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Research Article

Therapeutic Way to Predicting Acute Pancreatitis Non-Necrotising (Mild) and Necrotising (Severe) at 24 Hrs

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Abstract

Hypothesis: Intravenous fluid therapy is established in skin burn and acute pancreatitis. Surface area and depth decide the fluid volume in burn. Acute pancreatitis is a chemical burn of pancreas and retroperitoneum, can the volume of fluid therapy determine the severity?

Introduction: Acute pancreatitis can be mild to Severe. Currently available methods to predict severe pancreatitis are complex, cumbersome and inaccurate. A simple way is required.

Materials and Methods: Patients presenting clinically as acute pancreatitis with elevated serum amylase/lipase thrice than normal were managed by intravenous fluid similar to skin burnt injury, so as to have urine output of 0.5 - 1 mL per Kg. Based on the difference between the intake and output, they were predicted as necrotising/non-necrotising at 24 hrs. Confirmation was done on CT scan after 72 hrs or at autopsy.

Results: There was 63 patients with 9 females and 54 males, age ranging from 19 - 59 years. 38 were predicted mild and 25 as severe based on the fluid deficit ranged from 0.4 lit to 9.9L after 24 hrs. the prediction was accurate in 84.126% (severe 85.185% and non-severe 83.333%) cases and inaccurate in 15.873% of cases. And on ROC the area under the curve came out to be 88% at a confidence interval of 95% with a sensitivity and specificity of 88.416 and 82.857% respectively. Patients with fluid deficit > 8.7L died.

Conclusion: Fluid deficit at 24 hrs can predict severe AP 85.2%. Fluid deficit > 8.7 L at 24 hours were fatal.

Keywords: *Acute Pancreatitis; Predicting Severe; 24 Hrs Fluid Deficit; Non-Necrotising; Sterile Necrosis; Infected Necrosis*

Introduction

Acute pancreatitis (AP) is a common disease which can be mild and self-limiting or severe needing prolonged hospitalization, high morbidity and even mortality [1,2]. This is due to the intrinsic Trypsinogen activation [3], due to co-localization of zymogen and lysosomal granules inside the acinar cells. This action converts Trypsinogen to Trypsin [2,4]. Trypsin increases permeability, the proteolytic enzymes start digesting the pancreatic and peripancreatic tissue leading to the inflammatory cascades and initiating autophagy [4]. The inflammation leads to fluid exudation to peri-pancreatic area and later other parts. This leads to hypovolemia, low tissue perfusion, mitochondrial malfunction and organ systems failure. This whole process can be expressed in two words "chemical burn". These effects lead to a systemic inflammatory response Syndrome (SIRS).

Thermal skin burns cause local injury, which triggers SIRS in presence of imbalance in oxidation and antioxidation process [5] as well as lipid peroxidase activation [5,6]. The increasing evidence of oxidative stress leads to multiorgan failure after major burn [7]. Thus, the relation of skin burnt injury and acute pancreatitis are same in pathophysiological terms.

IV fluid therapy is the established truth in visible skin burn. One of the simple and commonly used is Parkland formula to calculate the total intra venous (IV) fluid needed in 24 hours, i.e. percentage body surface burn × weight (kg) × 4 = volume (mL). Half this volume is given in the first 8 hours and the second half of the fluid is given in the subsequent 16 hours [8,9]. Fluid resuscitation is a key component of both burn injury [8,9] and AP [8,10]. Thus, the proposed hypothesis sounds appropriate. Only exception is, burn is visible whereas AP invisible. Surprisingly, the skin and peritoneal skin surface is same [11]. So, the reverse formulation of fluid need can give an idea of severeness of the pancreatitis. In fact, in severe AP, it involves the pancreas, retroperitoneum from diaphragm till pelvis (Image A). The way, intravenous fluid therapy is given in skin surface flame burn/chemical burn, same is not taught in acute pancreatitis as a concept. Hence this hypothesis is proposed.

