Severe acute pancreatitis- it's diagnosis and management- A study of 20 cases

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Abstract

Background: Severe acute pancreatitis is defined as pancreatitis in which there is persistent organ failure that does not resolve within 48 hours. Severe acute Pancreatitis is characterized by pancreatic necrosis, a severe systemic inflammatory response and often multiorgan failure. Severe acute pancreatitis is a serious and life threatening disease. Mortality varies from 20 to 50 percent.

Objective: The objectives of this study are to develop our knowledge about presentation and diagnosis of severe acute pancreatitis, and to develop a standard management protocol to rescue that patient suffering from severe acute pancreatitis.

Methods: This observational study was carried out in Combined Military Hospital (CMH) Dhaka, during the period of August 2014 - Feb 2015. A total 20 patients of severe acute pancreatitis were studied prospectively, evaluated and managed.

Results: In this study, out of 20 patients 12(60%) male and 08(40%) female. Male: Female = 3:2. The youngest patient of this series was 03 years and oldest was of 55 years. First categorization of severity of acute pancreatitis was done on the basis of Ranson score. Those patients whose score is 3 or more are categorized as severe. After categorization subsequent management was planned on the basis of laboratory and CT findings. Out of 20 patients all have raised WBC, serum Calcium level decrease in 16 patients, LDH raised in 16 patients, PaO_2 decrease in 14 patients, Base deficitincreased in 12 patients, and blood urea nitrate raised in 14 patients. Contrast enhanced CT scan done in all patients and 12 patients were found with reduced enhancement in pancreas, peripancreatic edema and stranding of fatty tissue and remaining 8 patients have fluid collected in peri- and retro pancreatic space. Total 12 patients were given conservative treatment. Remaining 8 patients were operated whose CT findings were reduced enhancement in pancreas and these patients were suspected for infective pancreatic necrosis. In this study 3 patients were expired. Out of these three patients, 2 patients underwent operative intervention and 1 patient was given conservative treatment.

Conclusion: Severe acute pancreatitis is a life threatening condition. Its serious regional and systemic involvement causes multiple organ or system failure. Early diagnosis and effective treatment can significantly reduce the mortality and morbidity.

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Key words: Acute Pancreatitis, Clinical pattern and management.

Introduction

Acute pancreatitisis defined as an acute condition presenting with abdominal pain and is usually associated with raised pancreatic enzyme levels in the blood or urine as a result of pancreatic inflammation. Acute pancreatitis may be categorized as mild or severe.[1] Severe acute Pancreatitis is characterized by pancreatic necrosis,a severe systemic inflammatory response and often multiorgan failure.In those who have a severe attack of pancreatitis, the mortality

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varies from 20 to 50 percent.[1] Severe acute pancreatitis is associated with extensive and prolonged pancreatic and retroperitoneal inflammation with patchy or generalized areas of tissue necrosis and haemorrhage in and around the pancreas. The course can arbitrarily be divided into two successive phases(early toxaemic and late necrotic).[2]

It is important to identify patients with severe acute pancreatitis early. Ranson developed 11 objective criteria and postulated that the risk of death and /or developing major complications may be estimated objectively by five parameters on admission to hospital and six parameters within the initial 48 hours of admission. Imrie proposed the Glasgow criteria based on eight of Ranson's factors. APACHE II system is currently the best and easiest scoring system available to assess the severity of acute pancreatitis and can be used at any time in the course of the disease.[3]

In 1998 the British Society of Gastroenterology (BSG) published UK guidelines on the management of acute pancreatitis.[4] A planned revision after two years was anticipated. The BSG as the prime sponsor of these guidelines initiated a review process in 2000. Recently, a working party of the World Association of Gastroenterology has produced a further set of guidelines for the management of acute pancreatitis[5,7] and the International Association of Pancreatology has published guidelines on the surgical management of acute pancreatitis.[6]

The first stage of severe acute pancreatitis, the period within 2 weeks after the onset of the disease, is characterized by the systemic inflammatory response syndrome (SIRS), and pancreatic necrosis develops in parallel with that within the first 4 days after onset. The second stage begins 2 or 3 weeks after onset with the development of infectious pancreatic complications such as infected pancreatic necrosis (bacterial infection of the pancreatic necrosis) and pancreatic abscess. Infection of pancreatic necrosis is a major prognostic risk factor in severe pancreatitis, and sepsis-related multiple organ failure is the main life-threatening complication with a mortality rate of 20%-50%.[8]

