

How much does decompressive laparotomy reduce the mortality rate in primary abdominal compartment syndrome?

A single-center prospective study on 66 patients

Mircea Muresan, MD, PhD^{a,*}, Simona Muresan, MD, PhD^b, Klara Brinzaniuc, MD, PhD^c, Septimiu Voidazan, MD, PhD^d, Daniela Sala, MD, PhD^a, Ovidiu Jimborean, MD, PhD^a, Al Husseim Hussam, MD^e, Tivadar jr. Bara, MD, PhD^a, Gabriel Popescu, MD^a, Cristian Borz, MD, PhD^a, Radu Neagoe, MD, PhD^a

Abstract

Contribution of decompressive laparotomy within the framework of the complex therapeutic algorithm of abdominal compartment syndrome (ACS) is cited with an extremely heterogeneous percentage in terms of survival. The purpose of this study was to present new data regarding contribution of each therapeutic step toward decreasing the mortality of this syndrome.

This is a longitudinal prospective study including 134 patients with risk factors for ACS. The intra-abdominal pressure was measured every hour indirectly based on transvesical approach and the appearance of organ dysfunction. Specific therapy for ACS was based on the 2013 World Society of Abdominal Compartment Syndrome guidelines, which include laparotomy decompression. Management of the temporarily open abdomen included an assisted vacuum wound therapy.

Of 134 patients, 66 developed ACS. The average intra-abdominal pressure significantly decreased after therapy and decompression surgery. The overall rate of mortality was 27.3% with statistical significance in necrotizing infected pancreatitis. Surgical decompression performed within the first 24 hours after the onset of ACS had a protective role against mortality (odds ratio <1). The average time after which laparotomy decompression was performed was 16.23 hours. The complications occurred during TAC were 2 wound suppurations and 1 intestinal obstruction. Wound suppurations evolved favorably by using vacuum wound-assisted therapy associated with the general treatment, whereas for occlusion, resurgery was performed after which adhesions dissolved. The final closure of the abdomen was performed at a mean of 11.7 days (min. = 9, max. = 14). The closure type was primary suture of the musculoaponeurotic edges in 4 cases, and the use of dual mesh in the other 11 cases.

The highest mortality rate in the study group was registered in patients with necrotizing pancreatitis and the lowest in trauma group. Surgical decompression within the framework of the complex algorithm treatment of ACS contributed to the reduction of mortality by 8.7%. It is extremely important that the elapsed time since the initiation of the ACS until the surgical decompression is minimal (under 24 hours).

Abbreviations: ACS = abdominal compartment syndrome, CI = confidence interval, DL = decompressive laparotomy, IAH = intra-abdominal hypertension, IAP = intra-abdominal pressure, OA = open abdomen, OR = odds ratio, St. deviation = standard deviation, TAC = temporally abdominal closure, WSACS = World Society of Abdominal Compartment Syndrome.

Keywords: abdominal compartment syndrome, decompressive laparotomy, intra-abdominal pressure, open abdomen

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^a Surgery Clinic No. 2, ^b Department of Physiology, ^c Department of Anatomy, ^d Department of Epidemiology, ^e Cardiovascular Surgery Clinic, University of Medicine and Pharmacy of Targu Mures, Targu Mures, Romania.

* Correspondence: Simona Muresan, Associate Professor, Physiology Department, University of Medicine and Pharmacy of Targu Mures, Targu Mures 540139, Romania (e-mail: dr_muremir@yahoo.com).

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1. Introduction

Although abdominal compartment syndrome (ACS) has long been known, the true basis and definitions of this concept were revealed in 2004 at Noosa, Australia, when The World Society of the Abdominal Compartment Syndrome (WSACS) was founded.^[1] ACS is now a well-known entity defined by increased intra-abdominal pressure (IAP) that is >20 mmHg (with or without arterial perfusion pressure [APP] ≤60 mmHg) and is associated with organ dysfunction. Primary ACS is triggered by the condition located in the abdominal-pelvic region, and secondary ACS develops owing to certain diseases outside the abdominal-pelvic region (Table 1). Recurrent ACS develops following previous surgical or medical treatment of primary or secondary ACS.^[2] Additionally, IAP is sometimes associated with lower degree organ failure in critical patients. Therefore, in 1996, Burch et al^[3] concluded that even first stage of intra-abdominal hypertension (IAH:10–15 mmHg) involves organ failure leading to same clinical course as ACS.

