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Intraabdominal hypertension/abdominal compartment syndrome after pelvic fractures: How they occur and what can be done?

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ABSTRACT

Background: Limited data exist regarding intraabdominal hypertension/abdominal compartment syndrome (IAH/ACS) after pelvic fractures. We aimed to explore risk factors for IAH/ACS in pelvic fracture patients, assess the physiological effects of decompressive laparotomy (DL) on IAH/ACS, and generate an algorithm to manage IAH/ACS after pelvic fracture.

Materials and methods: Pelvic fracture patients were included based on the presence of IAH/ACS. Intraabdominal pressure (IAP) was measured through a Foley catheter. DL was performed in patients with refractory IAH or ACS. Multivariable linear regression was applied to assess associations between IAP levels (≥12 mmHg) and age, sex, injury severity score (ISS), pelvic fracture, volume of resuscitation fluids over 24 h and hemoglobin values. The Wilcoxon signed-rank test for paired samples was used to compare variables before and after DL.

Results: Among 455 pelvic fracture patients, 44 (9.7%) and 5 (1.1%) were diagnosed with IAH and ACS, respectively. The volume of resuscitation fluids over 24 h exhibited a significant positive correlation with IAP levels (≥12 mmHg) ($p = 0.002$). The main findings during DL were edematous bowel (11/20) and retroperitoneal hematoma (7/20). DL caused a significant decrease in the mean IAP from 24.4 ± 8.5 mmHg to 13.4 ± 4.0 mmHg ($p < 0.0001$). Physiological parameters (APP, PaO2/FIO2 ratio, PIP, arterial lactate and UOP) were significantly improved after DL. The mortality rate was 15% in patients who underwent DL and 40% in ACS patients.

Conclusions: IAH/ACS is common in pelvic fracture patients. The most effective method to decrease IAP in pelvic fracture patients is DL. Prophylactic DL is important for decreasing mortality as it prevents IAH from progressing to ACS. Massive fluid resuscitation is a significant risk factor for IAH/ACS. A pathway incorporating prophylactic/therapeutic DL and optimized fluid resuscitation to prevent and manage IAH/ACS after pelvic fractures may reduce morbidity and mortality.

Introduction

The terms intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS) represent pathological points on a spectrum of intraabdominal pressure (IAP) that can affect intra-abdominal tissue viability and organ function. IAP ≥12 mmHg is considered to represent IAH, and IAP >20 mmHg combined with new organ dysfunction/failure is diagnosed as ACS [1]. Previous reports of ACS within the major trauma population showed a prevalence higher than 30% and a mortality rate of more than 60% [2,3].

Although the understanding of IAH/ACS has greatly improved, few studies have focused on IAH/ACS after pelvic fracture [4–7]. ACS after pelvic fracture represents secondary ACS (also known as extraabdominal compartment syndrome) that can develop when a massive retroperitoneal hematoma emerges (due to delayed definitive hemorrhage control), when bowel edema occurs (due to capillary leak caused by massive crystalloid resuscitation) or when other complications of pelvic fractures present [8,9] (Fig. 1a–c).

Because secondary ACS can further increase the morbidity and mortality of pelvic fractures, the purpose of this article is thus to explore the risk factors of IAH/ACS in pelvic fracture patients,

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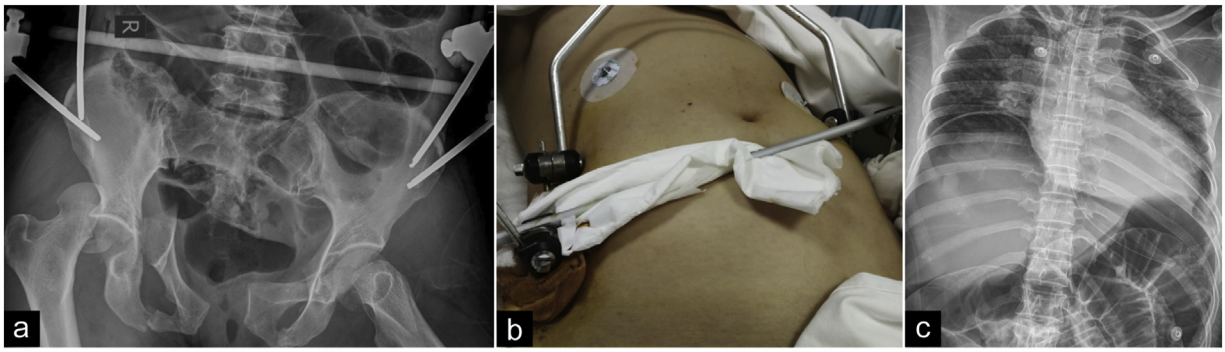

