

A Prospective Study of Evaluation of Preoperative Intra-Abdominal Pressure Monitoring and Its Association With Post Operative Morbidity and Mortality in Emergency Laparotomy

Dr.S. Thirunavukkarasu, Dr.S. Maithreyi
(Department of General surgery, Kilpauk Medical College)
(Department of General surgery, Kilpauk Medical College)

Abstract:

Introduction: Abdominal compartment syndrome and Intra-Abdominal Hypertension have been prevalently identified among patients with surgical emergencies such as pancreatitis and intestinal obstruction and among patients in intensive care unit.

Objective: To assess the relationship between pre-operative intra-abdominal pressure and postoperative patient morbidity (with special reference to kidney function and pulmonary atelectasis) and mortality.

Material and Methods: A prospective comparative study was conducted on 50 cases of Emergency Laparotomy and 50 Elective Laparotomy patients admitted in Government Royapettah Hospital. The study was conducted between May 2013 and April 2014. Preliminary details, Laboratory Test results were recorded. Intra-abdominal pressure and urine output were monitored. The results were documented and tabulated.

Results: The mean Intra-abdominal pressure of patients who underwent emergency laparotomy and those having elective surgery was 16.6mmHg and 4.22mmHg respectively. Urea and creatinine levels significantly raised in all cases of increased intra-abdominal pressure. There is a mild increase in the incidence of atelectasis postoperatively in cases of increase intra-abdominal pressure.

Conclusion: In patients with developing IAH, monitoring strategies and fluid resuscitation should be adjusted to prevent further increases of IAP. If despite these preventive measures full-blown ACS should occur, immediate decompressive laparotomy is warranted.

Keywords: Abdominal Compartment Syndrome, Intra-abdominal hypertension
Intra-abdominal pressure.

I. Introduction

The presence of severe sepsis, septic shock, trauma and emergency surgery are still associated with a very significant mortality despite of best efforts by clinicians and researchers worldwide. The pathway leading from shock following trauma or infection to death passes through various stages and degrees of organ dysfunction. This has been known for hundreds of years and most of critical care history has focused on sustaining and in some cases taking over organ function. Over the years, more experience in treating such patients has revealed that once the negative spiral of terminal organ dysfunction is reached, the process can become irreversible and death is unavoidable. This has led to a shift in focus to earlier stages of the disease and ways to prevent or treat organ dysfunction before irreversible damage occurs. Modern research has identified many mechanisms for organ dysfunction in sepsis and molecular and genetic biology are contributing more etiologic insights everyday. All these insights are yet to be combined into probably complex but useful model for the evolution of systemic infection to sepsis. It is important to realize that at every step in this evolution, there are other factors contributing and interacting with the inflammatory changes which may lead to organ damage and dysfunction. One of these factors is the intra-abdominal pressure. Intra-abdominal hypertension and abdominal compartment syndrome have been identified as a cause of organ dysfunction and mortality in several subsets of critically ill patients including patients with severe sepsis and septic shock and after trauma or emergency surgery. The mechanism of IAH induced organ dysfunction can be directly pressure related for organs lying inside or adjacent to the peritoneal cavity (such as the kidneys) or indirectly related to increased intrathoracic pressure in distant organs.

Several studies have shown that the incidence of IAH and ACS is significantly more when associated with sepsis and septic shock. It may be as high as 85% and 30% respectively. In cases of pancreatitis about 40-70% patients have been recognized to have IAH and 10-50% to have ACS. Post laparotomy incidence is variable but is generally low with elective surgeries and higher following emergency procedures.

II. Material And Methods

The study was conducted on 50 cases of Emergency Laparotomy and 50 elective laparotomy patients admitted in Government Royapettah Hospital attached to KILPAUK MEDICAL COLLEGE AND HOSPITAL. This study was conducted between May 2013 and April 2014. Before the study conducted informed consent was obtained from all the patients.

Study design : A Prospective, Comparative study.

