Predictors of exploration in patients at high risk of abdominal compartment syndrome

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Context
Abdominal compartment syndrome (ACS) is caused when an acute increase in intra-abdominal pressure (IAP) occurs sufficient to impair vascular inflow and thereby compromising the viability of the tissues and organs within the abdomen. Familiarity with the presentation of ACS is mandatory for all clinicians who care for critically ill patients to avoid its high mortality if not recognized and treated. The measurement of IAP is a vital part of clinical management of ACS. Early recognition is important, and the need for surgical decompression may be urgent.

Aim
The aim was to evaluate the cases at high risk for development of ACS trying to prevent its fatal adverse effects by defining its predictors.

Patients and methods
This prospective, interventional, non-randomized clinical trial was conducted at Mansoura University and emergency hospitals over the period from February 2016 to February 2019. This study was conducted on patients at high risk of intra-abdominal hypertension. Two groups were included. The first group was managed conservatively, and the second one received decompressive laparotomy depending on clinical parameters, including IAP, and general status. Data were analyzed using SPSS v-24.

Results
A total of 40 patients who had an increased IAP greater than or equal to 20 cmH_2O with several clinical presentations were included. These patients had respiratory distress (90%) and distended tense abdomen, whereas abdominal pain and oliguria were only present in ~25% of cases. Overall, 20 (50%) patients underwent conservative management, and all these cases passed normally. Moreover, 20 (50%) patients underwent decompressive laparotomy. Univariate and multivariate analyses showed central venous pressure, BMI, urinary bladder pressure, and postoperative organ failure were found to be significant independent risk factors that increased the rate of mortality. The complications of the first group were owing to the primary operation and those of the second of group were owing to the primary operation as well as owing to operative decompression procedure. The mortality of the studied groups was 25% in the second one only, and there was a significant difference between both groups in the hospital stay.

Conclusion
These results had suggested that early detection of cases with ACS and proper management may be curative, and they could decrease multiorgan dysfunction and mortality in such cases. The avoidance of early abdominal closure, which may be distressing to the patients, could be mandatory in such conditions.

Keywords:
abdominal compartment syndrome, exploration, high risk, predictors

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Introduction

Abdominal compartment syndrome (ACS) is caused when an acute increase in intra-abdominal pressure (IAP) occurs sufficient to impair vascular inflow and thereby compromising the viability of the tissues and organs within the abdomen. It can occur in correlation with abdominal operations, various infection, or other causes and is easily mistaken as adult respiratory distress syndrome (ARDS) or multiple organ dysfunctions (MODS) [1,2]. In the presence of ACS, decompressive laparotomy (DL) as well as the use of laparostomy is the management of choice, as this has been reported to lead to recovery of circulatory, respiratory, and renal problems. Various prosthetics have been proposed for temporary abdominal closure

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(TAC), for example, plastic bags, towel clips, vaccum assissted closure (VAC), zipper systems, and absorbable meshes [3].

Control of intra-abdominal infection and recompensation of the circulation eventually permits the definitive closure of the abdominal wall. In case intestinal edema is persistent, the need for the laparostomy might exceed 14 days, a period beyond which the formation of granulation tissue and adhesions is considered to make the definitive closure increasingly dangerous or even impossible. This condition occurs in up to 90% of surviving victims, which eventually leads to a large abdominal wall hernia requiring mesh-augmented repair [4].

The measurement of IAP is a vital part of clinical management of ACS. Early recognition is important, and the need for surgical decompression may be urgent [5,6]. This prospective study was conducted to define the predictors of exploration in patients at high risk of ACS, trying to prevent its morbidity and mortality.

Patients and methods

This prospective study was conducted on all cases at high risk of developing intra-abdominal hypertension (IAH) and ACS admitted to Mansoura University and Emergency Hospitals in the period from February 2016 to February 2019.

All patients of both sexes of all age groups with etiologic factors leading to IAH were recruited. The etiologic factors included three main categories:

(1) The postoperative group: postoperative intra-abdominal hemorrhage, any major abdominal surgery, tight abdominal closure, repair of giant abdominal hernia, and intra-abdominal collections or peritonitis;.
(2) The post-traumatic group: damage control laparotomy, polytraumatized patients with torso trauma, and intraperitoneal or retroperitoneal bleeding.
(3) The nontrauma group: abdominal infection, that is, peritonitis, intra-abdominal abscess, acute pancreatitis; and ileus of any origin.

All patients who developed ACS or had more than one organ failure were excluded from the study.

All patients were subjected to proper history taking and thorough clinical examination, including patient demography, information of associated co-morbidity, mode and time of trauma, and concurrent injury. All patients were investigated regarding complete blood count, urea, electrolytes, liver functions, coagulation profile, blood sugar level, and blood gases.

They also underwent the following:

(1) Chest radiography and radiograph of abdomen and pelvis, erect and supine, and pelviabdominal ultrasound.
(2) Computed tomography scan was done when indicated.
(3) Assessment of the patients according to MOD scoring system [7].
(4) Serial measurement of the IAP was done every 8 h.