Fig. 1. A pelvic fracture was stabilized by external fixation (a). Serious abdominal distension developed in the patient four days after the injury. Note the expanded abdominal wall against the connecting rod of the external fixation (b). Chest X-ray showing that the intestinal tympanites significantly elevated the diaphragm (c). IAP (Intraabdominal pressure).

assess the effects of prophylactic/therapeutic decompressive laparotomy (DL) on IAH/ACS, and summarize the latest, comprehensive algorithm for the management of IAH/ACS after pelvic fracture.

Materials and methods

Consecutive pelvic fracture patients who were diagnosed with IAH/ACS between May 2011 and May 2018 were identified through a database search. Data were retrospectively reviewed from electronic medical records (Table 1). Written informed consent was obtained from all participants and was approved by our Institutional Review Board.

The inclusion criteria were as follows: the patient had pelvic fracture; the IAP was measured through a Foley catheter rather than other devices; the patient had IAH or ACS. Patients younger than 18 years or those with a severe head injury (AIS > 3) were excluded. Patients who had a laparotomy before being transferred to our trauma center and patients with IAH/ACS due to causes unrelated to complications of pelvic fractures were excluded.

The pelvic fractures were classified according to the AO classification as type A, stable; type B, partially stable; or type C, completely unstable. IAP was measured indirectly via the bladder pressure using a Foley catheter. Detailed procedures for measuring the IAP and the corresponding key points were previously described elsewhere [1,10,11]. The IAP was measured at first contact in the intensive care unit (ICU) for screening and then measured every 4 h in pelvic fracture patients with elevated IAP. IAH was graded according to mean IAP (mean value of the last three measurements) levels as grade I, 12–15 mmHg; grade II, 16–20 mmHg; grade III, 21–25 mmHg; and grade IV, >25 mmHg [1]. Additional physiological parameters recorded included hemodynamic parameters (mean arterial blood pressure [MAP], abdominal perfusion pressure [APP]), ventilatory parameters (PaO2/FIO2 ratio, peak inspiratory pressure [PIP], arterial lactate level) and a renal parameter (urinary output [UOP]). The abovementioned parameters were compared between groups of patients who did and did not undergo DL and individually before and after DL. Other parameters recorded included injury severity score (ISS), volume of resuscitation fluids over 24 h and hemoglobin values at admission.

Table 1
Patient demographics and injury characteristics.

Variable	N = 49	%
Age in years	42.4 ± 12.8	
Gender: men/women	35/14	
Injury severity score	23.1 ± 7.4	
Hemoglobin at admission (g/dL)	9.5 ± 1.6	
Volume of IV fluids over 24 h (mL)	3965 ± 739	
Mechanism of injury		
Fall from height	15	30.6
Motor vehicle accident	29	59.2
Other	5	10.2
Pelvic fracture (AO classification)		
Type A	9	18.4
Type B	26	53.1
Type C	14	28.6
IAH/ACS condition		
Grade I IAH	23	46.9
Grade II IAH	11	22.4
Grade III IAH	7	14.3
Grade IV IAH	3	6.1
ACS	5	10.2

IAH (Intraabdominal hypertension); ACS (Abdominal compartment syndrome).

The nonoperative management of IAH in pelvic fracture patients

After recognition of IAH, patients underwent prompt nonoperative interventions to reduce IAP as recommended by the guidelines from the World Society of the Abdominal Compartment Syndrome (WSACS) [1,12,13]. The nonoperative interventions to reduce IAP included the following: evacuating abdominal fluid through paracentesis or percutaneous drainage; evacuating intraluminal contents through nasogastric and rectal decompression; correcting positive fluid balance by avoiding excessive fluid resuscitation and the use of diuretics; improving abdominal wall compliance by using sedation and analgesia; and improving organ function by optimizing ventilation and alveolar recruitment.

The operative management of IAH/ACS in pelvic fracture patients

Once a diagnosis of ACS was made, the patient rapidly underwent DL. Prophylactic DL was performed in patients with refractory IAH (sustained ≥24 h and/or unresponsive to nonoperative interventions and progressed to the next grade of IAH) [14,15]. All surgeries were performed by the corresponding author.

