ABDOMINAL COMPARTMENT SYNDROME AMONG CRITICALLY ILL SURGICAL AND TRAUMATISED PATIENTS: EXPERIENCE AT PIMS, ISLAMABAD

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Background: Raised intra-abdominal pressure (IAP) accompanied by evidence of organ dysfunction constitutes abdominal compartment syndrome (ACS). The ACS is now becoming an increasingly recognised fatal entity in the critically ill surgical and traumatized patients receiving critical care. The objectives were to determine the frequency of abdominal compartment syndrome (ACS) in critically ill surgical and traumatised patients and to identify the risk factors associated with its development in our patients. Methods: This descriptive study was conducted at Department of Surgery, Pakistan Institute of Medical Sciences (PIMS), Islamabad from July 2004 to February 2005. Two hundred critically ill adult surgical and traumatised patients who needed catheterisation were included in the study. Patients who had cardiac tamponade, tension pneumothorax, status asthmaticus, bladder outflow obstruction, pre-existing end organ failure and those not consenting to participate in the study were excluded. Diagnosis of the underlying surgical condition was made by history, physical examination and necessary investigations. The main diagnostic tool employed for detecting ACS was the measurement of intra-cvstic pressure (ICP) which was taken as an indirect measure of intra-abdominal pressure (IAP). It was measured four hourly by employing simple fluid column manometry method. Blood pressure, pulse rate, temperature, respiratory rate and urine output were recorded 4 hourly. Arterial blood gases (ABGs) and renal function tests (RFTs) were performed daily. ACS was diagnosed on the basis of raised IAP of >10 mmHg coupled with evidence of one or more end organ failure. A variety of risk factors that lead to ACS were studied among the patients. Results: Out of 200 patients, six had ACS. The overall frequency was thus 3%. The M:F was 2:1. Most of the patients were in the age range of 31-40 years. Severe peritonitis, severe gut oedema, SIRS and tense ascites were recognised as statistically significant risk factors for the development of ACS. All patients with ACS had features of multiorgan dysfunction. There was 80% in-hospital mortality among the ACS sufferers. Conclusion: ACS develops in a significant number of critically ill and traumatised patients developing quickly and proving fatal without ACS specific interventions. All such high risk patients should undergo serial ICP measurements as a screening test for early detection of ACS.

Keywords: Abdominal compartment syndrome, ACS, Intra-abdominal pressure, Intra-cystic pressure

INTRODUCTION

Raised intra-abdominal pressure (IAP) accompanied by evidence of organ dysfunction constitutes abdominal compartment syndrome (ACS). The ACS is now becoming an increasingly recognized fatal entity in the critically ill surgical and traumatized patients receiving critical care. Though the condition has been known for more than a century, there has been an explosive expansion of ACS literature over the last two decades.^{1–3}

Marey and Burt deserve to be acknowledged for their outstanding pioneering role in the understanding of ACS, who in 19th century described this condition and discussed the respiratory effects of raised IAP.⁴ The normal IAP is 0 mm Hg or slightly subatmospheric and typically approaches 10 mmHg following laparotomy.¹

The ACS can be seen in a variety of contexts particularly surgery and trauma. In case of primary ACS there is direct injury to the abdominal contents while in case of secondary ACS there is organ dysfunction caused by third space oedema and resuscitation.¹

ACS may be seen in patients with intraabdominal and retro-peritoneal hemorrhage^{5,6}, severe peritonitis⁵, severe acute pancreatitis⁵, ileus and intestinal obstruction⁷, severe gut oedema⁸, ruptured abdominal aortic aneurysm⁹, tense ascites especially in cirrhotics¹⁰, liver transplantation^{6,9}, perihepatic and other intra-abdominal packing¹¹, burn eschars¹², forced closure of non-compliant abdomen⁸, massive intravenous fluid resuscitation⁸, severe abdominal trauma accompanied by visceral swelling, haematoma or use of abdominal packs^{5,6} and septic shock² etc.