The key parameter in detecting the syndrome is the increased IAP. There are several methods for measuring IAP,^[7,8] both

Table 1**Risk factors for the occurrence of primary and secondary ACS.**
[2,4–6]

Primary ACS	Secondary ACS
Severe intra-abdominal infection	
Pancreatitis	
Blunt/penetrating trauma	Sepsis
Ruptured abdominal aortic aneurysm	Large-volume fluid replacement
Postoperative bleeding	Major burns
Retroperitoneal hemorrhage	Continuous ambulatory peritoneal dialysis
Postoperative closure of the abdomen	Morbid obesity
Undertension	
Ascites	
Ileus	
Pregnancy	

ACS=abdominal compartment syndrome.

directly and indirectly invasive. However, the transvesical IAP measurement has become the criterion standard owing to its simplicity, efficacy, and absence of side effects.^[9–12] According to the Guidelines of 2013—Definitions and Recommendations, IAP measurement is recommended whenever there is a risk of IAP development with possible development of ACS. The data from the literature are quite contradictory in terms of mortality rates after the occurrence of ACS. If not treated, >90% of the cases lead to death, and after treatment, the mortality is between 25% and 75%.^[13]

The purpose of this article was to present new data regarding terms of mortality rate and importance of decompressive laparotomy (DL), relying on interdisciplinary collaboration and the high addressability of the patients who are at risk of developing ACS. We mention that the Intensive Care department is the largest in Transylvania, with 75 beds and a regional center of medical and surgical emergencies, covering 5.8 million of Romania's population.

2. Materials

The study took place between January 2016 and September 2016 and included a total of 134 patients. The informed consent was obtained from each patient, and in case of comatose patients, it was signed by relatives according to the Romanian State legislation and the agreement of the Medical Research Ethics Committee of the University of Medicine and Pharmacy in Tîrgu-Mures, a document registered under no. 118/01.22.2016.

The study inclusion criteria were as follows:

- Patients with abdominal surgery for acute peritonitis caused by abdominal organ perforation or postoperative fistulas following digestive anastomoses, infected necrotic acute pancreatitis, traumas to the intra-abdominal organs as a result of polytrauma, and intestinal occlusion caused by adhesion processes of the abdominal scarring.
- Patients suffering from acute pancreatitis clinically confirmed by laboratory data and imaging, but without surgical indication.

The exclusion criteria were patients with secondary ACS, isolated IAH.

The data collected in the study were: biographical data (sex, age), primary disease, presence of surgery and its type, comorbidities, IAP after the primary surgery, after medical treatment of ACS, and after decompression laparotomy, organ dysfunctions, response to

conservative or surgical treatment, mortality rate, and how it is influenced by surgical decompression.

3. Methods

The measurement of IAP was determined indirectly, transvesically, by using a dedicated kit, Abviser ABV 331. Monitoring consisted of value recording every hour and was started as soon as the study was initiated. It was ended when the measurement of IAP repeated over 24 hours got <15 mmHg or in case of the patient's death.

3.1. Description of the IAP measuring technique

The autovalve of the kit is mounted between the Foley catheter and the collecting bag; the pressure transducer is fixed at the level of the midaxillary line on the iliac crest; the tubing is connected to a bag of saline solution; after emptying the air in the kit components, the monitor is calibrated by bringing the reference value to 0; the measurement of the abdominal pressure is started by aspiration of 25 mL saline solution into the kit's syringe and injecting it into the bladder. The value displayed on the monitor represents the IAP in millimeters of mercury (Fig. 1). After about 2 to 3 minutes, the saline solution injected is discharged by the autovalve into the collecting bag, and to perform a new reading, the syringe is refilled with 25 mL saline solution and the previous steps are repeated. Each new measurement adds 25 mL to the calculation of urine output every 24 hours.

Three average IAP values were defined (at least 3 determinations) as follows: IAP₁=average of the pressures recorded during the ACS diagnosing, before initiation of the specific treatment; IAP₂=average of the pressures recorded after initiation of the ACS specific medical treatment; IAP₃=average of the pressures recorded after the DL.

If the IAP exceeded 20 mmHg and patients have developed a de novo organ failure or a new one added to the existing one, the diagnosis of ACS was clear and we proceeded to implement the



Figure 1. Intra-abdominal pressure measurement using the Abviser ABV 331 kit.

medical therapy specific to this syndrome, according to the WSACS Guidelines of 2013.

