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Research Paper

Prophylaxis and treatment of intra-abdominal hypertension in patients with acute surgical pathology of abdominal organs

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ABSTRACT

Acute abdominal pathology may cause a progressive, unchecked increase of intra-abdominal pressure up to the development of the abdominal compartment syndrome (ACS). The last one is associated with organ failure. The aim of this study was to optimise the nonsurgical ways of abdominal decompression to prevent ACS development in patients with the acute diseases of abdominal organs that cause intra-abdominal hypertension (IAH). Fifty-nine patients of the control group with acute abdominal pathology associated with IAH were studied retrospectively. In the study group, 71 patients with the same pathology, managed according to the proposed three stage approach, were evaluated prospectively. Bladder pressure was measured using the modified system. It permits zeroing of the system without direct contact with a sterile surgical field. The main cause of IAH was the abdominal content volume increase. According to the proposed treatment approach, management was started from the least invasive methods and proceeded to the next step in case of ineffectiveness. In patients, mechanical ventilation adjustment of ventilator's parameters decreased abdominal pressure on average by 18.8 ± 2.6 mmHg; thus, in 19 patients, the average became less than 15 mmHg. First step measures permitted to stabilize the intra-abdominal pressure (IAP) on the target level in 39 (54.9%) cases. The second stage activities were effective in 19 (26.8%) cases. The most effective third step manoeuvre was the improvement of abdominal wall compliance. Described algorithm permitted to reduce IAP and escape surgical decompression in 67 (94.4%) cases. The incidence of complications was lower in the study group (22.5% vs 64.4% in control one, $p<0.05$). In general, postoperative mortality was 7.6% (all patients from the control group). To determine the optimal way of IAH/ACS management, it is necessary to clearly understand the causes of its development and underlying pathophysiological mechanisms in a particular case. Usage of the simplified algorithm for correction of intra-abdominal pressure has allowed a statistically significant reduction of the number of systemic and local complications in patients with urgent pathology of abdominal organs.

Key words: Abdominal compartment syndrome, acute surgical pathology, intra-abdominal pressure, intra-abdominal hypertension.

Abbreviations: ACS, The abdominal compartment syndrome; **IAH,** intra-abdominal hypertension; **IAP,** intra-abdominal pressure; **WSACS,** the world society of the abdominal compartment syndrome.

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INTRODUCTION

Despite intuitive comprehension of the role of increased abdominal pressure on the course of acute and chronic abdominal pathology, real understanding and scientific substantiation of the problem came only in recent years

(Malbrain and Waele, 2013; Balogh et al., 2014; De Waele et al., 2015; Hecker et al., 2015). Progressive, unchecked increase in intra-abdominal pressure may lead to intra-abdominal hypertension (IAH) and then to the abdominal compartment syndrome (ACS), which is defined by the consensus definitions of the World Society of the Abdominal Compartment Syndrome (WSACS) as a sustained intra-abdominal pressure (IAP) > 20 mmHg that is associated with new organ dysfunction/failure (Wise et al., 2015; Kirkpatrick et al., 2013, 2017).

The WSACS has created three algorithms detailing the current state-of-the-art diagnosis and management of IAH/ACS: the IAH Assessment (Wise et al., 2015), the IAH/ACS Management, and IAH/ACS Medical Management algorithms (Kirkpatrick et al., 2013, 2017). Elaboration of new technical and tactical approaches to prevent and treat ACS in patients with acute surgical pathology of abdominal viscus is of current importance.

The aim of this study was to optimise the nonsurgical ways of abdominal decompression to prevent abdominal compartment syndrome development in patients with the acute diseases of abdominal organs that cause intra-abdominal hypertension.

MATERIALS AND METHODS

Study design and setting

In this retrospective and prospective study, conducted in the Department of Surgery #2 of Bogomolets National Medical University (Kyiv, Ukraine), were enrolled 130 patients with acute abdominal surgical pathology, clinical signs of IAH and detected raised IAP. In the studied group, there were 68 (52.3%) males and 62 (47.7%) females. Their age varied from 21 to 85 years, average 46.2 ± 1.2 years. Depending on the tactical approach used, patients were divided into two groups: the main one and the control. The control group consisted of 59 (45.4%) patients who were treated in a standard way; mean different measures for IAP reduction were used on demand without a strict system. Case histories of these patients were analysed retrospectively. The main group included 71 (54.6%) patients to whom optimised therapeutic approach for IAP reduction and ACS prevention was used. These patients were admitted to the clinic in the order of the first aid between January 2014 and December 2016. This group was studied prospectively.

