

Silent Catastrophe: Necrotizing Pancreatitis

Sara Clark, PA-S, Susan McDiarmid, EdD, MS, PA-C

Westfield State University
Physician Assistant Program



Case

62-year-old female presented to the ED with sudden onset of severe epigastric pain beginning the day prior. Pain was rated 10/10 without radiation, with associated nausea, vomiting, and diarrhea. Her daughter accompanying her reported the pain typically occurs after consuming clam chowder, which the patient did the night prior.

Current medications included 10 mg Aripiprazole, 20 mg Atorvastatin, 25 mcg Cholecalciferol, 100 mg Gabapentin BID, 300 mg Glucosamine Chondroitin BID, 20-12.5 mg Lisinopril-Hydrochlorothiazide, 500 mg Metformin XR, 2 mg Semaglutide, 50 mg Trazodone as needed, 75 mg Venlafaxine XR, multivitamin, and a blood-glucose meter kit with test strips.

Past medical history included HTN, HLD, morbid obesity with a BMI of 47.14 kg/m2, uncontrolled T2DM with hyperglycemia, osteoarthritis of bilateral knees, depression, history of a urinary calculi, history of viral meningitis, leiomyoma of the uterus, low back pain, and a vitamin D deficiency. Social history included smoking 0.3 packs per day for 19 years (quit in 1995), and daily use of alcohol and marijuana. Family history included her mother having colon cancer, paternal grandfather having heart failure, and HTN in her father, mother, and sister

ED Physical Exam/Labs

Vitals	Temperature: 36.3°C (97.3°F) Heart Rate (HR): 114 Respiratory Rate (RR): 20	Blood Pressure (BP): 159/100 Oxygen Saturation (SpO ₂): 92%
CMP	Potassium: 3.3 Glucose: 247 eGFR: 58	AST: 368 ALT: 286 Alkaline Phosphatase: 202 Total Bilirubin: 2.1
Lipase	3,238	
Physical Exam	Abdomen: Hypoactive bowel sounds. Obese abdomen is soft, nondistended, tenderness to palpation (TTP) of epigastrium and milder TTP of left upper quadrant (LUQ) No guarding, rebound, or peritoneal signs Cardiac: Steady rate and rhythm, no murmur Pulmonary: Non-labored breathing, clear to auscultation bilaterally	