In recent years, treatment of severe acute pancreatitis has shifted away from early surgical treatment to aggressive intensive care. While the treatment is conservative in the early phase, surgery might be considered in the later phase of the disease. Surgical debridement is still the "gold standard" for treatment of infected pancreatic and peripancreatic necrosis.[9] surgical mortality rates in necrotizing pancreatitis have been dramatically reduced, from 60% to 80% in the early 20th century, to the 10% to 20% rates commonly reported today.[10]

Advances in radiological imaging, new developments in interventional radiology, and other minimal access interventions have revolutionized the management of many surgical conditions over the past decades.[9] The study will be designed with a view to have a glimpse to evaluate the patients with severe acute pancreatitis in regards to the mode of presentation, clinical assessment, method of diagnosis, nature of intervention and ultimate outcome of the patients treated subsequently.

Materials and Methods

Study design

Observational study

Place of study

Department of Surgery, Combined Military Hospital, Dhaka Cantonment. Study period August 2014 - Feb 2015.

Study population

People suffering from acute pancreatitis.

Sample definition

Patient with acute pancreatitis with minimum score on the basis of Ranson criteria. Minimum score 3.

Sample collection

All consecutive patient diagnosed as severe acute pancreatitis with minimum Ranson score 3.

Inclusion criteria

Patients diagnosed sever acute pancreatitis by clinical sign, laboratory investigations and imaging study (Ranson criteria).

Exclusion criteria's

- Patient with acute pancreatitis but Ranson score <3.
- Replacing acute pancreatitis
- Patient with acute on chronic pancreatitis.
- Patient with preexisting organ failure
- Any life threatening co-morbidity
- Patient those who are unwilling for the study

Procedures of preparing and organizing materials

Microsoft Office word 2007 /excel

Research Instruments

a. Interview schedule.
b. Predesigned Data Sheet.

Procedures of collecting data

By standard questionnaire.

Procedure of data analysis & interpretation

Data will be presented as percentage of total observation using SPSS version-15. Data presentation will be Table and Graph.

Observation and results

Total 20 consecutive cases of severe acute pancreatitis were analyzed. All patients were managed initially with conservative treatment. Out of 20 patients total 8 patients underwent operative intervention and 12 patients underwent conservative treatment. All patients were managed in Critical Care Centre which was recently established in CMH Dhaka with all modern equipments and technology. All the patients were managed under close supervision of doctors, nurses and paramedics and they were followed up for 06 months. In the present series, following observations were noted.

Age and Sex:

Age: The youngest patient of this series was 03 years and oldest was of 55 years.

Sex: Total patient: 20
Male: 12
Female: 08
Male : Female = 3:2

Table 2.5 : Age and Sex distribution

Age	Male	Female	Total
<20	01	01	02
20-30	02	02	04
31-40	05	03	08
41-50	03	01	04
51 years, & above	01	01	02
Age range	03 to 55	18 to 52	-
Mean age	36	34	-

Comment: Maximum number of the patients fall into 31-40 years. The mean age of male and female of this series were 36 and 34 years respectively. According to international study the disease may occur at any age, with a peak in young man and older women.

Clinical Presentation:

1. Pain: All patients presented with abdominal pain, specially diffuse abdominal pain. Sudden onset of severe abdominal pain in 16 patients (80%) and diffuse abdominal pain in 4 patients (20%). Pain radiated towards back in 11 patients (55%) and in 09 patients (45%), there was no radiation.

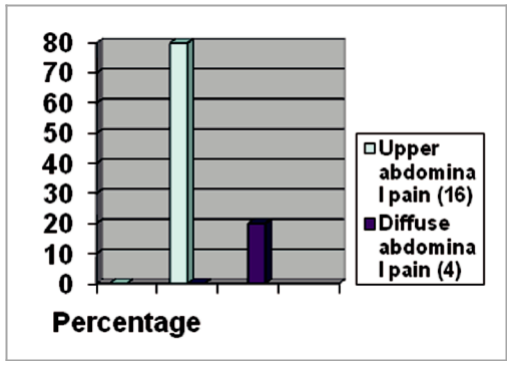

Figure 2.9: Site of Pain (Figure within bracket indicates no of cases)

