

Volume: 4 | Issue: 6 | Nov - Dec 2023 Available Online: www.ijscia.com

DOI: 10.51542/ijscia.v4i6.18

Relationship Between Degree of Solid Organ Trauma to Intra-Abdominal Pressure (IAP) In Blunt Abdomen Trauma at Prof. Dr. I G.N.G. Ngoerah General Hospital, Bali, Indonesia

I Wayan Suwarna^{1*}, I Ketut Sudiasa², and Tjokorda Gde Bagus Mahadewa³

¹Department of General Surgery, Faculty of Medicine, Udayana University Prof. Dr. IGNG Ngoerah General Hospital, Denpasar, Indonesia (80113)

²Division of Trauma Surgery, Department of Surgery, Faculty of Medicine, Udayana University, Prof. Dr. IGNG Ngoerah General Hospital, Denpasar, Indonesia (80113)

³Department of Neurosurgery, Faculty of Medicine, Udayana University Prof. Dr. IGNG Ngoerah General Hospital, Denpasar, Indonesia (80113)

E-mail: ucupsuwarna86@gmail.com; sudiasaketutdr@gmail.com; tjokmahadewa@unud.ac.id

*Corresponding author details: I Wayan Suwarna; ucupsuwarna86@gmail.com

ABSTRACT

Background: Blunt abdominal trauma is a common trauma case with a high mortality rate. Increased Intra-Abdominal Pressure (IAP) is one of the factors that affect the morbidity and mortality rates of blunt abdominal trauma. In this condition the authors are interested in examining the relationship between the Degree of Solid Organ Trauma and Intra-Abdominal Pressure (IAP) in Blunt Abdominal Trauma. Objective: To determine the relationship between the degree of hepatic, spleen and multi-organ trauma to intra-abdominal pressure (IAP) in patients with blunt abdominal trauma. Methods: This study used a prospective observational design, with an analytic cross-sectional design to assess the relationship between the degree of solid organ trauma and intra-abdominal pressure (IAP) in blunt abdominal trauma involving 45 samples of patients with blunt abdominal trauma at Prof. Dr. I.G.N.G. Ngoerah Denpasar Hospital in the period November 2021 - November 2022. Results: Intra-abdominal pressure (IAP) was significantly correlated with the degree of spleen trauma with an r value of 0.403 (p: 0.046) which indicated that intra-abdominal pressure (IAP) increased with increasing degree of splenic trauma. Conclusion: Splenic trauma is associated and significantly correlated with increased intra-abdominal pressure (IAP) and its degree in blunt abdominal trauma patients.

Keywords: spleen; intra-abdominal pressure; blunt abdominal trauma

INTRODUCTION

Trauma is an increasingly significant health problem with high mortality and morbidity worldwide. Trauma caused by blunt force is one of the most common medical cases experienced by adults and children. Trauma is also said to be the leading cause of death globally, with 90% of the health burden falling on low- and middle-income countries (LMICs). Based on data from the World Health Organization (WHO), about 16,000 people die from injuries every day, and many of them are permanently disabled. Around 90% of the total, these cases are most prevalent in low- and middleincome countries [1]. As in Egypt, road traffic injuries are the most common cause of trauma and blunt abdominal trauma is the most common associated injury. Patients who survive their injuries may die from sepsis or Multiple Organ Disfunction Syndrome, also known as MODS.

The 2018 Basic Health Research report shows that abdominal trauma ranks sixth among the most common traumas in Indonesia, with only 2.2% or around 2,045 trauma cases being abdominal trauma. In contrast to the data obtained from Egypt, in Indonesia itself, injury or trauma most often occurs in the home environment (44.7%) and road trauma ranks second (31.4%). Generally, road accidents are motorcycle accidents, either without passengers (72.7%) or with passengers (19.2%). Based on Indonesia basic research of health data, the incidence of trauma due to traffic accidents in Bali was 16,481 (2.5%) cases and was still above the national average of 2.2%. [2]. According to patient data obtained from the recapitulation of patients admitted to the emergency room of Prof. Dr. I.G.N.G. Ngoerah Hospital in 2020, abdominal blunt trauma cases were 1.59% of the total trauma cases, 54% required surgery and the mortality rate reached 4.3%.