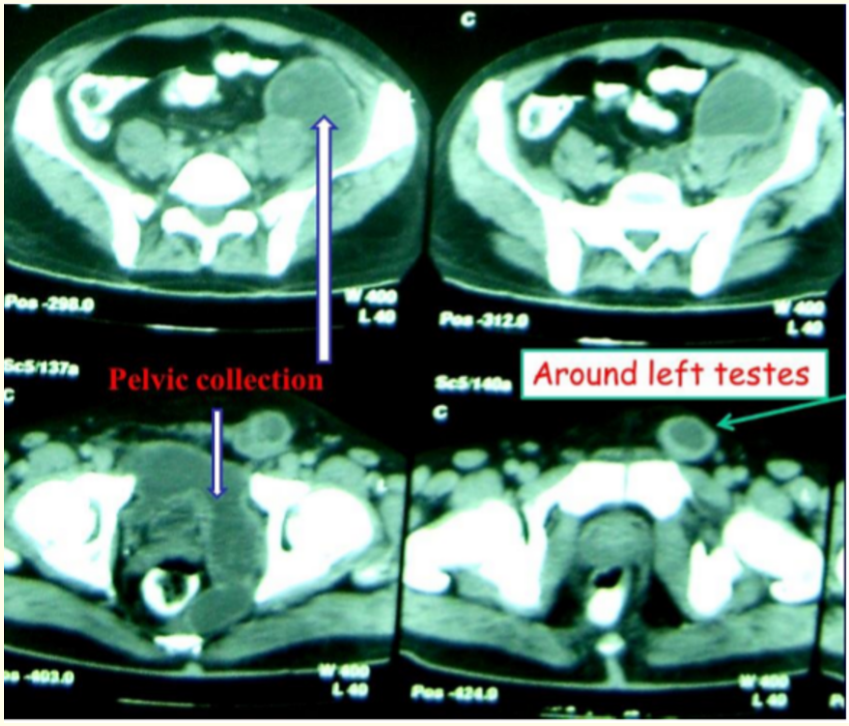

Image A

Methods

This work was done at two large military hospitals and patients were also referred from their feeder hospitals from December 2004 - August 2014. The inclusion and exclusion criteria are given below as table 1A and 1B respectively.

Inclusion criteria (1A)	Exclusion criteria (1B)
1. Age above 18 years and willing patient. 2. Clinical, biochemical acute pancreatitis. 3. Severity of pancreatitis were confirmed at CT scan, surgery/autopsy after >24 hrs of fluid management. 4. CT/autopsy confirmation of acute pancreatitis after 72hrs.	1. Age below 18 years 2. CAD and COPD (high volume IV fluid therapy may be detrimental) 3. CT scans done after 72hrs showing no AP. 4. CT scan with pancreatic calcification indicating chronic pancreatitis 5. Patients without proper fluid balance. 6. AP diagnosis laparotomy. (post-operative fluid requirement will be different) 7. Any death cases before 72hrs. 8. Cases reported after 24 hrs of onset of the disease Initial management

Table 1A and 1B: *Inclusion and exclusion criteria.*

On admission with a clinical and biochemical diagnosis of AP, weight of the patient is recorded, wide bore canulae were inserted, blood samples were collected. Oxygen inhalation was given so as to keep the saturation at 95 - 100%. Tablet Sorbitrate 2.5 mg given sublingually six hourly as the sphincter of Oddi relaxing agent (Max four dosages], [12] and a bolus of Ringer's lactate solution at the rate of 10 - 15 ml/Kg/hrs. (3 - 4L) with 2 units of heparin per ml till the passage of urine or up to 8 hours. If the patient had not passed/ no desire of passing urine by 8 hours, was catheterized. The IV fluid was adjusted, so as to get the urine output 0.5 - 1 mL/Kg/ hr. If urine output was less than 0.5 ml/kg/hr, the patient was shifted to ICU, central venous directed IV fluid was adjusted to keep the CVP 10 - 14 cm of saline. No routine prophylactic antibiotics were used. Intra-abdominal pressure was monitored with the help of urinary catheter and abdominal fasciotomy (Image B1, refined later B-2) was done with raised intra-abdominal pressure (36 - 38 mm Hg). When the saturation came down below 95%, Chest X-ray and USG of pleural cavities were done to look for effusion. Bilateral chest tubes were placed in presence of effusion so as to increase the tidal volume after removal of the pleural fluid. Patients detected to be having hyper-glycemia, were infused normal saline with insulin at a rate of 1.0 unit per every 100 mg with one hourly blood sugar to keep the blood sugar below 150 mg/dL and ketone bodies monitored to keep them on non-ketosis state. Nasogastric tube was not placed routinely, used only when there was repeated vomiting or for enteral feeding. Patients with history of alcohol abuse received 100 mg thiamine intravenously for 3 days. Other supportive therapies were used as and when necessary.

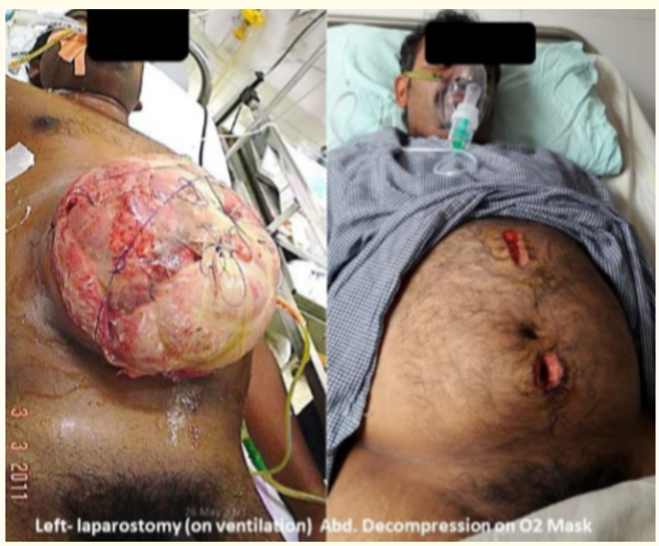

Image B: *B-1- Open laparostomy-left), [B-2- Minimum skin incision, divided linea alba and intact peritoneum- right).*

We stressed on enteral feeding by oral or tube at the earliest. Started oral water, oral rehydrating fluids (ORS), coconut water and fruit juice on day one. The correct intake (IV+ oral) and output (urine + vomitus) were measured. Insensible loss was not calculated because all were in the same environment and clinical situation, However, if the temperature was above 100°F IV fluid was adjusted accordingly. Primary and secondary outcome measures were given in the table 2A and 2B. All the patients were treated till finality including the excluded patients as control group. Computerised Tomography scan of abdomen were done only after 72hrs with haemodynamic stability and normal kidney function to see whether the necrosis and the fluid deficit were correlated. The correct intake (IV+ oral) and output (urine + vomitus) were measured. Computerised Tomography scan of abdomen were done only after 72hrs with haemodynamic stability and normal kidney function to see the necrosis and the fluid deficit data were co related [13,14]. Any deaths were recorded.

