Original Article

MEASUREMENT OF INTRA-ABDOMINAL PRESSURE AS AN INDICATOR OF MORBIDITY AND MORTALITY IN CRITICALLY ILL SURGICAL PATIENTS

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ABSTRACT

Intra abdominal pressure (IAP) is defined as the pressure in the peritoneal cavity and an acute rise in this pressure can result in adverse physiological consequences termed Abdominal Compartment Syndrome (ACS). This increase in abdominal pressure can affect multiple organ systems and once a certain stage has been passed, the effects are no longer reversible by decompression. So challenge is to be aware of the condition and act sufficiently early to reverse the effects.

Aims: 1) To assess whether intra abdominal pressure is an independent predictor of morbidity and mortality for patients undergoing emergency exploratory laparotomy.

2) To identify the hidden causes of abdominal compartment syndrome.

Settings and Design: this was a randomised controlled trial involving 50 patients, who were admitted and underwent emergency laparotomy.

Methods and Material: 50 patients who underwent emergency laparotomy due to various indications ,were included in the study. Intra abdominal pressure was measured through urinary bladder with Foley's catheter, which was connected to saline manometer. Readings were taken preoperatively and then post operatively at 0, 6, 24 and 72 hours. Grading of intra abdominal hypertension (IAH) was done as per the pressure. ACS was labelled if IAH was associated with at least one newly developed organ system dysfunction.

Results Pre operative IAH was seen in 62% of the patients. Raised IAP at 6 hours and 24 hours is significant predictor of morbidity ($p = 0.031$ and $p = 0.004$ respectively) whereas, raised IAP at 48 hours is a significant predictor of mortality. Similarly, raised IAP at 6 hours is also a significant predictor for longer hospital stay ($p = 0.031$).

Conclusions: **:** IAP is a significant indicator of morbidity and mortality in the patients undergoing emergency laparotomy. Thus a thorough monitoring and early decompression can decrease the morbidity and mortality rate.

Key-words: Abdominal compartment syndrome, intra-abdominal hypertension, intra-abdominal pressure

Key Messages: IAP is an easy to determine and vital indicator of morbidity in patients undergoing emergency laparotomy

INTRODUCTION

The term "intra-abdominal pressure" (IAP) is defined as the pressure in the peritoneal cavity. Recent studies have established that normal IAP is 5–7 mm Hg in critically ill patients.[1] Abdominal compartment syndrome (ACS) has been defined as the adverse physiological consequences that occur as a result of an acute increase in intra-abdominal pressure.[2]Affected patients experience splanchnic ischemia, central nervous system impairment and reduced cardiac output refractory to any means of support. If left untreated abdominal compartment syndrome is fatal.

Defined by IAP > 12 mmHg, IAH can be graded as: [2]

Grade I. IAH: IAP between 12-15 mmHg

Grade II. IAH: IAP between 16-20 mmHg

Grade III. IAH: IAP between 21-25 mmHg

Grade IV. IAH: IAP greater than 25 mmHg

Intra-abdominal pressure can be measured by direct and indirect methods.[3]

Direct methods:

1. Through a metal canula or by a wide bore needle that is inserted into the peritoneal cavity and attached to a saline manometer.
2. During laparoscopy.

Indirect methods:

1. Urinary bladder pressure (method of choice)
2. Gastric pressure
3. Rectal pressure
4. Pressure in vagina
5. Inferior vena cava pressure

Intra-bladder pressure (IBP) monitoring is considered the method of choice and is measured through the patient's indwelling urinary Foley catheter, utilizing the bladder wall as a passive transducing membrane.[4]

Identification of patients at risk of acute rise in intra- abdominal pressure :

Abdominal compartment syndrome can arise both in medical and surgical patients. Groups at risk include those with major trauma who are undergoing damage control surgery[19,] general surgical patients, especially those undergoing laparotomy for major bleeding or with already oedematous or ischemic bowel and patients having lengthy complex surgery such as abdominal vascular procedures. Abdominal compartment syndrome can partly reflect the systemic consequences of ischemia and reperfusion, with a tendency to oedema. Mechanical problems causing difficult abdominal closure, such as abdominal packs, distended bowel and hematoma are also predisposing factors. An important risk factor for the development of abdominal compartment syndrome is the volume of fluid used for resuscitation, the additional volume loading proving to be an independent factor for development of abdominal compartment syndrome.[5]

