Abdominal Compartment Syndrome and Infection

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11.1 Abdominal Compartment Syndrome in Patients with Intra-Abdominal Infections

A compartment syndrome is a condition of increased pressure in a confined anatomic space that adversely affects the circulation and threatens the function and viability of the tissues therein. This may arise in any closed compartment within the body.

Abdominal compartment syndrome (ACS) is a common consequence of severe intra-abdominal infections. Primary acute abdominal compartment syndrome occurs when an intra-abdominal injury or disease in the abdominopelvic region is directly responsible for the compartment syndrome. Secondary abdominal compartment syndrome occurs when sepsis and related-fluid resuscitation cause fluid accumulation in the abdomen in a scenario lacking primary intraperitoneal injury.

Abdominal sepsis is the host's systemic inflammatory response to bacterial or yeast peritonitis [1].

Sepsis from an abdominal origin is initiated by the outer membrane component of gram-negative organisms (e.g., lipopolysaccharide [LPS], lipid A, endotoxin) or gram-positive organisms (e.g., lipoteichoic acid, peptidoglycan), as well as toxins from anaerobic bacteria [1]. This leads to the release of proinflammatory cytokines such as tumor necrosis factor α (TNF-α) and interleukins 1 and 6 (IL-1, IL-6). TNF-α and interleukins lead to the production of toxic mediators [1], which may cause a complex, multifactorial syndrome that may evolve into conditions of varying severity and may lead to the functional impairment of one or more vital organs or systems.

Fluid therapy to improve microvascular blood flow is an essential part of the treatment of patients with sepsis. Crystalloid solutions should be the first choice because they are well tolerated and cheap. They should be infused rapidly to induce

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a quick response but not so fast that an artificial stress response develops. They should be interrupted when no improvement of tissue perfusion occurs in response to volume loading [1].

However, in patient with abdominal sepsis, excessive infusion of fluids may become a counterproductive strategy [1].

The systemic inflammatory response syndrome, increased vascular permeability, and aggressive crystalloid resuscitation predispose to fluid sequestration with formation of peritoneal fluid. Patients with ongoing sepsis commonly develop shock bowel resulting in excessive bowel edema. These changes and associated forced closure of the abdominal wall may result in increased intra-abdominal pressure (IAP) ultimately leading to intra-abdominal hypertension (IAH).

Elevated IAP commonly causes marked deficits in both regional and global perfusion that may result in significant organ failure [2]. An uncontrolled IAH, with an IAP exceeding 20 mmHg and a new organ failure onset, leads to abdominal compartment syndrome (ACS). This in turn has further effects on intra-abdominal organs as well as indirect effects on remote organ(s) and system(s). ACS is a potentially lethal complication characterized by effects on splanchnic, cardiovascular, pulmonary, renal, and central nervous systems. Ventricular filling is reduced as a result of decreased venous return caused by the compression of the inferior vena cava or portal vein. Preload measurements such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) may be falsely elevated. Critical clinical conditions play an important role in aggravating the effects of elevated IAP and may reduce the threshold of IAH that causes the clinical manifestations of ACS. In addition, IAH and ACS likely influence the clinical course of many critically ill patients with sepsis. This is a result of both primary intraperitoneal disease and the massive fluid resuscitation that is often required to stabilize hemodynamics in patients with ongoing sepsis or septic shock. The combination of IAH and the physiological effects of sepsis and septic shock may result in high morbidity and mortality rates. Especially in the case of severe peritonitis, the physiological effect of ACS to gastrointestinal tract may aggravate the abdominal sepsis. Specifically, the mucosal–barrier function is altered causing increased permeability and bacterial translocation.

11.2 Prevention of the Abdominal Compartment Syndrome in Patients with Abdominal Sepsis

Repeated intravesical measurements of intra-abdominal pressure should be frequently performed to identify patients at risk for intra-abdominal hypertension.