The exact approaches chosen were the midline approach for bowel edema or exploratory laparotomy and the ilioinguinal approach or the Stoppa approach for retroperitoneal hematoma. The primary goal of DL was to perform a complete fasciotomy to allow evisceration and allow ongoing evacuation of fluid from the abdominal cavity or retroperitoneal space using a vacuum sponge. Vacuum sealing drainage (VSD) was used for all patients for temporary management of the open abdomen (Fig. 2). The key points of DL have been elaborately described elsewhere [8].

Fig. 2. The vacuum sealing drainage (VSD) technique used for temporary management of an open abdomen.

Statistical analysis

SPSS version 21.0 (SPSS Institute, Chicago, IL, USA) was used for the statistical analyses. Continuous data are expressed as the mean ± SD (standard deviation). For multivariable linear regression, IAH/ACS was taken as the dependent variable, while age, sex, ISS, pelvic fracture, volume of resuscitation fluids over 24 h and hemoglobin values at admission were the independent variables. The Kruskal-Wallis test was used to compare IAP levels between the three pelvic fracture types. Patients were divided into DL and no-DL groups based on the presence of refractory IAH and ACS, respectively, and the Mann-Whitney test for unpaired samples was used to compare the physiological variables between the two groups. The Wilcoxon signed-rank test for paired samples was used to compare the physiological variables before and after the DL. A two-side p value less than 0.05 was considered statistically significant.

Results

The incidence and risk factors of IAH/ACS in pelvic fracture patients

Between May 2011 and May 2018, 455 consecutive patients with pelvic fractures were admitted to our trauma center, of which 44 (9.7%) patients developed IAH (by IAP mean), and 5 (1.1%) patients were diagnosed with ACS (Fig. 3). Among the factors entered into the multivariable regression model, the volume of resuscitation fluids over 24 h was the only factor significantly associated with IAP ≥ 12 mmHg (Supplementary Table 1). We found that each 1 L increment of resuscitation fluid over 24 h was significantly associated with a 5 mmHg increase in IAP (p = 0.002). Additionally, ISS (p = 0.11), age (p = 0.89), and hemoglobin values at admission (p = 0.34) had no statistically significant association

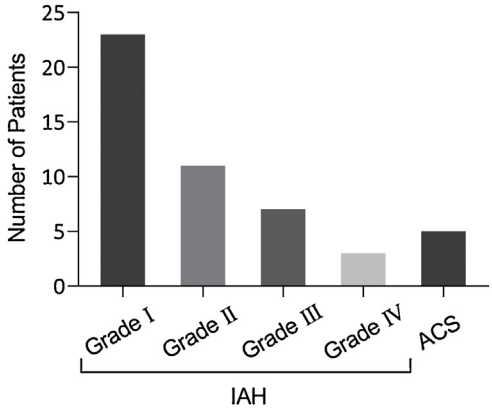

Fig. 3. The distribution of IAH/ACS in our patient population (N = 49).

with IAP ≥ 12 mmHg (N = 49). Interestingly, no clear association was observed between pelvic fractures and an IAP ≥ 12 mmHg in our population. Patients with type B pelvic fracture had a higher IAP by 4.09 mmHg (p = 0.15) than the IAP of those with type A pelvic fracture, and patients with type C pelvic fracture had a higher IAP by 2.08 mmHg (p = 0.51) than the IAP of those with type A pelvic fracture. However, neither of these associations reached statistical significance (Fig. 4).

The differences in physiological parameters between pelvic fracture patients who did and did not undergo DL

The IAH conditions of 29 patients were satisfactorily controlled by nonoperative management, and no further interventions were needed. In contrast, 4 out of 5 patients with ACS underwent DL, and 16 patients with refractory IAH underwent prophylactic DL. The mean IAP was significantly different between patients who did (24.4 ± 8.5 mmHg) and did not undergo DL (15.6 ± 5.4 mmHg) (p < 0.0001) (Supplementary Table 2). Except for MAP, the physiological parameters including APP, PaO2/FIO2 ratio, PIP, arterial lactate level and UOP were significantly different between patients who did (N = 20) and did not (except for PIP [N = 14], all N = 29) undergo DL (Fig. 5).