2. Abdominal distention: 15 patients (75%) presented with abdominal distention.

3. Fever: 11 patients (55%) presented with fever

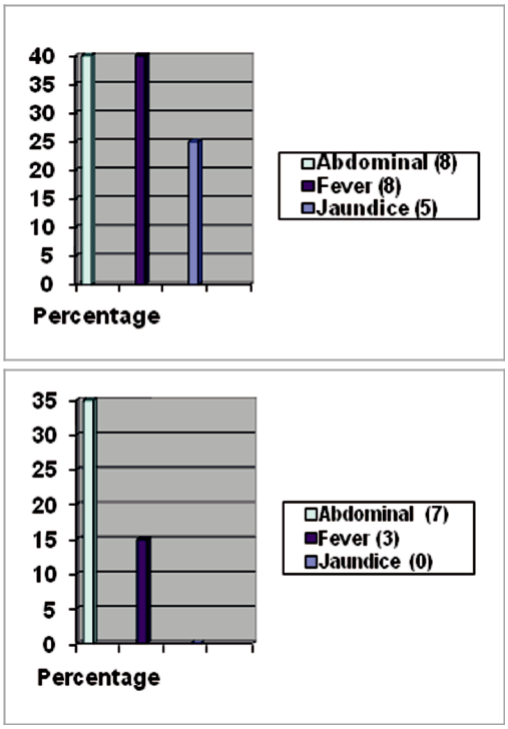

Figure 2.10 : Clinical presentation(Figure within bracket indicates no of case)

4. Associated symptoms: All patients of severe acute pancreatitis complains of anorexia (100%). 17 patients presented with nausea and vomiting (85%).

Physical signs:

All patients had abdominal tenderness. 16 patients had diffuse abdominal tenderness (80%) and 4 patients had upper abdominal tenderness (20%). Fluid thrill and shifting dullness were present in 08 patients (40%). Another 10 patients had features of paralytic ileus (50).

15 patients had (75%) pleural effusion, out of them ten patients had left sided and five patients had bilateral pleural effusion. Some common findings were present. Such as - tachycardia and tachypnoea. Jaundice was present in 5 patients (25%).

Table 2.7: Physical signs (Total no 20)

Signs	Medical management (12)	Surgical management (8)
Abdominal tenderness	12(60%)	8(40%)
Paralytic ileus	2(10%)	8(40%)
Ascites	1(5%)	7(35%)
Fever	5(25%)	6(30%)
Jaundice.	0%	5(25%)
Pleural effusion	7(35%)	8(40%)

Investigations

Investigations were aimed at confirming the diagnosis. Available investigations were done in most of the cases.

1) Laboratory investigations:

Laboratory test revealed leucocytosis and high serum and urinary amylase found in all patients. Abnormal liver function test in 05 patients (25%), C Reactive Protein (CRP) was high in all patients. Blood culture was done in every patients. Positive blood culture found in 3 patients(15%). Serum Calcium level was decreased in 09 patients(36%). Other tests were carried out including complete blood count, renal function test, blood group, blood sugar, chest X Ray, ECG, etc.

2) Radiological investigation

All the patients were investigated with

1. Plain X Ray abdomen
2. USG of whole abdomen
3. Contrast C.T. Scan of abdomen
4. ERCP

Table 2.9: Findings of Radiological study (Total no 20)

Name of the Investigations	Findings	Medical management(12)	Medical Management (12)
1. Plain X- Ray abdomen	Abnormal findings were present in 18 films	12 (60%)	6 (30%)
	a) Isolated dilated gas filled loop of Jejunum is seen (sentinel loop)	8 (40%)	2 (10%)
	b) Isolated gas filled duodenum is seen.	5 (25%)	0%
	c) Gas filled hepatic and splenic flexures are separated by gas less transverse colon (colon cut off sign)	2 (10%)	1 (5%)
2. X-Ray Chest (P/A view)	a) Left sided pleural effusion.	7 (35%)	1 (5%)
	b) Bilateral pleural effusion.	1 (5%)	4 (20%)
3. Contrast enhanced CT Scan done in all patients	Reduced enhancement in pancreas. Peripancreatic oedema Stranding of fatty tissue	0%	8 (40%)
	Fluid collected in peri- and retro pancreatic space.	12 (60%)	0%
	CT scan revealed gall stone.	0%	5%
4. ERCP Done in 05 patients	Dilated CBD	0%	3 (15%)