Inclusion criteria

- Patients with an acute pathology of abdominal organs with the clinical signs of IAH.
- Urinary bladder pressure ≥ 12 mmHg.
- Patients 18-85 years of age.
- Technical possibility to measure the urinary bladder pressure.
- Informed consent of the patients to participate in the study.

Exclusion criteria

- Impossibility to measure bladder pressure.
- Age less than 18 yrs. or more than 85 yrs.
- Refusal to give informed consent.

Ethical approval

The study protocol was approved by the institutional Ethics Review Committee. The research was carried out according to the principles of the Helsinki Declaration and the International Council for Harmonization of Good Clinical Practice guidelines.

Outcome measures

Intra-abdominal pressure was measured via an open bladder catheter after 25 mL of sterile saline instillation. Adding a laser pointer and a level to the system (Figure 1) permitted its zeroing without direct physical contact with the patient's trunk. This was especially useful during surgery to avoid contact with the sterile surgical field. Measurements were taken before surgery, at its different stages, especially before and after abdominal wall closure, and in the intensive care unit. For diagnosis of acute abdominal pathology ultrasound, CT and endoscopy, as well as physical examination and routine laboratory studies, were used.

The clinical outcome measures were: frequency of systemic and local complications, mortality and causes of death in both groups.

Statistical analysis

Statistica 8.0 (Statsoft Inc., Tulsa OK Oklahoma, USA) software was used for the data processing. Graphical assessment of normality was supplemented by Shapiro-Wilk test (Thode, 2002). Intra-abdominal pressure in patients with acute pancreatitis was presented as "mean ± SD". The means for two independent normally distributed groups were compared by an independent samples t-test, for two dependent groups – by dependent samples t-test. Differences between the samples were considered statistically significant when p-value < 0.05.

RESULTS

More than half of both groups consisted of patients with acute pancreatitis and abdominal trauma (Table 1).

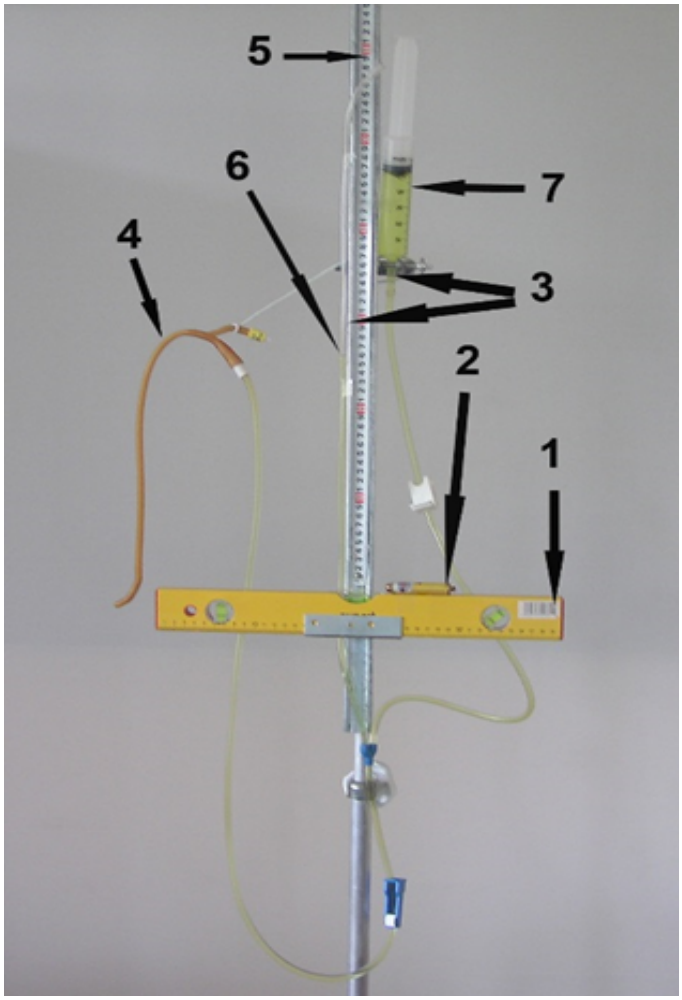

Figure 1: Modified system for IAP measurement.
1 – Level, 2 – Laser pointer, 3 – Y-tube, 4 – Foley catheter, 5 – Tape-line, 6 – Level of water, 7 – Syringe.

Table 1: The diagnosis in the patients of both groups.