Primary outcome measures (2A)	Secondary outcome measures (2B)
(a) Finding out fluid deficit after deducting the urine output and the vomitus from the total fluid intake (IV+ oral) at the end of 24hrs and give a prediction. (b) Contrast Enhanced CT (CECT) after 72 hours (c) Evidence of necrosis at CECT, surgery/autopsy after 72 hrs.	(a) Death and any autopsy finding (b) Patient needing support in complications related to the AP and any surgery for AP patient needing supportive care

Table 2A and 2B: *Outcome measures {Primary [A] and secondary [B]}.*

The excluded group of patients were managed in the same way after arrival to the hospital, with bolus of fluid and heparin. All were receiving antibiotics, nil per oral and Ryle's tube, were stopped and oral fluids were started. All others were adapted as in the study group to keep them as control just to see the effect of late staring of fluid therapy.

Data collection and statistical analysis

Data was collected prospectively and statistical analysis was carried out using SPSS-17.0 software. Continuous variables were expressed as mean and standard deviation (SD). The Mann Whitney's U test is done for other quantitative parameters like age in severe and non-severe group. Best cut off point of the fluid deficit was found out with ROC (Graph Image C) curve with a confidence interval of 95%. Categorical data were compared using Chi square, and continuous data were compared using T-test with confidence interval (CI) of 95%. P-values less than 0.05 were considered statistically significant.

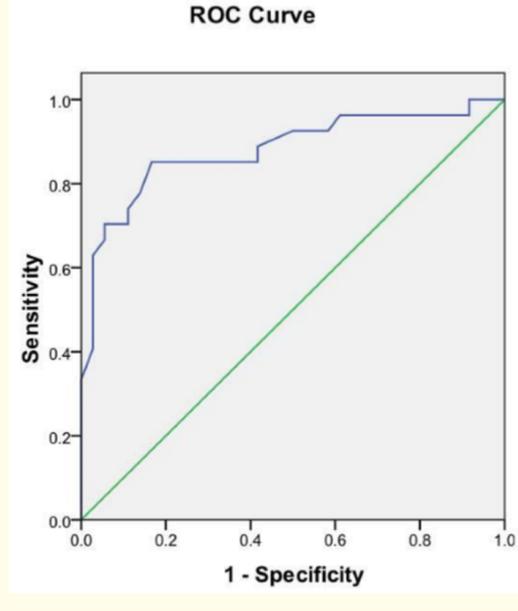

Image C: *ROC Curve.*

Results

There were 121 patients of AP. 58 were excluded as per exclusion criteria and taken as control group (Table 3) and 63 patients were included (Table 4). There were 9 females and 54 males. The fluid deficit varied from 0.4 lit to 9.9L (Table 5). As per the ROC and positive and negative predictive value (Chart Image D), the fluid deficit at 3.15L, the accurate predictions (non-severe and severe combined) came to a maximum of 84.126% (severe 85.185% and non-severe 83.333%) and inaccurate predictions were 15.873%. And on ROC the area under the curve came (Image C) out to be 88% at a confidence interval of 95% with a sensitivity and specificity of 88.416 and 82.857% respectively. There were three death and all three had fluid deficit > 8.7L. Autopsy findings confirmed the necrosis (Image E). Out of 25 necrotising AP, 17 had sterile necrosis and eight had infected. Five needed necrosectomy from the study group (Two open (Image F), two retroperitoneoscopic and one retroperitoneal drainage (Image G).

Condition of exclusion	n	Sex M: F	Aetiology	No necro	Necrosis/Sterile/Infected	
Age < 18 yrs.	02	2:0	Choledochal cyst and gall stone	02	00	00
Associated COPD and IHD	02	1:1	COPD IHD - Both biliary	02	00	00
Inadequate IV F On arrival[1]	29[4][2]	21:8	Alc.-18 Bil. -10 Alc+gall stone-1	11, 6 1	5 3 0	2 1 0
Inadequate[1] IVF+ Noradrenalin infusion[2]	21[5][2]	17:4	Alc.-16 Bil. - 4 Triglyceride1	5 1 1	7 2 0	4 1 0
Received Furosemide (Lasix) for no urine	02	2:0	Alc-2	00	02	00
Diagnosed at laparotomy- 2	02	2:0	2/0	02	00	00
Total	58	45:1 3		31 (48.82%)	19 (36.2%)	8 (18.96%)

Table 3: *Control-Excluded group.*

1: Haemoconcentration and/or elevated Blood Urea Nitrogen as indicator.

