



Research Article

AN EVALUATION OF INTRA-ABDOMINAL PRESSURES AND ITS OUTCOMES IN PATIENTS UNDERGOING EMERGENCY LAPAROTOMY

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The presence of IAH is associated with an 11-fold increase in mortality compared with patients without IAH.

ABSTRACT

Background: Intraabdominal hypertension (IAH) is defined as a sustained or repeated pathologic elevation of IAP of greater than 12 mm Hg. The presence of IAH is associated with an 11-fold increase in mortality compared with patients without IAH.

Aims and objectives: This single centre clinical prospective observational study aimed at determining the incidence of intra-abdominal hypertension in patients undergoing emergency laparotomy and to study the outcomes in patients with raised intra-abdominal pressures in terms of morbidity (occurrence of burst abdomen) and mortality.

Materials and methodology: The study was conducted in a tertiary care teaching hospital in 100 patients undergoing emergency laparotomy after obtaining approval from the institutional ethics committee, over a period of 2 years from January 2016 to December 2017. The abdominal pressure was indirectly determined by measuring urinary bladder pressure with a Foley catheter.

Results: Incidence of IAH in patients: Pre-op IAH: 79%, IAH at 6 hours post-op: 11%, IAH at 24 hours post-op: 7 %, IAH at 48 hours post-op: 1%. There was significant association between increasing grade of IAH preoperatively with increasing morbidity. There was significant association between increasing grade of IAH preoperatively and post-operatively at 6 hours and 24 hours with mortality ($P = 0.00$). All patients with Grade IV IAH preoperatively did not survive.

Conclusion: The identification of patients at risk of developing IAH/ACS by screening measures of IAP is important and helps undertake effective preventive therapeutic actions.

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INTRODUCTION

The effect of the increased intra-abdominal pressure (IAP) in various organ systems has been studied over the past century [1]. Emerson first noted the cardiovascular morbidity and mortality associated with elevated IAP in 1911[2]. However, the recognition of the abdomen as a compartment and the concept of intra-abdominal hypertension (IAH) resulting in abdominal compartment syndrome (ACS) have only recently received attention.

IAP is the steady state pressure concealed within the abdominal cavity [3, 4]. Although IAP can physiologically reach elevated values transiently up to 80 mm Hg (cough, Valsalva maneuver, weight lifting, etc.), these values cannot be tolerated for extended periods. Normal IAP is approximately 5-7 mm Hg in critically ill adults [5].

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IAP may be increased to 12-15 mm Hg in postoperative patients. Chronic IAP elevations can be seen in liver cirrhosis with ascites, large ovarian tumors, pregnancy, chronic ambulatory peritoneal dialysis (CAPD), or obesity. It is usually increased in abdominal surgical emergencies. This increased IAP leads to significant organ dysfunction; respiratory, cardiac, renal, gastrointestinal which inevitably leads to increase in morbidity and mortality.

IAH is defined as a sustained or repeated pathologic elevation of IAP of greater than 12 mm Hg [3]. The presence of IAH is associated with an 11-fold increase in mortality compared with patients without IAH[5]. Due to the elevated intra-abdominal pressure, the diaphragm becomes increasingly elevated, so reducing thoracic volume and compliance, and increasing intrapleural pressure. To maintain adequate ventilation, airway pressures must be increased. Compressive atelectasis eventually results in ventilation-perfusion mismatch with hypoxia, hypercarbia and acidosis and thus pulmonary dysfunction [6,7]. Impairment of venous return from the abdominal cavity to the heart, and specifically from the

inferior vena cava, begins to occur with IAPs ≥ 15 mm Hg. Once the pressure has reached ≥ 20 mm Hg, there is substantial collapse of the mesenteric and renal veins, as well as the vena cava, which results in a considerable drop in venous return. The second factor that decreases cardiac output is the elevated after load due to increased systemic vascular resistance mostly from the high intra-abdominal pressure and increased intrathoracic pressure. The increased after load in addition to the venous compression will in turn affect the central nervous system, renal and gastrointestinal system through ischemia [8]. Abdominal compartment syndrome is defined as a sustained intra-abdominal pressure (IAP) >20 mm Hg that is associated with new onset of organ dysfunction or failure [3]. The detrimental effects of IAH occur long before the manifestation of compartment syndrome. The ACS, therefore, should be viewed as the end result of a progressive, unchecked increase in IAP from a myriad of disorders that eventually leads to multiple organ dysfunction [1].

