DOI: https://doi.org/10.5114/ait.2019.89189

Acute primary abdominal compartment syndrome due to *Clostridium difficile* induced toxic megacolon: a case report and review of the literature

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Abstract

Background: Without timely diagnosis, acute primary abdominal compartment syndrome (ACS) is a potentially fatal syndrome and often goes unrecognized until severe symptoms appear. Early diagnosis may significantly improve the prognosis of these patients.

Case presentation: We present the case of a 54-year-old man, successfully treated for acute myeloid leukemia with cytosine arabinoside, admitted to the intensive care unit with severe shock, refractory to standard therapy with antibiotics, fluid resuscitation, and vasopressors. Early diagnosis of acute primary abdominal syndrome was made based on an intra-abdominal pressure of 20 mm Hg (3 kPa) with new onset organ failure, after which decompressive laparotomy was performed. Stool cultures grew *Clostridium difficile*. Despite abdominal decompression, the abdominal compartment syndrome persisted with the development of toxic megacolon and a total colectomy was performed with favorable evolution.

Methods: A systematic review of published case reports was performed describing a primary ACS due to *C. difficile* toxic megacolon. A PubMed database search was performed with the following search terms, single or in combination: 'clostridium difficile', 'toxic megacolon', 'abdominal compartment syndrome', and 'CDI'. The latest search was performed for March 2019; only case reports after 1998 were included.

Results: We found a total of 19 case reports with *C. difficile* toxic megacolon (including the present case). The male/female ratio was 12/7, and there were 3 children. The mean age was 48.7 ± 23.5 years. The reason for admission was sepsis in 6, trauma in 2, postoperative in 4, enterocolitis in 5, pregnancy in 1 and abdominal complaints after topical antibiotics in 1. Three patients did not develop diarrhea. Five patients presented with diarrhea on average 5.8 ± 5.1 (median 4, 1–14) days prior to hospital admission while 7 patients developed diarrhea on average after 10 ± 19.6 (median 3, 0–54) days during admission. The intra-abdominal pressure (measured in 6 patients, including ours) was 29.2 ± 11 (20–50) mm Hg (3–7 kPa). Treatment consisted of (a combination of) vancomycin (orally or via rectal enemas), metronidazole (orally or intravenously), and surgical intervention (with decompressive laparotomy). Three patients died (15.8%).

Conclusions: Monitoring of intra-abdominal pressure allows early detection of abdominal compartment syndrome and is warranted in patients with *C. difficile* infection and/or toxic megacolon. Early decompression can lead to improved outcomes in patients with severe shock and organ failure.

Key words: abdominal pressure, abdominal hypertension, abdominal compartment syndrome, sepsis, septic shock, *Clostridium difficile*, colitis, toxic megacolon.

Anaesthesiol Intensive Ther 2019; 51, 4: 273–282

Received: 28.06.2019, **accepted:** 23.08.2019

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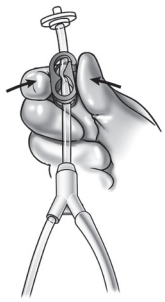
Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are increasingly recognized as causes of significant mortality and morbidity over the past decades [1]. Intra-abdominal pressure (IAP) is defined as the steady-state pressure concealed within the abdominal cavity and is affected by the volume of solid organs/hollow viscera, ascites, blood or other space-occupying lesions in combination with conditions that limit the expansion of the abdominal wall (e.g., edema). IAP is measured through a bladder catheter or nasogastric tube (Figure 1). IAH, defined as a sustained increase in IAP equal to or above 12 mm Hg (2 kPa), has numerous deleterious effects on end-organ function within and outside the abdominal cavity [2]. Detrimental consequences of IAH are reduced visceral perfusion and bowel ischemia, as well as decreased renal function due to transmission of elevated IAP on the kidney parenchyma and vascular structures leading to reduced arterial inflow and venous outflow. Reduced preload leads to activation of the renin-angiotensin-aldosterone system, further decreasing urine output [3]. Furthermore, IAH leads to elevated intrathoracic pressure, causing reduced venous return and cardiac preload, reduced end diastolic volumes and increased afterload. All these factors result in a decrease in cardiac output, leading to further organ failure. IAH can even worsen intra-cranial hypertension due to reduced cerebral venous return. It is clear that through these different mechanisms, ACS can lead to organ dysfunction and eventually death when left untreated [4]. Figure 2 summarizes the effects of IAH on end-organ function.