The technique of measurement of the IAP

The wall of the urinary bladder acts as a passive diaphragm when the bladder volume is between 50 and 100 ml. The pressure determination was done through a transurethral catheter.

Sterile saline (50 ml) was injected in the empty bladder through the indwelling Foley catheter. The sterile tubing of the urinary drainage bag was cross-clamped just distal to the aspiration port. The end of the drainage bag tubing was connected to the indwelling Foley catheter. The clamp is released just enough to allow the tubing proximal to the clamp to flow with fluid from the bladder and then reapplied. A 16-gauge needle is then used to Y-connect the infusion set after cleared from air bubbles through the aspiration port of the tubing to the drainage bag, and pressure was measured in centimeters or a pressure transducer when the column of saline in the infusion set stopped. The top of the symphysis pubis is used as the 0 point with the patient supine.

Different modalities of treatment

If the IAP increased above 20 cmH_2O and sustained for 12 h, the patients was given a diagnosis of IAH, and they were closely observed, and additional data were collected including hemodynamic variables [heart rate (HR), arterial blood pressure (ABP), and central venous pressure (CVP)], respiratory variables [respiratory rate (RR) and arterial blood gas (ABG)], renal variables (urine output and serum creatinine), and amount and composition of fluids of resuscitation before and after the increased IAP.

If there is no evidence of MODS, these patients were treated conservatively with close observation, and this is the first group of patients in our study. The second one included patients with evidence of MODS, and

early management of these patients was conducted by DL and exploration according to the patients' condition and clinical presentation. Closure of the abdomen can be done using a temporary method of closure, for example, towel clips, Bogota bags, and VAC. Then, delayed definitive closure can be done to prevent recurrence of IAH. All patients undergoing decompression laparotomy were observed in the postoperative period with respect to recording of morbidity and mortality (if occurred).

Conservative treatment of intra-abdominal hypertension

It was directed to definite goals, which were divided into five groups:

(1) Improvement of abdominal wall compliance (good analgesia, body positioning, and sedation).
(2) Evacuation of intraluminal contents (Ryle, Enema, and Prokinetics).
(3) Evacuation of peri-intestinal and abdominal fluids (tube drainage).
(4) Optimization of fluid balance.
(5) Specific treatment.

Decompressive laparotomy technique

Exploration and surgical management were tailored according to the patients' condition and clinical presentation. In the operative theater, under anesthesia (general or local), the incision of DL was done through the incision of primary laparotomy, and the specific treatment was done for each patient separately. Closure of the abdomen was done using a temporary method of closure, for example, Bogota bag (Bogota bag was simply a homemade urinary bags or a 3 l sterile plastic bag). The surgeon stitched it over the guts to the sides of the sheath of the abdominal incision to preserve the skin for subsequent closure.

This allowed the surgeon to check through the clear window of the plastic bag and see if the guts were healthy, and we put drains under the bag to drain fluid. Then, definitive closures were done either early (within 1 week) by anatomical repair or delayed (after 3–6 months) by mesh repair of planned incisional hernia. All patients undergoing decompression laparotomy were observed postoperatively with reporting of morbidity and mortality for 6 months after discharge. All of these data were collected in a special performed sheet and tabulated for statistical analysis (SPSS version 24; IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.). Univariate analysis was performed to assess the possible risk factors for mortality, including age, sex, IAP grade, and other clinical parameters. Variables with risk of mortality were assessed for odds ratio and 95% confidence interval. Significant factors with risk of mortality were assessed by multivariate logistic regression model to define independent predictors of mortality.

Results

This prospective study included 40 patients, with a mean age of 48.95 years. The BMI ranged from 22 to 36, with a mean of 31.66 (Table 1). The demographic characteristics of patients with IAH in this study were represented. It was evident that most of patients of our study were of old ages, and the predominant sex was the female sex, with BMI for most of the cases was more than average (>31.66).

Regarding the co-morbidities of patients with IAH (Table 2), four (10%) cases had a history of diabetes mellitus, two (5%) cases had a history of chronic obstructive pulmonary disease (COPD), three (7.5%) cases were associated with liver cirrhosis, and two (5%) cases had hypertension.

Regarding the causes of IAH, the most common causes of IAH in our study were huge abdominal hernias, peritonitis of different etiology, intestinal obstruction, and blunt abdominal trauma (Table 3).

The clinical presentations of patients of IAH are represented in Table 4. Most cases (≥90%)

Table 1 Demographic characteristics of patients with IAH

Variable	
Age (years)	
Range	17–80
Mean±SD	48.95±17.73*
Sex [*n* (%)]	
Male	17 (42.5)
Female	23 (57.5)
BMI	
Range	22–36
Mean±SD	31.66±5.24*

IAH, intra-abdominal hypertension.

Table 2 The comorbidities of patients with IAH

Comorbidities	Groups		Total (*n*=40)
	First (*n*=20)	Second (*n*=20)	
Diabetes mellitus	2 (10)*	2 (10)	4 (10)
Bronchial asthma	1 (5)	1 (5)	2 (5)
Liver cirrhosis	0	3 (15)	3 (7.5)
Hypertension	2 (10)	0	2 (5)
Total	5 (25)	6 (30)	11 (27.5)

IAH, intra-abdominal hypertension.