2: [x]fatality.

COPD: Chronic Obstructive Pulmonary Disease; IHD: Ischemic Heart Disease; Alc: Alcoholic; Bil: Biliary.

Factors	Non necrotising	Necrotising-severe	P value
N = 63	38	25	NS
Age range in years Mean SD	19-59 38.83SD +/- 13.547	21-57 41.07SD +/- 12.939	NS
F:M	7:31	4:21	NS
Range of fluid deficit	0.4 - 4.46L	1.0 - 9.9L	
Mean, SD of FD in L	2.28 +/- 0.996	4.688 +/- 19818	P < 0.0001
Alcohol	21 (60%)	12 (44.4%)	< 0.005
Biliary	7	10	NS
Idiopathic	5	4	NS
Hyper triglyceride	2	1	NS
Parathyroid adenoma	1	0	x
Necrosectomy	0	05	XX<05

Table 4: *Study group.*

SD: Standard Deviation; FD: Fluid Deficit.

Sr. No.	Study	Excluded	P value
Total cases	63	58	Not significant
Age	19 - 59	6 - 72	do
Sex M: F	52:11	45:13	do
Non-Necrotising	38 (60.32%)	31 (53.45%)	do
Necrotising	25 (39.68%)	27 (46.55%)	
Death	03 (4.7%)	9 (15.5%)	< 0.001
Sterile necrosis	17	19	
Infected necrosis	8	8	
Open necrosectomy	2	1	
Retro-peritoneoscopy	2	2	
Percutaneous tube drain	1	5	