Morbidity and Mortality

Emerson was the first who noted the morbidity and mortality associated with elevated IAP in 1911[4.] The presence of IAH was associated with an 11-fold increase in mortality compared with patients without IAH.[6]

Morbidity due to raised IAP is also high and related to wound closure leading to impaired wound healing, dehiscence and bowel fistulas.[7] Raised IAP significantly prolongs intensive care unit stay and causes more severe organ dysfunctions particularly respiratory and renal.[8]

In 1951 M.G. Baggot identified abdominal dehiscence as the main factor due to increased IAP and recommended avoiding closure under tension and leaving the abdomen open.[9]

In 1993 Lacey et al used Urinary bladder pressure as a guide to close abdominal wall defects in infants and found decreased morbidity by this method.[10]

Postoperative monitoring of intra-abdominal pressure can be used to determine the optimal time for definitive abdominal closure.

Conditions such as liver failure, decompensated chronic liver disease and liver transplantation are

complicated by the development of IAH and ACS.[11]

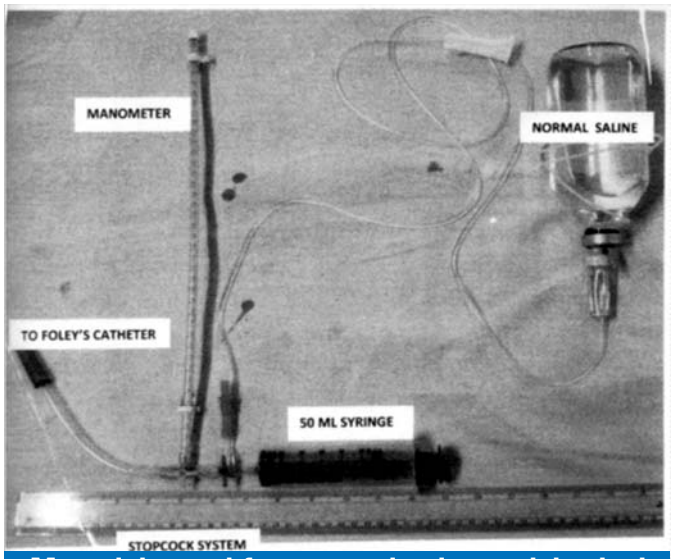
The detrimental effects of IAH occur long before the manifestation of compartment syndrome hence mortality despite timely and adequate treatment remains high and ranges 63-72%.[12]

The recognition of IAP as an independent prognostic factor will help in the "goal-directed" approach and reduce the associated morbidity and mortality in critically ill surgical patients.

Subjects and Methods :

The study was done on 50 patients, who were admitted and underwent emergency laparotomy. Pregnant patients and patients in whom Foley's catheterisation was not possible were excluded.

Intra-abdominal pressure was measured through urinary bladder with Foley's catheter which was connected to a saline manometer. Readings were taken preoperatively and then postoperatively at 0, 6, 24, and 72 hours. If IAP remained below 12 mm Hg, measurements were discontinued after 24 hours.



Materials used for measuring intra-abdominal pressure.

The term "abdominal compartment syndrome" was used when IAH was associated with at least one newly developed organ system dysfunction. In patients with ACS, the decision to proceed with decompressive laparotomy laid in the hands of the primary surgeon in-charge of the patient.

RESULTS

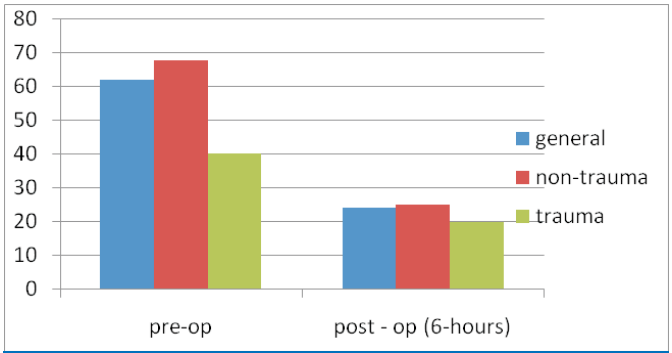
Pre-op IAH was seen in 62% of the patients.