Blunt abdominal trauma can cause damage to internal organs, resulting in internal bleeding, causing bruising, or injury to the intestines, spleen, liver, and bowel. Patients may also present with extra-abdominal injuries such as extremity injuries [3,4]. An examination that can be seen is by assessing intra-abdominal pressure (IAP). Some things that can affect the IAP are the volume of solid organs and intestines (which can be filled with air, fluid, or feces), space-occupying lesions (ascites, blood, tumors), and the extensibility of the abdominal wall itself. If the IAP is more than 12 mmHg on two consecutive measurements within 48 hours, the condition is called intra-abdominal hypertension (IAH). The rapid progression of IAH can cause acute compartment syndrome (ACS) which is a condition of IAP more than 20 mmHg with or without abdominal perfusion pressure (APP) less than 60 mmHg which, if not treated immediately, can cause multiple organ dysfunction syndrome (MODS) [5,6].

Increased IAP has significant systemic effects, including causing an increase in jugular venous pressure and interfering with venous return from the brain, thus increasing intra-cranial pressure (ICP) leading to decreased cerebral blood flow. High intra-thoracic pressure (ITP) will increase central venous pressure and pulmonary artery pressure. Simultaneously, left ventricular afterload pressure increases due to increased vascular resistance. Increased ITP also increases right ventricular afterload, which when very high, can lead to right ventricular failure. The pulmonary organs are also indirectly affected. Decreased compliancy of the chest cavity will increase the pressure required to perform adequate mechanical ventilation [7]. In addition, IAH also inhibits splanchnic perfusion due decreased cardiac output and increased peripheral and splanchnic vascular resistance. When severe, tissue ischemia may occur [5].

Elevated IAP has been identified as an independent predictor of mortality during the critical illness period. This suggests that comprehensive treatment of IAH and ACS is crucial for patient survival. Based on research conducted by Cheatham and Safesak (2010), the mortality rate in IAH and ACS patients who have been managed in the form of open abdominal procedures is 28%, this finding is lower when compared to previous studies which state the mortality rate of IAH and ACS patients without open abdominal procedure management is 53% and 80% respectively. [8]. The study shows that open abdominal procedures are one of the important factors that can improve the survival of IAH/ACS patients and early management with open abdomen can be the main choice for managing patients who are refractory to less invasive conservative interventions.

Blunt abdominal trauma can traumatize solid organs directly or indirectly. The lien organ is known to be traumatized quite often due to traffic accidents that cause abdominal trauma [9].

The pancreas is also commonly traumatized in abdominal blunt injuries with trauma mechanisms similar to those of the lien plus falls from height or direct strikes to the abdominal region can also cause trauma [10]. In addition, the liver is also very susceptible to injury due to increased IAP caused by blunt abdominal trauma. It is important to know the relationship between increased IAP and the degree of solid organ trauma to minimize immediate complications in patients and minimize mortality and morbidity in patients with blunt abdominal trauma.

There is still no research that discusses the importance of the relationship between the degree of solid organ trauma and intra-abdominal pressure. So the authors are interested in conducting research with the title "The Relationship of the Degree of Solid Organ Trauma to Intra-Abdominal Pressure (IAP) in Blunt Abdominal Trauma at Prof. Dr. I.G.N.G. Ngoerah Hospital". This study was conducted with the aim of serving as a basis for decision making regarding optimal treatment in patients with blunt abdominal trauma, so as to prevent delays in surgery.