3.2. Therapy protocol

Stage I comprise of nasogastric tube, adequate sedation of the patient, avoidance of the excess parenteral fluid intake, setting the fluid balance at zero or negative.

Stage II comprise of reducing enteral nutrition, evacuation enemas, parenteral administration of crystalloid or colloid hypertonic solutions to extract tissue edema, Trendelenburg position, administration of diuretics if the hemodynamic status allows it.

Stage III comprise of ceasing enteral feeding or discontinuing administration, colon decompression by inserting a rectosigmoid suction catheter with intermittent aspiration, curare administration to the patient, intubation and mechanical ventilation, evacuation puncture guided by ultrasound or computed tomography, hemodialysis.

Stage IV comprise of abdominal decompression by median laparotomy with specific management of open abdomen by the assisted vacuum wound therapy method using the Vivanotec abdominal kit system.

DL was performed in the following situations: the IAP increased despite the medical treatment (at least 3 measurements) and high levels of IAP remained (up to 20 mmHg) during the medical treatment for more than 24 hours.

We note that each stage of the ACS treatment was associated with the specific treatment of the primary disease.

3.3. Open abdomen management

DL was performed by a midline incision above and below the umbilicus, enough to release the loops and epiploon from the peritoneal cavity. From the beginning, the subsequent TAC management was dictated by the use of a negative pressure continuous suction system having dual roles: aspiration of collections and reducing intra-abdominal tissue edema, and avoiding musculo-aponeurotic retraction of the wound edges and the induction of granulation tissue. To this end, the Vivano system (Hartmann TM) was used from which the Vivano® Med Abdominal Kit was chosen. The entire system assembly consisted of the VivanoTec suction unit and consumables. The single use abdominal kit contains: mesh to protect the viscera with the diameter of 65 cm fitted with superior pockets to facilitate insertion, 2 polyurethane foams of 38 × 25 × 1.6 cm, 1 suction port, and hydrofilm strips for sealing.

Immediately after laparotomy, the intestines and epiploon were protected with mesh from the kit described above, by inserting it into the upper inter-hepato-phrenic spaces, laterally into the flaps toward the right and left parietocolic grooves, and lower into the posterior plane behind the urinary bladder. On top of the mesh, polyurethane foam was mounted in 2 or 3 layers, by adjusting their dimensions to the incision made. After installing the sealing strips over the entire wound without creating tension, the suction port connected to the collection tank was secured. The VivanoTech device was originally set to a gentle pressure of -105 mmHg. The dressings were changed every 2 to 3 days using the abdominal kit if the patient's condition was critical, maintaining ACS. After the third replacement of the abdominal kit, the pressure was lowered to -135 mmHg and continued in the same manner until the final closure. Subsequently, for the favorable developments, the reconstruction of the abdominal wall was performed by primary

fascial closure or using a substitute dual mesh (polyester + dimethyl siloxane—Cousin Biotech™), sutured on the musculoaponeurotic edges with Prolene 3.0 continuous threads secured with 10 separate suture points.

3.4. Statistical analysis

All statistical calculations were performed using Graph Pad Software (San Diego, CA). Continuous variables were tested for normal distribution with the Kolmogorov–Smirnov test. We characterized variables as mean and standard deviation (SD) or as median and range for variables with normal and abnormal distribution, respectively. We chose adequate statistical tests according to data distribution. Differences between the mean ages for each sex were determined by the Student *t* test. Intra-abdominal pressure, mortality, and decompression laparotomy results were analyzed using analysis of variance (ANOVA) test (associated with the Bonferroni multiple comparison test). In a Box-and-Whisker plot, the central box represents values from the lower to upper quartile (25–75 percentile). The middle line represents the median. A line extends from the minimum to the maximum value. All the tests were interpreted relative to the significance threshold *P*=0.05 and statistical significance was considered below the significance threshold value.

4. Results

Of the total of 134 patients included in the study with the risk of developing ASC, 76 of them developed IAH >20 mmHg, and 66 of them developed ACS (Fig. 2).