Table 3 Causes of IAH in the studied groups of patients

Causes	Groups		Total (*n*=40)
	First (*n*=20)	Second (*n*=20)	
Hernia	6 (30)*	6 (30)	12 (30)
Peritonitis	3 (15)	7 (35)	10 (25)
Intestinal obstruction	3 (15)	2 (10)	5 (12.5)
Blunt abdominal trauma	3 (15)	4 (20)	7 (17.5)
Intestinal fistula	0	1 (5)	1 (2.5)
Abdominoplasty	2 (10)	0	2 (5)
Postbariatric surgeries	3 (15)	0	3 (7.5)
Total	20 (100)	20 (100)	40 (100)

IAH, intra-abdominal hypertension.

Table 4 Clinical presentations of IAH

Clinical presentation	Groups		Total (*n*=40)
	First (*n*=20)	Second (*n*=20)	
Abdominal pain	3 (15)*	8 (40)	11 (27.5)
Tense abdomen	16 (80)	20 (100)	36 (90)
Respiratory distress	20 (100)	20 (100)	40 (100)
Oliguria	0	11 (55)	11 (27.5)
Refractory metabolic acidosis	0	3 (15)	3 (7.5)
Fever	6 (30)	12 (60)	18 (45)
Wound gap	0	1 (5)	1 (2.5)

IAH, intra-abdominal hypertension.

Table 5 Preoperative hematological and biochemical investigations

Variables	Groups	*N*	Mean±SD	*P* value
Hemoglobin (g %)	First	20	11.70±1.06	0.001
	Second	20	10.28±1.40	
Leukocytes ($\times10^3/\mu l$)	First	20	10.35±3.47	0.001
	Second	20	18.86±8.92	
Platelet ($\times10^3/\mu l$)	First	20	183.55±22.53	0.012
	Second	20	309.55±203.15	
Albumin (g %)	First	20	4.29±0.32	0.000
	Second	20	3.22±0.72	
Prothrombin time (s)	First	20	14.45±1.19	0.000
	Second	20	19.55±3.41	
International normalization ratio	First	20	1.15±0.08	0.029
	Second	20	1.40±0.48	
Creatinine (mmol/l)	First	20	1.12±0.10	0.000
	Second	20	1.58±0.42	

presented with respiratory distress and distended tense abdomen, whereas abdominal pain and oliguria were only present in ~25% of cases.

Laboratory and radiological investigations

The preoperative hematological and biochemical investigations of both groups of the study showed notable differences ($P \leq 0.05$) in hemoglobin, leukocytes, albumin, prothrombin time, and creatinine, whereas there was no significant difference in the rest of the investigations between both groups, as shown in Table 5. Radiological investigations were done for all patients trying to interpret the pathogenesis of IAH in our study, especially abdominal ultrasonography and plain radiography (abdomen and chest). The findings are tabulated in Table 6.

Assessment and grading of intra-abdominal hypertension patients: grading of IAH and ACS cases according to bladder pressure (cmH_2O) is represented in Table 7. Table 8 shows assessment of 40 patients of IAH and ACS by MOD score. The first group showed only changes in the respiratory variable, whereas the second group showed changes in all variables; so, the second

Table 6 Abdominal ultrasonography findings, radiograph of abdomen, radiograph of chest, and CT abdomen in patients with IAH

Radiological findings	Groups		Total (*n*=40)
	First (*n*=20)	Second (*n*=20)	
Abd US			
Free	7 (35)*	14 (70)	21 (52.5)
Free fluid	8 (40)	3 (15)	11 (27.5)
Distended loops	5 (25)	3 (15)	8 (20)
Radiograph of abdomen			
Free	10 (50)	14 (70)	24 (60)
Air fluid levels	10 (50)	6 (30)	16 (40)
Chest radiography			
Free	16 (80)	19 (95)	35 (87.5)
Pleural effusion	2 (10)	1 (5)	3 (7.5)
Consolidation	2 (10)	0	2 (5)
CT scan			
Free	4 (20)	0	4 (10)
Retroperitoneal hematoma	3 (15)	3 (15)	6 (15)

CT, computed tomography; IAH, intra-abdominal hypertension; US, ultrasound.

Table 7 Grading of IAH according to bladder pressure

Grades	Bladder pressure (cmH2O)	*n* (%)	Range	Mean±SD
I	10–15	0	–	–
II	16–25	20 (50)	17–22	19.50±0.95
III	26–35	15 (37.5)	25–35	30.50±3.81
IV	>35	5 (12.5)	35–40	36.50±3.11

IAH, intra-abdominal hypertension.