Initial set-up

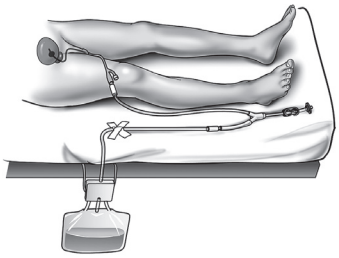
Open the Foley Manometer LV (Holtech Medical, Charlottenlund, Denmark, www.holtech-medical.com) or UnoMeter AbdoPressure (Convatec, Greensboro, North Carolina, USA) pouch and close the tube clamp.

Place the urine collection device under the patient's bladder and tape the drainage tube to the bed sheet.

Insert the FoleyManometer between catheter and drainage device.

Prime the FoleyManometer with 20 mL of sterile saline through its needle-free injection/sampling port.

Prime only once, i.e. at initial set-up, or subsequently to remove any air in the manometer tube.



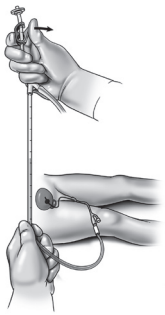
Urine drainage

Let the urine drain in between IBP measurements.

Urine sampling from the needle-free port is facilitated by temporarily opening the red clamp. Remember to close clamp afterwards.

Avoid a U-bend of the large urimeter drainage tube (which will impede urine drainage).

Replace the FoleyManometer whenever the Foley catheter or the urine collection device is replaced, or at least every 7 days.



Intravesical pressure monitoring

Place the "0 mm Hg" mark of the manometer tube at the midaxillary line at the level of the iliac crest (mark for future reference) and elevate the filter vertically above the patient.

Open the bio-filter clamp, and read IBP (end-expiration value) when the meniscus has stabilized after about 10 seconds.

Close clamp after IBP measurement and place the FoleyManometer in its drainage position. Normal IBP is 5–7 mm Hg (0.7–0.9 kPa).

FIGURE 1. Measurement of intra-abdominal pressure (IAP) through bladder catheter with the FoleyManometerLV.
This technique that uses the patient's own urine as pressure transmitting medium is a surprisingly simple, reliable, and cost-effective clinical tool. Based on a modified version of the IAP monitoring technique described by Kron *et al*. [17], the disposable Foley Manometer (or UnoMeter AbdoPressure) provides a closed sterile circuit which connects between the patient's Foley catheter and the urine collection device. Each IAP determination takes about 10 seconds, and no subsequent correction of urine output is required. The technique uses a low bladder infusion volume, has a needle-free sampling port and can measure IAP in a range of 0–40 mm Hg (0–5 kPa). Therefore, it is an ideal technique to screen critically ill patients for IAH

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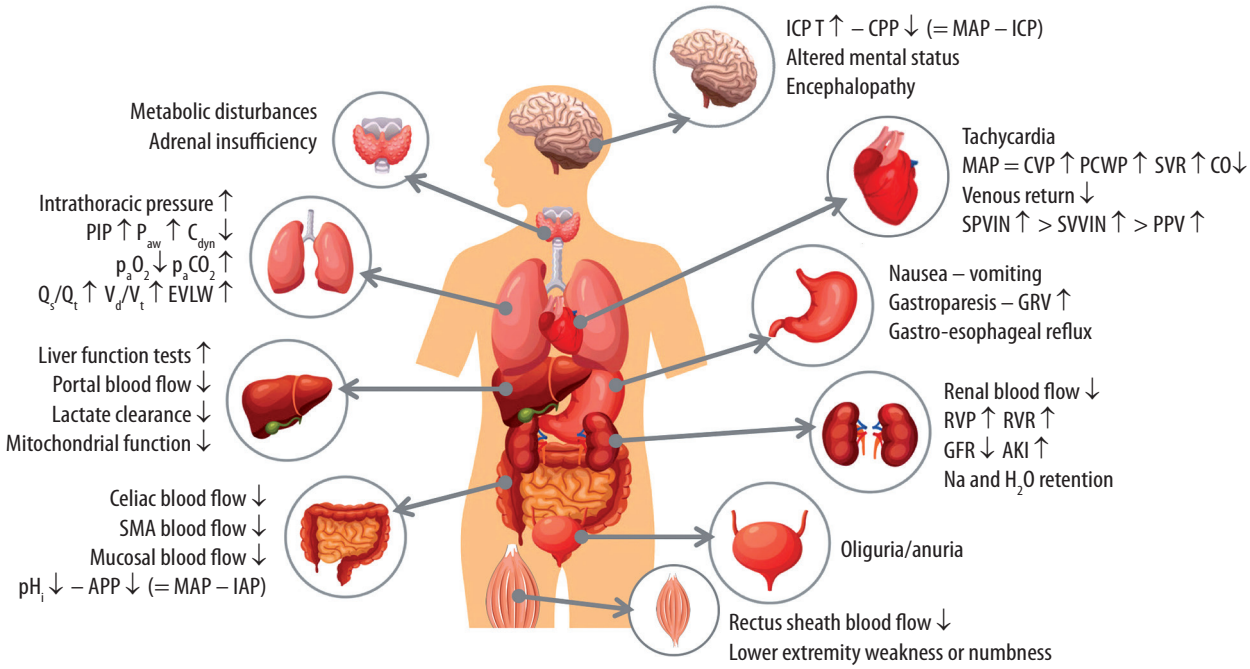