The significant prognostic value of elevated intra-abdominal pressure has prompted many intensive care units to adopt measurement of this physiologic parameter as a routine vital sign in patients at risk.

In this study we evaluated the intra-abdominal pressures in patients undergoing emergency laparotomy and then recognized intra-abdominal hypertension in these patients and study the outcomes such as burst abdomen and mortality associated with this condition.

Aims and Objectives of the Study

- To determine the incidence of intra-abdominal hypertension in patients undergoing emergency laparotomy.
- To study the outcomes in patients with raised intra-abdominal pressures:
 - a. Morbidity (occurrence of burst abdomen).
 - b. Mortality.

Definitions

Intra-abdominal pressure

The abdomen can be considered a closed box with walls both rigid (costal arch, spine, and pelvis) and flexible (abdominal wall and diaphragm). The elasticity of the walls and the character of its contents determine the pressure within the abdomen at any given time [3, 9]. Since the abdomen and its contents can be considered as relatively non-compressive and primarily fluid in character, behaving in accordance with Pascal's law, the IAP measured at one point may be assumed to represent the IAP throughout the abdomen (with the rare exception of upper ACS[10]). It is therefore defined as a steady-state pressure concealed within the abdominal cavity. IAP increases with inspiration (diaphragmatic contraction) and decreases with expiration (diaphragmatic relaxation)[11]. It is also directly affected by the volume of the solid organs or hollow viscera (which may be either empty or filled with air, liquid or fecal matter), the presence of ascites, blood or other space-occupying lesions (such as tumors or gravid uterus), and the presence of conditions that limit expansion of the abdominal wall (such as burn or third-space edema)[9].

Normal IAP ranges from sub-atmospheric to 0 mmHg. Certain physiological conditions such as morbid obesity and pregnancy may be associated with chronic IAP elevations [9].

In the critically ill, IAP is frequently elevated above the patient's normal baseline. Recent abdominal surgery, sepsis, organ failure, the need for mechanical ventilation and changes in body position are all associated with elevations of IAP. Normal IAP is approximately 5-7 mm Hg in critically ill patients [12,13].

Abdominal perfusion pressure

APP is calculated as the mean arterial pressure (MAP) minus the IAP. APP has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation [14]. By considering both arterial inflow (MAP) and restrictions to venous outflow (IAP), APP has been demonstrated to be superior to either parameter alone in predicting patient survival from IAH and ACS [14]. APP values of at least 60 mmHg have been associated with improved survival in patients with IAH and ACS [14].

Filtration gradient

The renal filtration gradient (FG) is the mechanical force across the glomerulus and equals the difference between the glomerular filtration pressure (GFP) and the proximal tubular pressure (PTP). In the presence of IAH, PTP may be assumed equal to IAP and thus GFP can be estimated as MAP minus 2IAP. Thus, changes in IAP will have a greater impact upon renal function and urine production than that caused by changes in MAP. As a result, oliguria is one the first visible signs of IAH [14,15].

Intra-abdominal hypertension

In healthy individuals, normal IAP is $<5-7$ mmHg [47]. The upper limit of IAP is generally accepted to be 12 mmHg by the WSACS, reflecting the expected increase in normal pressure from clinical conditions that exert external pressure to the peritoneal envelope or diaphragm, including obesity and chronic obstructive pulmonary disease [16]. In contrast, IAH is defined as a sustained or repeated pathologic increase in IAP >12 mmHg [49].

