Original Article

Intra-abdominal Hypertension and Abdominal Compartment Syndrome in patients undergoing emergency laparotomy

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Abstract

Objective: The objectives of the Prospective observational study were to identify the frequency of abdominal compartment syndrome and intraabdominal hypertension in patients undergoing emergency laparotomy for trauma and peritonitis and to determine the impact of raised intraabdominal pressure on the overall morbidity and mortality.

Materials and Methods: The study was conducted in the department of surgery Benazir Bhutto Hospital Rawalpindi. From June 2013 to May 2014 a total of 50 patients undergoing emergency laparotomy were included. IAP was measured preoperatively then postoperatively at 0, 6, 24 hours, and the findings were recorded on a specially designed preform. The patients having higher IAP were further evaluated for up to 72 hours. All vitals, urine output, oxygen saturation, serum urea, creatinine were noted. The main outcomes were duration of hospital stay, the occurrence of burst abdomen, and mortality.

Results: At the preoperative level the incidence of IAH was 86%. The mortality association with IAH at 6 hours postoperatively was quite significant (P<0.029). The incidence of postoperative ACS was 5% among the total patients and it was 15.6% in trauma patients. No significant association was found between IAP and occurrence of burst abdomen at any level (P values 0.4, 0.26, 0.53, 0.58 at intervals preoperatively, 0, 6, 24 hours postoperative respectively.

Conclusion: Intraabdominal pressure is an important factor that predicts the mortality of patients undergoing emergency laparotomy. It should be carefully monitored and managed accordingly to avoid the detrimental effects on virtually all organ systems. Abdominal decompression in significantly elevated intraabdominal pressure reverts the physiological derangement of Intrabdominal hypertension.

Keywords: Abdominal trauma, peritonitis, Intra-abdominal pressure (IAP), Intra-abdominal hypertension (IAH), abdominal compartment syndrome (ACS).

Introduction

Intra-abdominal hypertension is a potentially fatal condition of which all clinicians and surgeons in particular need to be aware.[1] Reports regarding IAH have been appearing increasingly throughout the literature; Emerson first noted the cardiovascular morbidity and mortality associated with elevated IAP in 1911 accompanied by a widening recognition of the potential for major morbidity and mortality should this condition go undetected.[1] Normal IAP is approximately zero to subatmospheric.[3] IAH is defined as a raised intra-abdominal pressure associated with organ dysfunction. The terms, IAH and ACS are often used synonymously.[3] Classically the compartment syndrome is defined as increased pressure within an Osseo facial compartment which approaches or exceeds the local venous pressure for a prolonged period, causing a critical reduction in intra compartmental perfusion pressure. Originally reported to occur in limb compartment syndromes have been diagnosed in the compartment of the abdomen.[3,4] Rapid progression of IAH leads to ACS, which is defined as an IAP greater than 20 mm Hg with at least one new organ system dysfunction/failure.[5]

IAH is likely to develop after an event that leads to an acute increase in the volume of abdominal contents sufficient to cause pressure related to organ dysfunction.[6] As such IAH can result from abdominal trauma, diffuse peritonitis, performing damage control surgery, peritoneal edema following resuscitation forceful abdominal closure under tension, and many numerous medical conditions.[7] The excess of pressure within the abdomen may be associated with significant cardiopulmonary and renal dysfunction and contributes to intestinal mucosal acidosis and bacterial translocation significant hemodynamic complications and compromise of IAH can translate to complications such as sepsis, multiple organ failure, and death.[8] Once the pressure is brought down by opening the abdomen these physiological changes can be reversed.[8,9]

Diagnostic suspicion of IAH can be confirmed by objective measurements of IAP since bedside manometer using a Foleys catheter provide a valuable estimate of IAP and are easy to perform.[6,9] In fact, IAH and ACS cannot be diagnosed solely based on the clinical picture and objective measurement of IAP is required for the diagnosis. IAH has been graded into four types based on IAP measurements and managed accordingly.[2]