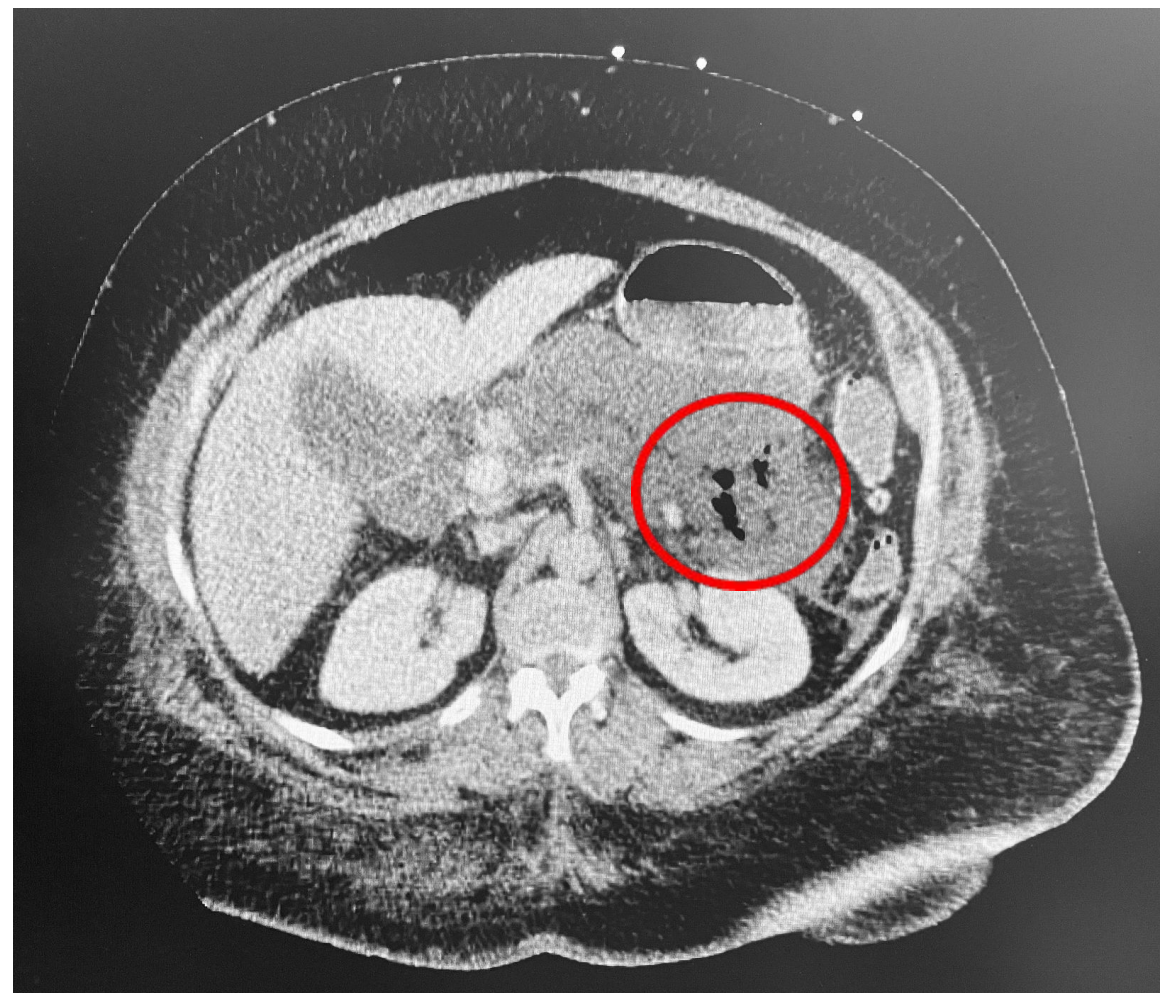


Figure 1. CTA on day 7 demonstrating scattered peripancreatic fluid collections and fat stranding with air locules (circled in red; 4.3 cm), along with minimal enhancement seen in the pancreatic head and uncinate process, with otherwise poor glandular enhancement.

Hospital Course

Day 1	Arrived at the ED US abdomen: heterogeneous pancreas without (w/o) pancreatic fluid collections, distended gallbladder w/o wall thickening, cholelithiasis w/o evidence of cholecystitis or biliary duct dilation Diagnosed with acute pancreatitis (AP) and admitted
Day 2	Epigastric/LUQ TTP HR 141, BP 151/51, Glucose 420, WBC 12.3 Lipase trending down: 3,238 → 747 Magnetic Resonance Cholangiopancreatography (MRCP) confirmed pancreatitis; tolerating clear fluids
Day 3	Additional lower abdominal TTP Shortness of breath (SOB) with long sentences, faint crackles HR 132, BP 153/90, RR 22, Glucose 390, WBC 12.4
Day 4	Reports improvement, no abdominal pain or TTP SOB with exertion HR 129, BP 147/77, RR 22, Glucose 373, WBC 14.5 Advanced to low fat diet
Day 5	Significant Event: Given Ativan night prior for anxiety/agitation HR 130s, BP 99/45, confused, hypoxic (SpO ₂ 80's on 4L) SVT with PVCs on telemetry HR 115, BP 137/87, RR 16, Glucose 391, WBC 16.4 BUN ↑ 15 → 29, Creatinine ↑ 0.68 → 1.08
Day 6	Significant Event: SVT (HR 190-200) on telemetry Drowsy, ill-appearing, dyspneic HR 127, BP 146/118, RR 22, Glucose 259, WBC 16.7 Tachypneic with shallow respirations, 2-3 word sentences
Day 7	Ill-appearing, somnolent but arousable, tachypneic Still denying pain HR 128, BP 123/71, RR 24, Glucose 217, WBC 15.7