Management:

All patients were managed in critical care centre in CMH Dhaka. First categorization of severity of acute pancreatitis was done on the basis of Ranson score. Those patients whose score is 3 or more are categorized as severe. After categorization subsequent management was planned on the basis of laboratory and CT findings. Initially all the patients were managed with conservative treatments. 8 patients were operated whose CT findings were reduced enhancement in pancreas and these patients were suspected for infective pancreatic necrosis. Remaining 12 patients whose were not suspected for infective pancreatic necrosis were given conservative treatment. Analgesic, aggressive fluid rehydration, oxygenation, nasogastric drainage were given to all the patients. 5 patients were kept in artificial ventilation due to acute respiratory distress syndrome. Antibiotic given to all patients. Initially patients were kept for brief period of NPO. Eight patients given enteral feeding by nasogastric tube and 12 patients were started with parenteral feeding. Gradually parenteral feeding was switched to enteral feeding. Eight patients were underwent operative intervention. Exploratory laparotomy with multiple drainage for 5 patients and exploratory laparotomy, necrosectomy and multiple drainage for 3 patients. One patient operated on the 2nd of admission and another 7 patients were operated after 3-4 weeks of conservative treatments. One patient died on 1st post-operative day and another patient died on 2nd post-operative day. Another patient died on 5th day of admission who was under conservative treatment. 01 patient was reoperated twice due to postoperative abdominal distention. 3 patients underwent cholecystectomy during exploratory laparotomy. Hospital stay was from 3 weeks to 2 months. Average hospital stay was 3 weeks. Total 03 patients were expired despite all efforts.

Table 2.11: Management (total no 20)

Option	No. of patient
Medical management	12 (60%)
Surgical management	8 (40%)

Table 2.12: Outcome of surgical and non-surgical group (total no 20)

Outcome	Surgical group (8)	Non-surgical group (12)
Death	2 (10%)	1 (5%)
Residual abscess	1 (5%)	2 (10%)
Burst abdomen	2 (10%)	0%
Fistula	2 (10%)	1 (5%)

Complications

15 patients had pleural effusion, 05 patients had acute respiratory distress syndrome, 03 patients developed septicaemia, 01 patient develops DIC and 03 patients developed renal failure. Acute fluid collection in 05 patients, pancreatic necrosis 12 patients, pancreatic pseudocyst formation occurred in 03 patients.

Table 2.13: Systemic complications

Complications	Surgical group (8)	Medical group (12)
Pleural effusion	8 (40%)	7 (35%)
ARDS	5 (25%)	0%
Septicaemia	3 (15%)	0%
DIC	1 (5%)	0%
Renal failure	3 (15%)	0%

Table 2.14 : Local complications

Complications	Surgical group (8)	Medical group (12)
Pancreatic necrosis (Abscess)	8 (40%)	4 (20%)
Pseudo cyst formation	3 (15%)	0%

Discussion

This observational study has been carried out in the department of surgery Combined Military Hospital, Dhaka cantonment during study period August 2014 - Feb 2015.

Acute pancreatitis can affect all age groups, although it is rare in children. The mean age at presentation is usually in the sixth decade.[28] The male / female ratio is close to unity or shows a slight female preponderance.[11]

In this series (table no 2.6) youngest patient of this series was 03 years and oldest was of 55 years. Maximum number of the patients fall into 31-40 years. The mean age of male and female of this series were 36 and 34 years respectively. In my study 12 (60%) male and 08 (40%) female.
Male:Female = 3:2.

Pain is the cardinal symptom. It characteristically develops quickly, reaching maximum intensity within minutes rather than hours and persists for hours or even days. The pain is frequently severe, constant and refractory to the usual doses of analgesics. Pain is usually experienced first in the epigastrium but may be localized to either upper quadrant or felt diffusely throughout the abdomen. There is radiation to the back in about 50 per cent of patients, and some patients may gain relief by sitting or leaning forward.[1]

In this series (Figure 2.10 and 2.11) all patients had pain abdomen. 16 (80%) patients out of 20 patients had upper abdominal pain and 4 (20%) patients had diffuse abdominal pain.15 patients presented with abdominal distension. 11 patients presented with fever (55%). Jaundice was present in 05 patients (25%).