Table 5: *Comparative table of fatality and necrotising pancreatitis).*

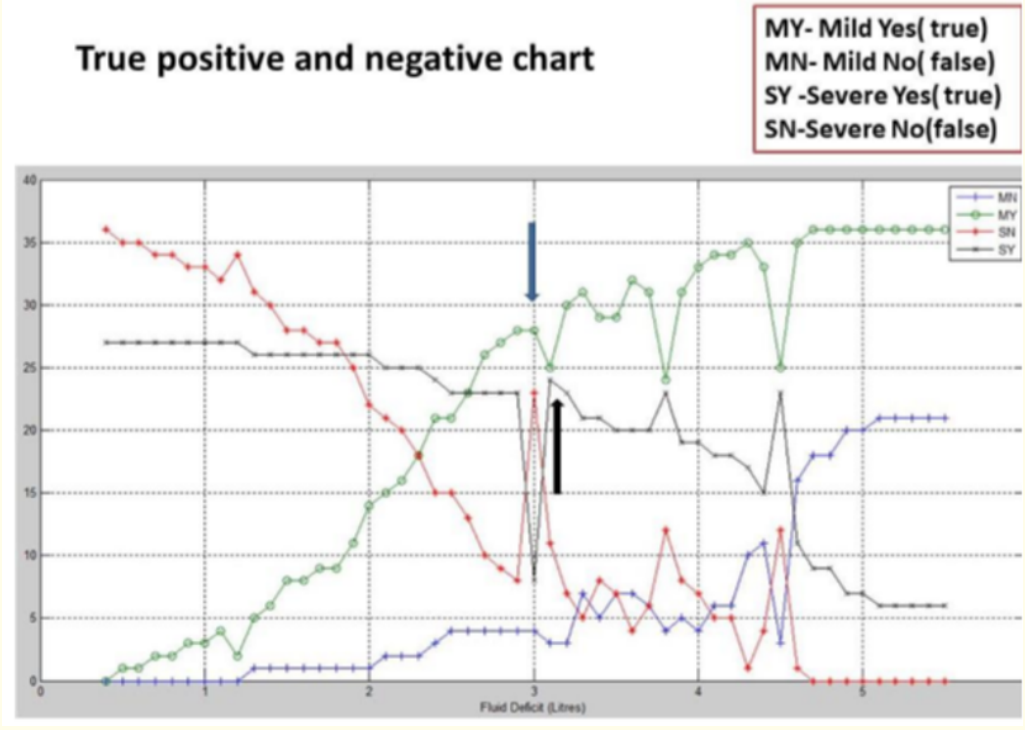

Image D: *True and false positive and negative curve.*

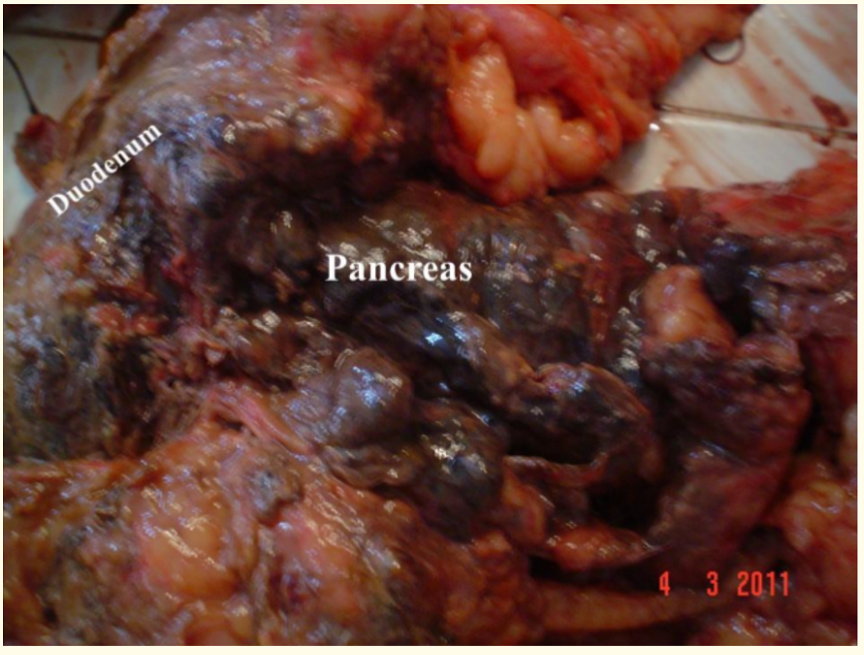

Image E: *Necrosis of duodenum and pancreas.*

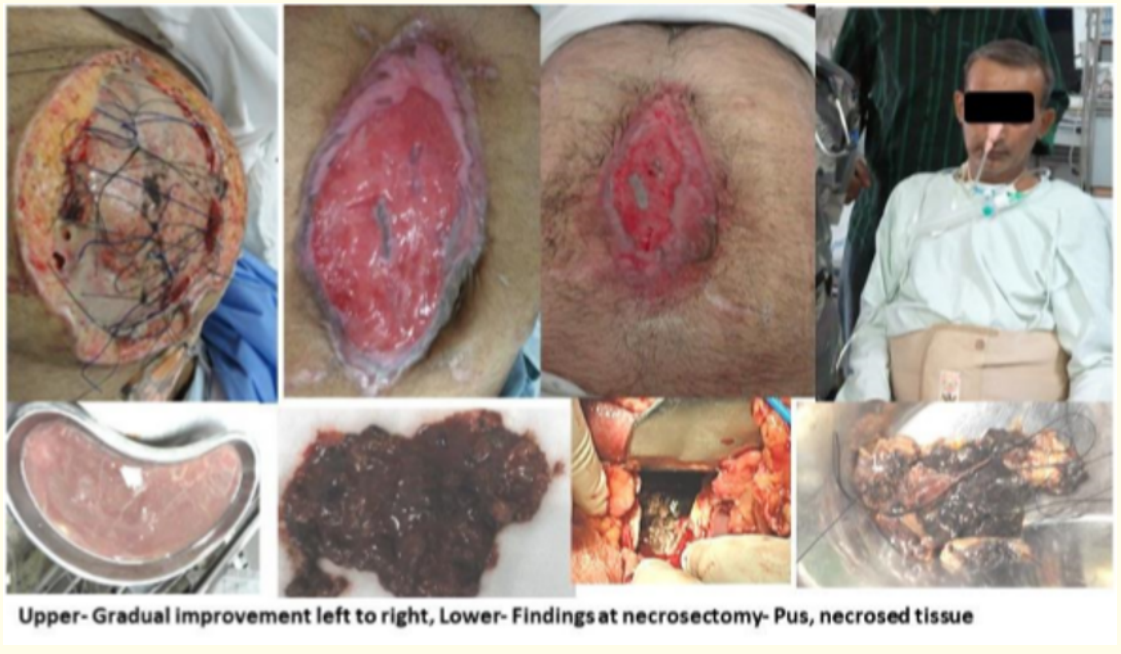

Image F

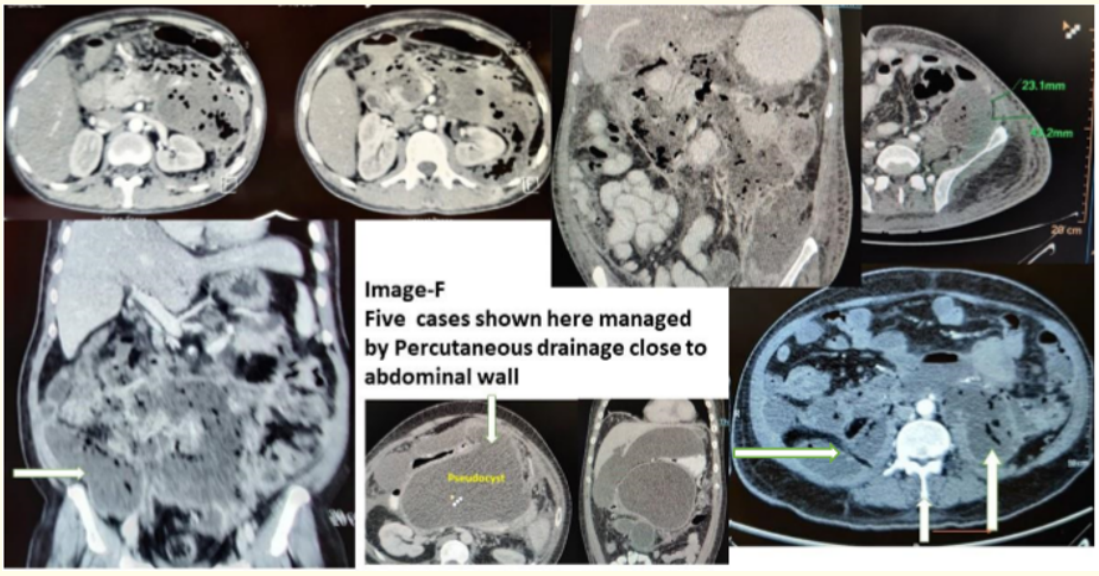

Image G: *Percutaneous drainage shown by arrow and distance in one image.*

In excluded (control) group there were nine fatality and three retroperitoneoscopic necrosectomy and five cases percutaneous tube drainage was done. After that once they were afebrile, on oral diet, ambulant and willing to go home, were discharged. Initially were on SOS review. The aetiological factors evaluated and removed or counselled if not possible to remove.

Discussion

Acute pancreatitis (AP) is a common inflammatory disease. Alcohol abuse and biliary stones are common causes [1,2,15]. It is known that activation of pancreatic proteoses, initiates the process of auto digestion causing a chemical burnt injury at and around pancreas [3]. Patho-physiologically AP is divided into two phases, early (initial two weeks), later phase (> two weeks) [15]. Sever AP leads to morbidity and mortality [16] and hence, after the diagnosis of AP is made based on clinical and laboratory pancreatic enzymes test, no further serum pancreatic tests are needed [17]. Early identification of patients at risk for severe AP is crucial by looking at the inflammatory markers [18]. Hydration is key in AP, due to inadequate intake with associated vomiting, the inflammatory response related fluid shift leading to dehydration and haemoconcentration [19] Inadequate hydration is directly related to system failure. This is because of exudation of fluid from the vascular space to the interstitium and third space. Initially, it occurs around the inflamed pancreas. This causes selective haemoconcentration of pancreatic micro vascular circulation and other organs later due to SIRS and become a systemic feature. Thus haemoconcentration, elevated Blood Urea Nitrogen (BUN) are the markers of degree of fluid deficit. Therefore, patients with elevation of these two parameters indicate urgency of fluid therapy during the early management [20]. Stressing on the intra-venous fluid therapy (IVFT), an editorial has written "Early fluid resuscitation in acute pancreatitis: a lot more than just fluids" [21]. During this initial 72 hours, besides the hydration with Ringer's Lactate (RL), enteral feeding is also an integral part of the therapy [19]. In the present study, hydration by RL and enteral feeding were given their due importance. Even though colloid have found to be better for longer retention capability in the vascular compartment [22], the need of the early diffusion to the tissue is not met with colloid. We used crystalloid only,