According to the level of IAP, IAH is graded as follows:

- Grade I: IAP 12-15 mmHg
- Grade II: IAP 16-20 mmHg
- Grade III: IAP 21-25 mmHg
- Grade IV: IAP >25 mmHg

Abdominal compartment syndrome

Critical IAP in the majority of patients appears to reside between 10 and 15 mmHg [44]. It is at this pressure that reductions in microcirculatory blood flow and the initial development of ACS occur. Classically, ACS is defined by the triad: (a) pathologic state caused by an acute increase in IAP $>20-25$ mmHg, (b) presence of adverse effects on end-organ function, and (c) abdominal decompression has beneficial effects [14].

ACS can also be subcategorized based on its causes as primary, secondary, or recurrent. *Primary* ACS or "surgical" or abdominal ACS is characterized by the presence of acute or subacute IAH resulting from an intra-abdominal cause (abdominal trauma or post-abdominal surgery). This is also considered to be "classic" ACS [3]. *Secondary* ACS or "medical" or extra-abdominal ACS is characterized by the presence of subacute or chronic IAH resulting from conditions requiring massive fluid resuscitation, such as septic shock or

major burns. This is a fundamentally unique entity because it occurs in patients without a primary intraperitoneal injury or intervention [3]. *Recurrent ACS* (tertiary) represents the resurgence of ACS following resolution of an earlier episode[14].

Pathophysiology of intra-abdominal hypertension/ abdominal compartment syndrome

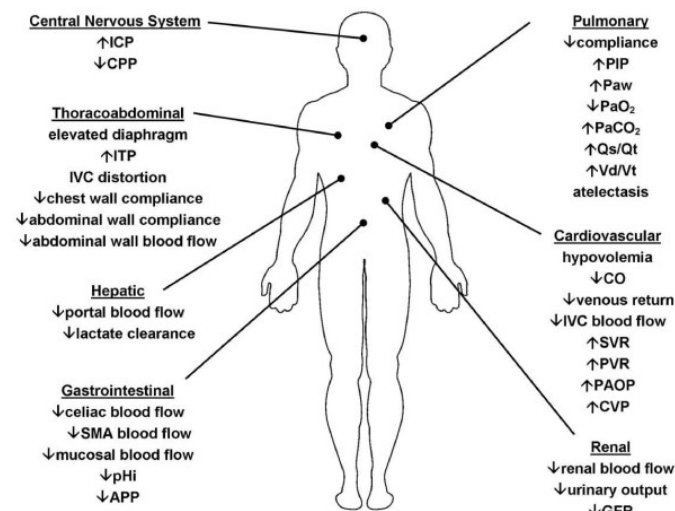


Figure 1

Pathophysiologic Implications of Intra-abdominal Hypertension: The effects of intra-abdominal hypertension are not limited just to the intra-abdominal organs, but rather have an impact either directly or indirectly on every organ system in the body. ICP - intracranial pressure; CPP - cerebral perfusion pressure; ITP - intrathoracic pressure; IVC - inferior vena cava; SMA - superior mesenteric artery; pH_i - gastric intramucosal pH; APP - abdominal perfusion pressure; PIP - peak inspiratory pressure; Paw - mean airway pressure; PaO₂ - oxygen tension; PaCO₂ - carbon dioxide tension; Qs/Qt - intrapulmonary shunt; Vd/Vt - pulmonary dead space ; CO - cardiac output; SVR - systemic vascular resistance; PVR - pulmonary vascular resistance; PAOP - pulmonary artery occlusion pressure; CVP - central venous pressure; GFR - glomerular filtration rate.

MATERIALS & METHODS

The study was conducted in a tertiary care teaching hospital in patients undergoing emergency laparotomy after obtaining approval from the institutional ethics committee. The study was done over a period of 2 years from January 2016 to December 2017.

Study Design

Single centre Clinical Prospective Observational Study.

Sample size

100 cases selected amongst patients undergoing emergency laparotomy.

Inclusion criteria

1. age ≥ 18 years and
2. all the patients undergoing emergency laparotomy.