METHODS

This type of research is a prospective observational study, with an analytic cross-sectional design to assess the relationship between the degree of solid organ trauma and Intra-abdominal Pressure (IAP) in blunt abdominal trauma. The study was conducted at the Emergency Department Surgical Triage Prof. Dr. I.G.N.G. Ngoerah Denpasar in the period November 2021 - November 2022. Inclusion Criteria: 1) Patients with complaints of pain throughout the abdominal area who are clinically and radiologically diagnosed as blunt abdominal trauma; 2) Patient age above 18 years; 3) IAP can be measured through an intra vesical urinary catheter. Exclusion Criteria: 1) Patients with penetrating wounds, polytrauma, bladder injury, urological pathology, intra-abdominal masses and pregnant women; 2) Patients with nonorganic bleeding or those in severe hypovolemic shock requiring resuscitation; 3) Patients with blunt abdominal trauma clinically and during laparotomy surgery found injury to hollow organs; 4) Subjects with incomplete data, damaged medical record files and others. Data analysis was performed using SPSS for Windows version 21.0 software. The statistical analysis was carried out in the form of univariate proportion analysis, bivariate analysis multivariate analysis.

RESULTS

A total of 45 patients with blunt abdominal trauma were included in this study with a mean patient age of 28.78±10.65 years and 80% of the subjects were male. More than half of the subjects underwent laparotomy with 51.1% of patients having liver trauma, 24 (53.3%) having lien trauma, and no patients with pancreas trauma were found.

In terms of degree, 5% of patients had severe hepatic trauma and 28.9% of patients had severe lien injury. Some patients had more than 1 organ trauma and were classified as multi-organ trauma group.

In this study, there were 9 (20%) patients with a history of multi-organ trauma. The mean IAP pressure examined was 12.44 (±2.77) mmHg (Table 1).

TABLE 1: Variable baseline characteristics of the study subjects.

Variables	Value
Age	28.78±10.65 years
Gender	
Male	36 (80,0%)
Female	9 (20,0%)
Action	
Conservative	5 (11,1%)
Laparotomy	40 (88,9%)
Hepar Trauma	
Yes	23 (51,1%)
No	22 (48,9%)
Degree of Hepar Trauma	
Weight	5 (11,1%)
Lightweight	18 (40%)
Pancreatic Trauma	
No	45 (100%)
Lien Trauma	
Yes	24 (53,3%)
No	21 (46,7%)
Degree of Lien Trauma	
Weight	13 (28,9%)
Lightweight	11 (24,4%)
Multi-Organ Trauma	
Yes	9 (20%)
No	36 (80%)
IAP Pressure	12.44±2.77 mmHg

Bivariate analysis was performed in two stages, namely distinguishing the mean IAP in each degree of trauma and then classifying IAP according to its degree and analyzing the risk of increasing IAP. The analysis began with a normality test of the IAP variable which showed an abnormal distribution (p: 0.000; Shapiro Wilk test) so that the comparative analysis of IAP values in the groups with/out with hepatic and splenic trauma.

Then IAP was classified into two categories according to the pressure, namely degree I (12-15 mmHg) and degree II (16-20 mmHg).

Descriptive results showed that there were 13 (28.9%) patients with an increase in grade II IAP and 32 (71.1%) patients with an increase in grade I IAP. The results of chi-square analysis showed that only splenic trauma and multi-organ injury were significantly associated with the degree of IAP elevation. The Odds Ratio for lien injury was 4.286 (p: 0.043; 95%CI: 1.988 - 18.586) and multi-organ trauma was 4.375 (p: 0.045; 95%CI: 1.946 - 20.238) In this analysis, there was no significant association between the degree of lien injury and the degree of IAP elevation but this has been analyzed in more detail in the previous mean comparison analysis (Table 2).

TABLE 2: Chi-Square test results and risk analysis of the association between the degree of IAP increase and hepatic trauma, degree of hepatic trauma, splenic trauma, degree of splenic trauma, and multi-organ injury.