The treatment of established ACS is abdominal decompression. The exact level of IAP at which decompression should be done is not established in the literature but significant cardiopulmonary dysfunction or oliguria in the presence of raised IAP is considered an indication for decompression.[10] Moderate elevations in IAP without significant cardiopulmonary compromise can be managed by fluid resuscitation, inotropic support, and diuretics Certain adverse consequences of IAH such as gut mucosal acidosis can appear at low pressures long before the adverse physiologic consequences i.e. respiratory cardiovascular, and renal become apparent, therefore IAH should be anticipated and prevented in high-risk patients.[3,11]

Thus, IAH leading to ACS is an important factor complicating the postoperative course of many surgical patients the clinical findings may be misleading as certain other conditions as acute respiratory distress, multiple organ failure, and septic shock may present with similar findings.[12,13] No study data available in our country before and we have underestimated the problem. Presently there is a great need to fully understand the clinical implications and management of this condition, which would have far-reaching clinical implications in surgical patients. The objectives of this prospective observational study were to identify the frequency of abdominal compartment syndrome and intraabdominal hypertension in patients undergoing emergency laparotomy for trauma and peritonitis and to determine the impact of raised intraabdominal pressure on the overall morbidity and mortality.

Materials and Methods

This is a prospective observational study that was carried out in the surgical department Benazir Bhutto Hospital Rawalpindi, which is a 500 beds teaching hospital attached to Rawalpindi medical college for one year from June 2013 to May 2014. A total of 50 cases of midline emergency laparotomies were included in this study. All adult patients above 18 years of age undergoing emergency exploratory laparotomy for abdominal trauma and peritonitis were included in the study. Patients in whom Foley's catheterization was not possible either due to external genital trauma or some other abnormality, trauma to urinary bladder patients with comorbidities such as

asthma, diabetes mellitus, cardiac failure, COPD, CRF, and CLD were excluded from the study. Intra-abdominal pressure measurement was done preoperatively. Patients were then admitted to ICU or HDU and intra-abdominal pressure measurements were done at 0, 6, 12, and 24 hours and continued afterwards 4 hourly if found persistently high. If IAP remained below 12 mmHg, measurements were discontinued after 24 hours. The regular routine monitoring of vital signs including pulse, blood pressure, respiratory rate, central venous pressure, arterial blood gases was done in the postoperative period. Grade 1 (IAP 12-15 mmHg) and Grade 2 (16-10 mmHg) were managed with Observation, normovolemic, and hypervolemia resuscitation respectively. A persistently high IAP (>20 mmHg) with organ system dysfunction defined as BP less than 90 mmHg systolic with a heart rate of >100 bpm, Respiratory rate > 20/min with Spo2 <90/min or the need for mechanical ventilation, Urine output <25 ml/min for 3 hours with deranged renal function tests was considered as an indication for decompression and temporary abdominal closure using Bogota bag.[11,13]

Measurement of IAP: IAP was indirectly determined by measuring urinary bladder pressure with a Foley catheter in the supine position. The patient was catheterized with a 16-guage Foley's catheter. The bladder was drained and then filled with 50 ml of sterile saline through the Foley catheter. The tubing of the collecting bag was clamped. The catheter was connected to a saline manometer. The symphysis pubis was the zero reference, and the pressure was measured in centimeters at the end of expiration.[14] The cm was converted to mmHg by multiplying with a factor of 1.36 .[15]

Statistical analysis: The data was analyzed using SPSS version 10 for windows. All the demographic parameters were categorized and analyzed accordingly. Carl Pearson's product-moment correlation was used to see the dynamic relationship between the hospital stay and IAP. The predictive role of IAP in-hospital stay was seen by linear regression analysis. The univariant regression method was used to identify this prediction. To test the significance of this regression the ANOVA statistics were used to see changes after the forced entry method. Point biserial correlation was used to study the relationship between IAP and the occurrence of burst abdomen and mortality. An independent sample *t*-test was used to see the effect of IAP on the various organ systems at a particular point.