Nosology	Patients with systemic complications (n=32)				
	Study group (n=71)		Control group (n=59)		Total (n=130)
	n	% of syst. compl.	n	% of syst. compl.	n
Strangulated ventral hernia	1	3.1	5	15.6	6
Acute pancreatitis	2	6.3	5	15.6	7
Acute bowel obstruction	2	6.3	5	15.6	7
Acute appendicitis	1	3.1	3	9.4	4
Acute cholecystitis	1	3.1	1	3.1	2
Blunt abdominal trauma	2	6.3	4	12.5	6
Total	9	28.1	23	71.9	32

Immediate surgery was undertaken in 92 patients (70.8%). Thirty-eight patients (29.2%) were managed medically: 29 with acute pancreatitis, 9 with peritoneal adhesions. For big size and giant ventral strangulated hernias, simple or mesh repair was performed, controlling intraoperatively IAP. All patients with acute appendicitis, acute cholecystitis and perforated duodenal ulcers were admitted with severe peritonitis and ileus. Patients with appendicitis and

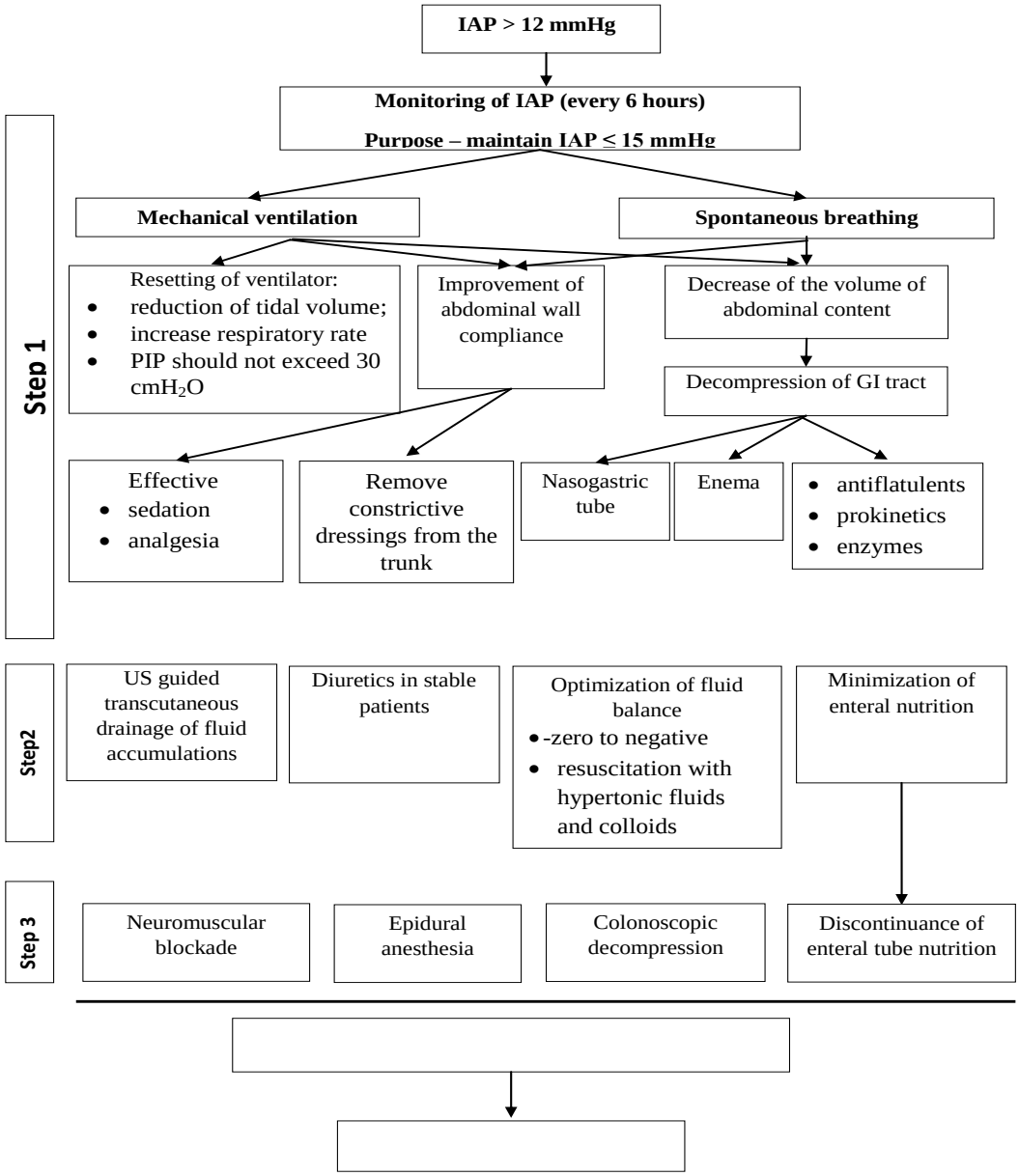

Figure 2: A working algorithm for nonsurgical correction of intra-abdominal hypertension for abdominal compartment syndrome prevention in patients with acute abdominal pathology.