Exclusion criteria

1. pregnant patients
2. Patients in whom Foley’s catheterization is not possible.
3. Patients with pelvic fractures, hematuria, neurogenic bladder, bladder rupture, cardiac, renal and respiratory conditions.

Parameters studied

A patient was included in the study only after a decision to operate upon him/her was taken. Patient specifics were noted along with the indication for surgery. Readings taken

preoperatively and postoperatively at 6hrs, 24 hrs. and 48 hrs. were noted.

Parameters Noted

Intra-abdominal pressures: Preoperatively, Postoperatively at 6 hrs. 24 hrs. and 48 hrs.

Preoperatively

1. Urine output
2. Serum creatinine

Postoperatively

1. Duration of surgery
2. Pulmonary inspiratory pressures at 6hrs, 24 hrs. and 48 hrs.
3. Duration of hospital stay
4. Morbidity (Burst abdomen)
5. Mortality

Measurement of Intra-Abdominal Pressure

The abdominal pressure was indirectly determined by measuring urinary bladder pressure with a Foley’s catheter. The patient was catheterized with a 16-gauge Foley’s catheter. The bladder was drained and then filled with 50 ml of sterile saline through the Foley’s catheter. The tubing of the collecting bag was clamped. The catheter was connected to a three-way stopcock and saline manometer. The symphysis pubis was the zero reference, and pressure was measured in centimeters of water at end-expiration. A conversion factor of 1.36 was used to convert the pressure into millimeter of Hg.

Interpretation of findings was based on the following criteria

Grading of intra-abdominal hypertension for adults

1. Grade I: 12-15 mmHg;
2. Grade II: 16-20 mmHg;
3. Grade III: 21-25 mmHg; and
4. Grade IV: >25 mm Hg

RESULTS

Characteristics of the Study Participants

A total of 100 patients were enrolled in this study. Of these 77 were males and 23 females. Patients’ age ranged from 13 to 90, the mean age being 33.77 (SD ± 14.682). Out of 100 cases, 38 were operated for exploratory laparotomy due to trauma (including both blunt and penetrating abdominal trauma). The remaining 62 were operated for a non-traumatic cause including exploratory laparotomy for perforative peritonitis and acute intestinal obstruction. Trauma: Non-Trauma distribution = 38:62.

Table 1 Pre-operative IAP grading

IAP grading	Trauma	Non-trauma	Total no. of patients
Normal	11	10	21
Grade I	14	22	36
Grade II	5	22	27
Grade III	6	7	13
Grade IV	2	1	3
	38	62	100

Pre-op IAP grading is as shown in Table 1. Normal IAP was present in 21% of patients preoperatively. Pre-op IAH was seen in 79% of the patients.

Table 2 Post-operative at 6 hours IAP grading

IAP grading	Trauma	Non-trauma	Total no. of patients
Normal	32	57	89
Grade I	1	4	5
Grade II	1	0	1
Grade III	1	0	1
Grade IV	3	1	4
	38	62	100

Post-op at 6hours IAP is as shown in Table 2. Normal IAP was present in 89% of patients. IAH was present in 11% of patients.

Table 3 Post-operative at 24 hours IAP grading

IAP grading	Trauma	Non-trauma	Total no. of patients
Normal	32	61	93
Grade I	1	0	1
Grade II	0	0	0
Grade III	0	0	0
Grade IV	5	1	6
	38	62	100

Post-op at 24 hours IAP is as shown in Table 3. Normal IAP was present in 93% of patients. IAH was present in 7% of patients.

Table 4 Post-operative at 48 hours IAP grading

IAP grading	Trauma	Non-trauma	Total no. of patients
Normal	18	51	69
Grade I	1	0	1
Grade II	0	0	0
Grade III	0	0	0
Grade IV	0	0	0
	19	51	70

Post-op at 48 hours IAP is as shown in Table 4. Normal IAP was present in 69% of patients. IAH was present in 1 % of patients.