Variables	IAP Category		OR	P-value	95% CI
variables	Drajat II	First degree	UK	r-value	95% CI
Hepar Trauma					
Yes	7	16	0,202	1,167	0,321-4,247
No	6	16			
Degree of Hepar Trauma					
Weight	3	2	5,250	0,142	0,629 - 43,138
Lightweight	4	14			
Lien Trauma					
Yes	10	14	4,286	0,043	1,988 - 18,586
No	3	18			

Variables	IAP Category		ΩD	P-value	050/ CI
	Drajat II	First degree	OR	P-value	95% CI
Degree of Lien Trauma					
Weight	6	7	1,500	0,697	0,290 - 7,753
Lightweight	4	7			
Multi-Organ Trauma					
Yes	5	4	4,375	0,045	1,946 - 20,238
No	8	28			

To see the relationship between IAP and the degree of hepatic and splenic trauma, Spearman correlation analysis was performed because the degree of trauma is an ordinal variable. From the correlation analysis, it was found that IAP was significantly correlated with the degree of splenic trauma with an p-value of 0.403 (p: 0.046) indicating that IAP increases with the degree of splenic trauma. However, since not all patients had lien trauma, only 25 samples were analyzed at this stage.

Table 3 Correlation analysis test results of the relationship between IAP and the degree of lien and hepatic trauma.

Variables	Correlation Coefficient (r)	P-value
Degree of Lien Trauma	0,403	0,046*
Degree of Hepar Trauma	0,271	0,211

To see the independent relationship between abdominal trauma and IAP pressure and control for confounding variables, a linear regression multivariate analysis was performed. Linear regression was chosen because IAP is a numerical variable and to illustrate the effect of the presence or absence of organ trauma on IAP elevation. The results of the linear regression analysis showed that

lien trauma was a significant factor for increased IAP pressure (Table 4). However, it was not possible to analyze the increase in IAP by degree of abdominal trauma due to the small amount of data. Referring to the Adjuster R value², it can be seen that splenic trauma influenced 44% of the changes in IAP and the rest was influenced by variables not examined in this study.

TABLE 4: Multivariate analysis of the association between Hepar Trauma, Lien Trauma, and Multi-Organ Trauma with Intra-abdominal pressure (IAP) to control for confounding variables.

Variables	В	Standard Error	P-value	95%CI
Constant	6,767	1,000	0.000*	2.449-4.951
Lien Trauma	3,700	0,620	0,000	2,449-4,951

DISCUSSION

Trauma is an increasingly significant health problem with high mortality and morbidity worldwide. Trauma caused by blunt force is one of the most common medical cases in adults and children [11,12]. Trauma is a leading cause of death globally, with 90% of the health burden falling on low- and middle-income countries (LMICs) with mortality rates reaching 16,000. As many as 90% of these cases occur in low- and middle-income countries [1].

Abdominal trauma can be divided into penetrating abdominal trauma and blunt abdominal trauma. Penetrating trauma occurs when a foreign object penetrates the skin and enters the body, causing injury. In penetrating trauma, the object remains in the tissue or passes through the tissue and out of the body. Meanwhile, blunt trauma is caused by blunt force that does not penetrate the skin and objects. Blunt abdominal trauma can cause damage to internal organs such as bruising, internal bleeding, which often occurs from injuries to the intestines, spleen, liver and pancreas. Patients may also present with extraabdominal injuries such as limb injuries [3,4].