cholecystitis had concomitant obesity (BMI ≥ 30 kg/m^2). Surgical approach to bowel obstruction management was determined considering its aetiology. To reduce the invasiveness of the acute pancreatitis surgical treatment to the maximum, repeated drainage of abdominal fluid accumulations under ultrasound control and sequestrectomy through small incisions were performed.

A retrospective assessment of the control group patients showed that from the perspective of the current understanding of this pathology, not all possibilities of conservative treatment were used. Thus the approach to IAH management was optimised in the following way. After IAH verification, a decision about the tactics of treatment was made. First of all, it is depended upon the aetiological factor. Excluding strangulated hernia, the main cause of abdominal pressure rise was an increase in the volume of abdominal content (bowel paresis, accumulation of free fluid, limited liquid formations). In elderly overweight patients with a hernia, IAH developed due both to decrease in the volume of the abdominal cavity and intestine paresis. According to the international recommendations, management of IAH/ACS started, beginning from medical and minimally invasive therapies (Kirkpatrick et al., 2017). For its optimization, we developed the following working

temporary algorithm, presented in Figure 2, which consists of three levels of medical measures. The main

purpose of all treatment affords was to maintain IAP ≤ 15 mmHg.

From the moment when bladder pressure was registered at the level of 12 mmHg or more, repeated measurements every 6 h were started. Management of the patients on mechanical ventilation started from the regulation of ventilator's parameters – tidal volume was reduced with a proportional increase of respiratory rate. Peak inspiratory pressure should not exceed 300 mmH_2O (22.2 mmHg). In 19 (59.4%) out of 32 patients who were on mechanical ventilation, the target pressure less than 15 mmHg was achieved after average decreases of IAP on 18.8 ± 2.6 mmHg. Thus there was no need to move to the next stage, only monitoring of IAP was continued. In the remaining 13 patients on mechanical ventilation, in whom this manoeuvre was not completely effective, and in all patients with spontaneous breathing, efforts were undertaken to improve abdominal wall compliance – all constrictive dressings and bandages were removed from the trunk and adequate analgesia and sedation were provided. After that, the number of patients in whom IAP achieved an acceptable level increased to 24.

Abdominal content volume decreased at this stage in 15 patients due to: 1) decompression of upper gastrointestinal tract with a nasogastric tube and of the colon – by enema; 2) reduction of meteorism – by antiflatulents, prokinetics and enzymes. All complexes of the first step measures were permitted to stabilize IAP on the target level with subsequent normalization in 39 (54.9%) patients.

In the case of ineffectiveness of the first stage activities, therapy was escalated to the second step which included: 1) transcutaneous drainage of fluid accumulations under ultrasound control in patients with acute pancreatitis; 2) optimization of fluid administration (zero to negative balance, utilization of hypertonic fluids and colloids, administration of diuretics in hemodynamically stable patients); 3) minimization of the tube enteral nutrition and avoidance of high concentration solutions. In 5 cases (3 mechanically ventilated patients and 2 with spontaneous breathing), IAP was reduced to the safe level by drainage of fluid accumulations. The above mentioned correction of infusion therapy and enteral nutrition demonstrated effectiveness in 4 from 13 patients on this stage. From another 13 patients in whom all three elements of second stage management were used to decrease IAP, lower 15 mmHg was registered in 10. In general, the activities of the second stage proved to be effective in 19 cases.

Patients who were refractory to conservative treatment had to pass to the third step. From our point of view, the most effective manoeuvre on this stage was the improvement of abdominal wall compliance. If mechanical ventilation was continued, preference was given to neuromuscular blockade, in spontaneously breathing patients – epidural anaesthesia. Neuromuscular blockade was effective in 5 cases and epidural anaesthesia – in 3 patients. Enteral tube feeding was completely discontinued in all patients. We suppose that on this stage colonoscopic decompression may also be considered, although in the group we observed that there were no such cases.

