# **Abdominal Compartment Syndrome Evaluation in Sever Acute Pancreatitis**

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#### **ABSTRACT**

Background: ACS (abdominal compartment syndrome) is a serious condition that affects critically ill people. Because it predominantly affects individuals who are already sick, it may go unnoticed. The aim of study to find a link between IAP and the development of problems in patients with severe acute pancreatitis. We also wanted to see if there was a link between IAP and the development of complications in patients with severe acute pancreatitis (SAP). Patients and methods: This prospective study was conducted on 18 patients with AP hospitalised to the Surgical Intensive Care Unit at Zagazig University's Faculty of Medicine's General Surgery Department (SICU). During the first week after admission, all patients were divided into two groups based on their IAP. Patients with IAP 20 mmHg were assigned to the IAH group, whereas those with IAP 20mmHg were assigned to the normal IAP group. Results: Age, serum Lipase and APACHE were significantly higher among ACS cases and also ACS cases were significantly associated with DM. ACS cases were significantly associated with longer hospital stay also with bleeding, septic shock and mortality. The mean Lipase level was 959.72±103.58 with rang (800-1200). The mean Intra-abdominal pressure of studied patients was 12.94±4.12 and 38.9% of patients had IAH while 2 cases 11.1% had ACS. Conclusion: In patients hospitalised to the ICU with severe acute pancreatitis, IAH and ACS are common findings. IAH could make severe acute pancreatitis worse. Early detection of this possibly treatable aggravating condition could lead to early management and, in turn, a better prognosis.

Keywords: Acute Pancreatitis; ACS; Abdominal Compartment Syndrome

# INTRODUCTION

Abdominal compartment syndrome (ACS) is a severe illness seen in critically ill patients. It may be underrecognized because it primarily affects patients who are already quite ill and whose organ dysfunction may be incorrectly ascribed to progression of the primary illness (1). Abdominal compartment syndrome can be divided and classified into two groups, primary and secondary ACS. Primary ACS causes include abdominal blunt or penetrating trauma, hemorrhage, abdominal aortic aneurysm (AAA) rupture, intestinal obstruction, and retroperitoneal hematoma. Secondary causes include pregnancy, ascites, ileus, burns, intra-abdominal sepsis, and large-volume fluid replacement (> 3 liters). Chronic causes of elevated intra-abdominal pressure include pregnancy, cirrhosis, obesity, intra-abdominal malignancy, and peritoneal dialysis. The presence of organ dysfunction in this setting due to compressive symptoms now confirms a diagnosis of abdominal compartment syndrome (2,3).

Acute pancreatitis (AP) is a well-established risk factor for intraabdominal hypertension (IAH) and abdominal compartment patients with severe acute pancreatitis is approximately 60 %, while ACS may occur in approximately 30 %. The mortality of patients with severe acute pancreatitis who develop ACS is high at 50–75 % (4,5). There have been significant advances about our knowledge of the natural course and underlying pathophysiology of severe AP and similarities between manifestations of fulminant acute pancreatitis (an early disease course characterised by pulmonary, cardiovascular, and renal insufficiency which may lead to rapidly progressive multiple organ dysfunction syndrome) and abdominal compartment syndrome (ACS) has increasingly drawn investigators to study relationship between the two (6.7).

While conservative treatment is still the gold standard in the early stages of severe AP, growing data suggests that a subset of patients with early severe disease may benefit from surgical/interventional procedures (8). While offering a previously elusive point of therapeutic intervention, evidence is still lacking as to whether intra-abdominal pressure (IAP) measurements should be routine in all patients with AP or if some selectivity can be maintained, and how patients at risk for developing IAH and ACS can be identified as soon as possible.

The aim of the present study was to determine the incidence of ACS in patients with acute pancreatitis who were diagnosed early on, to evaluate IAP as a marker of severity in acute pancreatitis, and to see if there was a link between IAP and the development of complications in patients with severe acute pancreatitis (SAP)

### PATIENTS AND METHOD

This prospective study was conducted on 18 patients with AP admitted to the Surgical Intensive Care Unit (SICU) between July 2019 and March 2020 in the General Surgery Department,

Faculty of Medicine, Zagazig University, with sample size determined by the IRB. During the first week after admission, all patients were divided into two groups based on their IAP. Patients with IAP ≥20mmHg were placed in the IAH group, while those with IAP 20mmHg were placed in the normal IAP group.

Inclusion criteria was patients with AP admitted to SICU with time interval between onset of typical abdominal symptoms and study inclusion of 72 hours or less. The presence of Systemic Inflammatory Response Syndrome (SIRS) manifested by two or more of the following conditions: temperature > 38°C or < 36°C; Heart Rate (HR) > 90 beats/minute; respiratory rate > 20 breaths/minute or PaCO<sub>2</sub>< 32 mmHg; WBC count > 12,000/mm³ or < 4000/mm³, or > 10% immature (band) forms; and at least 3-fold elevated serum amylase or lipase levels, or a APACHE II score >71, or a C-Reactive Protein (CRP) of  $\geq$  250 mg/L.