Blunt abdominal trauma can result in increased intraabdominal pressure (IAP) through mechanisms. By definition, intra-abdominal pressure or IAP is the pressure contained within the abdominal cavity. This pressure will increase when the diaphragm contracts (inspiration) and decrease when the diaphragm relaxes (expiration). Some things that can affect the high and low IAP are the volume of solid organs and intestines (which can be filled with air, fluid, or feces), space-occupying lesions (ascites, blood, tumors), and the extensibility of the abdominal wall itself [13]. However, physiologically IAP can also reach very high levels (up to 80 mmHg) but only temporarily during coughing, vasalva maneuvers, and heavy lifting or other conditions [5]. If the IAP measurement is more than 12, the condition is called intra-abdominal hypertension (IAH). The rapid progression of IAH can lead to acute compartment syndrome (ACS) a condition of IAP more than 20 mmHg with or without abdominal perfusion pressure (APP) less than 60 mmHg which, if not treated immediately, can lead to multiple organ dysfunction syndrome (MODS) [5,6].

Hepar is an important organ in the human body responsible for a series of functions that help support metabolism, immunity, digestion, detoxification, vitamin storage and other functions. Hepar is a solid abdominal organ that weighs about 2% of an adult's body weight, in addition, it has a dual blood supply that comes from the portal vein (about 75%) and the hepatic artery (about 25%) [14].

The liver is also highly susceptible to injury from increased IAP caused by blunt abdominal trauma. Animal and human studies have shown impaired liver cell function and liver perfusion even with a slight increase in IAP of 10 mmHg. In addition, complications of IAH and ACS also affect the liver such as acute liver function failure and decompensated chronic liver disease [15].

The relationship between hepatic trauma and IAP is bidirectional. On the one hand, increased IAP in patients may cause trauma to the hepatic tissue due to compression of the hepatic tissue and its vascularization. In particular, compression of the vena hepatica and inferior vena cava may cause restriction of hepatic reflux. Restriction is not only caused by increased IAP but also by hematoma in cases of intraabdominal bleeding [4].

On the other hand, hepatic trauma can also cause an increase in IAP itself. The dense vascularization of the hepatic can lead to massive bleeding, especially when the trauma causes rupture of the glison capsule, allowing blood to enter the peritoneal cavity [7]. This has been reported by Chen et al (2001) where it was reported that the increase in bleeding volume was directly related to IAP pressure. However, massive levels of bleeding are generally more common in grade IV-VI hepatic trauma, while grade I-III trauma very rarely causes bleeding and is generally treated conservatively [6].

Increased IAP pressure can cause several effects on the liver. Histologically, increased compression leads to increased leukocyte infiltration and an increased proportion of necrosis in hepatocytes which, if left untreated, can expand to cause hepatic failure. Macroscopically, clinical changes such as bile leakage, hemobilia, biliary peritonitis, hemoperitoneum, hepatic abscess, and delayed hemorrhage may appear and the risk of these complications appearing increases as the degree of hepatic trauma increases [6].

Despite the explanation of the relationship between hepatic trauma and increased IAP above, in this study, there was no significant relationship between the degree of hepatic trauma and IAP. This may be due to the fact that the hepatic trauma that occurred was generally grade I-III trauma and no massive intraabdominal bleeding occurred. In addition, hepatic trauma often occurs together with splenic trauma in the subjects of this study, and splenic trauma is a stronger factor in increasing IAP when compared to hepatic trauma.

The lien is the largest organ of the lymphatic system and is subject to traumatic injury in the form of penetrating or blunt trauma. Often, the resulting splenic trauma causes such an emergency that the patient must be transferred as quickly as possible to the operating room without the opportunity to evaluate the extent of splenic trauma prior to surgery. Splenic injury can be fatal not only at the time of admission to the Emergency Department (ED), but also due to delayed rupture of subcapsular hematoma or rupture of pseudoaneurysm [8].

In this study, there was a significant association between IAP pressure and lien trauma and multiorgan injury. So far, this is the first report of an evaluation of the factors influencing IAP elevation in patients with abdominal trauma. Although many patients suffered from hepatic trauma, this type of trauma did not appear to have a significant effect on IAP pressure changes. The degree of lien trauma was also found to have a significant effect, but was not included in the final analysis due to the small amount of data. In this regard, this study provides different results compared to previous studies where it was specifically found that splenic trauma was a major factor in increasing IAP pressure in abdominal trauma patients both in terms of mean pressure and degree of IAP pressure.

