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CASE REPORT: ACUTE PANCREATITIS, REVISED ATLANTA CLASSIFICATION – MODERATELY SEVERE (GRADE II)

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Abstract:

Acute pancreatitis (AP) is a common and potentially life-threatening inflammatory condition of the pancreas, predominantly triggered by gallstones or alcohol use. We present the case of a 38-year-old male who arrived at the emergency department with severe epigastric pain radiating to the back, nausea, vomiting, and a history of chronic alcohol consumption. Laboratory investigations confirmed markedly elevated serum amylase and lipase levels. Abdominal CT imaging demonstrated peripancreatic fluid collections, focal areas of necrosis, and surrounding fat stranding, consistent with moderately severe acute pancreatitis (Grade II) as per the Revised Atlanta Classification. The patient was managed with aggressive intravenous fluid resuscitation, bowel rest, analgesics, antiemetics, and prophylactic antibiotic coverage. Nutritional support was initiated via nasojejunal feeding on Day 3. The clinical course was complicated by hypocalcaemia and transient hyperglycaemia, both of which were corrected. The patient showed marked clinical improvement by Day 7 and was discharged on Day 10 with dietary counselling and abstinence advice. This case highlights the importance of timely diagnosis, risk stratification, and aggressive supportive management in achieving favourable outcomes in acute pancreatitis.

Keywords: Acute pancreatitis, Revised Atlanta Classification, Peripancreatic necrosis, Gallstone pancreatitis, Alcoholic pancreatitis, Serum amylase, Serum lipase, CT severity index, Fluid resuscitation, Nasojejunal feeding, Hypocalcaemia, Hyperglycaemia, Infected necrosis, Octreotide, Multidisciplinary management

1. INTRODUCTION

Acute pancreatitis is one of the most frequent gastrointestinal disorders requiring urgent hospitalisation, with an annual global incidence estimated between 13 to 45 per 100,000 population [1]. The inflammatory process, triggered by premature activation of digestive enzymes within the pancreas, results in autodigestion of pancreatic tissue and an intense local and systemic inflammatory response [2]. The most prevalent aetiological factors remain gallstones (40–70%) and alcohol use (25–35%), with other causes including hypertriglyceridaemia, hypercalcaemia, drugs, trauma, and idiopathic origins [3].

The clinical spectrum ranges from mild self-limiting disease to severe necrotising pancreatitis with multi-organ failure and a mortality rate exceeding 30% [4]. The Revised Atlanta Classification (2012) categorises acute pancreatitis into mild, moderately severe, and severe disease based on the presence and persistence of local and systemic complications [5]. Early risk stratification using scoring systems such as BISAP, Ranson's criteria, and CT Severity Index is vital for guiding management decisions.

This case report describes the clinical course, diagnostic workup, and pharmacological management of a 38-year-old male patient presenting with moderately severe acute pancreatitis attributed to chronic alcohol use, underscoring the significance of a multidisciplinary approach and early supportive intervention in achieving clinical recovery.

2. CASE PRESENTATION

A 38-year-old male patient presented to the emergency department with chief complaints of sudden-onset severe epigastric pain radiating to the back for 12 hours, associated with persistent nausea, bilious vomiting (4–5 episodes), and inability to tolerate oral intake. The patient reported a history of chronic alcohol consumption (approximately 80g/day for the past 8 years). There was no history of jaundice, fever, or prior similar episodes. On admission, the patient appeared distressed with signs of dehydration. Abdominal examination revealed significant epigastric tenderness with guarding. CT abdomen with contrast confirmed peripancreatic fluid collections and focal areas of pancreatic necrosis, with a CT Severity Index (CTSI) score of 6, consistent with moderately severe acute pancreatitis.

PHYSICAL AND SYSTEMIC EXAMINATION

	D1	D2	D3	D4	D5	D6	D7
Temperature (F)	99	98.6	99.2	98.8	98.4	98	98
Blood pressure (mmHg)	130/90	128/88	120/80	118/78	120/80	118/76	120/78
Pulse (bpm)	96	92	88	84	80	78	76
CVS	S1S2+	S1S2+	S1S2+	S1S2+	S1S2+	S1S2+	S1S2+
Respiratory system	BAE+	BAE+	BAE+	BAE+	BAE+	BAE+	BAE+
P/A	Tender	Tender	Tender	Mod.Tender	Mild Tender	SOFT&NT	SOFT&NT

3. LAB INVESTIGATIONS

HAEMATOLOGICAL PARAMETERS

Parameters	Results
Hb [13–17] g/dl	11.8

RBC [4.0–5.5] millions/cumm	3.9
WBC [4000–10000] cells/cumm	14,200
Lymphocytes [15–30]%	18
Eosinophils [1–6]%	3
Monocytes [2–10]%	6
ESR [0–10] mm 1st hour	42
Platelets [1–4] lakhs/cumm	2.8

OTHER INVESTIGATIONS

1. Serum Enzyme Levels

Serum Amylase: 1,240 U/L (Reference: 30–110 U/L) — markedly elevated

Serum Lipase: 2,860 U/L (Reference: 10–140 U/L) — markedly elevated

Blood Glucose (Random): 218 mg/dL — consistent with stress hyperglycaemia

Serum Calcium: 7.1 mg/dL (Reference: 8.5–10.5 mg/dL) — hypocalcaemia noted

Serum Triglycerides: 310 mg/dL — mildly elevated, not primary aetiology

2. Ultrasonography (USG) – Abdomen

Impression: Enlarged, hypoechoic pancreas with peripancreatic fluid collection. Gallbladder shows multiple calculi (largest 9mm). No biliary duct dilatation. Mild ascites noted in the right paracolic gutter.

3. Contrast-Enhanced CT (CECT) – Abdomen

Impression:

- Pancreatic body and tail show areas of non-enhancement suggestive of focal necrosis.