If, after using the entire arsenal of 3-step conservative treatment, it was not possible to achieve a reduction in IAP, on the contrary, it increased to 20 mmHg or more, there was a need for surgical decompression. The latter was performed in 4 patients. In other words, in 67 cases from 71 (94.4%) of the main group more optimised management permitted to reduce IAP by the nonsurgical method. On the contrary, not optimally organised conservative management of the control group patients gave the possibility to reduce IAP without decompressive laparotomy only in 49 patients from 59 (83.05%). The difference was statistically significant ($p < 0.05$).

It is clear that the stage-by-stage approach to the management of patients with such pathology is somewhat relative. For example, correction of ventilator's settings demonstrates greater efficiency against the background of the use of neuromuscular blockade. Set of activities may vary depending on the specific surgical pathology.

Keeping in mind that it is better to prevent some complication than make heroic efforts fighting with it, surgical interventions for strangulated hernia, mean interventions that reduce the volume of the abdominal cavity, were performed with the obligatory control of IAP after the closure of fascia and skin. Our previous experience of abdominoplasty and hernia repair demonstrated that increase of IAP to the level of 15 mmHg at the end of elective surgery did not lead to any problem in postoperative period if careful monitoring of the patient's condition was held. Increase of maximum inspiration pressure by 200 mm H_2O (14.8 mmHg) and more with unchanged parameters of artificial ventilation of the lungs during the operation may indicate a significant increase in IAP and require a mandatory check of the latter (Теплый и Колосович, 2014). The same approach was transferred to the treatment of patients with acute surgical pathology.

The dynamics of IAP reflect the effectiveness of used treatment methods. Very indicative it was in the patients of the most frequent diagnosis – with acute pancreatitis: 18 from the study group versus 17 from the control one (Figure 3). At admission, I grade IAH registered in six patients of each group, II grade – in nine patients of each group and III – in three patients of the study group and two patients of the control group. At the beginning of the treatment, average IAP in both groups exceeded 15 mmHg and was almost the same. A statistically significant decrease in IAP was noted in both the primary and control groups at the end of the first week. But from the third-day, pressure reduction was statistically faster in patients managed in accordance with proposed algorithm and on the seventh day it was lower (13.2 ± 2.62 vs 16.0 ± 4.37, $p = 0.025$). Abdominal pressure increase to the level of more than 20 mmHg on the second-third day of treatment was accompanied with worsening of the clinical picture,

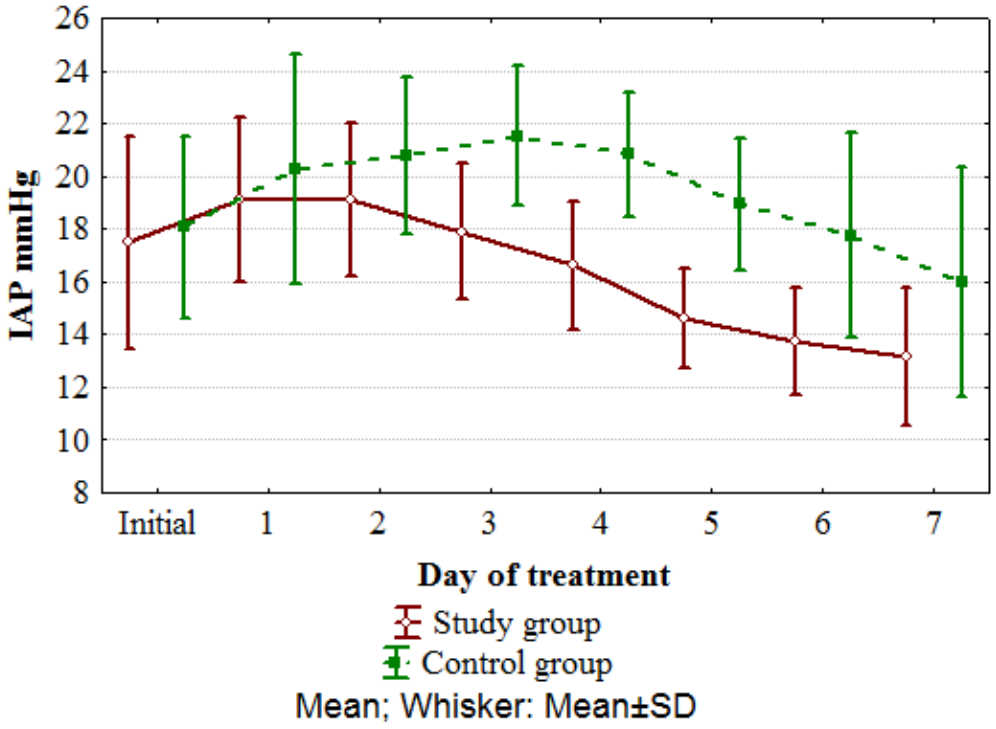

Figure 3: Dynamic of intra-abdominal pressure in patients with acute pancreatitis depending of treatment.

