

ORIGINAL RESEARCH PAPER

Medical Science

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

KEY WORDS: Intraabdominal pressure, acute severe pancreatitis

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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 aims 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.

INTRODUCTION

Intra abdominal hypertension is increasingly reported in patients with acute pancreatitis and is caused by several factors including visceral edema 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>25mmhg 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 performed1. 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 decompression² and ³ 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 Konaseema Institute Of Medical Sciences and Research Institute, Amalapuram. 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 transabdominal ultrasonography.

7.3) Inclusion Criteria:

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

7.4) Exclusion Criteria:

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

DIAGNOSIS OF ACUTE PANCREATITIS: RANSONS CRITERIA. At admission:

- 1. Age in years > 55 years
- 2. WBC count > 16000 cells/mm³
- 3. Blood glucose > 11.11 mmol/L (> 200 mg/dL)
- 4. Serum AST > 250 IU/L
- 5. Serum LDH > 350 IU/L

Within 48 hours:

- $1.\,Serum\,calcium\,{<}\,2.0\,mmol/L\,({<}\,8.0\,mg/dL)$
- 2. Hematocrit fall > 10%
- 3. Oxygen (hypoxemia $PaO_2 < 60 \text{ mmHg}$)
- 4. BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration $\,$
- 5. Base deficit (negative base excess) > 4 mEq/L
- 6. Sequestration of fluids > 6 L

CTSI

${\bf Grading\ Of\ Pancreatitis\ (Balthazar\ Score)}$

- A:normalpancreas:0
- B:enlargement of pancreas: 1
- C: inflammatory changes in pancreas and peripancreatic fat: 2
- D:ill-defined single peripancreatic fluid collection: 3
- E: two or more poorly defined peripancreatic fluid collections:4

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 H2O) 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 intraabdominal pressure in non-industrialised countries). A similar method has also been previously described by Sedrek (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 1st 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≥12 mmHg (World Society for the Abdominal Compartment Syndrome). In our study we define IAH as consistently increased IAP≥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 ,RANSONS 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	В	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%),

MildIAH(classB)-18(36%),

Moderate IAH-(class C)-16(32%),

Severe IAH-class D-8(16%)

Class Ei.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.

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

CONCLUSION:

In patients with acute pancreatitis when there is raised, intraabdominal pressure of >25mm of Hg are more prone to develop mods{multi organ dysfuntion sydrome}, which is leading to mortality of the patients.

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