because of its easy availability and lower cost. It is useful in maintaining tone in vascular, interstitial, third space and diluting inflammatory chemicals. But, in view of diffusion into three compartments, the volume of fluid required is higher. Normal saline (NS) and Ringer's lactate (RL) are two easily available fluids. We have used these two fluids for initial fluid therapy. RL is found to reduce the inflammatory response in comparison to NS [23]. We used RL universally and only changed to NS if the patient was hyperglycaemic. There is no consensus on the volume and rate of infusion of fluid. We have followed fluid therapy in burn as per Parkland's formula so as to compensate the initial deficiency, from the onset till arrival to the hospital and the ongoing chemical burn during the initial period, at the rate of 10 - 15 mL/ Kg /hr till the urine output increases to 0.5 - 1 mL/Kg /hr or eight hours, whichever is lesser. It falls in aggressive fluid therapy as per a Chinese study [24]. One study has used 20 ml/kg, as initial bolus like our study [25]. In fact, our patients received bolus therapy for eight hours or lesser time, till good urinary flow, which is as per our burn hypothesis, to cater for the time gap between the onset to hospital entry. It has been pointed out that there are equal number of studies in favour of aggressive and non-aggressive fluid resuscitation [26]. Our fluid management fall on the middle of aggressive and non-aggressive. We monitored only hourly urine output for the state of hydration and daily renal parameter and electrolytes. Adequate IVFT improves micro circulation, possibly avoiding necrosis of pancreas and system failure. When IVFT is ongoing, there is fluid loss to the third space like retro-peritoneum, peritoneum and pleura. The former two will raise the intraperitoneal pressure. Hence, periodic intra-abdominal pressure monitoring must go with fluid resuscitation for early detection of abdominal compartment syndrome [27]. Three of our patients had to undergo abdominal decompression (Image B1) from the study group and two from the excluded group. We modified, the procedure from laparostomy to only two skin cuts for cutting the linea alba, only leaving the intact peritoneum and with only two small skin incisions (Image B2). When IVFT is given, it will get distributed all over the body. Part of the fluid gets retained based on the degree of stress response and the balance of fluid comes out through the kidney as per the present hypothesis. And the intake-output difference gives us the severity. Obviously, more urine output is present in less stress and less urine with severe stress. Our hypothesis was accurate and fluid deficit was significantly higher ($p < 001$) in severe (necrotising) AP compared to the mild (non-necrotising) AP. Most severe AP had the highest deficiency and fluid deficit > 8.7 were fatal. The cut off level was found to be 3.15 L between mild and severe, as assessed by positive and negative prediction value charts (Image D), where both join after 3 L and from ROC.