Splenic trauma was also an independent factor of increased IAP after controlling for confounding factors. Although splenic rupture has the potential to produce intra-abdominal bleeding and increase IAP pressure, the literature has mostly positioned splenic rupture as a result of increased IAP and not as a cause. [16]. This is because the lien structure is softer but contains denser vasculature and blood than the liver, so an increase in IAP is more at risk of causing a lien rupture [7]. However, Fomin et al did not evaluate the clinical history to see if the lien rupture was due to increased IAP pressure or if the increased IAP was a result of the lien rupture.

Dilektasli mentioned splenic injury as one of the diagnoses in the group of patients with increased IAP but the prevalence of hepatic trauma was significantly more in this study. It was not mentioned or analyzed which injury contributed more to increased IAP in this study as the focus was on length of stay and mortality. Therefore, the status of splenic trauma as a major factor for increased IAP in this study is a novel finding that should be confirmed in future studies [11].

Multi-organ trauma emerged as one of the significant variables in this study at the bivariate level of analysis. However, on multi-variate analysis that controlled for confounding variables and evaluated independent effects, multi-organ trauma did not emerge as a variable that significantly affected IAP. This is likely due to the fact that in patients with multi-organ trauma, all patients had both lien and hepatic trauma. However, lien, which significantly affected IAP at both bivariate and multi-variate levels, was the dominant factor affecting IAP, while hepatic trauma had no impact.

Along with blunt abdominal trauma, solid organs in the abdominal cavity are also commonly affected by blunt trauma. As in the lien organ, which is known to be traumatized quite often due to traffic accidents that cause abdominal trauma [9]. The pancreas is also commonly traumatized in blunt abdominal injuries with a trauma mechanism similar to that of the lien plus falls from height or direct strikes to the abdominal area can also cause trauma [10]. In addition, the liver is also very susceptible to injury due to increased IAP caused by blunt abdominal trauma. In general, we know that currently the benchmark for assessing the degree of abdominal solid organ trauma uses the American Association for the Surgery of Trauma (AAST) which is assessed on postoperative laparotomy findings and through abdominal CT scan findings in conservatively treated patients. Animal and human studies have shown impaired liver cell function and liver perfusion despite only a slight increase in IAP of 10 mmHg. In addition, complications of IAH and ACS also affect the liver such as acute liver failure and decompensated chronic liver disease [17].

However, elevated IAP can affect many abdominal organs simultaneously and cause physiological and pathological damage. Elevated IAP (exceeding 20mmHg) can cause MODS (Multiple Organ Dysfunction Syndrome). This can occur because in blunt abdominal trauma there is compression of the vena cava, reducing venous return, and ultimately reducing cardiac output. In addition, increased IAP can also cause cardiac arrhythmias that worsen blood pressure conditions. In blunt abdominal trauma there is also increased diaphragmatic pressure, pulmonary atelectasis, and impaired ventilation. In addition, increased intra-abdominal pressure can compress the renal veins resulting in decreased urine output, which can eventually lead to acute renal failure. Increased IAP can also decrease vital organ perfusion through aortic compression and splanic hypoperfusion, which can eventually lead to metabolic acidosis and MODS [18-21].

Blunt abdominal trauma can also cause abdominal compartment syndrome (ACS). ACS is defined as a condition in which persistent IAP > 20 mmHg (with or without abdominal perfusion pressure < 60 mmHg) is associated with organ dysfunction or failure [19]. Increased IAP can result in physiologic and functional impairment of many abdominal organs due to limited abdominal wall compliances. In patients with severe trauma, the incidence of ACS has been reported to be 14% 15% after laparotomy. Another study that studied ACS in patients with hepatic injury in blunt abdominal trauma using IAP 25 cm H2O as a cut-off point compared the two groups with respect to transfusion estimates, PaO2/FiO2 and peritoneal signs. All patients with an IAP of 25 cm H2O or more had an exacerbation of peritoneal signs before surgery and a strong correlation was found between IAP values and the presence of peritoneal signs. The study also described the failure of non-operative therapy in cases with severe hemodynamic disturbances (systolic pressure < 90 mm Hg) despite REFERENCES

resuscitation, and in cases of patients who were hemodynamically stable but with an IAP of 25 cm H2O or greater [22].