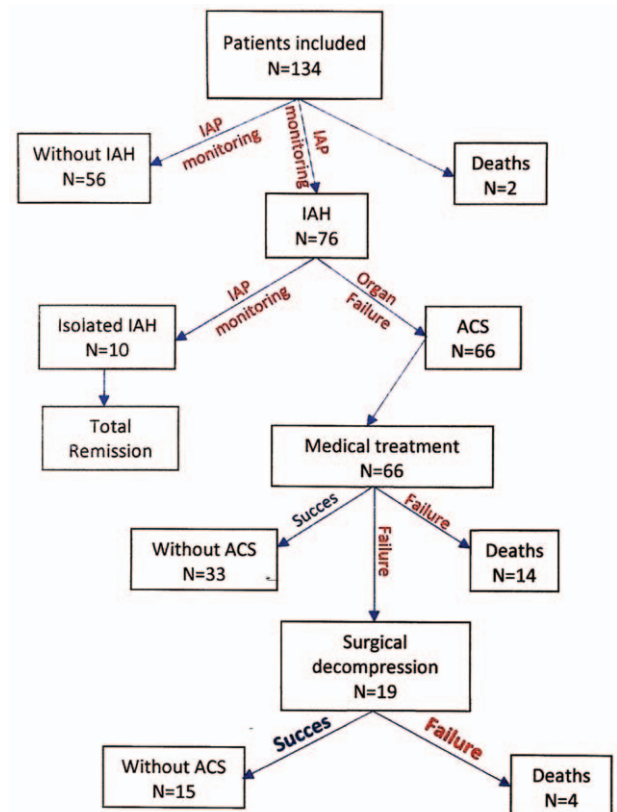


Figure 2. Study design.

Table 2**Primary abdominal diseases which led to ACS, and their surgical approach.**

Primary abdominal condition	Types of surgeries	No	Timing
Peritonitis after ileo-colic and colo-colic anastomoses leakage	Hartmann operation	6	Immediate emergency surgery
	Temporary ileostomy	2	
Peritonitis for perforated colon diverticulitis	Subtotal colectomy	4	
	Hartmann operation	5	
Peritonitis for perforated gangrenous appendicitis	Appendectomy	9	
Peritonitis for perforated gangrenous cholecystitis	Cholecystectomy	8	
Necrotizing infected pancreatitis	Computed tomography-assisted drainage	4	Delayed surgery
	Open surgery for drainage	7	
Trauma	Splenectomy	4	Immediate emergency surgery
	Atypical liver resection	2	
	Liver suture	5	
	Jejunal or ileal resection	2	
	Urinary bladder suture	1	
Intestinal occlusion	Adhesion removal	6	Immediate emergency surgery
	Jejunal and ileal resection	2	
	Colon resection	2	

ACS=abdominal compartment syndrome.

Table 3**Statistical comparison between the decrease in intra-abdominal pressure before and after the 2 stages of the treatment.****One-way analysis of variance**

P	<0.01		
	Mean difference	Significant? $P < 0.05?$	95% CI of difference
Bonferroni multiple comparison test			
IAP ₁ vs. IAP ₂	7.58	Yes	4.80–10.36
IAP ₁ vs. IAP ₃	17.87	Yes	14.09–21.66
IAP ₂ vs. IAP ₃	10.29	Yes	6.33–14.24

CI=confidence interval, IAP=intra-abdominal pressure.

The primary abdominal diseases who led to ACS were: peritonitis, necrotizing infected pancreatitis, noninfected pancreatitis (without surgery requiring), intestinal occlusion, and trauma. Each primary abdominal disease, which led to ACS, excepting noninfected acute pancreatitis, followed initial specific surgical treatment. Acute peritonitis, trauma, and intestinal occlusion benefited from immediate emergency surgery (in the first 24 hours). Infected necrotizing pancreatitis represented the unfavorable evolution of acute pancreatitis despite aggressive medical treatment and underwent delayed surgical treatment (after 3–8 days from initial diagnosis) (Table 2). In all cases, ACS installed after 3.62 days and decompressive laparotomies were performed in the first 16.23 hours.

The average age of the patients who developed ACS was 68.85 years (min–max=43–82, St. deviation=10.22), with a higher incidence in males (62.1%). Of the IAH-associated dysfunctions, kidney failure was present in 93.9%, liver failure in 80.3%, and cardiocirculatory failure in 66.7% of patients. IAP analysis of the early-stage ACS, after medical therapy and after surgical decompression, showed significant difference between the groups (ANOVA with Bonferroni multiple comparability test) (Table 3, Fig. 3).