Sample size : 100 admitted cases

Inclusion criteria:

1. patients admitted to GRH and undergoing emergency and elective laparotomy
2. Age group 18 yrs to 60 yrs

Exclusion criteria:

1. Age group < 18yrs and > 60yrs
2. Pregnancy
3. Morbid obesity
4. Spinal cord problems and fracture limbs who are unable to lie down supine
5. Bladder complains – neurogenic bladder, cystitis
6. Patients with established causes of co-morbidity such as renal failure, CAD, hypertension

Two groups of 50 surgical patients were selected. The experimental group had increased intra-abdominal pressure. The control group were selected from those undergoing elective surgery. In both groups, patients with factors which could cause increased intra-abdominal pressure like obesity were excluded. The preliminary details collected from patients included, name, age, sex, diagnosis, operative procedure planned, BMI. Presence or absence of diabetes mellitus, hypertension. Examination findings of pulse, blood pressure, respiratory rate, temperature and specific systemic examination of respiratory system, cardiovascular system and abdomen were noted. Laboratory tests done preoperatively and post operatively according to need were hemoglobin concentration, plasma urea, plasma creatinine and Chest X-Ray. Intra-abdominal pressure and urine output were monitored pre-operatively and at four hourly intervals in the post-operative period for 24 hours. The intravesical route of measuring the IAP was done by connecting the Foley's catheter to a three way tap which was then connected to a pressure transducer. The patient was placed in a supine position, catheterised using a Foley's catheter and the residual urine was drained. Later the Foleys catheter was clamped at a point distal to the point of pressure measurement. For every 20 degree head-up tilt, the IAP was found to increase by 2mm. The catheter was connected to a pressure transducer and the point of mid-axillary line at the iliac crest was taken as the reference point where the intra-abdominal pressure value was zeroed. Around 25ml (if weight <20kgs, 1ml per kg) of saline was instilled into the bladder, 30 to 60secs later the reading was taken, providing time for detrusor muscle relaxation. The measurements were taken in the absence of active abdominal muscle contraction and at the end of expiration. Measurements were made at regular intervals, usually varying from 4 to 6hrs. Depending on the IAP the treatment modality was adjusted. The Foleys catheter was clamped before each measurement. Operative findings during laparotomy and the surgical procedure done were noted. Post-operative mechanical ventilation and re-laparotomy were followed up if needed. Also, cause of mortality was noted if the patient expired in spite of maximal supportive medical care. The results were documented and tabulated. The statistical significance was found out using the Chi square test.

III. Results

The mean (S.D.) intravesical pressure of patients who underwent emergency laparotomy and those having elective surgery was 16.6 (4.16) and 4.22 (2.46) respectively. These groups are called Increased IAP group and Normal IAP group hereafter. The incidence of increased intra-abdominal pressure (emergency) is significantly more among men than in women ($p < 0.001$). The number of female patients is more in the elective surgery list. Hence more number of males suffered from intra abdominal hypertension in the study group. There is no significant difference in the prevalence of cases based on the BMI. This suggests that's the BMI and hence over weight of the patient does not significantly increase the intra abdominal pressure of the patient to critical level so as to cause intra abdominal hypertension. Urea is significantly raised in all the cases of increased intra-abdominal pressure as compared to the cases with normal intra-abdominal pressure in all time intervals (pre-operative, post – operative, 4th hourly, 12hourly, 24hourly), indicating renal dysfunction in cases of increased intra abdominal pressure. There is found to be a significant increase in the creatinine level in all cases of increased intra-abdominal pressure as compared to the normal group at all time intervals. This similarly indicates renal dysfunction in cases of increased intra abdominal pressure. There is no significant correlation between the urine output and the intra-abdominal pressure. This might indicate that urine output might not be

an ideal indicator of renal dysfunction in cases of intra abdominal hypertension or abdominal compartment syndrome. There is no significant difference in the incidence of atelectasis pre-operatively between the two groups. There is a mild increase in the incidence of atelectasis post-operatively in cases of increased intra-abdominal pressure as compared to the group with normal intra abdominal pressure. This might indicate that pulmonary atelectasis correlation is to the duration of intra abdominal hypertension as the post operative intra abdominal pressure is more in the emergency cases than the elective cases. There is no significant difference in the need for re-laparotomy among the two groups.

