



ORIGINAL RESEARCH PAPER

General Medicine

A DISEASE OF SEVERE SYSTEMIC INVOLVEMENT AND RECOVERY -ACUTE NECROTISING PANCREATITIS WITH MULTI ORGAN DYSFUNCTION SYNDROME -A CASE SERIES.

KEY WORDS: Multiorgan Dysfunction, Antibiotics

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ABSTRACT

Acute necrotizing pancreatitis (ANP) is a severe form of acute pancreatitis, where there is extensive inflammation of the pancreas along with tissue death (necrosis). It can lead to severe complications and is associated with high morbidity and mortality rates. The most common cause is gallstones or alcohol, but other factors include high cholesterol, high triglycerides, infections, trauma, and certain medications. Development of infection, pseudocyst and organ dysfunction are the most developed complications.

INTRODUCTION:

Acute necrotising pancreatitis, When progresses, it can lead to multiorgan systemic inflammation and complications stemming from pancreatic injury. ANP leads MODS by causing Systemic inflammatory recovery syndrome along with pancreatic enzymes activation that eventually leads to widespread infection leading to hypoperfusion and shock. The severity of MODS significantly affects the prognosis. In general, the more organs involved, the poorer the outcome. Mortality rates can be high (up to 50-50% or more) depending on the degree of necrosis, infection, and organ failure.

CASE SERIES:

CASE 1:

A 55 year old male presented to tertiary care centre with complaints of abdominal pain and vomiting for one day. He is a known case of diabetes and hypertension and was on regular medication. His hb was 19.8g/dl, Total counts 13850 cells/cumm with polymorphic predominance and PCV 57.4. BUN 12 and Creatinine was 1.4mg/dl with sodium 134mmol/lit, Potassium 6.3mmol/l. Amylase was 440 and Lipase 1614. Chest xray showed Bilateral CP angle blunting. Patient admitted to ICU and CECT abdomen done revealed acute necrotising pancreatitis with CT severity index 10/10 with splenic vein thrombosis and cholangitis. Patient was kept Nil per oral and nasojejunal tube was placed. Patient was started on Tab apixaban for splenic vein thrombosis. Dyselectrolytemia were corrected. Oral was started gradually.

CASE 2:

A 50 year old male came with complaints of abdominal pain for 2 days. Patient is known case of Dilated cardiomyopathy and diabetes on regular medications. Not an alcoholic or smoker. On arrival patient presented with hypotension and was resuscitated with fluids and was started on ionotropes. Serum amylase and lipase 1114 and 3336 and creatinine 1.5mg/dl. CECT abdomen showed acute necrotising pancreatitis with CT severity score of 10/10 with splenic vein thrombosis. MRCP done showed no evidence of biliary obstruction. Patient was started on Antibiotics and anticoagulants with ventilatory support due to tachypnea. Patient was initially conservatively managed. General surgery and medical gastroenterology were involved and Inj Octreotide were given to decrease secretions. Due to intolerance of pain, patient required IV sedation for a brief period. CT guided pigtail aspiration was done and sample sent grew klebsiella pneumoniae - pan susceptible. Due to persistent fever spikes, Repeat Cultures

were sent and was empirically started on meropenam. Blood culture sent grew Candida auris and was treated with caspofungin. Repeat CECT done was showing resolution of collection. After 6 weeks Patient was taken up for necrosectomy with diversion colostomy by surgical gastroenterology and drain placed was placed.

CASE 3:

A 36 year old male presented with abdominal pain for 3 days with vomiting. Patient is a alcoholic and smoker for 6 years. HB 12g/dl, TC 11950 cells/mm³, bun 72mg/dl and Creatinine 8.1mg/dl, amylase 560. CECT done showed pancreatic parenchymal enhancement with no evidence of necrosis with Severity index 5/10. Patient was started on meropenem and cultures showed no growth. Patient was initially admitted under surgery and was taken up by general medicine for management of AKI and dyselectrolytemia. Appropriate corrections were given and In view of persistent AKI with worsening oliguria, patient underwent 7 cycles of dialysis during in hospital stay and output improved with discharge creatinine of 1.3 mg/dl.

Table 1: Clinical Profile And Outcomes In Case Series:

DETAILS	CASE 1	CASE 2	CASE 3
HOSPITAL ID :	3070236	2670889	21894553
AGE/SEX	55/M	50/M	35/M
PRESENTATION:	ABDOMEN PAIN AND VOMITING	ABDOMEN PAIN AND SHOCK	ABDOMEN PAIN
CT SEVERITY SCORE	10/10	10/10	5/10
COMPLICATIONS	AKI	AKI/ ALI/ CANDIDEMIA	AKI/THROMBOCYTOPENIA
TREATMENT	SYMPTOMATIC	NECRECTOMY	HEMODIALYSIS

DISCUSSION:

Acute pancreatitis is the most common pancreatic disease worldwide. Necrotizing pancreatitis develops in approximately 20 to 30% of patients with acute pancreatitis. Pancreatic and peripancreatic necrosis that becomes infected nearly always leads to invasive intervention. When infected necrosis is suspected, initiation of broad-spectrum intravenous antibiotics with good penetration into the pancreas is recommended. These include carbapenems, quinolones, metronidazole, and third or higher-generation cephalosporins. Computed tomography-guided percutaneous biopsies of necrotic

collections with samples sent for Gram stain and cultures can be performed to confirm the presence of infection.

It was generally believed that patients with acute pancreatitis would be at risk for a worsening clinical course if the pancreas was stimulated by oral or enteral nutrition, and would benefit from “pancreatic rest” by receiving total parenteral nutrition (TPN) . However, these theories have been largely disproven. For patients without nausea, vomiting, or evidence of intestinal obstruction or ileus, a trial of oral nutrition should be commenced immediately. For patients unable to tolerate oral intake, early nutritional support should be prioritized within the first 24–72 hours.

Percutaneous drainage, alone or in combination with other minimally invasive approaches, remains an important treatment modality for patients with symptomatic WON.

In patients with infected pancreatic necrosis or those with sterile pancreatic necrosis who have persistent organ dysfunction or failure to thrive, operative debridement should be considered. Timing of intervention is paramount, as debridement during the early phase of acute pancreatitis, when the systemic inflammatory response is the driver of clinical morbidity (within 2–4 weeks of onset), carries a significantly higher mortality rate than supporting the patient through to the subacute period. The goals of operative debridement are to control the source of infection and decrease the burden of necrosis, while minimizing the pro-inflammatory insult of the intervention itself on the debilitated patient.

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