The outcomes after DL for refractory IAH or ACS in pelvic fracture patients

Among the 20 patients who underwent DL, the main findings were edematous small bowel and clear ascites (N = 11), retroperitoneal hematoma (N = 7), massive fluid collections (N = 1), and tympanites (N = 1). No patients in this study underwent bowel resection. The mean time from diagnosis of refractory IAH or ACS to laparotomy was 2.7 h (1.5–5.0 h). The mean IAP before DL was 24.4 ± 8.5 mmHg, which decreased to 13.4 ± 4.0 mmHg within 2 h after DL (p < 0.0001; N = 20) (Supplementary Table 2). Vital signs such as APP, PaO2/FIO2 ratio, PIP, arterial lactate level and UOP were significantly improved after DL, but the changes in MAP were not statistically significant (N = 20) (Fig. 6). The mortality rate was 15% in patients who underwent DL and 40% in patients with ACS. One patient with ACS died before the DL because of MODS, and the cause of his ACS was undetermined but was presumed to be bowel edema due to excessive fluid resuscitation (6.3 L over the prior 24 h). Another patient with ACS due to a retroperitoneal hematoma died after DL because of respiratory and circulatory failure. Surprisingly, one patient with grade II IAH died after DL, and the cause of death was presumed to be ARDS due to ischemia-reperfusion injury. The surviving patients successfully underwent delayed primary closure at 2.9 days (2–5 days) after DL.

Discussion

A limited number of studies have reported IAH/ACS in pelvic fracture patients. However, we failed to demonstrate pelvic fractures as a risk factor for IAH/ACS. The pelvic fractures were classified according to AO classification as type A, type B, or type C. We did not further classify the pelvic fractures into subgroups because of the relatively small sample size. Although fracture patterns cannot reliably predict associated vascular injury [16], they can indicate the transfusion requirements [17]. In accordance with previous studies, our results also showed that massive fluid resuscitation is a risk factor for IAH/ACS. Type C pelvic fractures are associated with higher transfusion requirements [17]; therefore, these patients are more likely to be treated with massive fluid resuscitation. We investigated whether the three pelvic fracture types had any effects on IAP levels. Unfortunately, the results we obtained were inconsistent with

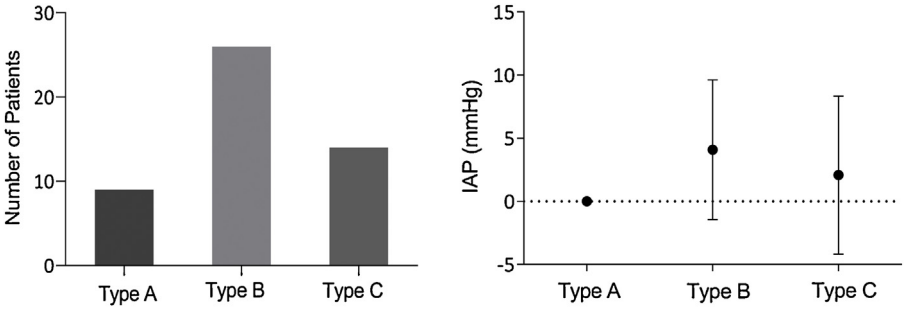

Fig. 4. The number of patients and IAP level of the three fracture types (N = 49).

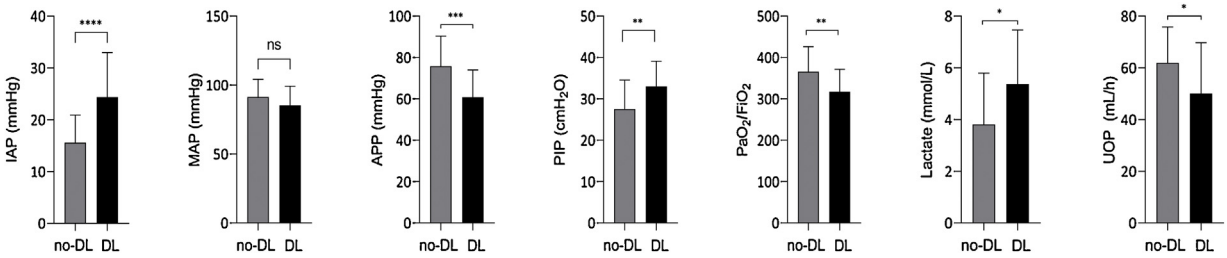

Fig. 5. Differences in the physiological parameters between pelvic fracture patients who did (DL, N = 20) and did not undergo DL (no-DL, except for PIP [N = 14], all N = 29).