Table 8 Assessment of patients by MOD score

Variables	Groups	Scores				
		0	1	2	3	4
Respiratory	First (*n*=20)	0*	0	20 (100)	0	0
	Second (*n*=20)	0	12 (60)	8 (40)	0	0
Renal	First (*n*=20)	20 (100)	0	0	0	0
	Second (*n*=20)	0	14 (70)	6 (30)	0	0
Hepatic	First (*n*=20)	20 (100)	0	0	0	0
	Second (*n*=20)	5 (25)	14 (70)	0	1 (5)	0
Cardio	First (*n*=20)	20 (100)	0	0	0	0
	Second (*n*=20)	20 (100)	0	0	0	0
Hematological	First (*n*=20)	20 (100)	0	0	0	0
	Second (*n*=20)	18 (90)	2 (10)	0	0	0
Neurological	First (*n*=20)	20 (100)	0	0	0	0
	Second (*n*=20)	19 (95)	1 (5)	0	0	0

group was assessed by the total scores of MOD assessment (Table 9), where greater than or equal to 60% of cases had a total score of greater than or equal to 4.

Modalities of treatment

The first group was treated conservatively via different options of medical treatment, as shown in Table 10, whereas the second group was treated by operative decompression. Table 11 shows the different types of anesthesia, incisions, TAC (using Bogota bag), definitive abdominal closure (either primary or using mesh repair for delayed planned incisional hernias), and outcome.

The postoperative complications and outcome of the studied groups are shown in Table 12. The complications of the 1st group were owing to the primary operation and those of the second of group were owing to the primary operation as well as owing to operative decompression procedure. The mortality of the studied groups was 25%, and there was a significant difference between both groups in the hospital stay; both of them are shown in Table 13. The possible causes of mortality of the dead patients are mentioned in Table 14.

Univariate and multivariate analyses (Table 15) show central venous pressure, body mass index, urinary

Table 9 The sum of MOD scores of patients of IAH

MOD score	Groups [*n* ()]	
	First (*n*=20)	Second (*n*=20)
2.00	20 (100)	2 (10)
3.00	0	6 (30)
4.00	0	8 (40)
5.00	0	3 (15)
6.00	0	1 (5)
Total	20 (100)	20 (100)
Range	2–6	
mean±SD	3.80±1.32	

IAH, intra-abdominal hypertension.

Table 10 Medical treatment of 20 patients of the first group

Procedure	First group (*n*=20) [*n* (%)]
Improve abdominal wall compliance	
Good analgesia	20 (100)
Body positioning	20 (100)
Evacuate abdominal fluid collection	6 (30)
Evacuate intraluminal contents	
Ryle	13 (65)
Enema	6 (30)
Prokinetics	9 (45)
Fluid balance	20 (100)

Table 11 Operative procedures and outcome of patients of the second group

Procedures	Outcome [*n* ()]		Total (*n*=20)
	Died (*n*=10)	Discharged (*n*=10)	
Anesthesia			
GA	9 (90)	10 (100)	19 (95)
Local	1 (10)	0	1 (5)
Incision			
Midline	8 (80)	10 (100)	18 (90)
Subcostal	1 (10)	0	1 (5)
Transverse	1 (10)	0	1 (5)
Temporary abdominal closure	10 (100)	10 (100)	20 (100)
Definitive abdominal closure			
Primary repair	0	6 (60)	6 (30)
Mesh repair	0	4 (40)	4 (20)

GA, gestational age.

Table 12 Postoperative complications of the studied groups

Postoperative complication	Groups [*n* ()]		Total (*n*=40)
	First (*n*=20)	Second (*n*=20)	
Wound infection	6 (30)	15 (75)	21 (52.5)
Acute hepatic failure	0	1 (5)	1 (2.5)
Acute renal failure	0	1 (5)	1 (2.5)
Recurrent abdominal compartment syndrome	0	1 (5)	1 (2.5)
Hematemesis	0	1 (5)	1 (2.5)
Pneumonia	0	1 (5)	1 (2.5)
Intestinal fistula	0	2 (10)	2 (5)

Table 13 Outcome and hospital stay of patients of the studied groups

Variables	Groups		Total (*n*=40)
	First (*n*=20)	Second (*n*=20)	
Outcome			
Died	0	10 (50)	10 (25)
Discharged	20 (100)	10 (50)	30 (75)
Hospital stay			
Mean±SD	9±1.89	17.8±15.22	
Range	7: 13	2: 64	*P*=0.002)

Table 14 The possible causes of mortality in patients of the Second group

Cause of mortality	Patients (*n*=10) [*n* ()]
Septicemia	1 (10)
Pneumonia	3 (30)
Acute hepatic failure	2 (20)
Acute renal failure	2 (20)
Hematemesis	1 (10)
Intestinal fistula	1 (10)

bladder pressure, and postoperative organ failure were found to be significant independent risk factors that increased the rate of mortality.