laboratory and ultrasound data in patients of the control group. Due to the ineffectiveness of conservative treatment, three patients in the control group underwent forced decompressive laparotomy. Uncontrolled multiple organ failure led to the death of 3 patients. At the end the seventh day, the average IAP in the control group was higher as 15 mmHg. The study group showed a gradual decrease of IAP during one week, and at the end of this period, abdominal pressure reduced to the target level (<15 mmHg). It is understandable that the subsequent dynamics of IAP was dependent on the severity of morphological changes in the pancreas and surrounding tissues. In three cases, increase of abdominal pressure promoted transcutaneous ultrasound-guided puncture of fluid accumulations or surgery. Systematic step by step approach in accordance with described algorithm allowed avoiding uncontrolled IAP rise and the development of multiple organ failure. Normalization of intestinal function (decrease of bloating, the spontaneous passage of flatus and stool) and essential reduction of IAP were achieved in 16 (88.9%) patients from the main group and only in 10 (58.8%) patients from the control group ($p < 0.05$). The same tendency of IAP dynamic was observed in patients with other urgent abdominal pathology.

Analysis of the results of management of patients with acute abdominal surgical pathology demonstrated a statistically significant difference in frequency of postoperative complications – 22.5% in the study group and 64.4% in control group, $p < 0.001$. Systemic complications (acute insufficiency of one or more systems, thromboembolic complications) arose in 9 patients from main group (12.7% of this group) versus 23 patients from control group (39.0% of the group). Difference was statistically significant ($p < 0.001$). The most unfavourable situation was developed in the case of fairly rapid increase of IAP. This happened due to the accumulation of fluid inside the intestine in acute bowel obstruction, the fast formation of liquid accumulations in acute pancreatitis, hernioplasty under tension. Detailed analysis showed that the most frequent complications were acute respiratory insufficiency, heart failure and multiple organ failure (Table 2).

The frequency of local complications also was lower in the study group – seven cases (9.9%) than in control one – 15 cases (25.4%, $p < 0.02$). Among them, the first surgical wound infection took place, that is, complicated postoperative period in four (18.2%) patients of the study group and nine patients (40.9%) in control one. In the study group, one case of gastrointestinal bleeding, acute thrombophlebitis of lower extremities and acute pneumonia also developed. One case of anastomosis leakage, two cases of acute thrombophlebitis of lower extremities and acute pneumonia, one case of the visceral ischemic syndrome developed in patients of the control group.

The occurrence of certain complications can be directly related to IAH. Thus distension of abdominal wall produces ischemia of wound edges which contribute to wound

Table 2: Frequency of systemic complications in patients with acute surgical pathology of the abdominal cavity, accompanied by IAH/ACS.

Complications	Groups of patients				
	Study group		Control group		Total
	n	% of syst. compl.	n	% of syst. compl.	n
Acute respiratory insufficiency	3	9.4	7	21.9	10
Heart failure	3	9.4	6	18.8	9
Acute liver failure	1	3.1	1	3.1	2
Enteral insufficiency	–	–	2	6.2	2
Multiple organ failure	2	6.2	6	18.8	8
Thromboembolic complications	–	–	1	3.1	1
Total	9	28.1	23	71.9	32

complications. Depletion of blood circulation in the walls of gastrointestinal tract secondary to high abdominal pressure against the background of bowels paresis and increased activity of the sphincters can directly explain the cause of the anastomoses leakage. Erosions and ulcers of upper gastrointestinal tract, which may be source of bleeding, also develop as a result of trophic changes in the mucosa and submucosal layer. Difficulties of venous outflow due to compression of the inferior vena cava contribute to the development of thrombophlebitis of lower extremities. Upward shift of diaphragm leads to increase of intrathoracic pressure, pulmonary arteriovenous shunting and alveolar hypoventilation. All these are the factors promoting development of pneumonia. Wound problems account for more than half of local complications.