Incidence of IAH in patients

1. Pre-op IAH: 79%
2. IAH at 6 hours post-op: 11%
3. IAH at 24 hours post-op: 7 %
4. IAH at 48 hours post-op: 1%

Incidence of IAH in Trauma patients

1. Pre-op IAH: 71.05%
2. IAH at 6 hours post-op: 15.78%
3. IAH at 24 hours post-op: 15.78%
4. IAH at 48 hours post-op: 5.2%

Incidence of IAH in Non-trauma patients

1. Pre-op IAH: 83.87%
2. IAH at 6 hours post-op: 8.06%
3. IAH at 24 hours post-op: 1.61%
4. IAH at 48 hours post-op: 0%

Pearson Chi-square test showed association between increasing grade of IAH preoperatively with increasing morbidity. This association was found to be significant. ($P = 0.00$)

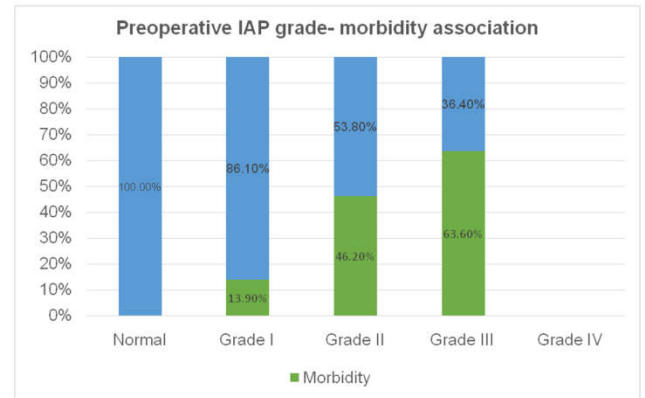


Figure 1

Figure 1 shows stacked column chart showing an association between increasing IAH grade preoperatively with morbidity.

Pearson Chi-square test showed association between increasing grade of IAH preoperatively with increasing mortality. This association was found to be significant. ($P = 0.00$)

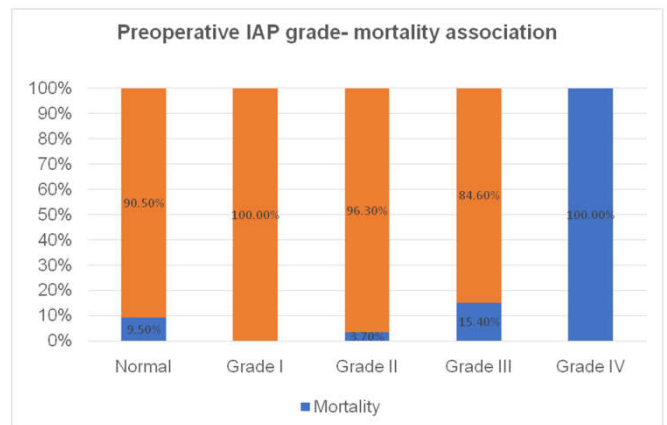


Figure 2

Figure 2 shows stacked column chart showing an association between increasing IAH grade preoperatively with mortality.

Pearson Chi-square test showed no association between increasing grade of IAH with increasing morbidity at post-op 6 hours ($P = 0.27$)

Pearson Chi-square test showed association between increasing grade of IAH with increasing mortality in post-op patients at 6 hours. This association was found to be significant. ($P = 0.00$)

Pearson's Chi-square was used and it showed no association between IAH grading at post op 24 and 48 hours and morbidity. However there was a significant association ($P=0.00$) between IAH grading at post op 24 hours and mortality.

ANNOVA test was used and the association between increasing trend of preoperative IAP and decreasing trend of preoperative urine output was found to be significant ($P = 0.00$) (Figure 10). There was no significant association between preoperative IAP and duration of surgery or serum creatinine ($P>0.1$).

