Pediatr Radiol (2002) 32: 319–322
DOI 10.1007/s00247-001-0569-3

ORIGINAL ARTICLE

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Abdominal compartment syndrome in children: CT findings

Received: 10 September 2000
Accepted: 5 July 2001
Published online: 15 February 2002

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Abstract Abdominal compartment syndrome (ACS) is caused by pathological elevation of intra-abdominal pressure (IAP) leading to multiple organ dysfunction syndrome. Since the condition is highly lethal, early diagnosis is imperative. We evaluated the pre-operative abdominal CT scans of three children with proven ACS to identify signs of elevated IAP. Findings common to these patients included narrowing of the inferior vena cava (IVC), direct renal compression or displacement, bowel wall thickening with enhancement and a rounded appearance of the abdomen. The aim of recognising the CT findings in such cases is to plan emergency surgical decompression. Although these findings are not specific for increased IAP, radiologists should be aware of this life-threatening condition and, in the proper clinical setting, should communicate the presence and significance of these findings to the referring clinician.

Keywords Abdominal compartment syndrome · Children · CT · Intra- abdominal pressure

Introduction

Little attention has been given to abdominal compartment syndrome (ACS) in the radiological literature, and the abdominal CT findings of this syndrome in adults have only recently been described [1]. We report the CT findings of ACS in three children, where the elapsed time from CT evaluation to diagnosis of ACS and surgical decompression was less than 6 h.

ACS is an uncommon entity resulting from increased intra-abdominal pressure (IAP). The term ACS was first used in 1984 by Kron et al. [2], who described the effects of intra-abdominal hypertension (IAH) following surgery for aortic aneurysm [2]. The various aspects of ACS have been well described and recently reviewed in the medical literature [3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16].

The leading cause of ACS is major abdominal surgery, most commonly for trauma or repair of aortic aneurysm. Although ACS is a well-recognised entity, the total number of patients described in the literature is only about 130 [3, 4] and most patients are adults. In our experience with ten patients treated for ACS in the Paediatric Intensive Care Unit at our institution, we have found that, in contrast to adults, children with ACS have diverse primary diagnoses, with a significant number of primary extra-abdominal conditions. The aetiologies included isolated head trauma, abdominal trauma and surgery, intussusception, bowel ischaemia/necrosis and meningococcaemia. Also, children may develop ACS at lower intra-abdominal pressures than adults, as low as 20 mmHg [7, 14].

Case reports

The following three cases were chosen (out of a total of ten) because in each one laparotomy was performed less than 6 h after abdominal CT, thereby confirming the CT diagnosis of ACS.

Case 1

A 3-year-old boy was admitted with multiple injuries due to a severe road traffic accident (head-on collision). His skull was fractured and was complicated by a CSF leak. CT of the head, chest and abdomen showed basal skull fractures, open and depressed frontal skull fractures, bifrontal cerebral haematoma, bilateral lung contusions, left haemopneumothorax and rib fractures, ruptured liver and spleen, and pelvic fractures. Almost immediately after arrival, his abdomen distended rapidly and he went into profound shock. Haemostasis was achieved after emergency laparotomy with splenectomy and liver packing.

Because of haemodynamic and respiratory deterioration with abdominal distension, an abdominal CT scan was performed revealing marked abdominal distension leading to a rounded morphology of the abdomen on axial CT scans, and a relative increase in the AP diameter compared with the transverse diameter. The inferior vena cava (IVC) had a slit-like appearance and active bleeding was noted in the splenic fossa (Fig. 1a).

Abdominal distension worsened and 1 h later the child developed oliguria and hypotension, and required very high ventilator pressures. IAP, measured by urinary bladder pressure (UBP), was markedly elevated at 27 mmHg (normal 0–10 mmHg). A bedside emergency laparotomy was performed. The abdomen was left open and the bowel was exteriorised for decompression. Within a short time, ventilatory parameters and blood gases improved, blood pressure normalised and urine output resumed. Two days later, the packs were removed and the abdomen was surgically closed.