Although decompressive laparotomy historically constituted the standard method to treat severe IAH/ACS and to protect against their development in high-risk situations, it has been reported to result in an immediate decrease in IAP and improvements in organ function. However, decompressive laparotomy is associated with multiple complications and overall reported patient mortality is considerable (up to 50%), even after decompression [2].

In addition to decompressive laparotomy for ACS, numerous medical and minimally invasive therapies have been proposed or studied that may be beneficial for patients with IAH or ACS. Approaches or techniques of potential utility include adequate fluid resuscitation strategies, sedation and analgesia, neuromuscular blockade, body positioning, nasogastric/colonic decompression, promotility agents, diuretics, and continuous renal replacement therapies, percutaneous catheter drainage [3].

Presumptive decompression should be considered at the time of laparotomy in patients who demonstrate risk factors for IAH/ACS. The decision to perform a laparostomy in patients with severe intra-abdominal infections is usually based on the intraoperative judgment of the surgeon without IAP measurements during the operation. In these patients, the open abdomen (OA) procedure may be a useful option [3].

OA procedure may allow early identification and draining of any residual infection, control any persistent source of infection, and remove more effectively infected or cytokine-loaded peritoneal fluid, deferring definitive intervention until the patient is appropriately resuscitated and hemodynamically stable and thus better able to heal [2].

The OA concept is closely linked to damage control surgery and may be easily adapted to patients with ongoing sepsis. Patients may progress to septic shock having progressive organ dysfunction, hypotension, myocardial depression, and then coagulopathy. These patients are hemodynamically unstable and clearly not optimal for candidates for immediate complex operative interventions. After initial surgery, the patient is rapidly taken to the ICU for physiologic optimization. Early treatment with aggressive hemodynamic support can limit the damage of sepsis-induced tissue hypoxia and may limit the overstimulation of endothelial activity. Following the early hemodynamic support, in principle after 24–48 h, reoperation may be performed with or without final abdominal closure [4].

Following reexploration, the goal is early and definitive closure of the abdomen, in order to reduce the complications associated with an open abdomen, such as entero-atmospheric fistulas, fascial retraction with loss of abdominal wall domain, and development of massive incisional hernias. Early definitive closure is the basis for preventing or reducing the risk of these complications and should be the goal when the patient's physiological condition allows. The literature suggests a bimodal distribution of primary closure rates. Early closure depends on postoperative intensive care management, and delayed closure depends on the choice of the temporary abdominal closure technique. The first mode is to close within 4–7 days and achieve a high rate of primary closure, the second mode has a delay (20–40 days) having lower overall closure rate. Temporary closure of the abdomen may be achieved by using gauze and large, impermeable, self-adhesive membrane dressings, both absorbable and nonabsorbable meshes, and negative pressure therapy devices. The first and easiest method to perform a laparostomy was the application of a plastic silo (the "Bogota bag"). This system is inexpensive. However, it does not provide sufficient traction to the wound edges and allows the fascial edges to retract laterally, resulting in difficult fascial closure under significant tension, especially if the closure is delayed. At present, negative pressure techniques (NPT) have become the most extensively employed means of temporary closure of the abdominal wall [2].

OA strategy presents a clinical challenge that is associated with significant morbidity and should be used in the right patients at the right time. Even with the lack of strong evidence in international literature, OA may be an important option in the surgeon's strategy for the treatment of selected physiologically deranged patients with abdominal sepsis. Well-designed prospective and randomized studies are required to adequately define the role of OA and negative pressure in managing patients with abdominal sepsis.

11.3 Conclusions

Surgeons should be aware of physiopathology of sepsis and always keep in mind the pathophysiology of ACS. A correct prevention and management of ACS, when it occurs, is crucial to avoid severe complications.

In addition to decompressive laparotomy for ACS, numerous medical and minimally invasive therapies have been proposed or studied that may be beneficial for patients with IAH or ACS.

Despite lack of strong evidence in international literature, open abdomen may be an important option in the surgeon's armamentarium for the prevention of abdominal compartment syndrome. Well-designed prospective studies are required to better define the role of open abdomen in managing patients with abdominal sepsis.

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