The overall mortality in 66 patients with ACS was 27.3%. By statistically analyzing the conservative treatment and the surgical treatment, we found that DL was protective against mortality (odds ratio [OR]<1). However, it was not statistically significant ($P > 0.05$) (Table 4).

The primary abdominal conditions were statistically compared regarding the mortality rate using the χ^2 test. The acute

peritonitis group consisted of 34 patients and mortality was 44.4% ($P=0.58$, OR=0.67, 95% confidence interval [CI]: 0.22–2.01). Necrotizing infected pancreatitis included 10 patients, mortality was 38.9% ($P=0.003$, OR=9.54, 95% CI: 2.10–42.9), and the bowel occlusion group comprised 9 patients with 16.7% mortality rate ($P=0.69$, OR=1.50, 95% CI: 0.31–6.31). In the groups of 4 cases of noninfected acute pancreatitis ($P=0.33$, OR=1.40, 95% CI: 0.20–1.62) and 9 cases of trauma ($P=0.04$, OR=0.46, 95% CI: 0.21–0.74), mortality was 0%. Therefore, in the study group, necrotizing acute pancreatitis was statistically significant correlated with a

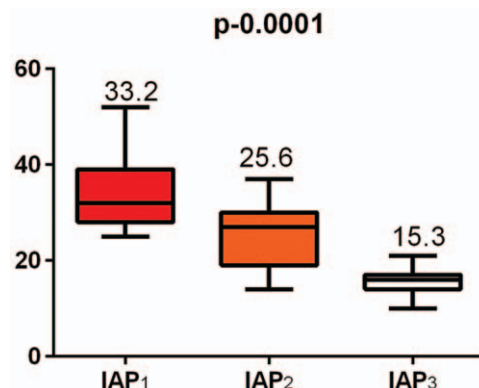
**Figure 3.** Intra-abdominal hypertension variation before and after the therapeutic stages.

Table 4
Statistical comparison between the 2 main stages of treatment, decompression laparotomy, and mortality rates.

Treatment	Mortality rate	OR value	P	95% CI
Medical treatment	29.8%	1.59	0.53	0.44–5.65
Medical treatment + decompressive laparotomy	21.1%	0.63	0.55	0.17–2.23

CI = confidence interval, OR = odds ratio.

high mortality rate, and trauma was statistically significant correlated with low mortality rate.

The causes of death were septic shock (in the third and fifth day after surgery) in 1 case (generalized peritonitis after perforated colon diverticulitis), respiratory failure in 2 cases, following bronchopneumonia in 2 patients with peritonitis associating chronic obstructive pulmonary disease (COPD) (on the 7th and 15th postoperative day), and severe bleeding in 1 case after necrotizing infected pancreatitis (after 6 weeks).

4.1. Postoperative complications

The complications occurred during TAC were 2 wound suppurations in patients who had undergone surgeries for generalized peritonitis after colorectal anastomosis fistula, and 1 intestinal obstruction owing to adhesions in a patient with frozen abdomen. Wound suppurations evolved favorably by using vacuum wound-assisted therapy associated with the general treatment, whereas for occlusion, resurgery was performed after which adhesions dissolved. The final closure of the abdomen was performed at a mean of 11.7 days (min.=9, max. 14). The closure type was primary suture of the musculoaponeurotic edges in 4 cases, and the use of dual mesh in the other 11 cases.

5. Discussion

After founding WSACS, the grounds for the definitions and recommendations regarding the epidemiology, diagnostic algorithm, and treatment of ACS were laid down.^[2] They remained largely valid; some new definitions were also added (Table 5).

Even the ACS was first described after abdominal traumatic injuries, WSACS identified 3 main risk factors for primary ACS developing: peritonitis, acute pancreatitis, and trauma.^[14] In different studies were added also intestinal obstruction, abdominal aortic aneurism, abdominal tumors, ascitis, complication of

pregnancy, and operated incisional hernias with high tension in abdominal wall.^[4,6]