However, within a day the patient developed ACS again and the abdomen had to be opened. His course was further complicated by bacterial and candida sepsis. CT of the brain showed multiple bilateral frontal abscesses and cerebral oedema. Abdominal CT revealed gastric emphysema and marked small-bowel wall thickening with enhancement (Fig. 1b). Laparotomy revealed a necrotic stomach, which had to be resected. He died a week later of multiple organ dysfunction syndrome.

Case 2

A 7-month-old girl was transferred from a peripheral hospital with a diagnosis of intussusception not reduced by barium enema. She arrived at our hospital in shock and at emergency laparotomy, intussusception was reduced. Following surgery, there was progressive distension of her abdomen with increased respiratory resistance, hypotension, oliguria and poor peripheral perfusion. The UBP was 23 mmHg, indicative of increased IAP. Abdominal CT showed dilated bowel loops, displacement of the right kidney with mild compression of the IVC (Fig. 2) and compression of the upper pole of the left kidney.

She underwent repeat laparotomy and the abdomen was left open using a mesh cover. Blood pressure, urinary output and peripheral perfusion returned to normal and ventilator pressures returned to their pre-ACS values. She was stable for 2 days, but again developed symptoms of ACS. She underwent emergency exteriorisation of her gut. There was immediate relief of all her symptoms, with blood pressure, urine output, peripheral body perfusion and ventilating pressures all returning to the pre-ACS level.

Her hospitalisation was further complicated by small-bowel perforation, but she eventually made a full recovery.

Case 3

A 3-year-old boy with cystic fibrosis presented to the emergency room in shock following 1 week of anorexia, abdominal pain and vomiting. His abdomen was bloated and tender without guarding or rebound sensitivity. He had marked leukocytosis (68,800 WBC/mm^3). An abdominal radiograph showed dilated bowel loops, particularly on the left side. An air enema excluded intussusception. During the procedure, however, the patient suffered a cardiorespiratory arrest.

Following resuscitation, he stabilised with supportive therapy, but a few hours later the abdomen became progressively distended and tense. Abdominal contrast-enhanced CT revealed massive ascites causing mild renal parenchyma compression, thickening and enhancement of the bowel walls, and the IVC and aorta showed diminished calibre (Fig. 3). One hour later he became anuric and acidotic and required very high ventilator pressures. IAP increased