Discussion

The incidence of IAH and ACS as causes of significant morbidity and mortality among the critically ill has

Table 15 Variables as a risk of mortality in patients of the studied group

Variables	Univariate analysis			Multivariate analysis		
	P value	OR	95%CI	*P* value	OR	95%CI
Age	1.000	1.000	0.167–5.985			
BMI	0.019	0.074	0.007–0.835	0.8970	1.154	0.131–10.174
CVP	0.051	9.000	0.809–100.139	0.4570	3.653	2.137–11.83
Urine output	0.6530	0.6670	0.113–3.919			
UBP	0.020	0.083	0.009–0.773	0.5510	2.049	0.193–21.726
Organ failure	0.010	0.167	0.015–1.879	0.5890	1.873	0.192–18.272
Bilirubin	0.531	0.4440	0.034–5.880			
Creatinine	0.502	0.8570	0.124–5.944			

CI, confidence interval; CVP, central venous pressure; OR, odds ratio; UBP, urinary bladder pressure.

increased exponentially over the past decade. Given the prevalence of elevated IAP as well as earlier diagnosis and appropriate therapeutic treatment of IAH and ACS, significant decreases in the morbidity and mortality had been gained in recent years. ACS has been identified as a complication of serious abdominal trauma for more than 5 decades. It occurs as an effect of increased IAP not only in abdominal trauma but also in intestinal obstruction with serious edema of the bowels or a chronically growing tense ascites, and in septic peritonitis [8,9].

ACS can lead to MODS by direct mechanical effects. Respiratory derangement occurs as the result of elevation of diaphragm, which leads to increased peak airway pressure, hypoxia, hypercapnia, and metabolic acidosis. Hemodynamic derangement is caused by the decreased preload. Compression on renal veins causes impairment of kidney function. Once detection of IAH and ACS has been done, they mandate urgent treatment [10].

The definite goal of this clinical research was to define the predictors of exploration in victims at high risk of ACS trying to prevent its morbidity and mortality by identifying the specific risk factors that facilitate early recognition of IAH before the start of frank MODS. In this study, all patients with IAP greater than or equal to 20 cmH_2O were selected, so that, all cases with even mild IAH were involved; they were monitored, and it was ensured that all cases with IAH were detected. The threshold of IAH was defined as sustained raised IAP greater than or equal to 20 cmH_2O for 12 h. For the aim of this study, we narrowed the definition of ACS as IAH associated with MODS which improved after surgery.

One author enumerated the most common causes of IAH and ACS as all causes of acute abdomen, which were neglected or had not been properly managed [11].

In our study, we found that that different causes of acute abdomen might lead to ACS as a fatal complication if not discovered early and managed properly. We had 10 (25%) patients with peritonitis owing to different causes, 12 (30%) patients owing to reduction of large hernia, seven (17.5%) patients presented with blunt abdominal trauma, three (7.5%) patients with postbariatric surgeries, five (12.5%) cases with intestinal obstruction of different causes, two (5%) patients with abdominoplasty, and one (2.5%) patient with postappendectomy intestinal fistula.

One study reported that 67% of cases developing ACS after trauma had abdominal packing. In another study, 20% of cases with severe abdominal trauma were in need of packing for homeostasis and 47% of these cases had ACS [12,13]. In the present study, 4 patients experienced liver injuries and three from internal hemorrhage and retroperitoneal hematoma but only two (5%) cases needed intra-abdominal packing for homeostasis.

Some authors stated that IAP is related to the patient's BMI and influenced by recent abdominal surgery. His average BMI was 27.6 kg/m^2 and the mean IAP was 6.5 mmHg. BMI is significantly related to the IAP. They found that sex, age, comorbidities, and medical and surgical histories did not significantly affect IAP. This correlates with a study that showed a positive relation between sagittal abdominal diameter and increased IAP. One should expect that an individual with larger sagittal abdominal diameter would have a greater BMI and high incidence of IAH and ACS [14,15].

In our study, we noticed that the more the BMI, the more the incidence of IAH and ACS, and this supports the previous studies. The IAP in the obese patients was greater than all. However, the age and co-morbidities represented in most patients reflected on the prognosis of IAH especially that of COPD and liver cirrhosis. We had four (10%) cases with history of DM, two (5%) cases had history of COPD, three (7.5%) cases associated with liver cirrhosis, two (5%) cases had hypertension, and one (2.5%) case of major depression.

Diagnosis of ACS required a high index of suspicion and familiarity with its presenting signs. However, clinical examination is not thought to be an accurate indicator of IAP. The suggestive features of ACS are progressive oliguria despite of adequate hydration, hypoxia with increasing airway pressures, tense or massively distended abdomen, and refractory metabolic acidosis [16,17].

In our study, the clinical presentations for patients of IAH were respiratory distress and distended tense abdomen in most cases (≥90%), whereas abdominal pain and oliguria were only present in ~25% of cases in addition to refractory metabolic acidosis and wound gap in minority of cases.

Research studies among physicians found that many of them use clinical examination for the detection of ACS; it was unreliable with a low sensitivity. Moreover, the use of abdominal perimeter is inaccurate. All radiologic investigations were also insensitive to the detection of increased IAP. However, they can be indicated to illustrate the cause of IAH (bleeding and hematoma) and may offer clues for management (paracentesis and drainage of collections) [18].

