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Decompressive laparotomy for abdominal compartment syndrome in children: before it is too late

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Abstract

Purpose: Abdominal compartment syndrome (ACS) in children is an infrequently reported, rapidly progressive, and often lethal condition underappreciated in the pediatric population. This under-recognition can result in a critical delay in diagnosis causing increased morbidity and mortality. This study examines the clinical course of patients treated for ACS at our institution.

Methods: A review of children requiring an emergency laparotomy (n = 264) identified 26 patients with a diagnosis of ACS. ACS was defined as sustained intraabdominal hypertension (bladder pressure >12 mm Hg) that was associated with new onset organ dysfunction or failure.

Results: Patients ranged in age from 3 months to 17 years old and were cared for in the pediatric intensive care unit (PICU). Twenty-seven percent (n = 7) were transferred from referring hospitals, 50% (n = 13) were admitted directly from the emergency department, and 23% (n = 6) were inpatients before being transferred to PICU. Admission diagnoses included infectious enterocolitis (n = 12), postsurgical procedure (n = 10), and others (n = 4). Patients progressed to ACS rapidly, with most requiring decompressive laparotomy within 8 hours of PICU admission (range, <1-96 hours). Preoperatively, all patients had maximum ventilatory support and oliguria, 85% (n = 22) required vasopressors/inotropes, and 31% (n = 8) required hemodialysis. Mean bladder pressure was 25 mm Hg (range, 12-44 mm Hg). In 42% (n = 11), cardiac arrest preceeded decompressive laparotomy. All patients showed evidence of tissue ischemia before decompressive laparotomy with an average preoperative lactate of 8 (range, 1.2-20). Decompressive laparotomy was done at the bedside in the PICU in 13 patients and in the operating room in 14 patients. Abdominal wounds were managed with open vacuum pack or silastic silo dressings. Physiologic data including fluid resuscitation, oxygen index, mean airway pressure, vasopressor score, and urine output were recorded at 6-hour intervals beginning 12 hours before decompressive laparotomy and extending 12 hours after operation. The data demonstrate improvement of all physiologic parameters after decompressive laparotomy except for urine output, which continued to be minimal 12 hours post intervention. Mortality was 58% (n = 15) overall. The only significant factor related to increased mortality was bladder pressure (P = .046; odds ratio, 1.258). Cardiac arrest

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before decompressive laparotomy, need for hemodialysis, and transfer from referring hospital also trended toward increased mortality but did not reach significance.

Conclusion: Abdominal compartment syndrome in children carries a high mortality and may be a consequence of common childhood diseases such as enterocolitis. The diagnosis of ACS and the potential need for emergent decompressive laparotomy may be infrequently discussed in the pediatric literature. Increased awareness of ACS may promote earlier diagnosis, treatment, and possibly improve outcomes.

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Abdominal compartment syndrome (ACS) can be defined as symptomatic organ dysfunction that results from an increase in intraabdominal pressure [1]. This diagnosis carries a very high morbidity and mortality and has become a growing concern in children who are critically ill.

Historically, the measurement of intraabdominal pressure began with Braune in 1865, who used rectal bougies, followed by Odebrecht in 1875, who measured intraabdominal pressure using an intravesicle device as is current practice [2]. The physiological consequences of increased abdominal pressure were first elucidated by Wendt in 1875 and again in a seminal paper published by Emerson in 1911 [2]. Wendt discovered that a rise in intraabdominal pressure was associated with decreased urine output. Using manometry studies in animals, Emerson demonstrated an inverse relationship between intraabdominal pressure and venous return to the heart. At elevated intraabdominal pressures, Emerson demonstrated death from cardiorespiratory failure. Leaping forward, the World Society on Abdominal Compartment Syndrome was founded in 2004 to define ACS, develop consensus guidelines, and improve survival in patients. The pathophysiologic effects of ACS are manifest as multiple end organ dysfunction. Upward displacement of the diaphragm reduces pulmonary compliance with progressive respiratory failure. Abdominal pressure exceeds intravascular pressure reducing hepatosplanchnic perfusion, renal blood flow, and collapsing the inferior vena cava reducing preload to the heart [3].

Treatment for ACS is emergent surgical decompression aimed at improving respiratory mechanics and restoring abdominal organ perfusion and venous return to the heart. Abdominal compartment syndrome has been well studied in adults; however, precise pressure thresholds at which organ hypoperfusion occurs in infants and children have not been defined.

1. Methods

A retrospective chart review from a tertiary care pediatric center was conducted of 264 patients who required emergency laparotomy between 2001 and 2009 after institutional review board approval (IRB#0030852). Only the 26 children who required emergency decompressive laparotomy (DL) for ACS were included in the study. Patient demographics, hospital course, treatment, and patient outcomes were reviewed. Children who did not have ACS and those less than 12 weeks old were excluded from the study. Those less than 12 weeks old typically lacked the needed bladder pressure measurements for inclusion.

The diagnosis of ACS was made if the patient had an intravesical pressure greater than 12 mm Hg associated with new onset end organ compromise including hypotension, increased ventilatory requirement, or decreased urine output less than 0.5 mL/kg per hour. Bladder pressure monitoring was evaluated with a needleless system. With the patient supine, a transducer is attached to a 3-way stopcock, a 60-mL Luer-Lok syringe is attached to the distal stopcock, and the transducer system is attached to the sampling port on the urine collection system using a needleless access device. The system is flushed, the transducer is leveled to the midaxillary line, and the urinary drainage tubing is clamped immediately distal to the sampling port. Normal saline at 1 mL/kg (maximum 20 mL) is injected into the bladder. The measurement of intraabdominal pressure is recorded from the monitor at "end-expiration" approximately 30 to 60 seconds after instillation. The system is unclamped, and the instilled fluid is allowed to drain from the bladder.

The technique of DL was variable according to surgeon preference as was the wound management device. A midline or transverse abdominal incision was made to gain access to the abdominal cavity, and the abdomen was explored. The bowel was run from the ligament of Treitz to the rectum and frankly necrotic bowel was resected, whereas at risk bowel was left for reexamination 24 to 48 hours later. An abdominal vacuum pack was created using sterile towels, an Ioban dressing, and Jackson-Pratt drains to wall suction. In survivors, abdominal wall closure was ultimately achieved primarily or with the assistance of a Whitman patch when the ansarca had resolved.

Collected data included age, sex, primary diagnosis, white cell count, bladder pressure, pre- and postoperative lactate, length of stay before operation, total length of stay, number of surgical procedures, surgical approach, days with open abdomen, days on ventilator, need for dialysis, need for vasopressor or inotropic medication, incidence of cardiac arrest, and mortality. In addition data including mean airway pressure (MAP), oxygen index (OI), vasopressor score, and urine output were recorded at 6 hourly intervals beginning 12 hours before DL and continuing for 12 hours after DL. Oxygen index was calculated as the product of the fraction of inspired oxygen (F_{IO_2}) and MAP, divided by the partial

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