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Review article

Current concepts of the role of abdominal compartment syndrome in acute pancreatitis – An opportunity or merely an epiphenomenon

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ABSTRACT

The association of acute pancreatitis (AP) with intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) has only recently been recognized. The detrimental effects of raised intra-abdominal pressure in cardiovascular, pulmonary and renal systems have been well established. Although IAH was associated with a higher APACHE II score and multi-organ dysfunction syndrome (MODS) in severe acute pancreatitis, a causal relationship between ACS and MODS in SAP is yet to be established. It is therefore debatable whether IAH is a phenomenon causative of organ failure or an epiphenomenon seen in conjunction with other organ dysfunction. This review systemically examines the pathophysiological basis and clinical relevance of ACS in AP and summarizes all the available evidence in its management.

1. Introduction

Acute pancreatitis (AP) is a dynamic inflammatory process involving the pancreas, peripancreatic tissue and less commonly remote organ systems. Currently, it is estimated to account for nearly 274,000 hospitalizations annually in United States, making it the most common GI hospital discharge diagnosis [1]. The recently revised Atlanta classification recognizes three degrees of severity of AP [2]. Mild disease is defined as AP without organ failure, local complications and systemic complications. Moderately severe AP is defined by the presence of transient organ failure (present for <48 h), and/or local/systemic complications [3]. Severe acute pancreatitis (SAP) is defined by the presence of persistent organ failure that is present for >48 h and is known to occur in 15–20% of the patients [2,4,5]. Despite the significant strides made in critical care management in the last few decades, the hospital mortality rate for severe AP remains high. Recent reports have indicated that multi-organ failure in some patients with AP resulted from untreated "abdominal compartment syndrome" [6].

The World Society for Abdominal Compartment Syndrome (WSACS), an international multispecialty consortium published a consensus definition of intra-abdominal hypertension/abdominal compartment syndrome based on current evidence and expert opinion [7]. Intra-abdominal hypertension (IAH) is defined as persistent increase of intra-abdominal pressure (IAP) > 12 mm, whereas acute compartment syndrome (ACS) is the combination of IAP>20 mm Hg and new-onset organ dysfunction. ACS has been described in a wide variety of clinical scenarios such as burns, small bowel obstruction, and hemoperitoneum, in patients who underwent emergency abdominal surgery or after abdominal trauma [8]. The first reports on the association of IAH and AP were published only in 2002 [9,10]. It appears that the number of AP patients with this complication has increased, probably due to a more aggressive resuscitation strategy, a major paradigm shift towards conservative or a minimally invasive approach, and efforts to delay open surgery [11]. It is associated with prolonged stay in the intensive care unit and higher mortality [12,13]. Early recognition and prompt treatment of these conditions is crucial to decrease morbidity and improve patient survival [12,14,15].

The purpose of this review is to give an insight on the pathophysiology, clinical relevance of ACS complicating AP and summarize the management options. It is hoped that review of this under recognized but lethal complication of AP, will further invigorate interest among clinicians to conduct carefully designed trials to lend evidence based support for management strategies.

1.1. Search criteria

In August 2013, an electronic database (MEDLINE, Scopus, and PubMed) search of the literature from 1980 to the present was

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performed using the MeSH (Medical Subject Headings) terms "acute pancreatitis", "severe acute pancreatitis", "necrotizing pancreatitis", "fulminant pancreatitis". They were combined with the Boolean operator "AND'' with studies identified by searching the keywords "intra-abdominal hypertension" and "abdominal compartment syndrome". It is to be noted that prior to the WSACS consensus, studies did not employ a consistent definition for ACS, as shown in Table 1. The limited and variable nature of the data provided by the available evidence precluded formal quantitative synthesis in the form of meta-analysis, thus only a narrative synthesis of evidence was possible.

2. Epidemiology of ACS in AP patients

The initial reports of association of IAH with AP monitored the IAP selectively [6,9,13]. This resulted in the overestimation of its incidence. After adopting the standardized definitions of the WSACS, IAH was known to occur in 59% and 61% in two studies [12,14]. A very recent prospective study from Scotland evaluating 218 patients with AP estimated that 14% had IAH on admission and another 3% developed IAH during hospitalization [16]. ACS has been reported in upto 27% of AP patients as per the current definition (which is lower than earlier reported incidence) [14,17,18]. IAH has consistently been reported to be an early phenomenon, but it could theoretically occur later in the course of the disease as a result of local complications such as infected pancreatic and peri-pancreatic necrosis [9,19].