Differentials / Diagnostic Testing

Differential	Pertinent Positives	Pertinent Negatives
Pneumonia	Dyspnea Tachypnea Tachycardia Crackles at lung bases Leukocytosis Hospitalized (risk)	No fever, cough, sore throat No chest pain (CP) Viral testing: negative CXR: congestion only CT chest: negative Blood culture: negative
Pulmonary Embolism	Tachypnea, tachycardia Dyspnea, hypoxia Immobile in hospital D-Dimer ↑ (6,244)	No CP or cough On prophylactic Lovenox Venous duplex: negative CTA: negative ECHO: negative
Obesity Hypoventilation Syndrome	Morbid obesity Dyspnea Somnolence, hypoxia	ABG: not retaining CO ₂
Sepsis	HR 115, WBC 12.6 Suspected infection on imaging Procalcitonin ↑ 2.25	Criteria only met on day 5 Blood culture: negative Lactic acid WNL at 1.7
Pancreatitis Complication	History of pancreatitis, History of hyperglycemia Somnolence, confusion ↑ work of breathing, tachycardia CT: peripancreatic fluid CTA: LUQ extraluminal gas CT abdomen: peripancreatic free air, colonic ileus, and a small bowel obstruction (SBO)	No abdominal pain No distention or bloating No fever, chills, night sweats Lipase normalized No Grey Turner No Cullen's sign

Diagnosis

The final diagnosis for this patient was necrotizing pancreatitis. This patient initially presented with a diagnosis of acute pancreatitis (AP), which subsequently progressed with radiographic evidence of extraluminal gas measuring 4.3 cm in the left upper quadrant. A concurrent peripancreatic fluid collection was also identified and later confirmed to be infected with *Klebsiella* species. Serial imaging over the following 10 days demonstrated progression to pancreatic necrosis, with enlargement of the gas-containing collection to 15.1 cm.

Management/Treatment

In the ED, the patient received a normal saline bolus, 1,000 mg PO Acetaminophen (Tylenol), 4 mg IV Ondansetron (Zofran), and 15mg IV Ketorolac (Toradol). She was made NPO, later advancing to clear fluids and then a low fat diet. Due to a SBO and ileus, peripheral parenteral nutrition was initiated as enteral feeds were contraindicated. Broad-spectrum antibiotics were started with IV Meropenem (Merrem), and changed to Piperacillin-Tazobactam (Zosyn) per Infectious Disease. A 12 Fr pigtail drain was placed, yielding nearly 4 liters of fluid within one week. Following culture results positive for *Klebsiella* species, antibiotics were narrowed to Ampicillin-Sulbactam (Unasyn) per Infectious Disease. She was ultimately discharged with a plan of prolonged IV antibiotics via a PICC line. Follow up was lost after this point with the patient.

Discussion

Necrotizing pancreatitis is a severe form of AP, accounting for 5% to 10% of cases.¹ The etiologies of necrotizing pancreatitis mirror those of AP, with gallstones and alcohol abuse accounting for 60%-75% of cases.^{2,3} The pathophysiology of AP progresses through three phases: (1) premature trypsin activation within pancreatic acinar cells; (2) intrapancreatic inflammation via multiple pathways; and (3) extrapancreatic inflammation.⁴ While most cases are mild, 10%-20% progress to severe disease with systemic inflammatory response syndrome (SIRS), which can predispose patients to multiple organ dysfunction and/or pancreatic necrosis.²