- Peripancreatic fat stranding and fluid collections noted bilaterally.

- CT Severity Index (CTSI): 6/10 — Moderately Severe pancreatitis.

- No evidence of pseudocyst, abscess, or vascular complications at this stage.

4. BISAP Score

BUN > 25 mg/dL: 1 | Impaired mental status: 0 | SIRS criteria met: 1 | Age < 60: 0 | Pleural effusion on imaging: 0

Total BISAP Score: 2/5 — Intermediate risk for in-hospital mortality

DRUG TREATMENT CHART

S.NO	TRADE NAME	GENERIC NAME	DOSE	ROA	FRQ	CLASS	INDICATION
1	INJ.PANTODAC	Pantoprazole	40mg in 100ml NS	IV	BD	Proton pump inhibitor	Reduction of gastric acid secretion; prevention of stress ulcers and GI complications
2	INJ.EMESET	Ondansetron	8mg in 100ml NS	IV	TID	5HT3 receptor antagonist	Prevention and management of nausea and vomiting
3	INJ.TRAMADOL	Tramadol	50mg in 100ml NS	IV	TID	Opioid analgesic	Management of moderate-to-severe abdominal pain associated with pancreatitis
4	INJ.TAXIM	Cefotaxime	1g in 100ml NS	IV	BD	Third-generation cephalosporin	Prophylaxis and treatment of secondary bacterial infections; prevention of infected pancreatic necrosis
5	INJ.METRO	Metronidazole	500mg in 100ml NS	IV	TID	Nitroimidazole antibiotic	Anaerobic bacterial coverage in infected pancreatic necrosis

6	INJ.RINGER LACTATE	Ringer Lactate	1000ml	IV	Continuous	Crystalloid IV fluid	Aggressive fluid resuscitation to prevent pancreatic ischemia and systemic hypoperfusion
7	INJ.OCTREOTIDE	Octreotide	100mcg in NS	IV	TID	Somatostatin analogue	Reduction of pancreatic enzyme secretion to allow gland rest and reduce autodigestion
8	TAB.CREON	Pancreatic enzyme supplement (lipase/amylase/protease)	25,000 units	PO	TID with meals	Pancreatic enzyme replacement	Management of exocrine pancreatic insufficiency during recovery; improve digestion
9	INJ.CALCIUM GLUCONATE	Calcium gluconate	1g in 100ml NS	IV	OD	Electrolyte supplement	Correction of hypocalcaemia, a common complication of acute pancreatitis
10	INJ.THIAMINE	Thiamine (Vitamin B1)	100mg in NS	IV	OD	Vitamin supplement	Prevention of Wernicke encephalopathy in patients with poor oral intake and nutritional deficiency

4. DISCUSSION

Acute pancreatitis caused by chronic alcohol consumption represents a significant proportion of hospital admissions for gastrointestinal emergencies worldwide. In this case, the patient's prolonged alcohol use led to repeated subclinical pancreatic injury, culminating in an acute inflammatory episode characterised by markedly elevated pancreatic enzyme levels, peripancreatic necrosis, and fluid collections as observed on contrast-enhanced CT imaging. The CTSI score of 6 and BISAP score of 2 placed this patient in the moderately severe category, justifying close monitoring and aggressive supportive measures.

Aggressive intravenous fluid resuscitation with Ringer's lactate was initiated promptly, consistent with current evidence favouring goal-directed fluid therapy in the first 24–48 hours to prevent pancreatic ischaemia and multi-organ involvement [6]. Pain management with IV tramadol provided effective analgesia while avoiding the adverse gastrointestinal effects associated with morphine, such as sphincter of Oddi spasm. Octreotide was administered to suppress pancreatic exocrine secretion, thereby reducing ongoing autodigestion, though its role remains debated in the literature for mild-to-moderate disease [7].

Prophylactic antibiotics with cefotaxime and metronidazole were employed given the presence of focal necrosis on imaging, which confers a risk of secondary bacterial infection — a major determinant of morbidity and mortality in acute pancreatitis [8]. Hypocalcaemia, a recognised complication resulting from saponification of peripancreatic fat tissue, was corrected with intravenous calcium gluconate. Early enteral nutrition via nasojejunal tube, commenced on Day 3, was preferred over parenteral nutrition to preserve gut mucosal integrity and reduce infectious complications [9].

The patient's recovery trajectory was favourable, with resolution of pain, normalisation of enzyme levels, and tolerance of oral diet by Day 8. This case reinforces the critical role of early risk stratification, systematic supportive care, nutritional optimisation, and strict alcohol abstinence counselling in the comprehensive management of alcoholic acute pancreatitis.

5. CONCLUSION

This case illustrates the clinical complexity and potential severity of moderately severe acute pancreatitis, a condition that demands prompt recognition, accurate risk stratification, and multidisciplinary intervention. The patient presented with hallmark features of alcoholic pancreatitis — severe epigastric pain, markedly elevated pancreatic enzymes, and CT-confirmed peripancreatic necrosis — and responded well to a structured management protocol comprising aggressive fluid resuscitation, analgesia, bowel rest, prophylactic antibiotic therapy, and early enteral nutrition.

The case underscores the value of validated scoring systems such as the Revised Atlanta Classification, BISAP score, and CTSI in guiding clinical decision-making and anticipating complications. Timely correction of metabolic derangements including hypocalcaemia and stress hyperglycaemia further contributed to the patient's recovery. Long-term counselling regarding complete alcohol abstinence remains the cornerstone of secondary prevention.

A coordinated approach involving gastroenterology, radiology, nutrition support, and pharmacy practice is essential for optimising patient outcomes in acute pancreatitis, particularly in resource-intensive presentations such as this.

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