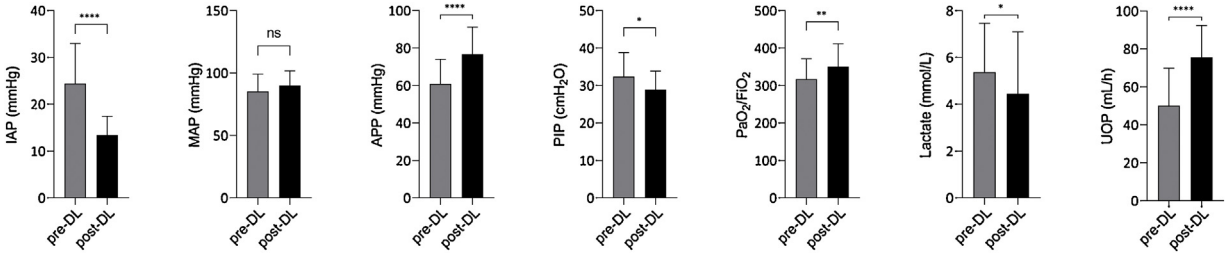

Fig. 6. Changes in the physiological parameters after DL for refractory IAH or ACS in pelvic fracture patients (N = 20).

the aforementioned conjecture, as no significant effect of fracture type on IAP level was found.

In pelvic fracture patients, arterial bleeding from the fracture site can be managed through transcatheter arterial embolization (TAE), and venous hemorrhaging can be controlled by pelvic packing. However, there have been no reliable or effective methods for managing retroperitoneal venous hemorrhaging. Uncontrolled retroperitoneal venous hemorrhaging not only produces space-filling hematomas that directly increase IAP but also contributes blood loss followed by resuscitation, which creates an ischemia-reperfusion injury to the bowel and leads to increased capillary permeability. Decreased oncotic pressure caused by massive crystalloid use combined with increased capillary permeability due to the inflammatory mediators all contribute to bowel edema, which leads to an increased IAP. As the IAP increases, upward pressure on the diaphragm increases central venous pressure, which decreases venous outflow, leading to abdominal venous hypertension. All of these factors, in turn, contribute to the growing bowel edema, and IAP continues to increase. This vicious circle ultimately leads to damage of the ventilatory capacity, hemodynamic instability, and decreased visceral perfusion [8].

As a general principle, anything should be attempted to help negate worsening IAH, but once a diagnosis of ACS (most often combined with respiratory failure, hemodynamic instability, and acute renal failure) is definitively made, DL should be rapidly performed [11]. There are a number of unresolved issues relating to IAH, in particular, which parameter should be the threshold for DL in patients with IAH. In our study, prophylactic DL was performed in patients with refractory IAH (sustained $\geq$ 24 h and/or unresponsive to nonoperative interventions and progression to the next grade of IAH) [14,15]. The rationale of this principle is that transient (approximately 24 h) IAH does not cause organ dysfunction; however, sustained IAH compromises visceral perfusion and causes organ dysfunction [14]. Patients with sustained IAH have the highest mortality [18]. Patients who manifest progressive IAP and organ dysfunction and are unresponsive to nonoperative interventions have been shown to benefit from early open-abdominal decompression [19]. In our study, the seriousness and fatality of the refractory IAH or ACS precluded us from dividing patients into DL and no-DL groups based on the intention to explore the effect of DL on critically ill patients. However, we divided patients into DL and no-DL groups based on the presence of refractory IAH and ACS, respectively. The mean IAP was significantly different between patients who did and did not undergo DL. In addition, except for MAP, the physiological parameters including APP, PaO2/FIO2 ratio, PIP, arterial lactate level and UOP were significantly different between the two groups. The mortality rate was 15% in patients who underwent DL and 40% in patients with ACS, which emphasizes the importance of prophylactic DL for blocking IAH from progressing to ACS.