The hallmark of ACS is a rising IAP affecting multiple organ systems. The raised IAP is transmitted to the pleural space which reduces lung compliance. A combination of raised intra-abdominal and pleural pressures leads to decreased venous return, direct cardiac compression and increased afterload. Critically impaired perfusion of intra-abdominal organs results in renal, hepatosplanchnic and gut ischemia. Reduced perfusion of abdominal wall may impair wound healing. Prolonged unrelieved raised IAP at greater than 20 mmHg can produce pulmonary compromise, renal impairment, cardiac failure, central nervous system dysfunction, shock and death.^{5,8} In fact the deleterious effects of raised IAP vary from patient to patient and depend on the type and severity of various abdominal and extra-abdominal injuries. The typical patient of ACS has a tight abdomen, raised IAP, progressively deteriorating pulmonary and renal functions in the context of trauma or surgical abdomen. Failure to recognise and treat ACS is inevitably fatal.²

The ACS has been classified into the following four grades on the basis of IAP levels.¹³

Grade-I	IAP of 10-15 mmHg
Grade-II	IAP of 16-25 mmHg
Grade-III	IAP of 26-35 mmHg
Grade-IV	IAP of >35 mmHg

Measurement of intra-cystic pressure (ICP) is the most helpful and reliable diagnostic modality for ACS. It has been confirmed that ICP closely parallels the pressure within the abdominal cavity up to 70 mmHg.^{12,14,15} Alternative methods of abdominal pressure measurement include direct estimation by inferior vena cava pressure, rectal and gastric pressure measurements and direct measurement of IAP by puncture of peritoneal cavity. However all these are invasive techniques of varying degree.^{16,17} The Division of trauma surgery and critical care of Cedars-Sinai medical centres Los Angeles USA, has devised simple fluid-column manometry method of determining ICP as an indirect measure of IAP.¹⁸ This latter method has gained much popularity over the others.

It would be much easier to prevent ACS particularly in high risk patients. At the end of a prolonged operation, when the abdominal closure is not tension free, a delayed or staged closure may be more appropriate. Various types of mesh closure and other alternative means of abdominal coverage have been described.

Even plastic of the intravenous drip bag has used with success in this regard.¹⁹⁻²¹ been Overenthusiastic fluid resuscitation should also be avoided as it often leads to secondary ACS.¹ Similarly early management of hypotension and hypoxia helps to prevent gut oedema. The active management of ACS is tailored according to the grade of ACS. In this regard Meldrum *et al*¹³ have devised a four stage ACS grading scheme which is a helpful ACS management tool. According to this Grade-I ACS is managed with maintenance of normovolemia, Grade-II ACS with hypervolemic resuscitation, Grade-III ACS with decompression and Grade-IV ACS with decompression abdominal exploration. Following and formal decompression immediate primary fascial closure is obviated and the alternative means of abdominal coverage are used. The resultant abdominal hernia is repaired with mesh after several months.^{17,20}

Internationally there is increasing awareness about the high morbidity and mortality associated with ACS, however our surgical community still seems to be largely unaware of it.

This study was conducted to determine the frequency of ACS in our critically ill surgical and traumatized patients and to identify the types and frequency of various risk factors among them.

MATERIALS AND METHODS

This descriptive study was undertaken at the Department of Surgery, Pakistan Institute of Medical Sciences (PIMS), Islamabad during the period from July 2004 and February 2005. A total of 200 consecutive patients were included by convenience sampling technique. All surgical and traumatized patients over 14 years of age with indication for urinary catheterization were included in the study. (None of the patients was catheterized for the sake of the study). Patients with cardiac tamponade, tension pneumothorax, status asthmaticus, bladder outflow obstruction, pre-existing end organ failure and those not consenting to participate in the study were excluded from the study.

All the patients underwent routine diagnostic work-up by thorough history, physical examination and ancillary investigations for their presenting surgical problem. All of them were managed on indoor basis. Majority of them presented as acute surgical emergencies while a small percentage were scheduled admissions for various gastrointestinal malignancies. Laparotomies were undertaken among 131 patients, 33 patients had tube thoracostomies while 36 received conservative management without any surgical intervention.

Upon inclusion of the patient in the study, four hourly recording of their ICP, blood pressure, pulse rate, temperature, respiratory rate and urine output was ensured. The arterial blood gases (ABGs) and renal function tests (RFTs) were performed twenty four hourly.