In this present study, the radiological investigations were done for all patients to interpret the pathogenesis of IAH in our cases, especially abdominal ultrasonography and plain radiography (abdomen and chest), and abdominal computed tomography for cases of abdominal trauma. However, these radiological investigations did not help us to accurately diagnose ACS.

MOD assessments of principal systems like pulmonary, cardiac, renal, hepatic, hematologic, and CNS were done and graded [7]. A previous study [5] did a grading system for IAH and settled that grade I and II need initial treatment aiming to restore splanchnic and renal perfusion by hypervolemic resuscitation, but grades III and IV may need urgent abdominal decompression.

In the study at our hands, grading of IAH and ACS cases was done according to bladder pressure (cmH_2O) and was done using the previous grading system [5], where grade I was absent in our study, whereas the other grades were represented (grade II representing the 1st group and grades III and IV representing the second group); moreover, the assessment of 40 patients of IAH and ACS was done by MOD score. The first group showed only changes in the respiratory variable, whereas the second group showed changes in all variables, so the second group was assessed by the total score of MOD assessment, where greater than or equal to 60% of cases had a total score greater than or equal to 4.

Although IAP between 10 and 15 mmHg was elevated and had been classified as grade I ACS, pressures in this range appears to be clinically insignificant [19]. The maintenance of normovolemia is advocated for victims with grade I IAH. Hypervolemic resuscitation may overcome the adverse hemodynamic of moderately increased IAP in cases with grade II. Most cases with grade III underwent decompression, whereas grade IV should have both decompression and re-exploration, as these cases are at risk for bowel ischemia and MOD [12].

In this study, we found that all patients with grade I ACS had no clinical significance, as it might be a normal elevation postoperatively and resolve spontaneously without any surgical intervention. All patients with grade II responded to nonoperative management. However, all patients with grade III and IV ACS needed laparotomy as a decompressive procedure as they were at high risk.

Although the life-saving DL will leave the victim with an open abdomen liable to complications such as bleeding, infection, enterocutaneous fistula, or excessive fluid losses, to protect against these complications, a form of TAC must be used. Any TAC technique used after DL should 1st and foremost protect against the development of recurrent ACS. Ideally, it should be cheap and applicable, control fluid losses, require minimal local care, and allow for easy re-exploration [8].

A recent randomized controlled research [20], reported that the type of TAC technique chosen makes a marked difference in outcome and cost of care. In spite of the cheaper option of a Bogota bag that exhibits the advantage of easy drainage and inspection of the bowel and abdominal contents, it has a markedly increased rate of fistula formation (50%), a long delay to closure, and high mortality (27%).

In our study, most of the patients were operated under general anesthesia and decompressed via midline laparotomy incision. All of them were closed by Bogota bag as TAC, and then the 10 survivors underwent definitive abdominal closure. The complications of the 1st group were owing to the

primary operation but those of the 2nd of group were owing to the primary operation as well as owing to operative decompression procedure. We had decreased rate of fistula formation at 10% and hospital stay of 17.8±15.22 days, and our successful closures reached to 50% but with high mortality (50%).

Of 311 patients, only 17 (5.5%) patients developed ACS. They all had RF, whereas cardiac and pulmonary dysfunctions were present in 86%. They stated that the renal functions are the most sensitive to increased IAP [13]. The most affected systems by ACS were cardiovascular, respiratory, and renal systems, and there was a disproportional relation between IAH and these physiologic parameters. The most sensitive parameters are renal function and serum, creatinine and they improved after DL [11].

In our study, physiologic parameters in the form of cardiac output and respiratory function and renal function were all suppressed by increased IAP, and all improved postoperatively. The hemodynamic parameters were affected in 50% of cases (20 cases). The most sensitive parameters were CVP and HR. However, mean arterial blood pressure was not significantly affected.

A direct relation was established between IAP and increased peak airway pressure, intra-thoracic pressure, CO_2 tension, and decreased O_2 tension. It was stated that the mechanism by which the IAP impaired pulmonary functions was mechanical via upward elevation of diaphragm leading to decreasing thoracic volume and increasing intrapleural pressure. *In vivo*, it is manifest as increased peak airway pressure, hypoxemia, hypercapnia, and acidosis [21]. Decompression should release the problem of the pressure exerted on the diaphragms by ACS, thus explaining the observed improvement in PaO_2/FiO_2 and O_2 saturation [22].

We found that respiratory embracement in such group of critically ill cases was in the form of severe hypoxia, hypercapnia, and acidosis. They all improved after laparotomy. Therefore, our results are in agreement with those of the previous authors and all literature studies in concern with respiratory derangement owing to elevated IAP.

Correlation between IAP and oliguria was confirmed in critically ill patients. Patients with IAP less than 25 mmHg had urine output more than 0.5 ml/kg/h. Those with IAP between 25 and 35 mmHg had urine output less than 0.5 ml/kg/h. In cases with IAP greater than 35 mmHg, anuria was uniformly encountered. DL led to establishment of urine output in all patients [12].