Pathophysiological mechanisms leading to increased abdominal pressure, regardless of the cause, include tissue edema, bowel and mesenteric edema, retroperitoneal space edema, and the accumulation of free abdominal fluid as a result of extracapillary extravasations. Intra-abdominally, the increased pressure leads to capillary compression exceeding the critical tissue perfusion threshold that translates as intestinal ischemia.^[17] Cardiac output decreases with an increase in the peripheral and abdominal hypoperfusion.^[18] In the lungs, it induces hypoxia and hypercapnia by decreasing the compliance of the thoracic wall and diaphragm, increasing pleural pressure and accumulation of intrapleural fluid.^[19] Renally, the arterial hypoperfusion and venous compression lead to progressive alterations of the glomerular filtration rate.^[18] Muftuoglu et al^[20] reported that in case of ACS caused by primary abdominal sepsis, the liver is rapidly and severely affected, and is considered the most severe hepatic tissue injury. The ischemia and infection from the portal blood flow activate Kupffer cells, which release inflammatory mediators affecting the activity of hepatocytes and sinusoidal cells. Thus, hepatocellular retention of bile, biliary acids, and exogenous substances develops.^[21–23] In our study, liver failure was present in 80.3% patients with no statistical correlations with the mortality rate. Betro and Kaplan have described 3 types of patients that are most likely to develop ACS. These types include those with massive transfusion during surgeries, medical patients that require large volumes of fluid resuscitation for severe sepsis, and surgical patients that require large volume resuscitation for an intra-abdominal underlying disease.^[24] Peritonitis, necrotizing acute pancreatitis, abdominal traumas, and aortic abdominal aneurisms are the main causes of primary ACS.^[17,25]

ACS therapy must be implemented immediately. The treatment algorithm is well established by the WSACS Guidelines of 2013.^[14] No therapeutic approach can improve the progression of the syndrome by itself. A multiple approach must be applied with most important points being sedation, setting the fluid balance to 0 =or negatively, nasogastric and rectal probe, and neuromuscular blockade.^[14] Surgical decompression reduces IAP, but it is not equally correlated with the mortality rate.^[26] In our study, the IAH significantly decreased after each therapeutically step. However, the same trend was not observed in the survival rate. The overall mortality depends on the underlying condition. If occurs after an already severe pathologic background, the mortality rate increases, but it is reduced after trauma, which suddenly occurs on healthy people. When we compared the groups of patients, we noticed the highest mortality rate from the study group after necrotizing pancreatitis and the lowest after trauma ($P < 0.05$) After Vidal et al,^[5] the respiratory and circulatory failure are the end-points of pathophysiological mechanism, being considered the main direct causes of death. The influence of surgical decompression on the survival rate during the complex treatment of ACS remains a topic with extremely

Table 5
Pressure parameters correction formula in case of intraabdominal pressure (MAP = diastolic AP + [systolic AP – diastolic AP]/3).^[2,14–16]

Parameter	New definition
Polycompartment syndrome	Condition where ≥ 2 anatomical compartments have elevated compartmental pressures
Open abdomen	Any abdomen requiring a temporary abdominal closure owing to the skin and fascia not being closed after laparotomy
APP	$APP_{corrected} = MAP - IAP$
CVP	$CVP_{corrected} = CVP - IAP/2$
PAOP	$PAOP_{corrected} = PAOP - IAP/2$
GFP	$GFP_{corrected} = MAP - 2 \times IAP$
CPP	$CPP_{corrected} = MAP - IAP$

MAP = diastolic AP + (Systolic AP – diastolic AP)/3. APP = arterial perfusion pressure, CPP = cerebral perfusion pressure, CVP = central venous pressure, GFP = glomerular filtration pressure, PAOP = pulmonary artery occlusion pressure. MAP = medium arterial pressure.

Table 6**The abdominal compartment syndrome mortality rates quoted by different authors in the literature and our study.^[13,31–35]**

Author	Year	No. of patients	Study type	Mortality
De Waele et al ^[13]	2006	250	Retrospective	49.2%
De Waele et al ^[33]	2010	18	Retrospective	36%
Davis et al ^[32]	2013	45	Prospective	24%
Divarci et al ^[34]	2014	150	Prospective	16%
Hwabejire et al ^[35]	2015	122	Retrospective	37.7%
De Waale et al ^[31]	2016	33	Prospective	36%
Our study	2016	66	Prospective	27.3%

heterogeneous data in the literature (Table 6). A very important aspect is represented by the time after which the DL was performed. There are studies that revealed a mortality rate close to 100% when DL was performed 48 hours following ACS diagnosis.^[13,26] In our study, the medium time between diagnosis and DL was 16.23 hours (min 6 hours, max 24 hours). However, improved survival parameters after surgical decompression are owing to the positive effects on the underlying disease, especially in acute pancreatitis and septic abdomen.^[27–30] In our study, DL was not significantly correlated with the mortality rate ($P > 0.05$) but was protective (OR < 1), reducing mortality by 8.7%.