Simple fluid column manometry method was employed to measure ICP. This was the most demanding of all the recordings and measurements and was undertaken with care and precaution. For its monitoring, each patient was catheterized with appropriate size Foley catheter under aseptic precautions and drainage tubing connected. A long strip of adhesive tape was calibrated with the help of ruler to mark 1cm increments onto the tape. The tape was adhered to the tubing a few millimetres away from the Y-junction of Foley catheter. The first mark was zero reference point when it was held at the level of pubic symphysis. For measuring ICP, the bladder was first emptied and then primed with 50 cc normal saline using a large piston syringe aseptically. The drainage tubing was connected to the Foley catheter and the tubing with bag was

elevated straight to vertical position so that the zero reference point came at the level of pubic symphysis. The column of fluid in the vertically held tubing was the ICP in cm H_2O . This reading was converted to mmHg by using the formula 1 mmHg=1.26 cm H_2O .

ACS was diagnosed on the basis of raised IAP of >10 mmHg coupled with an evidence of end organ failure such as oliguria, deranged RFTs, deranged ABGs, rising peak inspiratory pressures, cardiovascular collapse and shock. A variety of risk factors that lead to ACS were studied among the patients and included peritonitis, gut oedema, systemic inflammatory response syndrome (SIRS), tense ascites, intraperitoneal bleed, excessive intravenous fluid resuscitation, pneumoperitoneum, forced closure of tense abdomen, obesity, history of trauma and laparotomy.

Since it was an observational study, we did not institute any ACS specific interventions such as decompressive laparotomy or Baggota bag technique of temporary abdominal containment etc. Rest of the intensive care and surgical management was according to the established standard protocols.

The data were analysed through SPSS for Windows version 10. The nominal variables were reported as frequency and percentages. The numerical data was reported as Mean \pm S.D. Nominal variables were analysed using chi square test. The difference between two means was regarded if *p* was <0.05.

RESULTS

Out of 200 patients, 112 were male and 88 were female. The Male: Female was 1.27:1. The age of the patients ranged between 15–77 years with a mean of 38.7 ± 16.9 years. Half of the patients were in their third and fourth decades of life. One hundred thirty eight 69% of the patients were from the twin cities of Islamabad and Rawalpindi while the rest were referred complicated cases from Azad Jammu Kashmir and upper Punjab.

The disease-wise break-up of the included patients is depicted in Table-1. Six patients had ACS with an overall frequency of 3%. Their underlying pathologies included surgical severe acute pancreatitis, pseudomyxoma peritonei, gut gangrene due to midgut volvulous, intestinal obstruction, chest trauma and blunt abdominal trauma one each. All these patients had developed SIRS and sepsis. They had fulminant systemic illness. Their first ICP measurements were alarmingly high (>15 mmHg) and continued progressively increasing through the course of illness without any reducing trend. The Male:Female was 2:1. Fifty percent of the patients were in the age range of 31–40 years.

Table-2 shows the risk factors observed among the patients of ACS. The statistically significant risk factors for the development of ACS

included severe peritonitis, severe gut oedema, systemic inflammatory response syndrome (SIRS) and tense ascites. Other factors such as intraperitoneal bleed, excessive intravenous fluid resuscitation, large pneumoperitoneum, tight closure of non-compliant abdomen, obesity, history of trauma and surgery etc. were found in varying proportions but the association with ACS as isolated risk factors remained statistically insignificant. Most of the patients rather had a combination of more than three risk factors. All the patients with ACS had features of multiorgan dysfunction including low urine output with raised serum urea and creatinine, impaired breathing with respiratory acodosis, elevated central venous pressure and persistent hypotension.