Changes in IAP have significant systemic effects in the form of manifestations of IAH resulting in the diaphragm to lift upward and reduce the volume of the thoracic cavity and increase intra-thoracic pressure. This leads to increased jugular venous pressure and interferes with venous return from the brain, thereby increasing intra-cranial pressure and decreasing cerebral blood flow. As a result of IAH, venous return is also impeded, causing lower limb edema. The presence of high ITP increases central venous pressure and pulmonary artery pressure.

In addition, left ventricular afterload pressure increases due to increased vascular resistance. Increased ITP also increases afterload on the right ventricle, which when very high, can lead to right ventricular failure, this will burden the work of the heart. The lungs are also indirectly affected. Decreased compliancy of the chest cavity will increase the pressure required to perform adequate mechanical ventilation [7]. In addition, IAH also inhibits splanchnic perfusion due to decreased cardiac output and increased peripheral and splanchnic vascular resistance. When severe, tissue ischemia may occur [5].

Furthermore, elevated IAP has been identified as an independent predictor of mortality during the critical illness period which emphasizes the importance of comprehensive management of IAH and ACS is crucial for patient survival. Cheatham and Safesak (2010) showed that the mortality rate in patients with IAH and ACS who had management in the form of open abdominal procedures was 28% which was lower when compared to previous studies which stated 53% and 80% respectively without open abdominal management. [8]. The study showed that combining operative and nonoperative interventions to reduce IAP can significantly improve the survival of IAH/ACS patients. Although there are not many supporting studies, IAP measurement has an important role in preventing various complications that may occur, especially in patients with blunt abdominal trauma.

CONCLUSION

The conclusion that can be drawn from this study is that lien trauma is associated and significantly correlated with increased Intra-abdominal Pressure (IAP) and its degree in abdominal blunt trauma patients.

ACKNOWLEDGMENTS

The authors would like to thank the Department of Surgery, Prof. Dr. IGNG Ngoerah Hospital, for the support of the authors in this work.

DECLARATIONS

Funding: No funding sources Conflict of interest: None declared Ethical approval: The study was approved by the institutional ethics committee.

