



## ROLE OF INTRA-ABDOMINAL PRESSURE IN PREDICTING OUTCOME IN ACUTE SEVERE PANCREATITIS.

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**ABSTRACT** **Background:** Mortality in patients with severe acute pancreatitis (SAP) remains high. Some of these patients develop increased intra-abdominal pressure (IAP) Which may contribute to organ dysfunction. The aim of this study was to evaluate intra-abdominal pressure as a marker of severity in severe acute pancreatitis and to evaluate the relationship between intra-abdominal pressure and development of complications like SIRS, organ failure, pancreatic necrosis in patients with severe acute pancreatitis. A total of 50 patients with acute pancreatitis with no significant co-morbid conditions were studied in this group by measuring the intra-abdominal pressure with a Foleys catheter inserted into the bladder and calibrating the pressure with a pressure monitoring scale. This study deals with predictive value of intra-abdominal pressure in progression of acute pancreatitis as a cheaper but not an alternative for traditional predictive scores.

**KEYWORDS :** Severe acute pancreatitis , Intra-abdominal pressure, predictive value .

### INTRODUCTION

Intra abdominal hypertension is increasingly reported in patients with acute pancreatitis and is caused by several factors including visceral oedema and ascites associated with massive fluid resuscitation , paralytic ileus and retroperitoneal inflammation.

There is strong relation with early organ dysfunction and mortality in these patients , which makes intra abdominal hypertension an attractive target for intervention.

Although the typical symptoms of ACS (Abdominal Compartment Syndrome), IAP > 25 mmHg i.e. rapidly evolving multiple organ dysfunction syndrome (MODS) (most often a combination of respiratory failure, hemodynamic compromise and acute renal failure) are often found in patients with severe acute pancreatitis, it was not until recently that the importance of IAH has been recognised.

Insights into the true incidence of IAH in patients with acute pancreatitis are hampered by the lack of uniform definitions of IAH and ACS until recently. Also, guidelines for reliable IAP monitoring have changed, as instillation of larger volumes of saline for IAP monitoring may lead to falsely elevated values and in most studies selection bias cannot be completely excluded, as routine measurement of IAP is rarely performed.

Nevertheless, the high incidence rates of both IAH and ACS, and the mortality associated with both the problems are significant. A clear causal relationship could not be demonstrated, but the case reports and case series reporting excellent outcomes in pancreatitis patients undergoing abdominal decompression suggest that IAP may be a target for early intervention.

Thus there is a need for a study showing that a rise in IAP results in deterioration of patients condition.

### MATERIAL AND METHODS:

#### SOURCE OF DATA:

The total of 50 patients with acute pancreatitis presenting to ALLURI SITARAMA RAJU ACADEMY OF MEDICAL SCIENCES , ELURU.

Patients satisfying criteria of pancreatitis would be selected and prospective observational study conducted.

### METHODS OF DATA COLLECTION

The clinical definition of acute pancreatitis, whether in the presence or

absence of underlying chronic pancreatitis, requires two of the following three features:

- 1) Abdominal pain suggestive strongly of acute pancreatitis,
- 2) Serum amylase and/or lipase activity at least 3 times greater than the upper limit of normal, and
- 3) Characteristic findings of acute pancreatitis on trans-abdominal ultrasonography.

### INCLUSION CRITERIA:

- 1) All individuals greater than 18 years of age.
- 2) Duration of symptoms less than 72 hours.

### EXCLUSION CRITERIA:

- 1) Pregnant females.
- 2) Individuals with significant co morbid conditions like renal failure, cardiac disease, and immunosuppression.

### DIAGNOSIS OF ACUTE PANCREATITIS:

RANSON'S CRITERIA. At admission:

1. Age in years > 55 years WBC count > 16000 cells/mm<sup>3</sup> Blood glucose > 11.11 mmol/L (> 200 mg/dL)
2. Serum AST > 250 IU/L
3. Serum LDH > 350 IU/L

### Within 48 hours:

1. Serum calcium < 2.0 mmol/L (< 8.0 mg/dL) Hematocrit fall > 10%
2. Oxygen (hypoxemia PaO<sub>2</sub> < 60 mmHg) BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration
3. Base deficit (negative base excess) > 4 mEq/L
4. Sequestration of fluids > 6 L

### TECHNIQUE OF MEASUREMENT OF IAP:

To determine Intra Abdominal Pressure (IAP hereafter) we will use a Foley catheter inserted into the bladder and instilled with 25 ml sterile saline (1 mm Hg = 1.36 cm H<sub>2</sub>O) with symphysis pubis as level 0. We will use the low cost technique published by Adhish Basu (J.I.P.M.E.R. India) (*Ann R Coll Surg Engl* 2007; 89: 431–437 A low-cost technique for measuring the intra-abdominal pressure in non-industrialised countries). A similar method has also been previously described by Sedrak (Sedrak M, Major K, Wilson M. Simple fluid column manometry to monitor for the development of Abdominal compartment Syndrome. *Contemporary Surgery* 2002; 56: 6.).