In this study, we reported that 19 (47.5%) patients had oliguria and 1 (2.5%) patient anuria; all had elevated serum creatinine. Overall, 18 cases of them improved postoperatively owing to early diagnosis and decompression, and two cases did not improve and died because of development of acute renal failure owing to delayed diagnosis and increased MOD score at the time of presentation So, there was a clinically significant association between increased IAP and renal impairment in this studied group.

The use of urinary bladder pressure measurement was validated by directly comparing the finding pressures measured from the intra-abdominal drains, and a very high correlation was found [23]. In our study, we found that the bladder pressure had a high correlation with directly measured IAP in these cases.

MOD score and type of abdominal closure were not found to be predictive of ACS development; even prophylactic placement of a prosthetic closure did not preclude the formation of ACS [24]. Our study does not agree with the aforementioned authors, and we do not fully support their argument about the type of abdominal closure, as we noted that the closure of the abdomen under tension or abdominal packing may be a risk factor for development of ACS.

The studied 23 patients with ACS were treated conservatively. Their mean highest IAP was 29±8.2. Hemodynamic derangement was found in 87% (20 patients), pulmonary dysfunction in 96% (22 cases), and kidney dysfunction in 91% (21 cases). Mortality was 26%. Nolet *et al.* [25] concluded that the selected patients with ACS may not need DL but may improve with close observation and supportive therapy. In this study, we managed 20 cases by conservative treatment although they had high IAP (grade II), and they passed normally. We can assume that the pressures threshold that initiate decompression would be a matter of question, as not all patients with increased IAP will get benefit from surgical decompression except with MOD.

A total of 73 patients with trauma requiring celiotomy and had abdominal closure with absorbable mesh were retrospectively reviewed. Cases were divided into those who underwent mesh at initial laparotomy (47 patients) and those who had mesh at subsequent laparotomy (26 patients). The researchers found no

instance of ACS in cases with mesh at initial laparotomy compared with 35% incidence when mesh placement was delayed [26].

There was a decision in our study not to have the primary closure of the abdominal cavity in hazardous patient who would need postoperative IAP monitoring and early ACS diagnosis. So, we did application of Bogota bag as a TAC after DL of 20 patients (50%).

Morbidity in cases with ACS is commonly owing to sepsis and multiple organ failure (MOF). The presence of these clinical complications may be associated with the splanchnic hypoperfusion caused by increased IAP. High mortality rates were found in cases who had ACS, ranging from 40 to 60.5% of cases with ACS. Most deaths of victims are caused by the underlying insult and MOF [27,28].In this study, in patients with an IAP less than 20 cmH_2O, the mortality was 0% (hospital stay 9±1.89 days), and in cases with IAP greater than 20 cmH_2O, the mortality was 50% (hospital stay of 17.8±15.22 days). The complications of the first group were owing to the primary operation, but those of the second of group were owing to the primary operation as well as owing to operative decompression procedure.

The ACS has a potentially high mortality that must be detected early and treated effectively to optimize the outcome. Death associated with this condition was reported in 10.6–68.0% of patients [29]. The mortality of the studied groups was 25%, and there was a significant difference between both groups regarding the hospital stay. The cause of mortality was owing to pneumonia (7.5%), acute hepatic failure (5%), ARF (5%), septicemia (2.5%), hematemesis (2.5%), and intestinal fistula (2.5%).

In one previous study, it was concluded that mortality increased with increasing grade of disease. The mortality was 45.1% in cases with ACS, whereas it was 21% in victims with increased IAP alone. The presence of co-morbidity was a factor that increased mortality. They found that the primary etiology did not change the outcome and prognosis, which was mainly determined by the general clinical condition of the patient [30].

In our study, presence of co-morbidity was a factor that increased mortality; however, the primary etiology had a minimal change on the outcome and prognosis. CVP, BMI, urinary bladder pressure, and postoperative organ failure were reported to be independent significant risk factors that increased the mortality rate in the univariate and multivariate analyses.

IAP and physiological parameters aberrations can be considered as predictors of exploration in cases at high risk of ACS, especially following major blunt abdominal and/or pelvic trauma, or patients with late presentation with acute abdomen.

The sample size was small, which was considered as a significant limiting factor of the study. The scope of the study should be widened to involve several etiologic categories. Our hopes for the future are clinical awareness of ACS should be raised by all physicians of the different specialties of medicine.

(1) Determination of the critical level of IAP and the size of fascial defect would allow closure, without any adverse related to IAH.
(2) Minimally invasive maneuvers should be used to decompress the abdomen because of the complications associated with DL, for example, endoscopic techniques based on the component separation concept.
(3) Perfect and safe procedures should be used for abdominal wall reconstruction after laparostomy.

Conclusion

Early detection of cases with IAH and ACS and manage it properly may be curative and may decrease multiorgan dysfunction and mortality. Emergency surgery management should routinely include IAP measurement and the concept of the ACS as part of patient care. IAP and physiological parameters aberrations can be considered as predictors of exploration in patients at high risk of ACS. Diagnosis of ACS required a high index of suspicion and familiarity with its presenting signs. However, clinical examination is not thought to be an accurate indicator of IAP. We recommend routine bladder pressure measurement for patients at risk for IAH. Patients with increased IAP require careful monitoring, aggressive resuscitation, and early surgical decompression and correction of the intra-abdominal problems where appropriate.