The use of a wound vacuum system is one of the new recommendations in the WSACS Guidelines of 2013.^[14] The continuous aspiration of extravasated fluids decreases the IAP, removes the unwanted secretions, significantly reduces systemic effects of their toxicity, and significantly decreases the main open abdominal complications—lateral retraction of the wound edges.^[36–38]

After the procedure was performed by Brock et al and Barker et al in 1995,^[39,40] the development of vacuum therapy-dedicated kits started. TAC approach by vacuum-assisted wound therapy technique has also been the WSACS recommendation since the 2013 consensus.^[14] In our case, the chosen solution was VivanoTech (HartmannTM). Initially appearing as individual methods, the use of meshes to prevent lateral retraction was subsequently combined with the vacuum therapy techniques: Polydioxanone (PDS) mesh + vacuum therapy,^[41] polypropylene + vacuum therapy,^[42] or ABRA system—the combination of trans fascial elastomeric fibers tensed with buttons placed on the skin, associated with vacuum therapy.^[43] All these latter techniques have significantly increased the percentage of late fascial closure up to 100% according to some authors.^[44] Regarding the final closure of the abdominal wall, it should be done without tension. Depending on the TAC technique used, primary fascial closure varies. In our study, it could be carried out in 4 of the 19 cases. The closure without tension can be achieved by the use of meshes.^[45] If visceral protection with large omentum can be performed, or the granulation tissue is well enough developed after vacuum therapy, polypropylene meshes can be used.^[46] A safe alternative are the dual-meshes made from polypropylene, polyester, or expanded polytetrafluoroethylene (e-PTFE), which can be sutured to the aponeurotic edges and applied safely over the viscera.^[47] The modern meshes of cross-link and non-cross-link types, manufactured in the laboratory, are very expensive for the time being. As a result, they should only be used in the reserved cases.^[48] In our study, we had very good results with fewer complications using silicone polyester dual-meshes. Complications caused by VAC technique use include hemorrhage as a result of large vessels (portal vein, splenic vein) present in the open abdominal wound of necrotizing pancreatitis.^[29,37] Xiao et al^[49] reported kidney failure and numbers of laparotomies as main

predictive factors for bleeding occurrence in necrotizing acute pancreatitis. In our study, one of the deaths was caused by hemorrhagic shock through the splenic vein fistula. The patient underwent 4 laparotomies in 6 weeks. The general postoperative complications are usually the consequence of primary disease, and wound complications derive from open abdomen management. If the general ones sometimes led to death, the local ones were successfully treated using the vacuum wound therapy management with no related mortality. Regarding the advantage of VAC technique use, Cirocchi et al,^[50] analyzed all related studies, published until July 2015. In a systematic review and meta-analysis performed on 1225 patients from 8 articles of the most important research area, they compared the negative pressure technique with other 4 non-negative pressure techniques (Bogota bag, Mesh-foil laparostomy, Midline zip laparostomy and others). After statistical analysis was made, they observed that there are no significant differences in between VAC technique and all others, regarding the fascial closure, postoperative 30-day overall morbidity, postoperative enteroatmospheric fistulae rate, in the postoperative bleeding rate, and postoperative abdominal abscess rate. Instead, statistical significance was found in the postoperative mortality rate (28.5% vs. 41.4%) and in the length of stay in the intensive care unit.

6. Conclusions

Despite the new therapeutic protocols, ACS still has high mortality. Primary ACS most often occurs after events contaminating the peritoneal cavity. The highest overall mortality rate was after necrotizing pancreatitis. The specific medical therapy significantly reduced the mortality rate compared with no treatment of the syndrome. Decompression laparotomy was protective against mortality reducing it by 8.7%, and should be used as soon as possible in case of medical resuscitation failure. The prolonged duration (over 24 hours) between the occurrence of ACS and surgical decompression negatively influenced the prognosis. Recovery following decompression may depend on the severity of the primary disease. General postoperative complications are because of underlying disease and in some cases led to death, but local complications were easily reduced using the vacuum wound therapy system with no mortality correlated.

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