Nausea, repeated vomiting and retching are usually marked accompaniments. The retching may persist despite the stomach being kept empty by nasogastric aspiration. Hiccoughs can be troublesome and may be due to gastric distension or irritation of the diaphragm. On examination, the appearance may be that of a patient who is well or, at the other extreme, one who is gravely ill with profound shock, toxicity and confusion. Tachypnoea is common, tachycardia is usual, and hypotension may be present. The body temperature is often normal or even subnormal, but frequently rises as inflammation develops. Mild icterus can be caused by biliary obstruction in gallstone pancreatitis, and an acute swinging pyrexia suggests cholangitis.[1] In my study (table no 2.7) medical group presented with anorexia 12 (60%), nausea and vomiting 9 (45%), toxicity and confusion 12 (60%), tachypnoea, tachycardia 12 (60%) and hypotention 6 (30%) and in surgical group anorexia 8 (40%), nausea and vomiting 8 (20%), toxicity and confusion 8 (40%) , tachypnoea, tachycardia 8 (40%) and hypotention 8 (40%). In this series (table 2.8) on examination medical group has abdominal tenderness 12 (60%) , paralytic ileus 2 (10%), ascites 1 (5%), fever 5 (25%), jaundice 0%, pleural effusion 7 (35%) and surgical group has abdominal tenderness 8 (40%), paralytic ileus 8 (40%), ascites 7 (35%), fever 6 (30%), jaundice 5 (25%), pleural effusion 8 (40%).

The diagnosis of acute pancreatitis is traditionally based on clinical picture with some laboratory investigation. The serum amylase concentration rises to more than 2½ times normal within 6 hours after the onset of an acute episode. It generally remains elevated for several days. Many abdominal and non-abdominal conditions

can be associated with hyperamylasemia. In general, the serum amylase elevation in such cases is less marked than in acute pancreatitis. Serum amylase level in excess of 1000 IU/dl occur early in the attack in 95% of patients with biliary pancreatitis and 85% of patient with alcoholic pancreatitis.[12] The serum amylase level is insensitive in three uncommon situations: in delayed clinical presentation, because the serum amylase normalizes after several days of pancreatitis; in pancreatitis resulting from hyper triglyceridemia, which typically produces minimally or mildly elevated serum amylase levels, possibly because of the dilutional effects of the lipemia; and in acute-on-chronic alcoholic pancreatitis in which the amylase level rises only modestly because of pre-existing pancreatic injury.[13] CRP is an acute phase protein, synthesized by hepatocytes.

CRP raised in many condition eg. major trauma, sepsis, acute panecreatitis. Serum level rise within hours and usually peak after 24 - 48 hours. Serum calcium levels fall in about 25% of cases, and that signifies severe pancreatitis. When true hypocalcaemia does occur, it is not simply due to deposition of calcium in the calcium soaps of fat necrosis. Factor implicated include hyperglucagonemia, calcitonin secration and reduced levels of circulating parathormone.[14] Multiple factor prognostic scoring systems have proved valuable. Some important scoring system are Ranson system, Glasgow systems, APACHE ll score. C-reactive protein (CRP) levels have emerged as a reasonably reliable marker for the development of necrosis.[14]

In this series (table 2.9) medical group is found with leucocytosis 12 (60%), high serum and urinary amylase 10 (50%), high serum lipase level 10 (50%), abnormal liver function test 0%, raised CRP 12 (40%), blood culture 0%, decrease serum Calcium 8 (40%), normal serum amylase 0, LDH >700 unit/L 8(40%), PaO_2 < 60mmHg 6 (30%), base deficit >4mmol/L 6 (30%), blood urea nitrogen rise >5 mg% 6 (30%) and in surgical group leucocytosis 8 (40%), high serum and urinary amylase 8 (40%), high serum lipase level 8 (40%), abnormal liver function test 5 (35%), raised CRP 8 (40%), blood culture 3 (15%), decrease serum Calcium 8 (40%), normal serum amylase 2 (10%), LDH >700 unit/L 8 (40%), PaO_2 < 60 mmHg 8 (40%), base defecit >4 mmol/L 8 (40%), blood urea nitrogen rise >5 mg% 8 (40%).