The importance of prophylactic DL in pelvic fracture patients can be further validated by the fact that IAH/ACS develops in pelvic fracture patients more commonly due to 'polycystic' retroperitoneal hematoma or swollen bowel rather than due to fluid collection, as we found during laparotomy. Therefore, the most effective way to decrease IAP is via DL [18]. Ivatury was one of the first to advocate prophylactic decompression [3]. Indications for prophylactic DL have also been proposed [20]. Delayed decompression causes a marked decrease in gastric mucosal carbon dioxide. This decrease in carbon dioxide represents reperfusion injury, which is responsible for sudden deaths during or after late decompression of full ACS [9]. In our study, the respiratory function of 3 patients deteriorated after DL; two of them died, and ischemia-reperfusion injury may have contributed to these deaths. However, the UOP was universally improved after DL. This phenomenon is unsurprising because retroperitoneal hematomas commonly found in pelvic fracture patients can directly compress the kidney, and DL helps improve the APP.

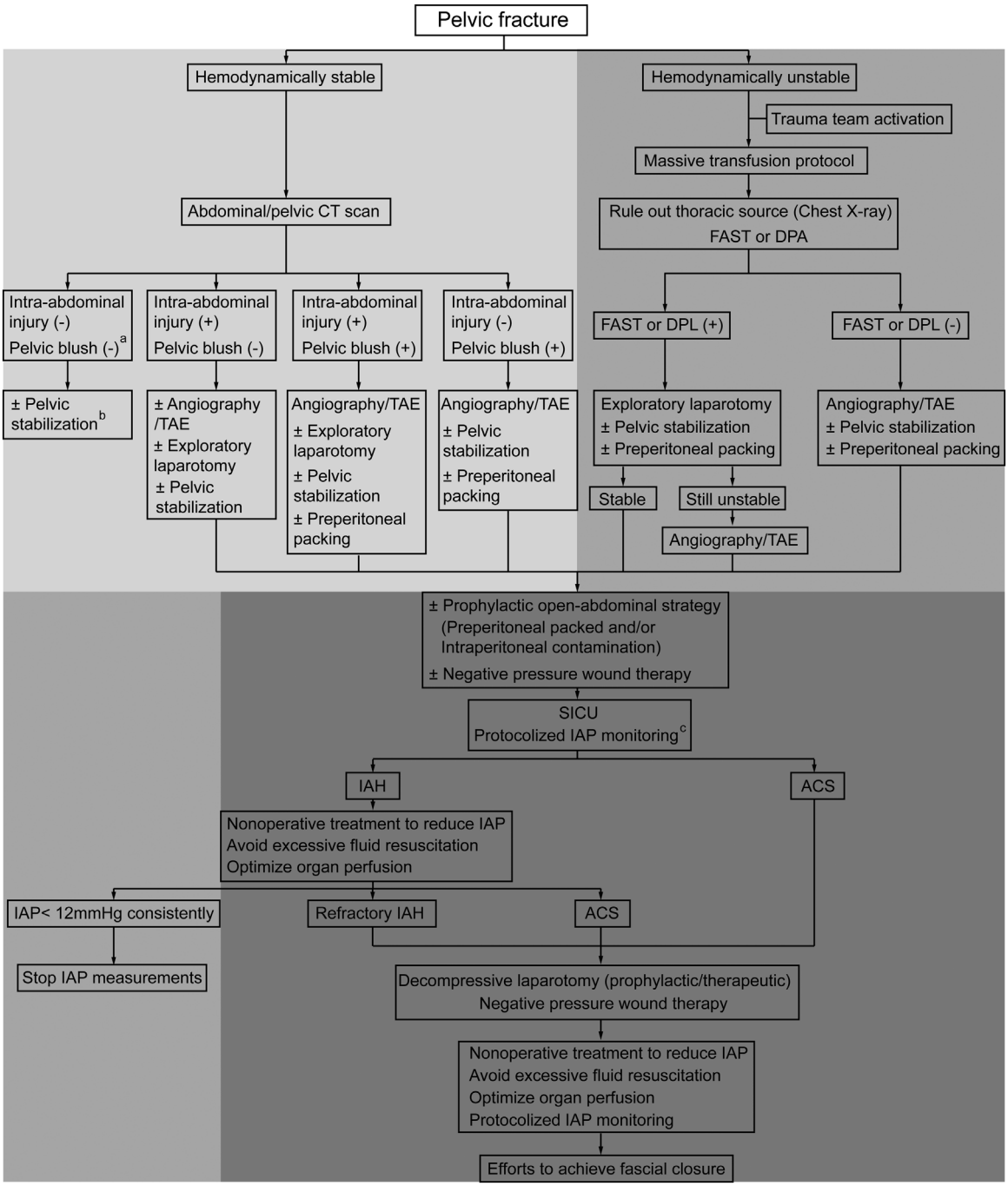

Fig. 7. The pathway for preventing and managing IAH/ACS in pelvic fracture patients. FAST (Focused assessment with sonography for trauma); DPL (Diagnostic peritoneal lavage); TAE (Transcatheter arterial embolization); SICU (Surgical intensive care unit); IAP (Intraabdominal pressure); IAH (Intraabdominal hypertension); ACS (Abdominal compartment syndrome).
[a]Pelvic blush: A contrast blush and the size of the blush or presence of a large pelvic hematoma may predict candidates for TAE.
[b]Pelvic stabilization was adopted for only biomechanically unstable pelvic fractures: injured anterior pelvic ring was stabilized by external fixation followed by TAE if necessary; injured posterior pelvic ring was stabilized by C-clamp followed by TAE if necessary.
[c]Protocolized IAP monitoring: Monitoring of IAP with serial measurements at least every 4 h.

We proposed a pathway to prevent and manage IAH/ACS after pelvic fractures to reduce morbidity and mortality based on existing evidence [1,21–24], the effectiveness of prophylactic/therapeutic DL, the risk of developing IAH/ACS due to massive fluid resuscitation, and our department's current clinical practice (Fig. 7). This pathway is divided into four quadrants as follows: quadrant I, management for hemodynamically stable pelvic fractures; quadrant II, management for hemodynamically unstable pelvic fractures; quadrant III, management for resolved IAH; quadrant IV, management for refractory IAH or ACS. Quadrant I and II have been extensively described elsewhere [21–24]. However, the transition from quadrant I and II to quadrant III and IV has not been emphasized, and this transition is a matter of life and death for pelvic fracture patients. If the patient has undergone preperitoneal packing and/or has intraperitoneal contamination, they should be treated with a