FIGURE 2. Summary of the most important pathophysiological effects of increased intra-abdominal pressure on end-organ function within and outside the abdominal cavity

Adapted from Regli *et al.* [36] with permission under the Open Access CC BY License 4.0.

ACS is defined as a sustained increased IAP > 20 mm Hg (> 3 kPa) with associated new organ failure and can be primary or secondary in origin (Table 1). The incidence of ACS in a mixed population of critically ill patients is estimated to be around 10–35% [5]. Average mortality of ACS is estimated to be around 50% [6]. Patients with ACS are often very ill, and organ dysfunction may be incorrectly ascribed to the progression of the primary illness. Furthermore, symptoms, clinical findings and imaging are insufficient or aspecific to diagnose ACS [7]. We present the case of a 54-year-old man with primary ACS [8] to emphasize the importance of early recognition of ACS since early therapy leads to improved organ function and survival, followed by a review of the present literature.

CASE PRESENTATION

A 54-year-old man with a history of acute myeloid leukemia (AML), in complete remission after cytosine arabinoside therapy, was admitted to the Intensive Care Unit from the hematology ward (ZNA Stuivenberg) because of progressive signs of shock. This case has been previously described in part [8]. At admission, he was awake but agitated with a Glasgow Coma Scale (GCS) of 14/15 (E4V4M6), blood oxygen saturation was 93% under oxygen therapy of 8 liters per minute with a respiratory rate of 32 per minute and hypoventilation of both lung bases. Blood pressure was 95/50 mm Hg with sinus tachycardia of 130 bpm with a pale aspect and a prolonged capillary refill time of 4 seconds. Central venous pressure (CVP) was 20 mm Hg (3 kPa). There was significant abdominal distention, diffusely painful at palpation without noticeable peristalsis. The most remarkable laboratory results were an acute kidney injury with creatinemia of 1.95 mg dL^{-1}, urea of 80 mg dL^{-1}, highly elevated inflammatory parameters and normochromic anemia of 8.9 g dL^{-1}. Lactate was only slightly elevated at 2.8 mmol L^{-1} at admission. The IAP at admission was 20 mm Hg (3 kPa). Despite aggressive fluid resuscitation, hypotension persisted with rising lactate levels and the patient developed progressive respiratory failure, after which he was sedated and intubated. Furthermore, the patient became oliguric with diuresis of < 0.5 mL kg^{-1} h^{-1}. Urgent bedside echocardiography showed low output with an estimated left ventricular ejection fraction of 30%, a mitral regurgitation of 2–3/4, a left ventricular end-diastolic pressure (LVEDP) of 25 mm Hg (3 kPa) and a dilated inferior vena cava of 21 mm but with respiratory variation. Inotropics (dobutamine) and vasopressors (norepinephrine) were initiated together with further fluid resuscitation. A computed tomography (CT) scan of the abdomen showed a dilated cecum of 16 cm, a diffusely thickened colon wall (4–6 cm), air in the cecum wall and extensive ascites (Figure 3). Given an IAP of 20 mm Hg (3 kPa), new onset organ failure (oliguria) and presence of extensive colitis on CT the diagnosis

TABLE 1. Summary of case reports in abdominal compartment syndrome in *Clostridium*-induced toxic megacolon since 1998