Contrast-enhanced computed tomography (CECT) is the gold standard in diagnosing pancreatic necrosis. Contrast-enhanced magnetic resonance imaging is a good alternative in patients with renal failure. The current guidelines recommend that CECT is indicated for patients with persistent organ failure, signs of sepsis, or clinical deterioration 6-10 days after admission.[15] The Balthazar's CT severity index (CTSI) is commonly used to stratify the severity and also helps predicting mortality.[16] CTSI modified by Mortele et al. differs from the Balthazar's severity index by the addition of a simplified evaluation of the presence and number of fluid collections and the extent of pancreatic necrosis, and assessment, with different weighting factors, of the presence of extrapancreatic findings such as pleural fluid, ascites, extrapancreatic parenchymal abnormalities (infarction, hemorrhage, or subcapsular fluid collection), vascular complications (venous thrombosis, arterial hemorrhage, or pseudoaneurysm formation), and involvement of the gastrointestinal tract (inflammation, perforation, or intramural fluid collection).[17] In severe acute pancreatitis pleural and lung changes are apparent radiologically in about 20% of cases.[14] The chest radiographs may reveal a pleural effusion that is more common on the left side.[18]

In this series (table 2.10) for medical group, plain X- Ray abdomen -abnormal findings were present in 18 films. Isolated dilated gas filled loop of Jejunum is seen (sentinel loop) 12 (60%), Isolated gas filled duodenum is seen 8 (40%), gas filled hepatic and splenic

flexures are separated by gas less 5 (25%), transverse colon (colon cut off sign) 2 (10%). X-Ray Chest (P/A view) - Left sided pleural effusion 7 (35%), bilateral pleural effusion 1 (5%), contrast enhanced CT Scan done in all patients- reduced enhancement in pancreas, peripancreatic oedema, stranding of fatty tissue 0%, fluid collected in peri- and retro pancreatic space 12 (60%), CT scan revealed gall stone 0%. ERCP Done in 05 patients, dilated CBD 0% and in surgical group plain X- Ray abdomen abnormal findings were present in 18 films- Isolated dilated gas filled loop of Jejunum is seen (sentinel loop) 6 (30%), Isolated gas filled duodenum is seen 2 (10%), gas filled hepatic and splenic flexures are separated by gas less transverse colon (colon cut off sign) 1(5%) , X-Ray Chest (P/A view) left sided pleural effusion 1(5%), bilateral pleural effusion 4 (20%), Contrast enhanced CT scan done in all patients, reduced enhancement in pancreas peripancreatic oedema, stranding of fatty tissue 8 (40%), fluid collected in peri- and retro pancreatic space 0%, CT scan revealed gall stone 5%. In the United Kingdom, 30-50% of cases of acute pancreatitis are related to gallstones and 15-29% to alcohol.[19] However, there was a higher percentage of idiopathic cases(44%) than in Bristol (27%), north west London (22%), and north east Scotland (20%). This figure is higher than the audit standard of 20-25%. Many years of alcohol abuse are required before acute alcoholic pancreatitis develops.[20] The clinical management of this pancreatitis generally is similar to that for pancreatitis of other causes.[21] The precise pathogenesis of gallstone induced pancreatitis continues to be debated. Since pancreatitis develops in only approximately 5 percent of alcoholics, other unexplained factors clearly must predispose patients to this disorder.[22]

In this series (table no 2.11) for surgical group gall stone 25%, chronic heavy alcohol 5%, hypertriglyceridemia 0%, previous surgery 0%, idiopathic 10% and for medical group gall stone 0%, chronic heavy alcohol 0%, hypertriglyceridemia 15%, Previous surgery 5%, Idiopathic 40%.