At times the patient comes in a state of haemoconcentration and pre-renal azotaemia indicating inadequate fluid therapy (Table 3, excluded group). Few received nor-adrenaline infusion to raise the blood pressure and even injection Furosemide for poor urine output. This bolus fluid becomes useful for some patients for correction of hypo-voluemia and for recovery of pre-renal azotaemia. If the dehydration phase persists or fluids were given at a slow pace for a long period, which Wu Bu., *et al*. used blood urea nitrogen to define fluid unresponsiveness after 8 - 12 hrs [23], fluids can be administered carefully to avoid fluid overload and fluid restriction and dialysis may be needed. Fatality is most likely, in a situation, where IVFT is needed but can-not be given due to renal failure. All the early fatalities were from this group due to MOS.

When IVFT is inadequate, there is haemoconcentration and concentrated blood with little plasma, leads to slow flow state; more so in small vessels leading to Disseminated Intravascular coagulation (DIC) [28,29]. Intimal damage takes place due to stress response and vasoconstriction seen in such situations and fits well to the Virchow's triad (low flow state, haemoconcentration, hypercoagulability and intimal damage) as well and multi organ failure is initiated. In such situations endothelial protector is recommended in the early period. We have used Heparin 2 units/mL from the first bottle of IVFT for the same. In addition, Heparin improves the microcirculation further due to its known anti-thrombotic property. Initially, there was a term haemorrhagic AP which made the use of heparin in AP controversial. Heparin and Insulin is found to activate lipoprotein lipase and thus reduces the triglyceride level and hence, is used in AP due to hypertriglyceridemia [30]. Others have used plasmapheresis and hemofiltration, besides insulin and heparin infusion during an acute attack [31]. In an RCT on LMWH no haemorrhagic complications were observed and mortality was not significantly lower than the non- heparin groups ($p = 0.056$). Low molecular weight heparin treatment is safe and provides better prognosis in moderate severe

AP [32]. In a meta-analysis, LMWH was noted to have improved the prognosis of severe AP. It was also found that the heparin group had lower mortality, organ failures, pancreatic and peripancreatic complications needing surgery, and lower duration of hospital stay [33]. Initially, pseudo-aneurism bleeding was over exaggerated, whose incidence is 0.7% with anecdotal mortality. Once fear is reduced and understanding of necrosis is understood to be due to small vessel thrombosis in the pancreas, low molecular-weight heparin is found to have lower incidence of necrosis [34].

Our basic aim of using heparin is multifactorial. They were to keep the canula and vein patent for a longer duration by avoiding thrombophlebitis, faster hydration, besides preventing thrombosis of pancreatic vessels. We have used regular heparin in intravenous drip during bolus fluid as well as all the fluids. We have not used heparin in the group of inadequate IVF patients with a view to keep them as control.

Any surgical intervention during the inflammatory phase should be avoided [19]. In the early phase, IL-6, TNF alfa and other inflammatory parameters are elevated and any surgical intervention leads to high mortality. A study reported 22 of 46 patients died, when IL-6 was > 1512 pg/mL (normal < 10 pg/mL) [35].

During this initial 72 hours, besides the hydration with Ringer's Lactate (RL), enteral feeding is also an integral part of the therapy [19]. The need of caloric increases in stress and disease. AP an inflammatory condition, causes protein catabolism and need more calorie for increased metabolic rate [36]. Enteral feeding is physiological. Intake of food initiate the peristalsis to propagate distally. The peristalsis helps absorption of food and prevent bacterial transmural translocation [37]. These bacteria cause sepsis of the necrosed pancreatitis. Given orally, nasogastric, jejunostomy are the same as all goes to the bowel. Only issue is vomiting due to duodenal ileus which surrounding the pancreas. That is why, patients having frequent vomiting a tube in jejunum serves the aim. In this study, we gave the enteral feeding orally, through nasogastric tube or a naso-jejunal tube. When a naso-jejunal tube is being placed, same sitting biliary pancreatitis were delt with. Nutrition could be started as early as possible. We have given, water/ORS in thirsty patient to add to hydration. Best practices in acute pancreatitis management focus on triage, hydration and enteral feeding [19]. All these give nutrition and correct dehydration. Many questions the timing. Few have started with in 24 hrs [36] and other only after 48 hours and found effective even in severe acute pancreatitis (SAP) [38]. Others wanted to give on day today basis on the need [39]. Severe Acute pancreatitis are at nutritional risk. Adequate nutrition lowers the mortality and septic necrosis [40]. We started with water, oral rehydration solution with little more of sugar to have protein sparing effect. Later started milk and supplement of protein. The patients with open abdomen after necrosectomy gave parenteral nutrition with immunogenic amino acids till out of pressure agents for 5 days. Giving only crystalloid IV make the patients nutritionally depleted and die of hunger. But starting early enteral feeding, reduces hospital stay and decreases the mortality in biliary AP [40,41].