No.	Author	Year	Reference	Patient age (years)	Gender	Reason for admission	Development of diarrhea (days before admission)	Development of diarrhea (days after admission)	IAP (mm Hg)	ACS diagnosis	Treatment	Outcome	Closure of abdominal fascia
1	Jennings *et al.*	1998	12	53	F	Burn victim (34% BSA) – no previous antibiotics; silver sulfadiazine dressings					Nasogastric tube, exploratory laparotomy with right hemicolectomy and ileostomy	Full recovery	Not mentioned
2	Dobson *et al.*	2003	31	69	M	Pneumonia with need for invasive ventilation – cefotaxime/clarithromycin	Diarrhea but day of onset not mentioned	Diarrhea but day of onset not mentioned		IAP not mentioned	Enteral metronidazole – subtotal colectomy	Full recovery	
3	Shaikh *et al.*	2008	20	36	M	Polytrauma with traumatic brain injury – antibiotherapy for scalp wound		7	30	IAP 30 mm Hg (4 kPa), oliguria	Oral metronidazole, neostigmine and nasogastric tube	Full recovery	
4	Oud *et al.*	2010	9	61	F	Respiratory sepsis		54	30	IAP 30 mm Hg (4 kPa), oliguria and increased respiratory failure	Oral vancomycin, nasogastric and rectal tube	Death	
5	Sayedy *et al.*	2010	13	64	F	Nausea, abdominal pain and diarrhea – treated for urinary tract infection 14 days earlier with ciprofloxacin	3				Oral vancomycin, IV metronidazole, vancomycin enemas; no surgical management	Full recovery, discharge day 23	
6	Candiotto *et al.*	2010	33	39	F	Week 29 of pregnancy, 14 days of diarrhea – no previous antibiotherapy	14			IAP not mentioned	IV metronidazole, oral vancomycin; C-section on week 32	Full recovery	
7	Mann *et al.*	2011	25	54	M	Abdominal pain, diarrhea and fever. 10-day therapy with clindamycin for oral abscess 2 weeks prior				IAP not mentioned	IV metronidazole, total colectomy with ileostomy; vancomycin enema later	Full recovery	

TABLE 1. Cont.

No.	Author	Year	Reference	Patient age (years)	Gender	Reason for admission	Development of diarrhea (days before admission)	Development of diarrhea (days after admission)	IAP (mm Hg)	ACS diagnosis	Treatment	Outcome	Closure of abdominal fascia
8	Griniatsos *et al.*	2011	34	92	M	Total nephrectomy for adenocarcinoma. 3 days of ampicillin/sulbactam postoperatively	No diarrhea	No diarrhea		IAP not mentioned	IV metronidazole and oral vancomycin, surgery declined	Death	
9	Cheng-Ming *et al.*	2011	28	24	M	Infectious enterocolitis	7			IAP not mentioned	Exploratory laparotomy with subtotal colectomy and ileostomy	Full recovery	
10	Castillo *et al.*	2012	29	4	M	Heart transplant patient, septic shock	No diarrhea	No diarrhea	20	20 mm Hg, persistent shock	Vancomycin rectally and IV metronidazole, exploratory laparotomy with TAC, terminal cecostomy and ileostomy	Full recovery	Closure 1 month after admission ICU
11	Tan *et al.*	2012	11	60	M	Abdominal pain and bloating – no antibiotic use though topical silver sulfadiazine for pemphigus vulgaris		2		IAP not mentioned	IV metronidazole and oral vancomycin, subtotal colectomy with ileostomy	Death on day 12 of admission	
12	Patel *et al.*	2012	32	10	F	Heart transplant – 3 weeks earlier ceftriaxone for MSSA bacteremia		3	25	25 mm Hg and acute kidney injury	Metronidazole (oral then IV), subtotal colectomy and ileostomy, vancomycin enemas	Full recovery	Continuity repair six months later
13	Thai *et al.*	2014	21	49	M	Postoperative inguinal hernia repair – single shot cefazolin		4	50	IAP 50 mm Hg, AKI and respiratory failure	Oral vancomycin and metronidazole	Full recovery	
14	Costello *et al.*	2014	22	69	F	Fever, abdominal pain and diarrhea – treated for CDI 3 weeks earlier	4				Oral vancomycin and IV metronidazole, surgery refused – treated with fecal transplantation	Full recovery	

TABLE 1. Cont.