Results

A total of 57 patients who underwent laparotomy for abdominal trauma and peritonitis were included in the study. Seven patients with inadequate monitoring data were later excluded. The percentage of trauma and peritonitis patients was 32% and 69% respectively. Out of 50 patients, 36 (72%) were males and 14(28%) were females. The mean age was 38.26 years range (18-82 years) with SD 15.66 and variance of 245.2. The average duration was 119 min which was spreading from 70 min to 200 min maximum.

Overall, pre-operative IAH was seen in 86% (n=43) of the patients Grade 1 in 34% (n=18), grade II in 24% (n=12) grade III in 18 % (n=9) grade 4 in 10 % (n=5). At 0 hours post-operative 4% (n=2) patient had IAH (18 mmHg) while IAH was noted in 85% (n=42) mean IAP 14 mmHg range 10 mmhg to 20 mmHg, 8% (n=4) mean IAP 15 mm Hg range 12 to 18 mmHg, 6% (n=3) mean IAP 20 mmHg range 18 to 26 mmHg of patients at 6, 24 and 72 hours postoperatively.

In trauma group (n=25) pre-operative IAH was seen in 93% (n=23) patients and at 6 hours post-operative the IAH was seen in 14% (n=4) of cases. While in non-trauma group the pre-operative IAH was detected in 84% (n=42) patients and at 6 hours post-op the IAH is 6% (n=3). Frequency of ACS was 6% (n=3) among peritonitis and 16% (n=8) in trauma patients.

Table 1: Various Grades of Intraabdominal Hypertension

	Total Patients	Trauma	Non-Trauma
No IAH	7	1	6
Grade I	17	8	9
Grade II	12	3	9
Grade III	9	3	6
Grade IV	5	1	4

Four patients (8%) had burst abdomen and the same no of patients expired in our study due to various complications.

The average duration of hospital stay was 7.96 days with a range of 28 and a span of 4-32 days.

Pearson's correlation between a hospital stay and different readings of abdominal pressures is showed in Table 4 indicates that no significant negative relationship exists at any level of the IAP readings in our data. All the readings for r value are positive. The only value which came close to 0 was 0.006 which was at 24-hour post-op and the *p*-value was 0.969. Rests of

the readings were not significant too. The ANOVA was also not significant too for the correlation between a hospital stay and IAP measurements.

The point biserial correlation between the IAP and burst abdomen showed no significant results and so we can say that the burst abdomen is not dependent on raised IAP to occur, at least in our study. The highly significant and strong association arose between the death of a patient and raised IAP as is shown in Table 5. Pearson's correlations for the IAP and occurrence of death are more significant at the level of immediate postop and 6-hour postop. The binary logistic regression between the occurrence of death and IAP indicates a strong predictive role of IAP (*p* values are 0.001 and 0.000 for the IAP at 0 postop and 6-hour postop readings.

The effect of IAP on various organ systems at a particular point was quite obvious when we divided the sample into two categories. One with IAH and others with no IAH, the t-test was applied to various physiological systems comparisons. The major changes were in the cardiovascular, renal, and respiratory systems in which the p-value was much significant i.e. less than 0.001, 0.03, and 0.002 respectively.

The mortality rate was equally complying with raised IAP. Patients who developed the postoperative ACS (2 patients) i.e. 100% went for decompression but none survived.

Table 2: Pearson correlation between the hospital stay and IAP

Pearson correlation between IAP and hospital stay		
IAP	**R-value**	**P-value**
Preop	0.124	0.411
0 hr postop	0.089	0.555
6 hr postop	0.089	0.555
24 hr postop	0.006	0.969

Table 3: Pearson correlation between the hospital stay and IAP

IAP	Pearson correlation	P-value
Preop	-0.111	0.443
0 hr postop	0.473	0.001
6 hr postop	0.452	0.001
24 hr postop	-0.109	0.466
72 hr postop	-0.027	0.983