AP is not a bacterial disease. So, no antibiotic will cure AP. But the usage of antibiotics in AP has been ongoing as a prophylaxis to prevent the infection [36]. The duration of the antibiotic prophylaxis was variable. With change of time, opinions also keep changing. Next indication was to prevent/reduce the infection of necrosed pancreas [43,44]. By 2010, the conclusions changed towards preventing mortality from pancreatic necrosis with the use of imipenem [45]. The present knowledge is, there is no requirement of antibiotics in early phase of the disease [16]. Recently a Chinese study has reported multidrug resistant organism in severe acute pancreatitis (SAP) with septic necrosis [46]. The only indication of antibiotics is infected necrosis, after culture directed antimicrobial therapy [16]. We have been following this practice. We have seen multidrug resistance *Klebsiella* in a transferred patient. One such patient transferred after amputation of the left leg (Image H). It was resistant to all available antibiotics. We used 1% acetic acid irrigation through all the tubes as acidic pH is inimical to most of the organism and acetic acid is physiological and removed all drain and started oral food. Fortunately, this elderly lady survived.

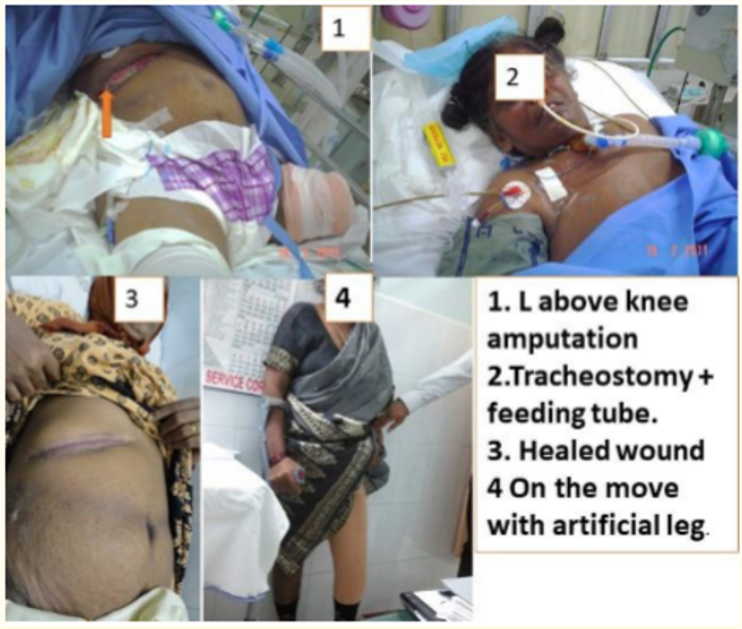

Image H

We have not used any antibiotics in the early phase nor done any needle aspiration from the necrosed pancreas with a fear of giving the infection to sterile necrosis and puncturing the bowel, which can carry bowel organism to sterile necrosis. We decided the necrosectomy clinically and on a contrast enhanced CT scan [47].

Mortality in AP is bimodal, early death is due to severe cytokines storm with most severe body response leading to SIRS and multisystem organ failure (MOF) [17]. The second phase is due to infected necrosis caused by septicaemia [49]. One Indian study has found 50% death rate in each both the phases [48]. The overall death rate was reported to be 4.8% (55 of 1135) and death from severe AP was 13.5%. 50.9% occurred in the first two weeks [49].

We had three of 63 (4.76%) from the study group and nine of 58 (15.51%) deaths from the excluded group, but still lesser then reported [35]. Death rate is higher in the excluded group, mostly due to delays in their referral. All death were in the early phase. All deaths from the study group happened from 5 - 10 days, all three had fluid deficit > 8.7L at 24 hrs, hyper-glycemia, raised intra-abdominal pressure needing decompression, ventilatory support and renal compromise. The autopsy finding (Image F) show necrosis of the whole pancreas, second and third part of duodenum, part of transverse colon and greater omentum. Fortunately, we had no mortality after necrosectomy. It is possibly due to small number of cases. No routine antibiotics possibly avoided multidrug resistance bacteria including our minimal approach of retroperitoneoscopic necrosectomy. In fact, at the later part, we started draining the retroperitoneal pus under local anaesthesia, when presenting close to abdominal wall (Image G). We used a Ryle's tube, easily available in various sizes, wide bore in relation to outer diameter and multiple holes. We perceived a concept that retro-peritoneum is a single space and one drain is sufficient to drain the pus. Patient position was changed to keep the drain location more dependant. Once the tract is formed, the necrosed tissue came out slowly after removal of the drain. However, we had morbidity after open necrosectomy with open abdomen and a prolonged hospital stay

(Image F). The low mortality can be explained by adequate hydration, early oxygen therapy, use of heparin and early institution enteral nutrition either by oral/tube feeding.

Conclusion

Burnt injury like fluid therapy in AP predicted severe and non-severe correctly 85.2% and 83.33% AP respectively at 24hrs. Fluid deficit > 8.7L leads to fatality with organ necrosis. Possibly no early antibiotics, with use of oxygen inhalation, heparin, enteral feeding, and retroperitoneal necrosectomy can prevent second phase death. Further study is needed.

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