No.	Author	Year	Reference	Patient age (years)	Gender	Reason for admission	Development of diarrhea (days before admission)	Development of diarrhea (days after admission)	IAP (mm Hg)	ACS diagnosis	Treatment	Outcome	Closure of abdominal fascia
15	Gweon *et al.*	2015	23	74	M	Transfer from another hospital for septic shock		0		IAP not mentioned	IV metronidazole, oral vancomycin, nasogastric tube; fecal microbiotic transplantation	Full recovery, discharge day 29	
16	Kerstens *et al.*	2016	30	58	M	Planned sigmoidectomy after complicated diverticulitis – 48 h of amoxicillin-clavulanic acid postoperatively	No diarrhea	No diarrhea		IAP not mentioned	IV metronidazole (oral and rectal) and blowhole colostomy	Full recovery	
17	Lorenzo *et al.*	2016	26	9	M	Cystic fibrosis patient, admitted for fever and abdominal pain after recent antibiotherapy for respiratory infection				IAP not mentioned	Decompressive laparotomy with Bogota bag	Full recovery	
18	Oguri *et al.*	2019	27	46	F	Infectious enterocolitis	1			IAP not mentioned	Oral vancomycin and IV metronidazole	Full recovery	
19	Our patient	2019	8	54	M	Sepsis, immunocompromised due to chemotherapy for AML		0	20	IAP > 20 mm Hg, oliguria and abdominal distension	Vancomycin-switch linezolid – decompressive laparotomy, TAC with Bogota bag; relapse of ACS and total colectomy with VAC as TAC	Full recovery	
Mean				48.7	M		5.8	10.0	29.2			Mortality ($n = 3$)	
SD				23.5	12		5.1	19.6	11.1			15.8%	
Min				4	F		1	0	20				
Max				92	7		14	54	50				

of acute primary ACS was made. Urgent surgical decompression was performed, and confirmed ACS and cecal hypoperfusion. Temporary abdominal closure (TAC) was performed with a Bogota bag. Stool cultures showed a toxin-producing *Clostridium difficile*; abdominal fluid cultures grew vancomycin-resistant *Enterococcus* and *Candida* infection, after which antibiotherapy was adapted to meropenem, fluconazole, and linezolid. Immediately after abdominal decompression, urine output increased, and the need for vasopressors decreased. However, on the next day, there was ventilatory failure (increasing $paCO_2$), hypotension despite high dose vasopressors and again a drop in urine output. Because of rising IAP despite the open abdomen urgent surgical reassessment of the abdomen was made, showing severe transmural colitis, and a total colectomy was performed with a VAC dressing as TAC. Afterwards, preload and cardiac output increased as well as urine output, followed by normalization of IAP and lactate levels. On day 5, the patient developed acute respiratory distress syndrome (ARDS), as shown in Figure 4, for which lung protective ventilation was initiated in combination with de-resuscitation, initially with hyperoncotic albumin

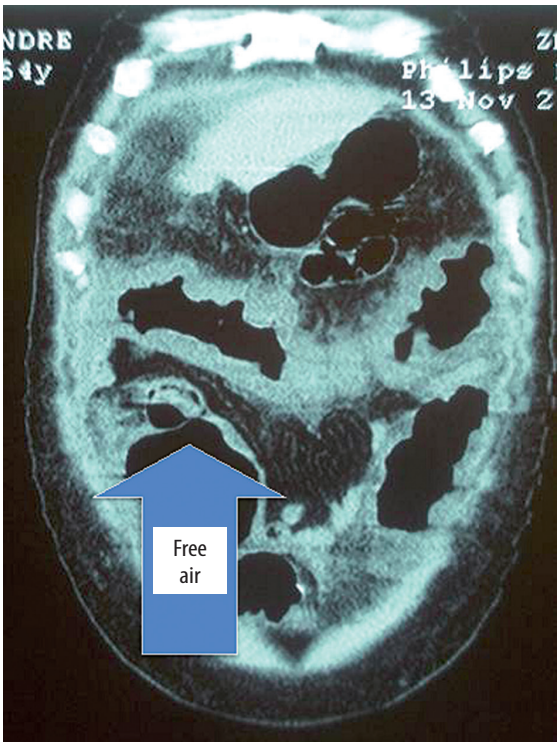

FIGURE 3. Coronal view of computed tomography abdomen showing dilatation of the cecum (16 cm), thickened colon wall (4–6 cm), free air in the cecum and ascites