Patients with history of chronic pancreatitis, admission after resuscitation for cardiac arrest, or patients with significant comorbid conditions like renal failure, cardiac disease, chronic abdominal pathology, and immunosuppression are excluded from this study

Intravesical Pressure (IVP) measurement had been described as a standard and validated technique of indirectly reflecting IAP and rapidly recognizing ACS (9).

Hemodynamics and other physiologic parameters, including HR, Mean Arterial Pressure (MAP), Central Venous Pressure (CVP), Urine Output (UO), Peak Airway Pressure (PAP), arterial carbon dioxide partial pressure (PaCO $_2$ ), Modified Respiratory Index (MRI) expressed as the the arterial oxygen partial pressure (PaO $_2$ ) to fraction of inspirated oxygen (FiO $_2$ ) ratio (PaO $_2$ /FiO $_2$ ), arterial pH, arterial base deficits (BE) and arterial lactic acid, were registered before and after decompression of ACS.

Acute Physiology and Chronic Health Evaluation (APACHE) II score and Ranson scoring system during the first 24 or 48 hours of SICU stay were recorded respectively in the identification of the severity of pancreatitis. Total Marshall scores were computed daily and for the entire duration of the intensive care stay. All patients with AP were detected using contrast-enhanced Computed Tomography (CT) on admission.

The incidences of pancreatic infection, septic shock, MODS, and the in-hospital mortality were also recorded. Pancreatic infection included infected necrosis and pancreatic abscess. Necrosis formation of the pancreas was assessed by contrast-enhanced CT. Patients with pancreatic necrosis or large peripancreatic fluid collections who manifested clinical signs of sepsis with a non-improving or deteriorating clinical course despie a reasonable time of medical therapy underwent Fine-Needle Aspiration (FNA) of the necrotic areas or fluid collections under CT or ultrasound guidance to determine the presence of bacterial contamination or pancreatic infection.

# Statistical analysis:

Microsoft Excel software was analyse data using Social Sciences (SPSS version 20.0) software. The following tests were used to test differences for significance based on the type of data: qualitative data was represented as a number and percentage, quantitative data was represented as a mean and SD, and the following tests were used to test differences for significance based on the type of data: Chi square test (X2) was used to determine the difference and association of qualitative variables. t test for differences between quantitative independent groups. P value was set at <0.05 for significant results &<0.001 for high significant result.

#### **RESULTS**

Age was distributed as 39.66±7.69 with minimum 27 and maximum 52 years, regard sex distribution.(table 4)APACHE was distributed as 3.94±1.35 as 50% were 71> (low) and 50% were <71 (high) (**Table 1**).

About 72.2% of studied group had no morbidity and 27.8% had comorbidities as 2 cases with HTN and 3 cases had DM, regard previous surgery only 5 cases had history of surgery (**Table 2**).

Majority were Biliary with 72.2% followed by alcohol 16.7% and trauma 11.1% the type of trauma Abdominal penetrating trauma Lipase level was distributed as Lipase mean of 959.72±103.58 with rang (800-1200). Intra-abdominal pressure was distributed as 12.94±4.12 and 38.9% had IAH and only 2 cases with 11.1% had ACS. (**Table 3**).

Age, serum Lipase and APACHE were significantly higher among ACS cases and also ACS cases were significantly associated with DM (**Table 4**).

ACS cases were significantly associated with longer hospital stay also with Bleeding, Septic shock and Mortality. One case needed for surgical decompression because inter in A.C.S. complication. Bleeding occure in 2 cases is internal bleeding in serous cavities caused by abdominal trauma regard septic shock only one case is affected by septicemia especially gram –veseptiomia lead two damage of intestinal mucosa and acute erosive gastritis (**Table 5**).

Table1: APACHE distribution among studied group

S-O-I							
		APACHE					
Mean± SD		3.94±1.35					
Median (Range)		3.50 (2-8)					
		N	%				
	< 71	9	50.0				
APACHE	> 71	9	50.0				
	Total	18	100.0				

Table2: Cause of AP distribution among studied group

		N	%
	Biliary	13	72.2
Cause	Alcohol	3	16.7
	Trauma	2	11.1
	Total	18	100.0

Table3: Intra-abdominal pressure distribution among studied group

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		IAP	IAP		
Mean± SD		12.94±4.12	12.94±4.12		
Median (Range)		11.0 (9-25)	11.0 (9-25)		
		N	%		
IAH	Normal	11	61.1		
	IAH	7	38.9		
ACS	Negative	16	88.9		
	Positive	2	11.1		
Total		18	100.0		