	Total	Non-trauma	Truma
No IAH	19	13	6
Grade I	15	13	2
Grade II	15	14	1
Grade III	0	0	0
Grade IV	1	0	1

Pre-operative IAP grading

	Non-trauma	Trauma	Total
No IAH	30	8	38
Grade I	9	2	11
Grade II	1	0	1
Grade III	0	0	0
Grade IV	0	0	0

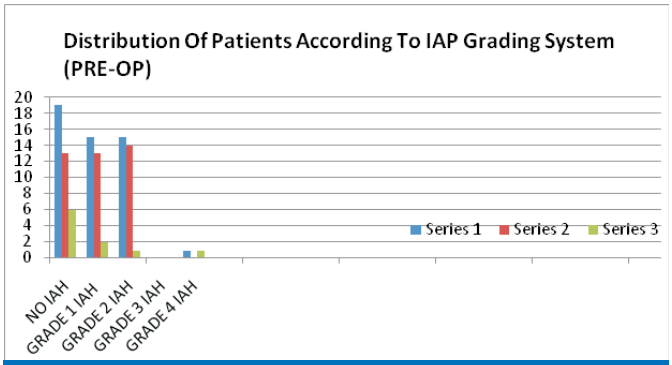
Post-op (6 hours) IAP grading



Incidence of IAH(%)

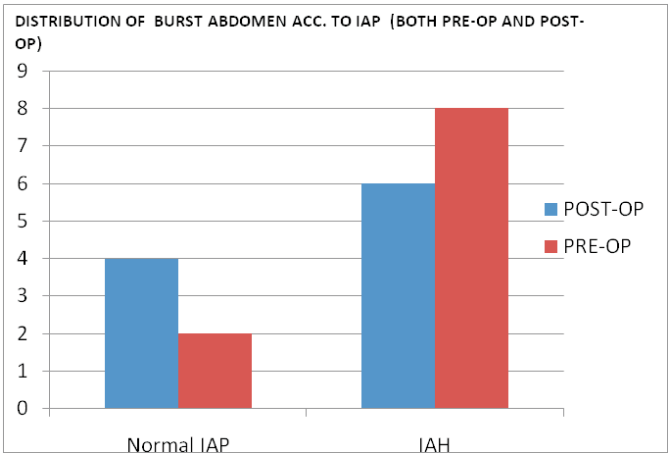
Only 56% of the patients had intra-abdominal pressure values within normal limits in post operative period. Intra Abdominal Pressure in most of the patients had reached Grade 1 IAH value (34% patients) and in 2% of patients intra-abdominal pressure values reached Grade 2 IAH value. None of the patient reached grade 3 IAH value or higher intra-abdominal pressure values. 2 patients had newly developed organ dysfunction along with intra-abdominal hypertension in the post operative period

hence they were diagnosed to have developed abdominal compartment syndrome .



Morbidity and mortality

Ten (20%) patients developed burst abdomen during post-operative hospital stay. Four (8%) deaths were recorded. All of these patients died during their hospital stay, due to post-operative severe chest infection. Out of these four patients three developed grade 1 intra- abdominal hypertension and one had developed abdominal compartment syndrome in the early post operative period.



IAP at 6hr	No morbidity	Morbidity	Total
Not raised	33(86.8%)	5 (13.2%)	38
Raised	7 (58.3%)	5 (41.7%)	12
Total	40 (80%)	10 (20%)	50

x^2 = 4.633; df = 1; p = 0.031; Significant

IAP at 6 hr	No Mortality	Mortality	Total
Not – raised	35 (92.1%)	3 (7.9%)	38
Raised	11 (91.7%)	1 (8.3%)	12
Total	46 (92%)	4 (8%)	50

x^2 = 0.002; df = 1; p = 0.961; Not significant

IAP at 24 hr	No morbidity	Morbidity	Total
Not – raised	40 (83.3%)	8 (16.7%)	48
Raised	0 (0%)	2 (100%)	2
Total	40 (80%)	10 (20%)	50

x^2 = 8.333; df = 1; p = 0.004; Significant

IAP at 24 hr	No Mortality	Mortality	Total
Not – raised	45 (93.8%)	3 (6.3%)	48
Raised	1 (50.0%)	1(50.0%)	2
Total	46 (92%)	4 (8%)	50

x^2 = 4.993; df = 1; p = 0.025; Significant

From the above tables it is evident that raised IAP at 6 hour and 24 hours post-operatively were significant predictor of morbidity. Whereas raised IAP at 24 hour post-operatively was significant predictor for mortality.

Similarly raised IAP at 6 hour post-operatively was also significant predictor for longer hospital stay.