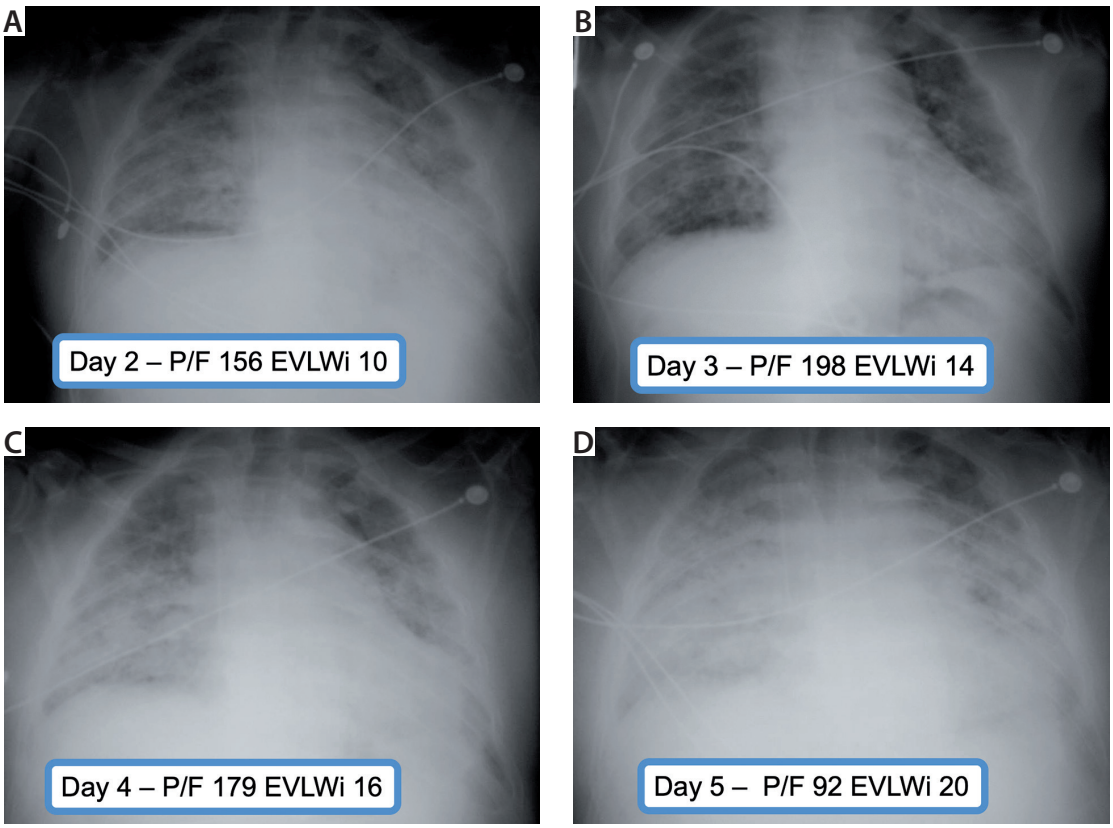

FIGURE 4. Evolution the plain chest radiograph during intensive care unit stay. The patient developed progressive respiratory insufficiency during treatment. Echocardiography showed no signs of congestion, compatible with acute respiratory distress syndrome (ARDS). Primary abdominal compartment syndrome is often associated with secondary ARDS

P/F – pO_2 over FiO_2 ratio, EVLWi – extravascular lung water index (normal < 7 mL kg^{-1} predicted body weight)

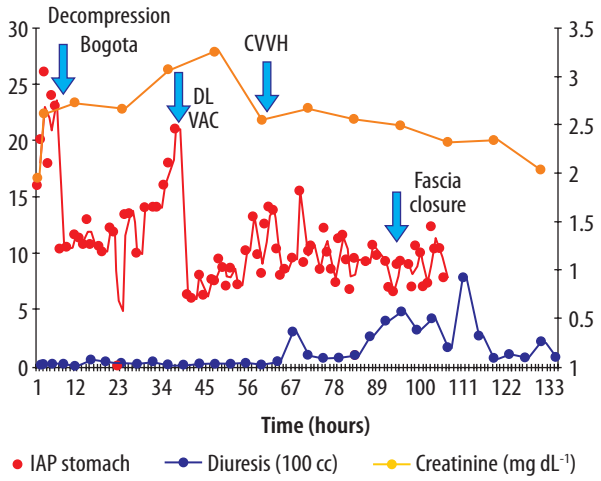

FIGURE 5. Evolution of serum creatinine, intra-abdominal pressure and dialysis during intensive care unit stay. Serum creatinine decreases (secondary Y-axis in mg dL^{-1}) and diuresis increases when intra-abdominal pressure (IAP) is normalized (primary Y-axis in mm Hg)

CVVH – continuous veno-venous hemofiltration with net ultrafiltration, DL – decompressive laparotomy, VAC – vacuum therapy

20% and diuretics and afterwards with continuous veno-venous hemofiltration (CVVH) with ultrafiltration. On day seven the patient was extubated, and he was discharged from the ICU to the hematology ward on day 10. Figure 5 shows the evolution of the physiologic parameters.