3. Pathophysiology of ACS in AP

The development of IAH in AP can be attributed to multiple factors, although their relative contribution varies from patient to patient and changes over time [19]. In AP, pro- inflammatory cytokines contribute to increased capillary permeability in various organs [25]. Subsequently, this results in exudation with inflammatory fluid accumulations in intra-abdominal and retroperitoneal cavities (acute fluid/necrotic collections) [2]. This is often accompanied by ascites which further increases the intra-abdominal volume. Paralytic ileus is another common risk factor for IAH in patients with AP. Massive fluid resuscitation, particularly with crystalloids in the early course of the disease, along with severe retroperitoneal inflammation worsens the evolving visceral edema further increasing the IAP [19]. In patients with SAP, there is pancreatic hypoperfusion, with IAH further compromising the pancreatic microcirculation, which expedites the onset of necrosis. IAH also causes splanchnic ischemia, resulting in hypomotility of the gut which facilitates luminal bacterial translocation into the sterile portal system [26,27]. This could further result in infected necrosis which can cause IAH in later stages of the disease or worsen already present IAH [19].

Table 1
Showing the definition and incidence of ACS in AP across studies.

Study and year	Number of AP patients	Definition of ACS	Incidence of ACS
Pupelis (2002) [9]	37	IAP > 25 cm of H_2O	29.7%
Keskinen (2007) [6]	37	IAP > 20 mm of Hg + MOD	49%
Al-Bahrani (2008) [12]	18	IAP > 15 mm of Hg + MOD	56%
Chen (2008) [14]	74	IAP > 20 mm of Hg + MOD	27%
Dambrauskas (2009) [17]	44	IAP > 20 mm of Hg + MOD	13.6%
Mentula (2010) [20]	26	IAP > 20 mm of Hg + MOD	NA
Ke. (2012) [18]	58	IAP > 20 mm of Hg + MOD	12%
Bezmarevic (2012) [21]	15	IAP > 20 mm of Hg + MOD	NA
Boone (2013) [22]	12	IAP > 20 mm of Hg + MOD	NA
Davis (2013) [23]	45	IAP > 20 mm of Hg + MOD	35%
Bhandari (2013) [24]	40	IAP > 20 mm of Hg + MOD	7.5%

AP = Acute Pancreatitis, MOD = Multi-Organ Dysfunction, NR = Not Reported, NA = Not Applicable.

4. Clinical consequences of intra-abdominal hypertension

There is growing evidence in the literature that development of abdominal compartment syndrome (ACS) in patients with severe acute pancreatitis (SAP) has a strong impact on the course of disease [11,12,14,17]. A recent study further concluded that the presence of IAH/ACS was associated with significantly increased extent of pancreatic necrosis, multi-organ failure, longer hospitalization and mortality among patients with SAP [24]. In patients with SAP and deteriorating organ function, the degree to which IAH contributes to the progression of MODS is still unknown [20]. Although IAH was associated with a significantly higher APACHE II score and MODS score in patients with SAP, a causal relationship between ACS and MODS in SAP has not been established [12,17,20]. Thus, according to some experts it is debatable whether intra-abdominal hypertension is a phenomenon causative of organ failure or an epiphenomenon, occurring in conjunction with other organ dysfunction [16]. Further the duration of IAH is of greater significance than the absolute increase in IAP [28]. Comorbid conditions such as chronic kidney disease, pre-existing heart and lung disease may further accentuate the deleterious effects of IAH and further lower the threshold of IAP at which organ damage occurs [28].

IAH causes elevation of the diaphragm, leading to a decline in lung and chest wall compliance, decrease in functional residual capacity, total lung capacity and residual volume. Oxygenation is further diminished by ventilation-perfusion mismatch which leads to respiratory failure necessitating mechanical ventilation. IAH also impairs venous return from the periphery to the right heart, simultaneously it increases left ventricular afterload. Both the effects lead to diminished cardiac output, reduced arterial pressure and lower organ perfusion pressure, which predisposes to arrhythmias especially in the setting of pre-existing ischemic heart disease. IAH also results in decreased renal blood flow, perfusion pressure and filtration gradient, besides direct compression of the parenchyma of the kidneys resulting in renal dysfunction. It has been shown that IAP >20 mm of Hg is associated with oliguria and significant reduction in cardiac output [27]. There is decreased hepatic arterial and portal venous blood flow which results in ischemic injury to liver and therefore diminished lactate clearance, altered glucose metabolism and disrupted mitochondrial function [29]. Increased IAP has an adverse effect on splanchnic hemodynamics, resulting in gut mucosal ischemia which in animals has been shown to facilitate bacterial translocation [27]. This further contributes to development of sepsis and accelerates multi-organ dysfunction in ACS. Thus IAH has an impact on almost every organ of the body which if unrecognized or untreated culminates into ACS and death.

5. Diagnosis of IAH

An essential first step in management is early recognition of IAH. The WSACS recommends that IAP should be assiduously measured, every 4–6 h in critically ill patient who demonstrate risk factors for the development of IAH or ACS [28]. IAP can be measured either directly (through needle puncture of the abdomen during peritoneal dialysis treatment or laparoscopy), which is rare and is typically restricted to patients undergoing continuous abdominal peritoneal dialysis. It can also be measured indirectly (using intravesicular pressure as measured through a bladder catheter or gastric pressure through a balloon catheter). The bladder route is widely adopted, as the intravesicular pressure accurately reflects