead to poor patient outcomes if not identified. As a direct response to intra-abdominal hypertension, renal function is reduced with remarkable impairment from pressures of around 10 mmHg, oliguria developing at 15 mmHg and anuria developing at 30 mmHg. Intestinal micro-circulation is significantly reduced by up to 50% with intra-abdominal pressures as low as 15 mmHg. Mucosal and submucosal tissue hypoperfusion causes considerable damage to the intestinal cells, potentially resulting in bacterial translocation, endotoxin release, sepsis and multiple organ failure. The critical care nurse plays an important role in the early identification of intra-abdominal hypertension however, without this essential knowledge base and comprehension of intra-abdominal hypertension, clinical signs and symptoms may go unnoticed or be misinterpreted as signs of other critical illnesses.

Contents

Implications for Clinical Practice

- Patients who present to the intesive care unit, especially the overweight, may have chronic undiagnosed intra-abdominal hypertension;
- Secondary intra-abdominal hypertension has a high mortality rate, is insidious in its development and is often missed in routine nursing assessment especially in the over-weight and obese critically ill patient;
- The renal effects of intra-abdominal hypertension is oliguria and is often the first sign that renal compressions is occurring despite adequate cardiac indices such as mean arterial pressure and cardiac output;
- Bacterial translocation can occur at intra-abdominal pressures of 14 mmHg;

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Introduction

The prevalence of intra-abdominal hypertension (IAH) in the intensive care unit (ICU) is high at around 50% (Gallagher, 2010). Studies have found that 20%–30% of critically ill patients present to the ICU with IAH, which increased to 30%–55% during their ICU-stay (Atema et al., 2014; Kim et al., 2012; Vidal et al., 2008). Approximately 8% of patients then went on to develop abdominal compartment syndrome (ACS) as a result of increasing and sustained IAH (Cresswell, 2016; Gallagher, 2010). This is a concern, as mortality rates of ACS are as high as 38%–80%, due to subsequent multiple organ failure (MOF) (Cheatham, 2010; Gallagher, 2010). Intra-abdominal pressure (IAP) within the abdominal cavity is the dynamic pressure exerted by the interaction between abdominal contents and the mechanical properties of the abdominal wall (Cresswell, 2016; Gallagher, 2010). The World Society of Abdominal Compartment Syndrome defines IAH as an IAP of 12 mmHg and over, the grades of IAH are: one – IAP of 12–15 mmHg; two – IAP of 16–20 mmHg; three – IAP of 21–25 mmHg; and four – IAP of more than 25 mmHg (Kirkpatrick et al., 2013). IAH is usually classified as either primary or secondary. Primary arises as a result of intra-abdominal or retro-peritoneal pathophysiology, such as abdominal trauma or pancreatitis, whereas, secondary is a result of generalised inflammation, capillary leakage and massive fluid resuscitation causing oedema of the bowel or ascites and is usually seen in patients with post-resuscitation severe shock or burn injuries (An, 2008; Gallagher, 2010; Atema et al., 2014). Most commonly IAH is multifactorial, such as, primary IAH being exacerbated by secondary IAH as a result of massive fluid resuscitation to treat the primary cause (An, 2008). Risk factors for the development of IAH are extensive and varied (Table 1). Upon development of IAH the renal artery blood flow and the mesenteric blood flow are preferentially reduced in comparison to other abdominal organ systems, with evidence of reduced perfusion occurring with as little as 10 mmHg–12 mmHg; therefore, stringent monitoring for early IAH is essential (Cheatham, 2009; Villa et al., 2016).

Table 1
Risk factors associated with intra-abdominal hypertension.

	Examples of Risk Factors for Primary and Secondary IAH
Decreased Abdominal Wall Compliance	Obesity Abdominal Surgery Older Age Liver Dysfunction
Increased Abdominal Intraluminal Contents	Ileus Abdominal Aorta Aneurysm Gastroparesis Peritoneal/Retro-peritoneal Haematoma
Related to Abdominal Corrections of Fluid, Air or Blood	Massive Fluid Resuscitation – at least 5 L over the past 72 h Multiple Transfusions Mechanical Ventilation
Related to Capillary Leak Syndrome	Acidosis Sepsis Shock Various Diseases such as Pancreatitis Coagulopathy Hypothermia Major Burns Abdominal Trauma

An (2008), Atema et al. (2014), Balogh et al. (2007), Blaser et al. (2011), Holodinsky et al. (2013), Hunt et al. (2017), Madigan et al. (2008) and Malbrain and De laet (2009).

Intra-abdominal hypertension

Enclosing the abdominal compartment is 1) the non-distensible posterior wall and the inferior bony pelvis, 2) the partially distensible lateral and anterior abdomen and 3) the diaphragm (Cresswell, 2016). Increased pressure of around 10 mmHg–15 m mHg within this relatively non-expandable compartment leads to initial microvascular irregularities in blood flow to visceral organs progressing to a reduction in venous return and arterial inflow (An, 2008; Mohmand and Goldfarb, 2011; Papavramidis et al., 2011; Patel and Connor, 2016).

Blood flow is initially impeded as a result of direct transmission of pressure on vessels causing extrinsic compression (Cresswell, 2016; Papavramidis et al., 2011). Compression of abdominal veins may result in venous hypertension, congestion and organ oedema, which perpetuates IAH and organ dysfunction (Mohmand and Goldfarb, 2011; Patel and Connor, 2016). Venous congestion and impaired venous return to the heart decreases preload, and extrinsic compression of the aorta increases afterload, thus diminishing cardiac output and further decreasing abdominal perfusion pressure (APP) (Christensen and Craft, 2017; Papavramidis et al., 2011; Scheppach, 2009). APP is measured as the difference between the mean arterial pressure (MAP) and the IAP (APP = MA P – IAP), resulting in the net pressure available to perfuse intra-abdominal organs. As IAP increases to around 20 mmHg the significant reduction in capillary bed perfusion results in tissue ischaemia, which leads to an immune response that activates inflammatory mediators and cytokines. This further increases IAP as increased capillary permeability and hydrostatic pressure, which cause fluid shifts from the intravascular to interstitial space, increase intra-abdominal volumes (An, 2008; Balogh et al., 2007; Cheng et al., 2013; Gallagher, 2010; Malbrain and De laet, 2009). At this stage, the abdominal lymphatic system is overwhelmed and is unable to drain excess fluids, perpetuating oedema and IAH (An, 2008; Balogh et al., 2007). Accumulation of extravascular fluid in the tissues causes local ischaemia leading to complications such as acute respiratory distress syndrome, renal failure and acute intestinal distress syndrome (Cheng et al., 2013; Malbrain and De laet, 2009).

Complications may occur with a ‘first hit’ physiological insult or illness, the prior administration of large volume resuscitation fluids and the reperfusion injury causing a systemic inflammatory response syndrome (Balogh et al., 2007; Gallagher, 2010; Malbrain and De laet, 2009). Upon progression of IAH, a threshold is reached where microvascular derangement increases venous outflow resistance, which impedes venous return, leading to venous congestion and further increases to IAP (An, 2008; Mohmand and Goldfarb, 2011). Decreased capillary perfusion, impaired venous drainage and subsequent progressive increases in IAP leads to decreased perfusion to the gastrointestinal system,

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