Table-1: Break-up of the patients (n=200)

	No. of		
Pathology	patients	percentage	
Intestinal obstruction	36	18.0	
Pancreatitis	23	11.5	
Chest trauma	43	21.5	
Gut perforation	30	15.0	
Blunt abdominal trauma	29	14.5	
Gut gangrene	14	7.0	
Rectal carcinoma	8	4.0	
Penetrating abdominal trauma	5	2.5	
Gastric carcinoma	5	2.5	
Esophageal carcinoma	2	1.0	
Pancreatic carcinoma	2	1.0	
Cholangio carcinoma	2	1.0	
Pseudomyxoma peritonei	1	0.5	

Five out of 6 of the ACS sufferers had inhospital mortality while one patient with pseudomyxoma peritonei underwent laparotomy but left the hospital against medical advice following receiving information regarding the prognosis and plan for chemotherapy. This later patient was lost to further follow-up. Thus the in-hospital mortality among our ACS patients remained 80%.

 Table-2: Risk factors found amongst the patients

	Pati	ents	Patients		
	with ACS		without ACS		
	(n=6)		(n=194)		
Risk factors	No.	%	No.	%	р
Severe peritonitis	5	83.3	11	5.7	< 0.05
Severe gut oedema	5	83.3	13	6.7	< 0.05
Systemic inflammatory					
response syndrome	6	100	7	3.6	< 0.05
Tense ascites	2	33.3	4	2.0	< 0.05
Intra-peritoneal bleed	2	33.3	11	5.7	>0.05
Trauma	2	33.3	75	38.66	>0.05
Laparotomy	5	83.3	126	64.9	>0.05
Excessive fluid					
resuscitation	1	16.6	5	2.5	>0.05
Large pneumoperitoneum	1	16.6	3	1.54	>0.05
Tight/forced closure of					
non-compliant abdomen	1	16.6	4	2.0	>0.05
Obesity	1	16.6	9	4.63	>0.05

DISCUSSION

The exact incidence of ACS is yet to be established, however it is certainly high among certain patients such as those with severe blunt and penetrating abdominal aortic abdominal trauma, ruptured aneurysms, retro peritoneal haemorrhage, pneumoperitoneum, pancreatitis, massive ascites, liver transplantation, extrinsic compression of abdominal wall by burn eschars, and also patients undergoing massive fluid resuscitation, forced closure of a noncompliant abdominal wall or intra-abdominal packing.8,12,14

In our present study the frequency of ACS in our critically ill surgical patients was 3%. Published studies mostly from the West have reported the incidence to be in the range of 4% to 40%.^{12,22} The incidence is relatively higher among surgical patients admitted to intensive care units.^{3,13}

In the present study ACS was found among the patients who had severe peritonitis, severe gut oedema, systemic inflammatory response syndrome and tense ascites. A variety of other risk factors as described in the introduction have been reported by various studies.^{1-3,23}

The in-hospital ACS mortality in our series was 80%. All of them had multiple organ failure with SIRS and sepsis. All the dying patients had fulminant course of illness and death ensued within the first 48 hours of admission. The patient with pseudomyxoma peritonei who left hospital against medical advice also had multiple organ failure and hence a potential nonsurvivor. None of these patients could receive ACS specific interventions, however as a routine policy of our unit, now we employ decompressive laparotomy and Baggota bag technique of temporary abdominal containment in all patients with raised IAP or fullblown ACS. Most of the authorities have recognized the high mortality associated with ACS and have emphasised rather preventive measures among the high risk patients.^{12,13,24}

We employed simple fluid column manometry method¹⁸ for measuring ICP and found it to be simple, rapid and inexpensive method requiring no special device such as pressure transducer. Methodological issues regarding abdominal pressure measurement have long been debated. Traditionally IAP has been measured by measuring ICP using a Foley catheter and connecting it to a pressure transducer. This technique was popularized by Kron *et al*¹² in 1984 in order to avoid other direct invasive techniques.^{25,26}

CONCLUSION

ACS develops in a significant number of critically ill and traumatised patients. It develops quickly and

proves almost inevitably fatal without ACS specific interventions. A high index of suspicion is imperative for prompt recognition of this fatal condition. All such high risk patients should undergo serial ICP measurements as a screening test for early detection of ACS. Moreover no local published study on ACS is available to date in Pakistani literature, the present study would emphasise the need for further epidemiological studies so as to elucidate the exact association between various risk factors and ACS in our patients. This would not only contribute to ACS research but also help to improve the existing standards of patient care.

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