Discussion

Intra-abdominal hypertension leading to abdominal compartment syndrome is a poorly appreciated complication of raised intra-abdominal pressure. Abdominal surgery, Trauma, and peritonitis being the most common causes of IAP.[6] Risk factors associated with IAH include vigorous fluid resuscitation,>10L of crystalloid. Duration of surgery >2 hours with the evisceration of gut, visceral edema, abdominal closure under tension, and placement of intra-abdominal packs to stop bleeding. Most of the studies which contain the data regarding IAH and ACS usually revolve around the non-surgical causes of these conditions. We encounter many patients dying of cardiopulmonary and renal sepsis, multiple organ failure, and complications of anesthesia. I have intended to find out the frequency of IAH in surgical patients who undergo laparotomy for peritonitis and abdominal trauma. We gained data of 50 patients undergoing emergency laparotomy due to trauma or non-trauma cause. There were 34 (68%) males and 16 (32%) females in the study. The similar gender ratio was seen in some other studies like Khans et al. (76% males, 24% females), Hong et al. (72% males), and Meldrum et al. (70% males) but Cheatham et al. and Surge et al. both reported 60% males in their studies.[3,14,15] The mean age in our study was 38.26±15.66 with a range of 64, 18-82 years was the spread. The similar features were in the Khans et al. study in which the mean age was 34.78±14.9 and the range was 18-85 years. Other studies have the mean age higher than out study like Meldrum et al. 39±9 years and Hong et al. 42 years.[3,10,16,17] Out of 50 patients the trauma patients were 32%. Other studies either show trauma percentage below this (Khan.S et al. 19%) or above this (Cheatham et al. 68%).[3,14]

In the trauma group, we had 20% blunt abdominal trauma and 12% FAI of total patients. Meldrum et al. reported 60% blunt abdominal trauma patients. These are the demographic variations as we have abdominal trauma usually due to some kind of assault. While in the developed countries the major share is the blunt abdominal trauma caused by road traffic accidents.

The IAP measurements improved dramatically after the decompression. Anyhow the decompression was not the prime aim, as all patients were operated for regular surgical treatment. IAP mean pre-op was 8.96 ± 1.91 mmHg with a range of 7-18 mmHg. Immediate post-op mean was 11.96 ± 4.28 and the range was 8-30 mmHg. The value at 24 hours postop was somewhat similar to pre-op i.e. mean 8.68 ± 3.67 mm Hg. The 72

hours postop values are high (mean 23.33 ± 10.50 mm Hg) as we did not consider further monitoring the patients having IAP <12mm Hg at 24-hour postop. These findings are quite different when we compare it with Khan et al. In which the pre-op IAP was 18mmHg and the postop mean IAP was 6 mm Hg. Some studies even showed quite higher value as in Meldrum et al. the pre-op and postop mean IAP was 27 ± 2.3 and 14 ± 4.6 mm Hg respectively. Initially, we were expecting that duration of the surgery will affect the mortality of the patient and can be a good predictor of mortality because the more the duration of surgery means more complicated the procedure is and hence more complications. But Pearson correlation between the duration of surgery and mortality is not significant (r=0.037, p=0.799). A similar correlation exists between the hospital stay and the duration of surgery. (r=0.165, p=0.273). IAP measurements at any point were not correlated significantly with the duration of surgery too. Ivatuary et al. in their study have described that elevation of IAP to 12 mmHg i.e. Grade I IAH after surgery should be considered normal and is not associated with significant complications such patients should be closely observed and managed by normovolemic resuscitation.[18]