Table 4: Relation between ACS and other demographic and clinical parameters

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		Normal	ACS	t/X <sup>2</sup>	P		
Age		38.56±6.33	48.50±4.94	2.189	0.035*		
Serum Lipase		932.81±71.6	1175.0±35.3	4.621	0.00**		
APACHE	,		3.50±1.16	7.50±0.70	3.996	0.001**	
	Male	N	10	1			
Com		%	62.5%	50.0%			
Sex	Female	N	6	1	0.11	0.73	
	remaie	%	37.5%	50.0%			
_	A	N	4	1			
	A	%	25.0%	50.0%			
Blood	В	N	2	0			
group	В	%	12.5%	0.0%	0.69	0.706	
	0	N	10	1			
		%	62.5%	50.0%			
	<71	N	7	2			
APAC		%	43.8%	100.0%			
HE	>71	N	9	0	2.25	0.13	
		%	56.2%	0.0%			
	No	N	13	0			
		%	81.2%	0.0%			
Comor	HTN	N	2	0			
bidities	HTN	%	12.5%	0.0%	11.25	0.004*	
	DM	N	1	2			
	DM	%	6.2%	100.0%			

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Cause	Alcohol	N	3	0		
		%	18.8%	0.0%		
	Biliary	N	12	1	3.59	0.166
		%	75.0%	50.0%		
	Trauma	N	1	1		
		%	6.2%	50.0%		
	Normal	N	11	2		
		%	68.8%	100.0%		
D	CS	N	3	0		
Previo		%	18.8%	0.0%	0.86	0.08.3
us surgery	Laparotom	N	1	0		
	y	%	6.2%	0.0%		
	Appendect	N	1	0		
	omy	%	6.2%	0.0%		
Total		N	16	2		
		%	100.0%	100.0%		

Table 5: Relation between ACS and outcome

			NO	ACS	t/X <sup>2</sup>	P
Stay			11.56±3.03	18.0±4.24	2.749	0.014*
	WE	N	13	1		
	-VE	%	81.3%	50.0%		
Infection	+VE	N	3	1	1.41	0.12
	+VE	%	18.7%	50.0%		
	WE	N	15	2		
Acute Pseudo	-VE	%	93.8%	100.0%		
cyst	WE	N	1	0	0.13	0.71
	+VE	%	6.2%	0.0%		
	-VE	N	16	0		
Dlooding		%	100.0%	0.0%		
Bleeding	+VE	N	0	2	18.0	0.00**
		%	0.0%	100.0%		
	-VE	N	16	1		
Contin about		%	100.0%	50.0%		
Septic shock	+VE	N	0	1	8.47	0.004*
		%	0.0%	50.0%		
	Survive	N	16	1		
Montolity	d	%	100.0%	50.0%		
Mortality	Died	N	0	1	8.47	0.004*
		%	0.0%	50.0%		
Total N/%		N	16	2		
		%	100.0%	100.0%		

#### DISCUSSION

Intra-abdominal pressure was distributed as  $12.94 \pm 4.12$  and 38.9% had IAH and only 2 cases (11.1%) had ACS. **Smit et al.** (7) summarized IAP measurements. IAP measurement was performed in 29 out of 59 patients (49.2 %).

In our study, hospital stay was distributed as  $12.27 \pm 3.67$  with minimum of 8 and maximum of 21 days. In agreement with our study, **Smit et al.** (7) found that the mean interval between admission to a (regional) hospital and the tertiary ICU was 13.4 days (SD 20.7, range 0–123).

Although the exact mechanisms are not completely understood, IAH in severe acute pancreatitis is usually an early phenomenon and may have an important role in the development of early organ failure (10).

In our study, ACS cases had significantly higher age, serum lipase, and APACHE, and ACS cases were also significantly associated with DM. **Smit et al.** (7), on the other hand, found that age and Apache II scores did not differ significantly between the groups where IAP was measured and those

who did not. Patients who developed ACS and those who did not had similar age, gender, and APACHE II scores upon admission.

ACS cases were found to be associated with a longer hospital stay, bleeding, septic shock, and mortality in our study. According to van **Santvoort et al.** (11), SAP mortality is divided into two phases: an early phase in which multiple organ failure is caused by SIRS, IAH, or ACS, and a later phase in which mortality is caused by secondary infection and necrosis.

In the setting of pancreatitis with organ failure, measurement of IAP and protocol zed monitoring and management of IAPs recommended ,IAP can be measured easily and reliably. in patients through the bladder using. IAH may affect all organ systems, but. respiratory, cardiovascular, and kidney function are affected most often. Even slightly elevated abdominal pressures lead to signs of systemic inflammation and acute kidney injury. article describes that death occurs in the early phase of the disease as a result the development of organ failure that appears to be related to the development of sirs (12).