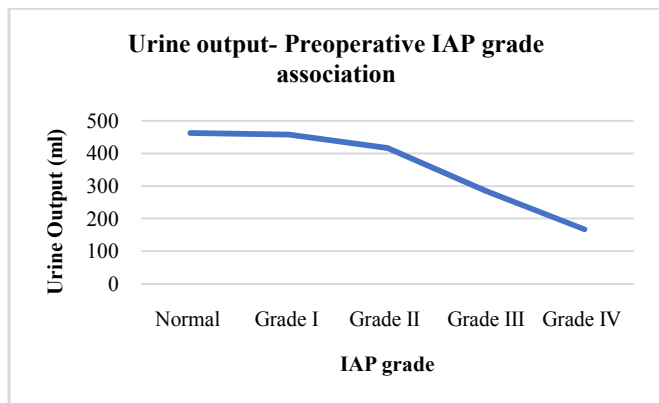


Figure 3

ANNOVA test was used and the association between increasing trend of preoperative IAP and increasing trend of duration of hospital stay. However, a sudden dip in this trend is seen in patients with Grade IV IAP. This association was found to be significant ($P = 0.00$)

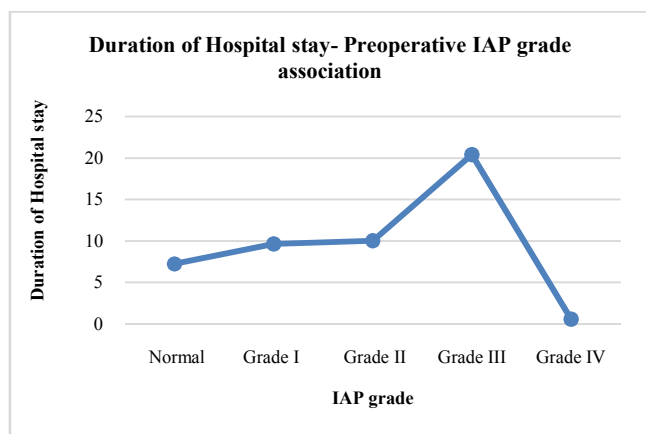


Figure 4

DISCUSSION

Basic characteristics of the study participants

There were 77% males and 23% females. A similar ratio was seen in the studies by Khan et al (76% males); Hong *et al.* (72% males) and Meldrum *et al.* (70% males), but Sugrue *et al.* and Cheatham *et al.* reported about 60% males in their study groups[6,45,61-62,63]. The mean \pm SD (range) age in our study was 33.77 ± 14.682 (range 13-90) years. This was similar to the mean age reported by Khan et al of 34 ± 14 [17]. Cheatham *et al.* have reported a mean age of 51 ± 19 years, Meldrum *et al.* 39 ± 9 years, and Hong *et al.* 42 years [14, 18-19]. Of the 100 patients, there were 38 trauma patients. This was in contrast to the study by Khan et al who had 19% trauma patients and to the study by Cheatham *et al.* who had 68% trauma patients in their study group [14,17]. Our sample included all those who underwent emergency laparotomy, hence a predominance of general surgical non-trauma patients as compared to trauma patients.

Intra-abdominal pressures

The mean (SD) IAPs before and post laparotomy at 6 hours were 14.9 (5.043) mm Hg and 8 (2.942) mm Hg respectively. This finding was similar to the mean (SD) IAPs in the study group of Khan et al before and after laparotomies which were 18 (4.8) mm Hg and 6 (1.7) mm Hg, respectively, in the patients who had IAH at admission [63]. The mean (SD) IAPs

in the study group of Sugrue *et al.* before and after decompressions were 16.6 (9.4) mm Hg and 10.3 (3.1) mm Hg, respectively[6]. Meldrum *et al.* reported higher values of IAP (SD) pre- and post-op: 27 (2.3) and 14 (4.6) mm Hg, respectively[61]. This can be explained by the observation that in our study, 62% of the patients had a non-trauma cause for exploratory laparotomy including perforative peritonitis and acute intestinal obstruction leading to elevated IAP which, after decompression and removal of liters of fluid and gas, returned to normal level immediately.