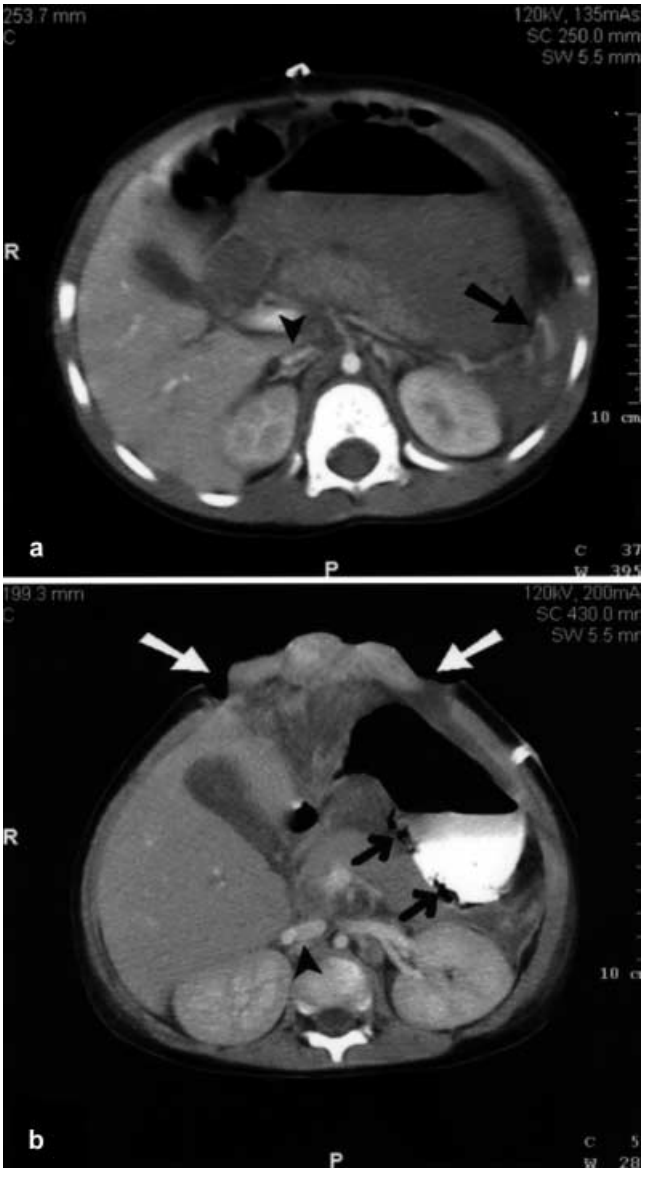

Fig. 1a, b. A 3-year-old boy with ACS after emergency laparotomy subsequent to motor vehicle accident. **a** CT performed after surgery shows active bleeding in the splenic fossa (*arrow*), narrowing of the IVC (*arrowhead*) and a rounded morphology of the abdomen. **b** CT performed 3 days later because of sepsis. Note the decompressive effect of the open wound with exteriorisation of bowel loops (*white arrows*), normal calibre of the IVC (*arrowhead*) and gastric emphysema (*black arrows*)

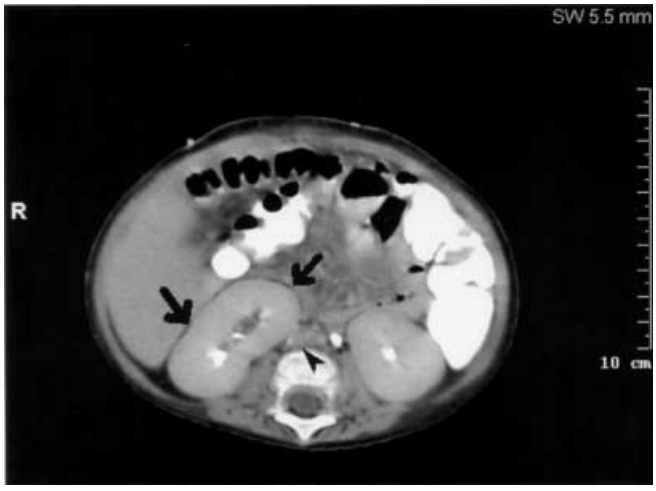

Fig. 2. A 7-month-old girl with ACS after surgery for unreduced intussusception. CT performed a few hours before decompressive laparotomy shows displacement of the right kidney (*arrows*) with mild compression to the IVC (*arrowhead*)

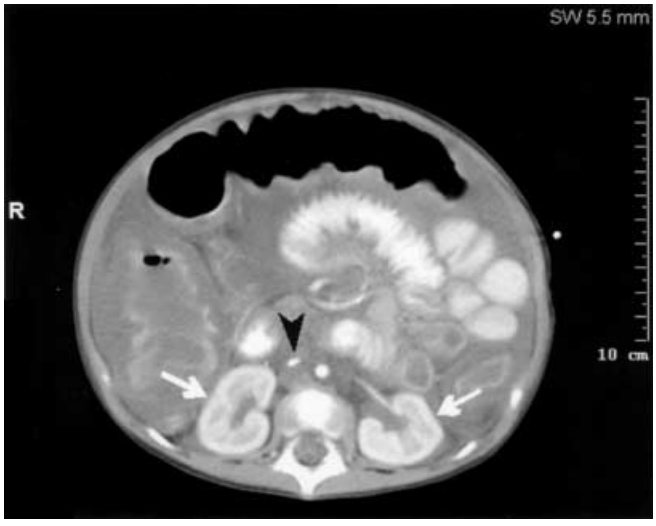

Fig. 3. A 3-year-old boy with ACS as a complication of cystic fibrosis and sepsis. CT shows marked large-bowel wall thickening and enhancement, ascites, mild pressure on the parenchyma of both kidneys (*arrows*), a slit-like IVC (*arrowhead*) and a rounded morphology of the abdomen

from 15 to 22 mmHg. Emergency laparotomy was performed and disclosed massive bowel oedema and ascites. The abdomen was left open using a mesh cover. Despite immediate improvement, with haemodynamic stability, improved respiratory functions and return of urine production, the patient eventually developed severe multiple organ dysfunction syndrome and died 3 days later.