METHODS

A systematic search of PubMed and NCBI databases was performed for studies describing cases of *Clostridium difficile* infection (CDI) causing toxic megacolon and primary ACS. Search terms used were: 'clostridium difficile', 'toxic megacolon', 'abdominal compartment syndrome', single or in combination. Related articles were used to broaden the search and citations were scanned for relevance. The last search was performed for March 2019.

RESULTS

We found 18 case reports relevant to the subject. Table 1 summarizes the 19 cases of CDI with ACS. The male/female ratio was 12/7, and there were 3 children. The mean age was 48.7 ± 23.5 years. The reason for admission was sepsis in 6 patients (of whom 2 were immunocompromised after heart transplant and with cystic fibrosis), trauma in 2 (one with severe burns), postoperative in 4 (one heart transplant, one nephrectomy), enterocolitis in 5, pregnancy in 1 and abdominal complaints after topical antibiotics in 1. Three patients did not develop diarrhea. Five patients developed diarrhea on average 5.8 ± 5.1 (median 4, range 1–14) days before hospital admission while 7 patients developed diarrhea on average after 10 ± 19.6 (median 3, range 0–54) days during admission. The mean IAP (measured in 6 patients, including ours) was 29.2 ± 11 (range 20–50) mm Hg (3–7 kPa). Treatment consisted of (a combination of) vancomycin (orally in 10 and rectal enemas in 4), metronidazole (orally in 4 and IV in 11), and surgical intervention (with decompressive laparotomy) in 12 (surgery was refused in 2). Neostigmine was added as adjuvant treatment in 1 patient while fecal transplantation was performed in 2. Three patients died (15.8%).

DISCUSSION

Incidence of *C. difficile* colitis has been increasing in recent years [9] and severe *C. difficile* infection (CDI) can cause severe morbidity and mortality. Severe CDI is defined as fulminant colitis, toxic megacolon or perforation and is thought to occur in 8% of cases, with an estimated mortality of 30–80% [10]. Toxic megacolon often leads to IAH. We describe a case of IAH leading to abdominal compartment syndrome with the need for exploratory laparotomy and temporary abdominal closure with a Bogota bag. Despite the presence of an open abdomen, the patient developed ACS, for which he subsequently received total colectomy. To our knowledge, this is the first case report which describes a new ACS despite decompressive laparotomy (excluding the trauma setting). We performed a review of current literature for case reports of abdominal compartment syndrome in the setting of CDI.

Since the symptoms and clinical and radiological findings of IAH are not very specific and patients with ACS are usually very ill, there can be a delay in diagnosis and adequate treatment. Severe CDI with toxic megacolon can occur without diarrhea and thus diagnosis is sometimes postponed [8]. Failure to recognize IAH before the development of ACS

TABLE 2. Primary versus secondary intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS)

	Primary IAH/ACS	Secondary IAH/ACS
Definition	IAH or ACS due to abdominopelvic injury or disease	Due to conditions that do NOT originate in abdominopelvic region but which require extensive fluid resuscitation (–> splanchnic reperfusion)
Synonyms	Surgical, abdominal	Medical, extra-abdominal
Example	Liver, spleen or hollow viscus trauma, hemoperitoneum, pancreatitis, perforated viscus, peritonitis, ruptured AAA	Fluid resuscitation, severe burns, sepsis

causes tissue hypoperfusion, which may lead to multisystem organ failure, and potentially death. Monitoring IAP for early detection of IAH and ACS in critically ill patients not only can be helpful in diagnosis, leading to early intervention and an improved outcome for the patient, but also in management (e.g., oliguria due to ACS should not lead to more aggressive fluid resuscitation, but to fluid removal or decompression). Since treatment can improve organ dysfunction, it is essential that diagnosis is considered in the appropriate clinical situation. Studies show that ACS will not develop under 10 mm Hg (1 kPa), while ACS is mostly sure at an IAP of 25 mm Hg (3 kPa) or above [5]. ACS should be suspected when IAP is in the grey zone in between, and there is new onset organ failure. Acute kidney injury (AKI) with oliguria is often one of the first findings of ACS as AKI is considered as the canary in the coalmine for IAH. The most important step in the treatment of IAH/ACS is prevention or early recognition through IAP monitoring.