IV. Figures And Tables



Intra abdominal pressure monitoring – GRH



Equipment used

Table 1: Sex * Group Crosstabulation

		Group		Total	P value	
		Normal IAP	Increased IAP			
Sex	Male	Count	21	41	<0.001	
		% within Sex	33.9%	66.1%		100.0%
		% within Group	42.0%	82.0%		62.0%
	Female	Count	29	9		
		% within Sex	76.3%	23.7%		100.0%
		% within Group	58.0%	18.0%		38.0%
Total		Count	50	50		100

Table 2: Age *Group Crosstabulation

	Group	N	Mean	Std. Deviation	P value
Age in years	Normal IAP	50	46.20	13.737	0.149
	Increased IAP	50	41.98	15.236	

Table 3: Group Statistics-Urea levels in patients with normal and increased abdominal pressure

	Group	N	Mean	Std. Deviation	Std. Error Mean	P value
Urea – pre	Normal IAP	50	33.54	5.970	.844	<0.001
	Increased IAP	50	47.82	11.261	1.593	
Urea – op	Normal IAP	50	32.44	4.807	.680	<0.001
	Increased IAP	50	44.78	10.533	1.490	
Urea - 4h	Normal IAP	50	32.96	5.103	.722	

	Increased IAP	50	44.04	10.103	1.429	<0.001
Urea - 12h	Normal IAP	50	33.28	4.481	.634	<0.001
	Increased IAP	50	42.90	9.179	1.298	
Urea - 24h	Normal IAP	50	32.74	3.848	.544	<0.001
	Increased IAP	50	42.50	9.554	1.351	

Table 4: Group Statistics- Creatinine levels in patients with normal and increased IAP

	Group	N	Mean	Std. Deviation	Std. Error Mean	P value
Cre – pre	Normal IAP	50	1.052	.2667	.0377	<0.001
	Increased IAP	50	1.838	1.2660	.1790	
Cre – op	Normal IAP	50	.980	.2740	.0388	<0.001
	Increased IAP	50	1.594	1.1474	.1623	
Cre - 4h	Normal IAP	50	1.064	.2776	.0393	0.001
	Increased IAP	50	1.588	1.0578	.1496	
Cre - 12h	Normal IAP	50	1.042	.2425	.0343	0.001
	Increased IAP	50	1.548	.9725	.1375	
Cre - 24h	Normal IAP	50	.996	.2432	.0344	0.001
	Increased IAP	50	1.460	.8947	.1265	

Table 8 : Post-operative * Group Atelectasis - Crosstabulation

		Group			Total	P value
		Normal IAP	Increased IAP			
Atelectasis – Post	Yes	Count	4	12	16	0.027
		% within Atelectasis – Post	25.0%	75.0%	100.0%	
		% within Group	8.0%	24.0%	16.0%	
	No	Count	46	38	84	
		% within Group	92.0%	76.0%	84.0%	
		Count	50	50	100	