Discussion

A compartment syndrome may be defined as a condition in which an acute increase in pressure within a confined anatomical space adversely affects the viability and function of the tissues within. A well-known instance of this is the compartment syndrome that takes place within the non-compliant fascia of the extremities. Correspondingly, ACS is a clinical entity that develops from an acute and rapid (within hours) elevation of IAP, and may result in harmful physiological consequences [7].

The physiological alterations of increased IAP classically involve the cardiovascular, pulmonary and renal systems. In addition, visceral blood flow, the central nervous system and wound healing may also be compromised by increases in IAP. Significant decreases in cardiac output consistently occur as the result of increased IAP. Compression of the IVC and other large veins results in decreased preload and impaired cardiac performance, venous stasis and thrombosis. Elevation of intracranial pressure as a result of impeded cranial venous return is also a well-known complication [8, 9]. Finally, increased IAP causes elevation of the diaphragm, which in turn increases intrathoracic pressure, further depressing cardiac output. Renal dysfunction associated with IAP results from impaired cardiac output, renal arterial and venous obstruction, and direct compression of the renal cortex [11].

ACS can develop in both non-surgical and surgical conditions, and either pre- or post-operatively in the latter [3]. Increased intra- or retroperitoneal volume is the leading cause of increased IAP, and intraperitoneal bleeding, bowel distension, abdominal packs, tense ascites, peritonitis, mesenteric venous obstruction and tumour are common associated conditions [3, 9]. Most cases, however, are multifactorial. In the paediatric population, ACS is a known complication of repair of abdominal wall defects (gastroschisis or omphalocoele). Omphalocoele has been called ''a prototype for ACS'' [13, 14].

The clinical diagnosis of ACS is not difficult once considered. The typical patient has a tensely distended abdomen, elevated intra-abdominal and peak airway pressures, depressed cardiac output, hypoxia and hypercarbia with progressively increasing airway pressures, and reduced renal function. Confirmation of this diagnosis is commonly made by measuring the UBP, an indirect method to estimate the IAP.

UBP is considered the gold standard [2], and with UBP above 25 mmHg decompression is necessary for patient salvage [14]. However, in children, ACS may develop at a lower IAP [7, 14]; therefore, the most important factor is a high index of suspicion in the appropriate clinical situation. Decompression may be strongly considered for pressures between 20 and 25 mmHg [14].

CT can provide evidence of elevation of the IAP in patients at risk of developing ACS. Findings suggesting

IAH may include narrowing of the IVC [17] that may be the result of extrinsic compression or hypovolaemia. The rounded appearance of the abdomen on axial CT scans [7], analogous to the gross physical appearance of massive abdominal distension, may be an imminent sign of IAH. Direct compression of the renal parenchyma may also be found and may be the cause of oliguria [7, 11]. Other findings of IAH are bowel-wall enhancement and thickening and bowel dilatation. Most of these findings were present in our patients. Some of these findings may be evocative of the features of the hypoperfusion complex [18].

US was not performed in our patients. Moreover, in searching the literature, we found no reports describing sonographic findings in ACS. We suppose, however, that once familiar with the morphological changes as seen by CT, the radiologist will probably be able to identify them by US as well. Further investigation is needed to verify our assumption.

The only treatment for ACS is immediate decompression. Silastic sheets or other material, such as irrigation bags, may be sutured to the fascia to provide temporary closure of the abdominal contents.

In conclusion, ACS is a life-threatening condition requiring emergent decompressive laparotomy that can be life-saving in this setting. Although ACS has been well documented in the surgical literature, it has received little attention in the imaging literature, with only one report describing the CT findings of ACS in adults [1]. The imaging findings described in adults did not differ significantly with those found in children. Despite the fact that ACS is an entity that should be diagnosed clinically, radiologists should still be aware of the CT findings suggesting increased IAP. Due to the increased use of imaging in medical care, the radiologist encounters more and more findings of conditions that were previously diagnosed clinically. By promptly recognising and reporting these findings of ACS, non-specific as they may be, the radiologist can play an important role in the care of the critically ill patient.

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