prophylactic open-abdominal strategy and negative pressure wound therapy rather than definitive abdominal closure because this type of patient is at high risk of developing IAH/ACS. The standard surgical treatment algorithm in our study is DL combined with VSD. The goals of a DL include negating the worsening of IAH to prevent or stop organ dysfunction and allowing room for continued expansion of the abdominal viscera during the disease process [8]. VSD is a type of negative pressure wound therapy that represents the recommended method for temporary abdominal closure because it provides coverage and prevents excessive fascial retraction, which facilitates subsequent definitive abdominal closure. In addition, the VSD can remove inflammatory mediators and excess fluid from the peritoneal space [25]. Finally, efforts should be made to attempt early fascial closure because fascial closure within 2 days was associated with improved survival compared with later fascial closure [26].

Limitations

First, because of the study's retrospective design, it was susceptible to possible selection, performance, and recall biases. Patients were not randomized to either group, and selection bias may have influenced the results in a manner difficult to predict. In addition, the study's retrospective nature also precludes providing a more comprehensive set of physiological parameters to reflect all the important physiological changes related to IAH/ACS. Second, although we included only pelvic fracture patients with IAH/ACS, the patient population in our study was fairly heterogeneous due to the presence of polytrauma, which complicates interpreting the outcomes.

Conclusions

IAH/ACS is not rare in pelvic fracture patients. However, the pelvic fracture type does not appear to significantly affect the IAP level. Importantly, IAH/ACS that develops in pelvic fracture patients is much more commonly due to 'polycystic' retroperitoneal hematoma or swollen bowel rather than due to fluid collection; therefore, the most effective way to decrease IAP is DL. Despite a good physiological response to DL, the outcome for ACS was still poor, which emphasizes the importance of prophylactic DL for preventing refractory IAH from progressing to ACS. In addition, massive fluid resuscitation is a significant risk factor for IAH/ACS and should be optimized in the management of pelvic fracture patients. Following a pathway that incorporates prophylactic/therapeutic DL and optimized fluid resuscitation to prevent and manage IAH/ACS after pelvic fractures may reduce patient morbidity and mortality.

Ethical approval

Written informed consent was obtained from all participants and was approved by the Institutional Review Board of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology.

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Conflicts of interest

No any conflict of interest exists for all the authors.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.injury.2019.03.037.