Chen et al. confirmed our findings that ACS was found in severe acute pancreatitis (13). IAH was discovered in roughly 60% of the patients studied.

We strongly advocate for protocolized IAP monitoring in high-risk patients, such as those with severe acute pancreatitis, in order to detect and treat IAH and ACS early. We also recommend that international guidelines for the treatment of pancreatitis patients be updated to reflect this recommendation.

The British Society of Gastroenterology, the American College of Gastroenterology, and the American Gastroenterological Association have all published guidelines that do not mention the need for routine IAP measurements. Once IAH is detected, IAP and organ functions must be carefully monitored, and measures must be taken to prevent further organ dysfunction and irreversible damage (12).

Patients with acute pancreatitis who underwent surgery for abdominal compartment syndrome at a tertiary referral centre were studied by **Boone et al. (4).** For abdominal compartment syndrome, twelve patients had a decompressive laparotomy. The median time between the onset of pancreatitis and the insertion of a laparotomy was 4.5 days. Within seven days of the onset of pancreatitis, nine patients had a laparotomy. Four decompressive laparotomies were performed in the intensive care unit due to cardiopulmonary instability. Cardiopulmonary improvement was observed in 11 patients. Multiple physiologic parameters showed statistically significant improvements. Despite this initial improvement, six patients (50%) died from multisystem organ failure. Two patients survived without need for pancreatic débridement. Abdominal compartment syndrome is an uncommon but likely underrecognized and highly lethal complication of acute pancreatitis that should be considered in patients who become critically ill early in the course of their pancreatitis.

In our study measurement of i a p ,in all cases by ct scan and intravesicular design on 18 patients, one of patient needed surgical decompression because increased in I a p and multi organ failure .most commen cause of death in this patient is adult respiratory distress (12).

# **CONCLUSION**

In patients hospitalised to the ICU with severe acute pancreatitis, IAH and ACS are common findings. IAH could make severe acute pancreatitis worse. Early detection of this possibly treatable aggravating condition could lead to early management and, in turn, a better prognosis.

# No conflict of interest.

# REFERENCES

- 1. Luckianow G M, Ellis M, Governale D, and Kaplan LJ. (2012): Abdominal compartment syndrome: risk factors, diagnosis, and current therapy. Critical care research and practice, 2012. Vol (3): p133.
- 2. Gray S, Christensen M, Craft J. (2018): The gastro-renal effects of intra-abdominal hypertension: Implications for critical care nurses. Intensive Crit Care Nurs48, 69-74.
- 3. Miranda E, Manzur M, Han S, Ham SW, Weaver FA, and Rowe VL. (2018): Postoperative Development of Abdominal Compartment Syndrome among Patients Undergoing Endovascular Aortic Repair for Ruptured Abdominal Aortic Aneurysms. Annals of vascular surgery, 49: 289-294.
- 4. Boone B, Zureikat A, Hughes S J, et al. (2013): Abdominal compartment syndrome is an early, lethal complication of acute pancreatitis. Am Surg. 79(6), 601-607.
- 5. Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, and Sugrue M. (2013): Intra-abdominal hypertension and the abdominal compartment syndrome:

- updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive care medicine, 39(7), 1190-1206.
- 6. Bhandari V, Jaipuria J, Singh M, Chawla AS. (2013). Intra-abdominal pressure in the early phase of severe acute pancreatitis: canary in a coal mine? Results from a rigorous validation protocol. Gut Liver. 7(6), 731
- 7. Smit M, Buddingh KT, Bosma B, Nieuwenhuijs VB, Hofker HS and Zijlstra JG (2016): Abdominal compartment syndrome and intra-abdominal ischemia in patients with severe acute pancreatitis. World J Surg; 40: 1454-1461.
- 8. Ikeda S, Kagami T, Tani S, et al. (2019): Decompressive laparotomy for abdominal compartment syndrome resulting from severe acute pancreatitis: a case report. BMC Gastroenterol. 19(1), 1-5
- 9. Fusco MA, Martin R S, and Chang MC. (2001): Estimation of intra-abdominal pressure by bladder pressure measurement: validity and methodology. Journal of Trauma and Acute Care Surgery, 50(2), 297-302.
- De Waele JJ, Leppaniemi AK (2009): Intra-abdominal hypertension in acute pancreatitis. World J Surg; 33: 1128–1133.
- 11. van Santvoort HC, Besselink MG, Bakker OJ (2010): A stepup approach or open necrosectomy for necrotizing pancreatitis. N Engl J Med; 362: 1491–1502.
- 12. Tenner S, Baillie J, DeWitt J (2013): American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol. 108: 1400–1415.
- 13. Chen H, Li F, Sun JB, Jia JG (2008): Abdominal compartment syndrome in patients with severe acute pancreatitis in early stage. World J Gastroenterol. 14: 3541–3548.