A total of 8 patients died (6.2%). One main group patient (1.4%), conservatively treated for acute pancreatitis, died from multiple organ failure. From seven deceased control group patients (11.9%) four died from multiple organ failure, one from acute respiratory insufficiency, one from heart failure and one from pulmonary embolism. The difference in mortality between groups was statistically significant ($p < 0.02$). Among the deceased patients from control group, three were with acute pancreatitis, two were treated for bowel obstruction and two for blunt abdominal trauma (1 – pancreatic damage and 1 – spleen rupture). Thus, overall postoperative mortality was 7.6% (7 patients from 92 who underwent surgery). All operated patients were from the control group. Among the causes of death, the first place unconditionally took multiple organ failure – 5 cases (62.5%).

DISCUSSION

The first understanding of the negative pathophysiological influence of increased abdominal pressure on blood circulation and external breathing after blunt abdominal trauma became a stimulus for future evaluation of IAP in critically ill patients with other diseases (Papavramidis et al., 2011). Soon investigators found that ACS may complicate the course of acute pancreatitis (van Brunschot et al., 2014; Park et al., 2011), peritonitis (Muresan et al., 2017) and acute bowel obstruction (Papavramidis et al., 2011). Abdominal hypertension did not obligatorily lead to ACS but the tendency of its growth in surgical patients was found (Svorcan et al., 2017).

For successful management of IAH, clear understanding of etiological factor is essential. The main idea of management is to maintain IAP ≤ 15 mmHg. The rationales for choosing this level of pressure were the following considerations. First of all, at this pressure reduction, microcirculatory blood flow occurs (Cheatham et al., 2004) and oliguria develops (Richards et al., 1983; Cheatham, 2009). Then, we took into account our previous experience of patients managing after hernioplasty (Теплый и Колосович, 2014). Finally, this level was proposed in the second consensus of WSACS (Kirkpatrick et al., 2013).

Decrease of IAP may be achieved by the normalisation of ratio between the volume of the abdominal contents and the capacity of the abdominal cavity. Gastrointestinal decompression, reduction of meteorism, transcutaneous drainage of fluid accumulations, optimization of fluid administration and tube enteral nutrition contribute to the decreasing volume of the abdominal content. On the other hand, an increase of abdominal wall compliance by the refusal of constrictive dressings and bandages from the trunk, adequate analgesia and sedation, in refractory cases neuromuscular blockade or epidural anaesthesia increase the abdominal cavity capacity.

It should not be neglected the possibility of IAP correction by ventilator settings adjustment – very fast and effective procedure to which not often attention is paid. To prevent pulmonary barotrauma, peak inspiratory pressure should not exceed 30 cmH_2O. This easy manoeuvre leads to a quick effect without great efforts.

Recognizing that the proposed by WSACS IAH/ACS management and IAH/ACS medical management algorithms encompass all currently known aspects of the IAH correction and treatment of ACS, we have created a slightly modified and shorter conservative treatment algorithm for our daily work. It includes therapeutic

methods available to us. Also, in the above-mentioned algorithms, interventions were applied in a stepwise fashion. In the case of no response to a particular intervention, therapy escalated to the next step in the algorithm. The proposed algorithm consists of 3 levels of increasing complexity. The differences between our algorithm and the one proposed by the WSACS are as follows. Since many of the patients with registered IAP > 12 mmHg were on artificial ventilation of the lungs, resetting of a ventilator was introduced at the first stage. Thus, it made possible to quickly normalize IAP in some patients. We shifted enema to the first step of treatment as a cheap, not time consuming but effective procedure. Antiflatulent drugs and enzymes were also added to the first step. Surgical evacuation of lesions was excluded from the third step since this is a surgical procedure. Epidural anaesthesia was introduced instead. The proposed algorithm has demonstrated convenience for routine use.

Conclusions

1. To determine the optimal method of IAH/ACS management, it is necessary to clearly understand the causes of its development and underlying pathophysiological mechanisms in a particular case – this may be as a result of an increase of abdominal content volume or a reduction in the volume of the abdominal cavity.
2. At the first step of the correction of the IAH, it is fundamental to know whether the patient is on mechanical ventilation or not.
3. Usage of the working temporary algorithm for correction of intra-abdominal pressure has allowed a statistically significant reduction of the number of systemic and local complications in patients with urgent pathology of abdominal organs.
3. The main cause of the lethal outflow in patients of both groups with the ACS was multiple organ failure.

REFERENCES

Balogh ZJ, Lumsdaine W, Moore EE, Moore FA (2014). Postinjury abdominal compartment syndrome: from recognition to prevention. Lancet. 384: 1466–1475.