V. Discussion

The importance of IAH and ACS are still not widely known. The present study (called GRH study hereafter) is an attempt to explore the incidence and importance of these conditions in the morbidity of surgical patients. Very few experimental studies have been made in these patients and since each person used different criteria, comparison is sometimes difficult. While analysing the results, it was found that the incidence of increased intra-abdominal pressure (emergency) was more among men when compared to women. The number of females was more in the elective case list. Hence more males were suffering from intra abdominal hypertension. There was no significant difference between the two groups of increased and normal intra abdominal pressure with regard to mean age distribution. Urea was significantly raised in the increased intra-abdominal pressure group when compared to the normal intra-abdominal pressure group at all time intervals (pre-operative, post – operative, 4th hour, 12th hour and 24th hour), thus indicating renal dysfunction in cases of increased intra abdominal pressure. Further, the intra abdominal pressure and urea of each of these patients were monitored serially at regular intervals. There was no correlation between the two. This reinforces the concept that the duration of IAH is more important than the actual pressure. Similarly a significant increase in the creatinine level in all cases of increased intra-abdominal pressure was found when compared to the normal group, at all time intervals. Similarly when monitored serially there was no correlation between the increased intra abdominal pressure and creatinine. Hence it reinforces the concept that, the duration of increased intra abdominal pressure is more significant than the actual pressure probably due to renal dysfunction due to alteration in the renal blood supply. There was no significant correlation between the urine output and the intra-abdominal pressure. This might indicate that urine output might not be an ideal indicator of renal dysfunction in cases of intra abdominal hypertension or abdominal compartment syndrome. There was no significant difference in the incidence of atelectasis pre-operatively between the two groups which was tested clinically and with the help of chest X-Ray. There is a mild increase in the incidence of atelectasis post-operatively in cases of increased intra-abdominal pressure as compared to the group with normal intra abdominal pressure. This might indicate that pulmonary atelectasis is related to the duration of intra abdominal hypertension since the post operative intra abdominal pressure is more in the emergency cases than the elective cases. Mechanical ventilation was not needed for both the groups post operatively. Mortality Rate. Mortality of 0% was observed in GRH group. In the Shehtaj khan et al study most patients were in Grade I intra abdominal hypertension (96%), while in GRH most cases were prevalent in Grade II intra abdominal hypertension (48%). Also relatively more cases presented with Grade III intra abdominal pressure as compared to the other study which are 16% and 1.5% respectively. Regarding the age of incidence, the GRH study did not find any significant difference in the mean age group of elective and emergency patients. The large standard deviation indicates the large scatter around the mean. Other studies reported mean age groups varying between 30 and 50 yrs. This difference could be due to differences in experimental design. When the sex difference was analysed, incidence of the disease in men was

seen to be significantly higher than in females in all studies. This could be due to the higher incidence in accidents in men. Increase in the intra-abdominal pressure is believed to affect renal function especially renal filtration. The commonly performed glomerular function tests are blood urea and creatinine. A much more sensitive test is creatinine clearance but this test cannot be done properly in patients undergoing emergency surgery. GRH and other studies show a significant increase in blood urea and creatinine levels. GRH study did not find any significant difference in the urine output. The reasons for this could be that tubular function may not be affected by IAH and one more important factor viz., fluid balance is another influence which can affect urinary output. A comparison of the renal dysfunction which has been calculated using the RIFLE'S criteria, revealed a renal dysfunction of 44% in GRH pre-operatively. On comparing it to other studies, Shehtaj Khan series had a high incidence of renal dysfunction upto 78% while Sugrue had 69%. The fall in the post operative creatinine as compared to pre operative creatinine was similar in all the three case series. The ratio of renal Dysfunction and non-dysfunction both pre and post operatively seem to be similar in both the studies. It was found to be 47.5% and 54% in the studies conducted by Shehtaj Khan and GRH respectively. The increase in intra-abdominal pressure could affect pulmonary function due to the upward displacement of the diaphragm. The commonly expected pathology is atelectasis of the bases of lungs. Post operatively, a small percentage of patients with increased IAH had atelectasis although there was no difference pre - operatively. None of the groups needed the use of ventilator. The cases of IAH with Respiratory dysfunction, both pre and post operatively seem to be more prevalent in the Shehtaj Khan (74.7%) studies where as in GRH the patients with No-respiratory dysfunction pre and post operatively seem to be more prevalent (78%).

VI. Conclusion

ACS/ IAH is associated with profound physiological abnormalities both outside and within the abdomen. While treating these patients it is essential to identify the signs of increased abdominal pressure early and start the management accordingly. It is also important to monitor the intra-abdominal pressure of the affected patients and those with (more than two) risk factors either continuously or intermittently. Understanding the pathophysiology of ACS/IAH is of prime importance for applying patient tailored treatment. If needed, appropriate surgical intervention should be done at the stage of IAH itself and should not be postponed till the development of ACS.