Incidence of IAH

The incidence of IAH Preoperatively was 79% and at 6 hours post-operatively was 11%. This was similar to the incidence of IAH in the study of Khan et al which was 80% at admission and 3.55% at 6 hours post-operatively [17].

Out of 79% Patients who had IAH Preoperatively, 36% had Grade I IAH, 27% had Grade II, 13% had Grade III and 3% had Grade IV IAH.

Post operatively at 6 hours out of 11% patients who had IAH, 5% had Grade I, Grade II and III were present in 1% and 4% had Grade IV IAH.

The incidence of IAH in trauma patients were 71.05% preoperatively and 15.78% at 6 hours post-operatively as compared to non-trauma patients in whom 83.87% had IAH preoperatively and 8.06% at 6 hours post-operatively.

Outcome factors associated with IAH

There was significant association between increasing grade of IAH preoperatively with increasing morbidity ($P = 0.00$). Patients who died eventually were excluded from morbidity. However no significant association was found between increasing grade of IAH postoperatively and morbidity.

There was significant association between increasing grade of IAH preoperatively and post-operatively at 6 hours and 24 hours with mortality ($P = 0.00$). All patients with Grade IV IAH preoperatively did not survive. Additionally, post operatively at 6 hours patients with Grade II, III and IV patients did not survive, emphasizing the need to objectively measure post-operative IAP especially at 6 hours leading to early recognition which can proceed with effective intervention whenever possible. No significant association, however, was found between increasing grade of IAH postoperatively at 48 hours and mortality, further stressing the fact that most of patients with increased grade of IAH had expired. Only one such patient with increased preoperative IAP had died after 48 hours.

Thus, even though there was significant association between increasing grade of IAP both preoperatively and postoperatively, it was beyond the scope of this study to describe it as a predictor of morbidity and mortality, as other factors responsible for burst abdomen like anemia, malnutrition, obesity, diabetes mellitus, underlying disease, sepsis, surgery related factors like, and type of incision, type of closure, suturing material, and suturing method influence were not considered [20-21]. Similarly, other factors responsible for mortality like dependent functional status, advanced age, malignancy, comorbidities including concurrent diabetes, cardiovascular disease, and lung disease and thus corresponding high ASA score (American Association of

Anesthesiologists physical status classification) were not studied [22].

There was significant association between increasing trend of preoperative IAP and decreasing trend of preoperative urine output.

Preoperative IAH grade was also significantly associated with increasing trend of duration of hospital stay. However, a sudden dip in this trend due to mortality of the patients with Grade IV IAH, thus causing a short duration of hospital stay.

Limitation

The measurements may have varied depending on whether if they were taken at end of inspiration or at the end of expiration which may have led to under estimation of the readings.

At 48 hours, the IAP measurements were omitted if patient had no episode of IAH, pre-op and post-op up to 24 h. This was done for the concern of causing urinary tract infections in the patients, hence the reading 48 h was to be assumed as normal (but this was not analyzed) hence lesser number of subjects at 48 h.

Postoperatively at 6hours, 24 hours and 48 hours, pulmonary inspiratory pressures were to be measured. However due to technical difficulties this could not be measured and was thus excluded from the study.

CONCLUSION

A single centre clinical prospective observational study was conducted in a tertiary care teaching hospital for a period of 24 months (January 2016 to December 2017) on 100 cases (77 men and 23 women) selected amongst patients undergoing emergency laparotomy

Significant association was found between preoperative IAP grade and morbidity and mortality of patients. Also, the grade of IAP was found to be significantly associated to urine output and duration of hospital stay.

Thus, clinical examination as a method itself is insufficient for determining the presence of IAH. A standardized measurement of the intra-abdominal pressure is fundamental for the establishment of intra-abdominal hypertension and abdominal compartment syndrome. The identification of patients at risk of developing IAH/ACS by screening measures of IAP is important and helps undertake effective preventive therapeutic actions.