ACS was 5% postoperatively in our studies and most of the patients developed major changes in the CVS, respiratory, and renal systems. The renal function disturbances were in 21 (42%) patients who were having the IAH at presentation. These values were less when we see the derangements in renal functions in other similar studies as Khan S et al. (49%) and Sugrue at el. (69%).[3,15] The McNemar statistics were quite helpful when we ascertained the role of decompression in the improvements of organ systems. The urine output at the pre-op level is 29.10 ± 15.90 ml/hr. which increase quite significantly up to 48.80 ± 16.15 ml/hr. after decompression. Whether or not IAH was there pre-operative and improvements in urine output shows that using the binomial distribution, the Mcnemar test is 0.021 with a p-value <0.001 (from Pearson Chi-Square). These results verify the negative role of IAH in the urine output. The measurements of serum creatinine were 1.56 and 1.32 mg/dl pre and post-op respectively, which also validates further renal function improvements after decompression. Khan S et al. found similar results in his study (urine output increase from 48.7 ml/hr. to 53.9 ml/hr. pre to post-op respectively. Creatinine change from 1.5 to 1.3 mg/dl), Ma et al. found a negative correlation between IAH and urine output (r = -0.747, p <0.01). Sugrue et al. showed in study results that the value of urine output pre to post-op were 1399 ml/24 hours and 1770 ml/24 hours and creatinine change was 151 to 128 micromol/L.[3,15,19]

The incidence of IAH was 86% at admission and ACS was 5% in the general population and 15.6% in trauma patients. The various studies like Khan et al. and Cheatham et al. shows the incidence of IAH 2 to 78% and ACS 0.5 to 36% respectively.[3,14] These large range variations are due to the targeted population upon which the studies were conducted. Ball et C.G their study found the incidence of IAH is 30 to 80% and the incidence of ACS is 4% to 30% respectively in intensive care patients.[20] Which is contrary to my study as well as the studies discussed before but these authors carried out their study on critically ill patients who were operated upon for abdominal trauma, peritonitis, elective major surgeries as well as patients who were critically ill and had certain medical problems whereas my study was carried out only on patients who had abdominal trauma and peritonitis.

No significant association was found at any point between the IAP and the occurrence of burst abdomen (p >0.1). A similar association was found between the IAP and hospital stay but Khan et al. showed the IAP at 6 hours have shown quite a negative relationship with the hospital stay (r=-0.234, p<0.001), and the duration of hospital stay was calculated by the regression equation.[3] The occurrence of IAH and mortality was quite significant at 6 hours post-op (p<0.029) while no significant relationship was found between the IAH at pre-op, 0 hour postop, 24 hours postop and mortality.(p>0.379, 0.268, 0.792 respectively). The study of Khan et al. validated findings with a p-value of <0.001 at 6 hours postop, while Cheatham et al. found that IAP alone is neither sensitive nor specific for the predictor of mortality at any point.[3,14] The linear regression analysis at 6 hour post-op IAP and mortality show the beta coefficient of 0.452 which shows that every unit increase in the 6 hour postop IAP the probability of death increases by a factor of 0.452 provided the other conditions remains constant. Hence when the IAP is more in the early postoperative period then aggressive monitoring is needed to avoid fatal complications of the raised IAP. Chiara et al. described that after 30 mmHg the medical treatment has no role and surgery is the only option for IAH.[21]

Finally, I would like to discuss the technique of IAP measurement. Luckianow et al. compared various techniques of indirect measurement of IAP with direct measurement of IAP and found it to be the most

reliable technique and it most closely reflected the IAP.[9] In my study all IAP measurements were done by measuring urinary bladder pressure. Direct measurements of IAP were not done as it was impractical and unethical to insert a cannula into the patient's abdomen for measuring the IAP, putting the patient at a greater risk of developing infection and therefore, no comparison of the two techniques was done. The temperature of saline and the volume of saline inserted into the bladder are important In my study 50 ml of saline was used to measure IAP. Chiumello et al. described that 50 ml of normal saline for bladder pressure is reasonable as below 50 and after 100 ml instillation of normal saline does not show true IAP. He also showed that normal saline colder than body temperature shows an increased level of IAP.[22]

Conclusion

Intraabdominal pressure is an important factor that predicts the mortality of patients undergoing emergency laparotomy. It should be carefully monitored and managed accordingly to avoid the detrimental effects on virtually all organ systems. Abdominal decompression in significantly elevated intraabdominal pressure reverts the physiological derangement of Intrabdominal hypertension. Frequent monitoring and timely abdominal decompression may help improve the mortality rate.

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