The UK guidelines for the management of acute pancreatitis were formulated and released by the British Society of Gastroenterology (BSG) in 1998. Firstly, acute pancreatitis is stratified according to severity. Glasgow-Imrie scoring together with Creactive protein are recommended by the BSG for stratification of severity of acute pancreatitis. The next step is to determine aetiology. Imaging to find aetiology should be performed within 24 hours, in contrast with the BSG recommendations of a CT scan between three and 10 days. The rationale behind imaging within 24 hours is to facilitate early endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy, as there is strong evidence that ERCP and sphincterotomy performed less than 72 hours decreases the complication rate in acute severe gall stone pancreatitis.[23] "Surgery has little use in the early management of acute pancreatitis. Interventional endoscopy with sphincterotomy is indicated for acute cholangitis and/or obstructive jaundice in gall stone associated acute pancreatitis".[24] In this series (table 2.12) 12 (60%) patients were given with conservative treatment and remaining 8 (40%) patients were underwent operative intervention after giving conservative treatment with a certain period. Analgesic, aggressive fluid rehydration, oxygenation, nasogastric drainage were given to all the patients. Antibiotics given to all patients (Injection ceftriaxone, injection metronidazole and injection meropenam those who were not responding to ceftriaxone). Initially patients were kept NPO. Eight patients given enteral feeding by naso gastric tube when their abdominal distention reduced and appearance of bowel sound.[12] patients were started with parenteral feeding due to abdominal distention and absence of bowel sound. Gradually parenteral feeding was switched to enteral feeding. 08 patients were underwent operative intervention due to appearance of fever, contrast CT scan

findings of pancreatic necrosis and haemodynamic unstability. Laparotomy with multiple drainage for 05 patients and Laparotomy, necrosectomy and multiple drainage for 03 patients and these options of surgery had decided during laparotomy.

Enteral nutrition starting in the early phase of severe acute pancreatitis (SAP) is superior to total parenteral nutrition unless paralytic ileus is present.[25] Tube feeding is possible in the majority of patients, but may need to be supplemented by the parenteral route. Continuous tube feeding with peptide-based formulae is possible in the majority of patients; the jejunal route is recommended if gastric feeding is not tolerated. In SAP, it is also possible to combine total parenteral nutrition and enteral nutrition when adequate caloric support cannot be obtained by the enteral route alone.[26]

The use of prophylactic broad-spectrum antibiotics reduces infection rates in CT-proven necrotizing pancreatitis, but may not improve survival.[27] However, broad-spectrum antibiotics with good tissue penetration are necessary to prevent infection in SAP.[25] Commonly used antibiotics are carbapenems, quinolones, and third-generation cephalosporins. How long the antibiotics should be given is a debate, although they are generally continued for 2 weeks. Prolonged antibiotic therapy for >4 weeks increases the prevalence of fungal infections.[28]

Patients with sterile pancreatic necrosis should be managed conservatively and undergo intervention only in selected cases, such as those patients with MOF who do not improve despite maximal therapy in the intensive care unit.[27]

The benefits of ERCP with endoscopic sphincterotomy (ES) has been studied in 3 randomized trials 29-31 and 2 meta-analyses 32,33 patients with predicted mild ABP in the absence of cholangitis have not shown benefits from an early ERCP. The decision on management of patients with predicted severe ABP is still debatable. The United Kingdom guidelines recommend that urgent therapeutic ERCP should be performed within 72 h of admission in all patients with predicted severe ABP, whether or not cholangitis is present.[15] To decompress the common bile duct ERCP was done in 05 patients who had gall stone. 03 patients had dilated CBD and sphincterotomy done during ERCP.

Surgery is the gold standard treatment for infeceted pancreatic necrosis. The current recommendation is to delay the surgery for 4 weeks after the onset of pancreatitis. This may lead to prolonged use of antibiotics causing more antibiotic resistance and secondary fungal infections.[34] In this study one patient operated on the 2nd day of admission due to haemodynamic unstability, appearance of fever, CT scan findings of pancreatic necrosis. Remaining 7 patients were operated after 3-4 weeks of conservative treatment as these patients were not indicated for operative intervention initially. When these patients developed fever with their CT scan findings of pancreatic necrosis, they underwent laparotomy.

In those who have a severe attack of pancreatitis, the mortality varies from 20 to 50 per cent. About one-third of deaths occur in the early phase of the attack, from multiple organ failure, while deaths occurring after the first week of onset are due to septic complications.1 Mortality occurs in two peaks. In the early phase, it is due to severe SIRS and in the late phase due to multi organ failure (MOF), secondary to infective local complications or systemic sepsis.[35,36]

In this study (table 2.13) one patient died on 1st post operative day and one patient died on 2nd post operative day. Another patient died on 5th day of admission who was under conservative treatment due to SIRS and multi organ failure. Total 03 patient were expired despite all efforts. In this series post operatively patient died due to septicaemia and multiorgan failure. The median hospital stay for the severe cases was 16 days (range 2-304) in a study of 186

patients.[19] In this study average hospital stay was 3 weeks. Prolongation of hospital stay was due to development of complications. In this series (table 2.13) other outcomes of surgical group were residual abscess 1 (5%), burst abdomen 2 (10%), fistula 2 (10%) and other outcomes of medical group were residual abscess 2 (10%), burst abdomen 0%, fistula1 (5%).