Risk factors for development of severe CDI are recent antibiotic therapy and an immunosuppressed status. Two case reports describe the development of toxic megacolon due to CDI after administration of topical antibiotics in burn patients [11, 12]. Clindamycin, cephalosporins and fluoroquinolones are the most common provoking antibiotics [13].

Treatment of IAH consists of measures to increase abdominal wall compliance, amongst other treatment modalities. Careful fluid management is essential to avoid fluid overload due to aggressive fluid resuscitation, which may lead to increased IAP due to second and third space fluid accumulation. Therefore, advanced hemodynamic monitoring (e.g. with transpulmonary thermodilution) should be performed in these patients to guide fluid, inotrope and vasopressor therapy. Barometric based assessment of intravascular volume (such as CVP) has been shown to be erroneous in IAH [14]; volumetric-based parameters (such as global end-diastolic volume) are better to guide fluid therapy [15]. The evacuation of intraluminal fluid contents (through a nasogastric or rectal tube) and percutaneous drainage of intra-abdominal fluid collections have also been proposed to reduce IAP. Furthermore, administration of diuretics or even the use of renal replacement therapy with ultrafiltration (UF) in patients with profound capillary leak are recommended to reduce IAP in hemodynamically stable patients. Dabrowski *et al*. found that in septic shock patients continuous venovenous hemofiltration with net UF successfully reduced IAP [16]. However, it remains unknown whether strategies that target a neutral or even negative fluid balance may be correlated with improved clinical outcome [17].

The most effective and definite treatment (in the setting of primary ACS), with the most supporting evidence, is urgent abdominal decompression through median laparotomy [18]. Though several techniques exist, some form of temporary abdominal closure should be performed. Urgent decompression should be considered in patients with ACS refractory to conservative medical measures or patients with an IAP > 20 mm Hg (3 kPa) and worsening organ failure. This decision should be made on an individual basis with an assessment of risks and benefits for the patient. Even after decompression, monitoring of IAP should be continued, since ACS can still occur in patients with an open abdomen and temporary closure and should warrant new surgical exploration (as in our case presented herein). Patients undergoing decompressive laparotomy should have early (within 7 days), or at least same-hospital-stay abdominal fascial closure. Early fascial closure is associated with reduced mortality and complications [19].

Although there are no current guidelines for the management of severe CDI, most authors suggest initial conservative management with IV metronidazole and vancomycin enterally [20]. There is no consensus as to whether vancomycin should be administered orally or rectally. Some authors suggest that this should depend on the most dominantly affected part of the intestine (enema for transverse and distal colon, oral route for ascending colon or small bowel). We found 1 case report with small bowel disease related to CDI [21]. The role of newer agents (e.g. fidaxomicin) has yet to be determined. Two case reports describe treatment with fecal microbiota transplantation, with full recovery [22, 23].

For patients with severe CDI combined with selected host factors (age > 70 years) and laboratory test results (WBC > 15 G L^{-1}; creatinine > 1.5 × baseline; albumin < 3 g L^{-1}) and the clinical triad of abdominal pain, abdominal distension, and diarrhea, or toxic megacolon, surgical management is indicated. The prior standard procedure was colectomy, but more recently an alternative procedure has been a diverting ileostomy with colonic lavage using vancomycin and metronidazole [24].

CONCLUSIONS

In conclusion, IAP should be monitored early in critically ill patients at risk for IAH, so that measures can be undertaken to prevent ACS. The incidence of ACS in CDI is low, with only 19 cases reported in the literature. We recommend monitoring of IAP in patients with toxic megacolon caused by CDI. If ACS occurs, hemodynamic monitoring should be used to guide fluid therapy through volumetric-based parameters to avoid fluid overload and fluid accumula-

tion. Early surgical decompression with TAC should be performed if there is progressive organ failure under medical management. Early fascial closure should be attempted if the outcome is favorable.

ACKNOWLEDGEMENTS

1. Special thanks to Rita Jacobs for reading the final manuscript and suggesting some minor edits.
2. Financial support and sponsorship: none.
3. Conflicts of interest: none.

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