Renal dysfunction is the most common complication of abdominal compartment syndrome. Pre-operative renal dysfunction was found to be high in all the case series ranging from around 40% to 80%. The fall in the post operative creatinine as compared to pre operative creatinine was also observed in all the case series. In many of the studies renal dysfunction became evident as oliguria and later progressed to anuria. Compression of the renal vein and parenchyma and reduced renal perfusion, lead to reduced microcirculation to the functioning glomeruli and cortex. This results in tubular and glomerular dysfunction and substantially reduced urine output since $FG(\text{Filtration Gradient}) = \text{MAP}(\text{Mean Arterial Pressure}) - 2 \times \text{IAP}$.

Thus the IAH induced renal dysfunction and prerenal azotemia will neither be responsive to fluid resuscitation nor vasopressors. It improves dramatically by appropriately and promptly reducing the elevated IAP. The awareness of the entity called Intra abdominal hypertension and abdominal compartment syndrome is spreading in recent times. Yet in many of the centers it is still under-diagnosed as strict protocols to monitor intra abdominal pressure in critical care patients both in the medical and surgical side have not been laid down. As recommended by the World Society of Abdominal Compartment Syndrome, all cases in the critical care wards should be assessed for intra abdominal pressure immediately following admission and serially in cases of elevated initial pressure. Though the mortality rate is zero in this case series, it is probably due to the choosing patients with increased abdominal pressure selectively rather than monitoring all the patients in the critical care ward, some of whom may have died because of undiagnosed Intra abdominal hypertension. Hence abdominal compartment syndrome is a treatable condition when it is timely diagnosed and appropriately managed. Both medical and surgical treatments play equally important role in their management. Awareness and recognition of this entity will go a long way in reducing the mortality of many critically ill patients and all it takes is a simple bedside test to make the difference between probable death and survival.

References

- [1]. 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;32:1722–32. [PubMed]
- [2]. Emerson H. Intra-abdominal pressures. *Arch Intern Med.* 1911;7:754–84.
- [3]. Baggot MG. Abdominal blowout. *Curr Res Anesth Analg.* 1951;30:295–9. [PubMed]
- [4]. Fietsam R, Jr, Villalba M, Glover JL, Clark K. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. *Am Surg.* 1989;55:396–402. [PubMed]
- [5]. Moore EE, Burch JM, Franciose RJ, Offner PJ, Biffl WL. Staged physiologic restoration and damage control surgery. *World J Surg.* 1998;22:1184–90. [PubMed]

- [6]. De Laet IE, Malbrain M. Current insights in intra-abdominal hypertension and abdominal compartment syndrome. *Med Intensiva*. 2007;31:88–99. [PubMed]
- [7]. 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. [PubMed]
- [8]. 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. [PubMed]
- [9]. Sugrue M. Abdominal compartment syndrome. *CurrOpinCrit Care*. 2005;11:333–8. [PubMed]
- [10]. Lambert DM, Marceau S, Forse RA. Intra-abdominal pressure in the morbidly obese. *Obes Surg*. 2005;15:1225–32. [PubMed]
- [11]. Carlotti A, Carvalho W. Abdominal compartment syndrome: A review. *PediatrCrit Care Med*. 2009;10:115–20. [PubMed]
- [12]. Malbrain ML, Deeren D, De Potter TJ. Intra-abdominal hypertension in the critically ill: it is time to pay attention. *CurrOpinCrit Care*. 2005;11:156–71. [PubMed]
- [13]. Balogh Z, Moore FA, Moore EE, Biffi WL. Secondary abdominal compartment syndrome: A potential threat for all trauma clinicians. *Injury*. 2007;38:272–9. [PubMed]
- [14]. Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Expertson Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med*. 2007;33:951–62. [PubMed]
- [15]. Malbrain ML. Abdominal pressure in the critically ill. *CurrOpinCrit Care*. 2000;6:17–29.
- [16]. Malbrain M. Different techniques to measure intra-abdominal pressure (IAP): Time for a critical re-appraisal. *Intensive Care Med*. 2004;30:357–71. [PubMed]
- [17]. Cheatham ML. Nonoperative management of intraabdominal hypertension and abdominal compartment syndrome. *World J Surg*. 2009;33:1116–22. [PubMed]