Complications following necrosectomy remain common; however, recent data have shown that the overall morbidity and mortality have declined. These complications include pancreatic fistula, enterocutaneous fistula, colonic strictures, intestinal obstruction, secondary infections, wound infection, dehiscence, and incisional hernia. Bleeding is a rare complication usually managed by topical haemostatic agents, suturing, packing, and angioembolization. Fistulae occur in up to 30% of patients undergoing laparotomy, and initially it should be managed conservatively, with surgical repair deferred until such time as the pancreatitis has resolved completely. Colonic complications in the form of perforation or strictures are not uncommon. Patients with extensive necrosis extending in the left paracolic gutter are more prone to these complications. A defunctioning ileostomy followed by resection with primary anastomosis later may be required in these patients. Wound infection and dehiscence must be treated more aggressively. Endocrine and exocrine insufficiencies of the pancreas are also relatively common. [37]

Fluid collections are common and are termed as acute fluid collections before 4 weeks. Fluid collections after 4 weeks are termed as pancreatic pseudocyst. Late phase of AP is characterized by local complications. Infection of pancreatic necrosis occurs in 25–70% of patients with necrotizing pancreatitis and is believed to occur as a result of bacterial translocation due to failure of intestinal barrier.[38,39] In this study (table 2.14) systemic complication including pleural effusion 8 (40%), ARDS 5 (25%), septicaemia 3 (15%), DIC 1 (5%), renal failure 3 (15%) for surgical group and pleural effusion 7 (35%), ARDS 0%, septicemia 0%, DIC 0%, renal failure, 0% for medical group. In this series (table 2.15) local complications including pancreatic necrosis 8 (40%), pseudo cyst formation 4 (20%) and for surgical group and pancreatic necrosis 3 (15%), pseudo cyst formation 0%.

In summary from the beginning of this study patient were selected meticulously. Acute pancreatitis patient were selected from all patients of acute abdomen. Then by Ranson criteria patients were categorized to severe acute pancreatitis. Those who scored 3 or more would be selected for severe acute pancreatitis. After categorization subsequent management were planned on the basis of laboratory and CT findings. Analgesic, aggressive fluid rehydration, oxygenation, nasogastric drainage were given to all the patients. Antibiotics given to all patients (Injection ceftriaxone, injection metronidazole and injection meropenam those who were not responding to ceftriaxone). Initially patients were kept NPO. 8 patients given enteral feeding by naso gastric tube when their abdominal distention reduced and appearance of bowel sound. 12 patients were started with parenteral feeding due to abdominal distention and absence of bowel sound. Gradually parenteral feeding was switched to enteral feeding. 08 patients were underwent operative intervention due to appearance of fever, contrast CT scan findings of pancreatic necrosis and hemodynamic instability. Laparotomy with multiple drainage for 5 patients and Laparotomy, necrosectomy and multiple drainage for 3 patients and these options of surgery had decided during laparotomy. In this study total 03 patients were expired. Post operatively patients developed residual abscess, burst abdomen and fistula. By maintaining proper nutrition, fluid electrolytes balance, early administration of broad spectrum antibiotics, early mobilization at post operative period, deep breathing exercise, regular wound care, maintaining movement of the visitors we can

reduce these complications. Severe acute pancreatitis is a challenge although there is existing protocol of management with high mortality. In this study it was proved that early detection, categorization, proper management and timely decision for operative intervention can reduce mortality.

Limitations

1. Sample size small and single centre study.
2. Unable to randomize
3. Follow up period is short

Summary and Conclusion

Management of acute necrotizing pancreatitis has changed significantly over the past years. Early management is nonsurgical and solely supportive. Today, more patients survive the early phase of severe pancreatitis due to improvements in intensive care medicine. It is clear that although the era of minimally invasive necrosectomy has arrived, there is a limited body of evidence. The selection of treatment must be guided by the need to ensure the availability of true multidisciplinary expertise in a specialist unit. Techniques should not be selected simply because of the expertise of an individual clinician.

In this study it was proved that early detection; categorization, proper management and timely decision for operative intervention could reduce mortality.

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