- [1] Ibrahim W, Mousa G, Hirshon JM, El-Shinawi M, Mowafi H. Non-operative management of blunt abdominal solid organ trauma in adult patients. African Journal of Emergency Medicine: Revue Africaine de La Medecine d'urgence 2020; 10:123–6. https://doi.org/10.1016/j.afjem.2020.02.002.
- [2] Kementerian Kesehatan RI. Laporan Riskesdas 2018. Laporan Nasional Riskesdas 2018 2018; 53:154–65.
- [3] So, H-F, Nabi H. Handlebar hernia A rare complication from blunt trauma. International Journal of Surgery Case Reports 2018; 49:118–20. https://doi.org/10.1016/j.ijscr.2018.06.003.
- [4] Tarchouli M, Elabsi M, Njoumi N, Essarghini M, Echarrab M, Chkoff MR. Liver trauma: What current management? Hepatobiliary & Pancreatic Diseases International: HBPD INT 2018; 17:39–44. https://doi.org/10.1016/j.hbpd.2018.01.013.
- [6] Kanlerd A, Nakornchai K, Auksornchart K, Watkwaw W. Incidence, Outcomes, and Factors Associated with Intra-Abdominal Hypertension and Primary Abdominal Compartment Syndrome in Abdominopelvic Injury Patients. Anesthesiology Research and Practice 2020; 2020:1982078. https://doi.org/10.1155/2020/1982078.
- [7] Regli A, Pelosi P, Malbrain MLNG. Ventilation in patients with intra-abdominal hypertension: what every critical care physician needs to know. Annals of Intensive Care 2019; 9:52. https://doi.org/10.1186/s13613-019-0522-y.
- [8] Cheatham ML, Safcsak K. Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival? Critical Care Medicine 2010; 38:402-7. https://doi.org/10.1097/ccm.0b013e3181b9e9 b1.
- [9] Oelhaf R, Sugumar K, King K. Splenic Trauma. StatPearls [Internet], Treasure Island (FL): StatPearls Publishing; 2021.
- [10] Kumar A, Panda A, Gamanagatti S. Blunt pancreatic trauma: A persistent diagnostic conundrum? World Journal of Radiology 2016; 8:159–73. https://doi.org/10.4329/wjr.v8.i2.159.
- [11] Dilektasli E, Inaba K, Haltmeier T, Wong MD, Clark D, Benjamin ER, et al. The prognostic value of neutrophil-to-lymphocyte ratio on mortality in critically ill trauma patients. Journal of Trauma and Acute Care Surgery 2016; 81:882–8. https://doi.org/10.1097/ta.000000000000000980.

- [12] Koirala U, Thapa PB, Joshi MR, Singh DR, Sharma SK. Systemic Inflammatory Response Syndrome following Gastrointestinal Surgery. Journal of Nepal Medical Association 2017;56. https://doi.org/10.31729/jnma.3144.
- [13] Malbrain M, Cheatham M. Definitions and Pathophysiological Implications of Intraabdominal Hypertension and Abdominal Compartment Syndrome. The American Surgeon 2011;77 Suppl 1:S6-11.
- [14] Coccolini F, Coimbra R, Ordonez C, Kluger Y, Vega F, Moore EE, et al. Liver trauma: WSES 2020 guidelines. World Journal of Emergency Surgery 2020;15:24.
- [15] Malbrain MLNG, De laet IE. Intra-abdominal hypertension: evolving concepts. Clinics in Chest Medicine 2009;30:45–70, viii.
- [16] Coccolini F, Montori G, Catena F, Kluger Y, Biffl W, Moore EE, et al. Splenic trauma: WSES classification and guidelines for adult and pediatric patients. World Journal of Emergency Surgery 2017;12:40.
- [17] Malbrain MLNG, De Laet IE, De Waele JJ, Kirkpatrick AW. Intra-abdominal hypertension: definitions, monitoring, interpretation and management. Best Practice & Research Clinical Anaesthesiology 2013;27:249–70. https://doi.org/10.1016/j.bpa.2013.06.009.
- [18] Scheppach W. Abdominal compartment syndrome. Best Practice & Research Clinical Gastroenterology 2009;23:25–33. https://doi.org/10.1016/j.bpg.2008.11.009.
- [19] Malbrain MLNG, De laet I, Viaene D, Schoonheydt K, Dits H. In vitro validation of a novel methodfor continuous intra-abdominalpressure monitoring. Intensive Care Medicine 2007;34:740–5. https://doi.org/10.1007/s00134-007-0952-0.
- [20] KRON IL, HARMAN PK, NOLAN SP. The Measurement of Intra-abdominal Pressure as a Criterion for Abdominal Re-exploration. Annals of Surgery 1984;199:28–30. https://doi.org/10.1097/00000658-198401000-00005.
- [21] Malbrain M. Abdominal compartment syndrome. F1000 Medicine Reports 2009. https://doi.org/10.3410/m1-86.
- [22] Barnes GE, Laine GA, Giam PY, Smith EE, Granger HJ. Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology 1985;248:R208–13. https://doi.org/10.1152/ajpregu.1985.248.2.r2 08.