References

1. Saggi BH, Sugeran HJ, Ivatury RR, Bloomfield GL. Abdominal compartment syndrome. *J Trauma*. 1998; 45:597-609.
2. Mohapatra B. Abdominal compartment syndrome. *Indian J Crit Care Med*. 2004;8:26-32.
3. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppäniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Johansson K, Kolkman K, Wilmer A. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med*. 2006;32:1722-32.
4. Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, Balogh Z, Leppäniemi A, Olvera C, Ivatury R, D'Amours S, Wendon J, Hillman K, Wilmer A. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med*. 2007; 33:951-62.
5. Sugrue M, Buist MD, Hourihan F, Deane S, Bauman A, Hillman K. Prospective study of intraabdominal hypertension and renal function after laparotomy. *Br J Surg*. 1995;82:235-8.
6. Moore AF, Hargest R, Martin M, Delicata RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. *Br J Surg*. 2004;91(9):1102-1110. doi: 10.1002/bjs.4703.
7. Michael L Cheatham Abdominal Compartment Syndrome: pathophysiology and definitions. *ScandJ TraumaResuscEmerg Med*. 2009;17:10
8. Rezende-Neto JB, Moore EE, Melo de Andrade MV, Teixeira MM, Lisboa FA, Arantes RM, et al. Systemic inflammatory response secondary to abdominal compartment syndrome: stage for multiple organ failure. *J Trauma*. 2002;53:1121-8.
9. Papavramidis TS, Duros V, Michalopoulos A, Papadopoulos VN, Paramythiotis D, Harlaftis N. Intra-abdominal pressure alterations after large pancreatic pseudocyst transcuteaneous drainage. *BMC Gastroenterol*. 2009;9:42.
10. De Laet IE, Malbrain M. Current insights in intra-abdominal hypertension and abdominal compartment syndrome. *Med Intensiva*. 2007;31:88-99.
11. Ogilvie WH. The late complication of abdominal war wounds. *Lancet* 1940;2:253-256.
12. Sanchez NC, Tenofsky PL, Dort JM, Shen LY, Helmer SD, Smith RS. What is normal intra-abdominal pressure? *Am Surg*. 2001;67:243-8.
13. Lerner SM. Review article: the abdominal compartment syndrome. *Aliment Pharmacol*. 2008;28:377-84.
14. Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF. Abdominal perfusion pressure: A superior parameter in the assessment of intra-abdominal hypertension. *J Trauma*. 2000;49:621-6.
15. Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. *Arch Surg*. 1999;134:1082-5.
16. Lambert DM, Marceau S, Forse RA. Intra-abdominal pressure in the morbidly obese. *Obes Surg*. 2005;15:1225-32
17. Shehtaj Khan, Akshay Kumar Verma, Syed Moied Ahmad, and Reyaz Ahmad. Analyzing intra-abdominal pressures and outcomes in patients undergoing emergency laparotomy. *J Emerg Trauma Shock*. 2010 Oct-Dec; 3(4): 318-325.
18. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM. Prospective characterization and selective management of the abdominal compartment syndrome. *Am J Surg*. 1997;174:667-73.
19. Hong JJ, Cohn SM, Perez JM, Dolich MO, Brown M, McKenney MG. Prospective study of the incidence and outcome of intra-abdominal hypertension and the abdominal compartment syndrome. *Br J Surg*. 2002;89:591-6.

20. Mahey R, Ghetla S, Rajpurohit J, Desai D, Suryawanshi S. A prospective study of risk factors for abdominal wound dehiscence. *IntSurg J* 2017;4:24-8.
21. Madsen G, Fisher L, Wara P. Burst abdomen clinical features and factors influencing mortality. *Danish Med Bulletin*. 1992; 39(2):183-5.
22. Sørensen L, Malaki A, Wille-Jørgensen P, Kallehave F, Kjaergaard J, Hemmingsen U, *et al*. Risk factors for mortality and postoperative complications after gastrointestinal surgery. *J GastrointestSurg* 2007 Jul; 11 (7):903-10.

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