IAP		N	Hospital stay Mean ± SD	't' value	P value
Pre-op	Raised	31	14.258 ± 9.949	0.695	0.490^{NS}
	Not-raised	19	12.526 ± 5.480		
At 0 hr	Raised	18	14.500 ± 10.639	0.557	0.580^{NS}
	Not-raised	32	13.094 ± 7.181		
At 6 hr	Raised	12	18.167 ± 13.874	2.217	0.031*
	Not-raised	38	12.158 ± 5.450		
At 24 hr	Raised	2	6.500 ± 2.121	1.210	0.232^{NS}
	Not-raised	48	13.896 ± 8.551		

Relation of IAP with Hospital stay

NS: p > 0.05; Not significant; * p < 0.05; Significant

DISCUSSION

Our study analyzed IAH and ACS, in general surgical patients, who underwent laparotomy for various indications, including traumatic as well as non-traumatic causes. The incidence of IAH in our study was 62% at admission and 24% at 6 hours post-op. The incidence of post-op ACS was 4% in our study population.

The incidence of IAH and ACS reported by various studies ranges from 2 to 78% and 0.5 to 36%, respectively, and depends on the population and the values used to define these entities. 13 The lower incidence observed was because this study includes low risk as well as high-risk patients, whereas most of the previous studies confined data collection to high-risk patients.

The mean (SD) IAPs before and after laparotomies were 16.83 (3.17) mm Hg and 12.1 (2.96) mm Hg, respectively, in the patients who had IAH at admission. The mean (SD) IAPs in the study group of Sugrue et al in 1995 before and after decompressions were 16.6 (9.4) mm Hg and 10.3 (3.1) mm Hg, respectively. 14 Meldrum et al in 1997 reported higher values of IAP (SD) pre- and post-op: 27 (2.3) and 14 (4.6) mm Hg, respectively. 15

This can be explained by the observation that in our study sample had predominance of general surgical patients who underwent laparotomy. 56% of the patients had perforation peritonitis leading to elevated IAP which, after decompression and removal of litres of fluid and gas, returned to near normal level immediately. Their study had predominance of trauma patients who required ICU care.

In 34% of patients, IAH was seen in immediate post-op reading (at 0 hours post-operatively). At 6 hours post-op, tweleve (24%) patients were found to have IAH. Of these two had newly developed organ system failure and hence a diagnosis of ACS was made and one of them died during the hospital stay.

We observed that raised IAP at 6 hour and 24 hours post-operatively was significantly associated with morbidity (occurrence of burst abdomen in 20% of patients). Raised

IAP at 6 hour post-op was also significant predictor for longer hospital stay.

In our study, raised IAP at 24 hour post-operatively was found to be significant predictor of mortality. IAH was present in 62% of patients at admission which remained in only 4% of patients at 24 hours post-op. Only 4% patients developed ACS in the post-operative period. Overall mortality rate in our study group was 8%, mortality rate of 12.9% in patients who had IAH pre-operatively, while 50% in patients with IAH at 24 hours post operatively and also in those who developed ACS.

In 2000 Cheatham et al had found that elevated IAP alone does not have sufficient sensitivity or specificity to be useful as a predictor of mortality but reported mortality rate of 64-68% in patients who developed ACS. 16

In 2005 Malbrain ML et al reported 12.9% prevalence rate of ACS and overall mortality rate of 38.8% in patients with IAH in the multiple centre study on mixed population

of critically ill patients. They concluded that mean intra abdominal pressure on admission was not an independent risk factor for mortality; however, the development of intra abdominal hypertension during the intensive care unit stay was an independent predictor of mortality and associated with high rate of organ dysfunction. 17

In 2008 Vidal et al showed an independent association of IAP with mortality in critically ill patients. Overall mortality in patients who had IAH was 53%, while 80% in patients

who developed abdominal compartment syndrome. 25

In 2010, Shehtaj et al reported mortality rate of 13.2% in critically ill surgical patients with pre-op IAH, and 3% incidence of ACS after laparotomy with 100% associated mortality. They found elevated IAP pre-op, post-op at 0 and 6 hours to independently predict the occurrence of death but not at 24 hours.

No significant correlation between IAP and duration of hospital stay, occurrence of burst abdomen was found at any point of time in their study. 18

CONCLUSION

IAP is concluded to be significant predictor of morbidity and mortality in patients with acute surgical abdomen, more so after surgery. Raised IAP at 6 hour and 24 hours post-operatively is significant predictor of morbidity. Whereas raised IAP at 24 hour post-operatively is significant predictor of mortality. The duration of hospital stay is also dependent upon the IAP and ACS. Thus a thorough monitoring and early decompression can decrease the morbidity and mortality rate.

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