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References

1 Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am 1996; 76:833–842.

2 Cheatham ML, Safcsak K. Intraabdominal pressure: a revised method for measurement. J Am Coll Surg 1998; 186:594–595.

3 Töns C, Schachtrupp A, Rau M, Mumme T, Schumpelick V. Abdominelles Kompartmentsyndrom: Vermeidung und Behandlung [Abdominal compartment syndrome: prevention and treatment]. Chirurg 2000; 71:918–926.

4 Tremblay LN, Feliciano DV, Schmidt J, Cava RA, Tchorz KM, Ingram WL, *et al.* Skin only or silo closure in the critically ill patient with an open abdomen. Am J Surg 2001; 182:670–675.

5 Midwinter MJ. Abdominal compartment syndrome. Recent Adv Surg 2004; 27:13–23.

6 Saggi BH, Sugerman HJ, Ivatury RR, Bloomfield GL. Abdominal compartment syndrome. J Trauma. 1998; 45:597–609.

7 Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit Care Med 1995; 23:1638–1652.

8 Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, *et al.* Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 2005; 33:315–322.

9 Pleva L, Sír M, Mayzlík J. Abdominal compartment syndrome in polytrauma. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 2004; 148:81–84.

10 Hong JJ, Cohn SM, Perez JM, Dolich MO, Brown M, McKenney MG. Prospective study of the incidence and outcome of intra-abdominal hypertension and the abdominal compartment syndrome. Br J Surg 2002; 89:591–596.

11 Wittmann DH. Compartment syndrome of the abdominal cavity. In: Irwin RS, Rippe JM, (editors). Intensive Care Medicine. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003. p. 1694.

12 Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM. Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 1997; 174:667–672.

13 Ertel W, Oberholzer A, Platz A, Stocker R, Trentz O. Incidence and clinical pattern of the abdominal compartment syndrome after 'damage-control' laparotomy in 311 patients with severe abdominal and/or pelvic trauma. Crit Care Med 2000; 28:1747–1753.

14 Sanchez NC, Tenofsky PL, Dort JM, Shen LY, Helmer SD, Smith RS. What is normal intra-abdominal pressure? Am Surg 2001; 67:243–248.

15 Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 1997; 241:71–79.

16 Kirkpatrick AW, Brenneman FD, McLean RF, Rapanos T, Boulanger BR. Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients? Can J Surg 2000; 43:207–211.

17 Tiwari A, Haq AI, Myint F, Hamilton G. Acute compartment syndromes. Br J Surg 2002; 89:397–412.

18 Sugrue M, Bauman A, Jones F, Bishop G, Flabouris A, Parr M, *et al.* Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg 2002; 26:1428–1431.

19 Moore EE, Burch JM, Franciose RJ, Offner PJ, Biffl WL. Staged physiologic restoration and damage control surgery. World J Surg 1998; 22:1184–1190.

20 Kaplan MJ, Banwell P, Orgill DP, Ivatury RR, Demetriades D, Moore AF, *et al.* Does the method of temporary abdominal closure affect outcomes in trauma patients managed with an open abdomen? Am Surg 2011; 77:112.

21 Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. J Trauma 1995; 39:1071–1075.

22 Chang MC, Miller PR, D'Agostino R Jr., Meredith JW. Effects of abdominal decompression on cardiopulmonary function and visceral perfusion in patients with intra-abdominal hypertension. J Trauma 1998; 44:440–445.

23 Iberti TJ, Kelly KM, Gentili DR, Hirsch S, Benjamin E. A simple technique to accurately determine intra-abdominal pressure. Crit Care Med 1987; 15:1140–1142.

24 Raeburn CD, Moore EE, Biffl WL, Johnson JL, Meldrum DR, Offner PJ, *et al.* The abdominal compartment syndrome is a morbid complication of postinjury damage control surgery. Am J Surg 2001; 182:542–546.

25 Nolet J, De Wael J, Hoste E, Colpaert K, Bolt S, Roosense C, Decruyenaere J, *et al.* Nonoperative management of patients with abdominal compartment syndrome. Crit Care 2004; 8(Suppl 1):175.

26 Mayberry JC, Mullins RJ, Crass RA, Trunkey DD. Prevention of abdominal compartment syndrome by absorbable mesh prosthesis closure. Arch Surg 1997; 132:957–961.

27 Schein M, Wittmann DH, Aprahamian CC, Condon RE. The abdominal compartment syndrome: the physiological and clinical consequences of elevated intra-abdominal pressure. J Am Coll Surg 1995; 180:745–753.

28 Morris JA Jr., Eddy VA, Blinman TA, Rutherford EJ, Sharp KW. The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg 1993; 217:576–584.

29 Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 1998; 44:1016–1021.

30 Parsak CK, Seydaoglu G, Sakman G, Acarturk TO, Karakoc E, Hanta I, *et al.* Abdominal compartment syndrome: current problems and new strategies. World J Surg 2008; 32:13–19.