IAP will be measured at admission after control of severe acute pain by optimal use of analgesics including tramadol, morphine, pethidine, and fentanyl to minimize the confounding effect of pain on IAP measurement. Further IAP measurements will be done every 8 hourly on 1<sup>st</sup> day. Diagnosis of Intra Abdominal Hypertension will be done by a fixed protocol discussed below. IAP measurements will be done till Foley catheter is required in situ for patients. IAP measurements will be done every 4 hourly for those with abdominal compartment syndrome. 'Maximum IAP' is defined as the maximum pressure recorded in all readings and 'mean IAP' is defined as the mean of all pressure values recorded within the first 5 days.

Intra Abdominal Hypertension (IAH) is defined by a sustained or repeated pathologic elevation of IAP $\geq$ 12 mmHg (World Society for the Abdominal Compartment Syndrome). In our study we define IAH as consistently increased IAP $\geq$ 12 recorded by first 3 readings during at least 8 hours. Early severe pancreatitis mostly determines the outcome. IAH is classified into FIVE groups class-A= no IAH Class B-Grade one =12-15mmHg, Class C- Grade two= 16-20mmHg, Class D-Grade3= 21-25mmHg, Class E-Grade= more than 25mmHg. Class E is equivalent to abdominal compartment syndrome (Severe Acute Pancreatitis).

Each patient is classified into one class according to IAP and is followed up using a standard proforma. For each class vitals of day one, SIRS status, RANSON'S scores, presence of any organ failure, is measured further data will also be collected about length of hospital stay, development of SIRS, Multi organ failure, presence of septic complications, intra abdominal collections needing aspiration and/or percutaneous drainage apart from standard demographic and clinical data and ultimately condition of the patient at the of follow up period.

All patients will be treated by our standard management of pancreatitis protocol and practice guidelines in acute pancreatitis.

OUTCOME	CLASS A	B	C	D	E
TOTAL RANSONS	4.7	5.1	5.75	5.25	8
SIRS	5	8	16	7	1
ONE ORGAN FAILURE	1	6	6	0	0
MODS	0	1	3	5	1
MORTALITY	0	0	1	0	1
TOTAL	7	18	16	08	01

## RESULTS:

50 patients were included in the study, the mean age was 43 years. The mean time interval between onset of acute pancreatitis and admission on average was 24 hours.

No IAH noted in 7 patients (14%), mild IAH (class B) – 18(36%), Moderate IAH-(class C)-16(32%), severe IAH- class D- 8(16%) Class E i.e ACS in 1 patient -2% 13 patients had one organ failure (renal/hepatic/ respiratory) and 10 patients had MODS.

5 patients with no IAH had SIRS, only one had one organ failure and none had MODS.

2 deaths (4%) were seen one belonging to class C (moderate IAH) and the other belonging to class E(ACS).

## DISCUSSION:

One of the main problems in management of acute pancreatitis is to anticipate complications that may occur during its evolution.

Acute pancreatitis should be considered as a dynamic process and such its follow-up and reassessment must follow the same principles.

Prognostic markers have been created with either single (eg-C-reactive protein) or multiple variables (Ransons, Glasgow), but the later are more difficult to use.

The ideal marker should not only be easy, reproducible, inexpensive but can also detect patients with worrisome evaluation requiring diagnostic/therapeutic maneuvers.

Intra abdominal pressure was therefore proposed as a parameter to predict the evaluation of acute pancreatitis.

The mean IAP on day of admission appeared to be a more accurate marker of severity of pancreatitis.

Intra abdominal pressure was significantly related all these other prognostic parameters.

IAP is not indicated as an initial prognostic marker because the acute abdominal pain in early stages of the disease and unidentified detrusor instability may influence the measurement of IAP. Therefore measurement of IAP should not be performed until pain control was achieved.

To determine the sensitivity and specificity of IAP to detect the severity of acute pancreatitis, linear regression analysis were performed to establish cut-off values to classify mild or severe pancreatitis.

IAP measurement may be particularly useful in patients considered as having severe pancreatitis according to the traditional markers at admission (Ranson=3, or a serum C-reactive protein conc-150mg/dl) because it can predict which patients are prone to poor outcome of acute pancreatitis.

However we found significant relationship between IAP and the variables related to development of SIRS, organ failure, pancreatic necrosis.

An increase in IAP causes a decrease in splanchnic flow.

According to Diebel et al,9 the flow to mesenteric artery decreases by 37% with IAP >20mmHg and this decrease is even higher in intestinal mucosa, with baseline flow decreasing to 39% with only 10mmHg IAP.

This decrease in mesenteric blood flow secondary to an increase in IAP may cause two phenomenon that alters the integrity of intestinal barrier-ischemia and reperfusion.

The structural alteration that develops secondary to decreased flow may allow passage of microorganisms through the epithelium into the lymph nodes and blood stream by bacterial translocation, thereby leading to super-infection of pancreas and pancreatic necrosis.

The result of this study demonstrate that, although IAP will not replace the currently accepted scoring systems in patients with acute pancreatitis. IAP measurement may be useful as an easy, inexpensive prognostic marker of the evolution and complications of acute pancreatitis.

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