References

[1] Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, et al. 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 Med 2013;39(7):1190–206.
[2] Morris Jr. JA, Eddy VA, Blinman TA, Rutherford EJ, Sharp KW. The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg 1993;217(5)576–84 discussion 584-576.
[3] 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(6)1016–21 discussion 1021-1013.
[4] Hessmann M, Rommens P. Bilateral ureteral obstruction and renal failure caused by massive retroperitoneal hematoma: is there a pelvic compartment syndrome analogous to abdominal compartment syndrome? J Orthop Trauma 1998;12(8):553–7.
[5] Hagiwara A, Fukushima H, Inoue T, Murata A, Shimazaki S. Brain death due to abdominal compartment syndrome caused by massive venous bleeding in a patient with a stable pelvic fracture: report of a case. Surg Today 2004;34 (1):82–5.
[6] Ali SR, Mohammad H, Sara S. Evaluation of the relationship between pelvic fracture and abdominal compartment syndrome in traumatic patients. J Emerg Trauma Shock 2013;6(3):176–9.
[7] Knight CJ, Wanko Mboumi I, Thompson EC. Severe pelvic fracture with profound hypotension: a case report and treatment algorithm. J Surg Case Rep 2017(6) rjx093.
[8] Carr JA. Abdominal compartment syndrome: a decade of progress. J Am Coll Surg 2013;216(1):135–46.
[9] Balogh ZJ, Lumsdaine W, Moore EE, Moore FA. Postinjury abdominal compartment syndrome: from recognition to prevention. Lancet 2014;384 (9952):1466–75.
[10] Malbrain ML. Different techniques to measure intra-abdominal pressure (IAP): time for a critical re-appraisal. Intensive Care Med 2004;30(3):357–71.
[11] Rogers WK, Garcia L. Intraabdominal hypertension, abdominal compartment syndrome, and the open abdomen. Chest 2018;153(1):238–50.
[12] Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. Intensive Care Med. 2006;32(11):1722–32.
[13] Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. II. Recommendations. Intensive Care Med 2007;33(6):951–62.

[14] Balogh ZJ, Martin A, van Wessem KP, King KL, Mackay P, et al. Mission to eliminate postinjury abdominal compartment syndrome. Arch Surg 2011;146(8):938–43.
[15] Cheatham ML, Safcsak K. Percutaneous catheter decompression in the treatment of elevated intraabdominal pressure. Chest 2011;140(6):1428–35.
[16] Cook RE, Keating JF, Gillespie I. The role of angiography in the management of haemorrhage from major fractures of the pelvis. J Bone Joint Surg Br 2002;84(2):178–82.
[17] Agri F, Bourgeat M, Becce F, Moerenhout K, Pasquier M, Borens O, et al. Association of pelvic fracture patterns, pelvic binder use and arterial angio-embolization with transfusion requirements and mortality rates; a 7-year retrospective cohort study. BMC Surg 2017;17(1):104.
[18] De Waele JJ. Abdominal compartment syndrome in severe acute pancreatitis – when to decompress? Eur J Trauma Emerg Surg 2008;34(1):11–6.
[19] Cheatham ML, Safcsak K. Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival? Crit Care Med 2010;38(2):402–7.
[20] Sugrue M. Abdominal compartment syndrome and the open abdomen: any unresolved issues? Curr Opin Crit Care 2017;23(1):73–8.
[21] Biffl WL, Smith WR, Moore EE, Gonzalez RJ, Morgan SJ, Hennessey T, et al. Evolution of a multidisciplinary clinical pathway for the management of unstable patients with pelvic fractures. Ann Surg 2001;233(6):843–50.
[22] Durkin A, Sagi C, Durham R, Flint L. Contemporary management of pelvic fractures. Am J Surg 2006;192(2):211–23.
[23] White CE, Hsu JR, Holcomb JB. Haemodynamically unstable pelvic fractures. Injury-Int J Care Injured 2009;40(10):1023–30.
[24] Eckroth-Bernard K, Davis JW. Management of pelvic fractures. Curr Opin Crit Care 2010;16(6):582–6.
[25] Miller PR, Meredith JW, Johnson JC, Chang MC. Prospective evaluation of vacuum-assisted fascial closure after open abdomen: planned ventral hernia rate is substantially reduced. Ann Surg 2004;239(5)608–14 discussion 614-606.
[26] Ramirez JI, Sen S, Palmieri TL, Greenhalgh DG. Timing of laparotomy and closure in burn patients with abdominal compartment syndrome: effects on survival. J Am Coll Surg 2018;226(6):1175–80.