Cheatham ML (2009). Abdominal Compartment Syndrome: pathophysiology and definitions. Scand J Trauma Resusc Emerg Med. 17: 10.

Cheatham ML, Safcsak K, Llerena LE, Morrow CE, Jr, Block EF (2004). Long-term physical, mental, and functional consequences of abdominal decompression. J Trauma. 56: 237–241.

De Waele JJ, Malbrain ML, Kirkpatrick AW (2015). The abdominal compartment syndrome: evolving concepts and future directions. Critical Care. 19(1): 211-211.

Hecker A, Hecker B, Hecker M, Riedel JG, Weigand MA, Padberg W (2015). Acute abdominal compartment syndrome: current diagnostic and therapeutic options. Langenbecks Arch Surg. Oct 30: http://www.ncbi.nlm.nih.gov/pubmed/ 26518567.

Kirkpatrick AW, Roberts DR, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, Duchesne J, Bjorck M, Leppaniemi A, Ejike JC, Sugrue M,

Cheatham M, Ivatury R, Ball CG, Blaser AR, Regli A, Balogh ZJ, D'Amours S, Debergh D, Kaplan M, Kimball E, Olvera C, The Pediatric Guidelines Sub-Committee for the World Society of the Abdominal Compartment Syndrome (2013). Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Medicine; 39 (7): 1190–1206.

Kirkpatrick AW, Sugrue M, McKee JL, Pereira BM, Roberts DJ, De Waele JJ, Leppaniemi A, Ejike JC, Reintam Blaser A, D'Amours S, De Keulenaer B, Malbrain MLNG (2017). Update from the Abdominal Compartment Society (WSACS) on intra-abdominal hypertension and abdominal compartment syndrome: past, present, and future beyond Banff 2017. Anaesthesiol Intensive Ther. 49(2): 83-87.

Malbrain M, De Waele J (2013). Intra-abdominal Hypertension. Cambridge, Cambridge Univer-sity Press: 470 p.

Muresan M, Muresan S, Brinzaniuc K, Voidazan S, Sala D, Jimborean O, Hussam AH, Bara T Jr, Popescu G, Borz C, Neagoe R (2017). How much does decompressive laparotomy reduce the mortality rate in primary abdominal compartment syndrome? A single-center prospective study on 66 patients. eMedicineMedicine, 96 (5): e6006.

Papavramidis TS, Marinis AD, Pliakos I, Kesisoglou I, Papavramidou N (2011). Abdominal compartment syndrome – Intra-abdominal hypertension: Defining, diagnosing, and managing. Journal of Emergencies, Trauma, and Shock. 4 (2): 279–291.

Park S, Lee S, Lee HD, Kim M, Kim K, Jeong Y, Park SM (2014). Abdominal compartment syndrome in severe acute pancreatitis treated with percutaneous catheter drainage. Clinical endoscopy. 47(5): 469–472.

Richards WO, Scovill W, Shin B, Reed W (1983). Acute renal failure associated with increased intra-abdominal pressure. Ann Surg. 197: 183–187.

Svorcan P, Stojanovic M, Stevanovic P, Karamarkovic A, Jankovic R, Ladjevic N (2017). The influence of intraabdominal pressure on the mortality rate of patients with acute pancreatitis. Turk. J. Med. Sci. 47(3): 748–753.

Thode HJ (2002) Testing for normality. Boca Raton, CRC Press: 368 p. https://doi.org/10.1201/9780203910894.

van Brunschot S, Schut AJ, Bouwense SA, Besselink MG, Bakker OJ, Goor van H, Hofker H, Gooszen HG, Boermeester MA, van Santvoort HC & Dutch Pancreatitis Study Grp (2014). Abdominal compartment syndrome in acute pancreatitis: a systematic review. Pancreas. 43 (5): 665-674.

Wise R, Roberts DJ, Vandervelden S, Debergh D, De Waele JJ, De Laet I, Kirkpatrick AW, De Keulenaer BL, Malbrain ML (2015). Awareness and knowledge of intra-abdominal hypertension and abdominal compartment syndrome: results of an international survey. Anaesthesiol Intensive Ther. 47(1): 14–29.

Теплый ВВ, Колосович АИ (2014). Диагностика и профилактика внутрибрюшной гипертензии у больных с вентральными грыжами. Хирургия Восточная Европа. 4(12): 38-44.

[Teplyi V, Kolosovych A (2014). Diagnosis and prevention of intra-abdominal hypertension in patients with ventral hernias. Surgery. Eastern Europe. 4 (12): 38–44.