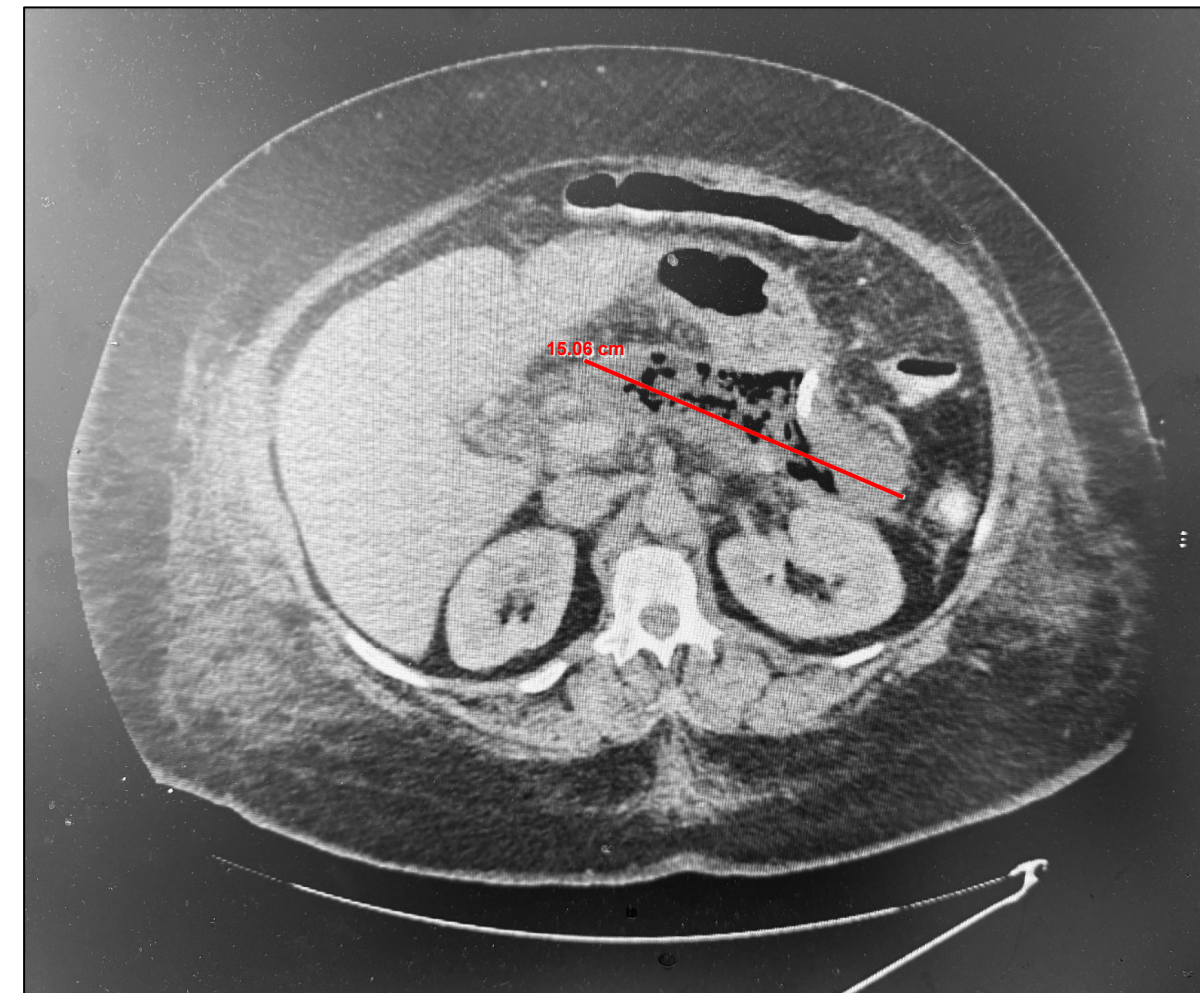


Figure 2. Repeat CTA on day 18 after interval drainage, demonstrating marked increase in intraluminal air (measured along the red line; 15.1 cm) and absence of discernible normal pancreatic parenchyma, consistent with progression to extensive pancreatic necrosis.

As necrotizing pancreatitis is a severe form of AP, it is essential to understand the diagnostic criteria and image findings of AP. Diagnosis of AP requires two of the following three criteria: (1) acute onset of severe epigastric pain, often radiating to the back; (2) serum lipase $\geq 3\times$ greater than the upper limit of normal; and (3) characteristic findings on a contrast-enhanced computed tomography (CECT), or less commonly, MRI or US.⁵ In the early course of AP, CECT may show patchy pancreatic perfusion, with necrosis becoming more apparent over several days.⁵

After the first week, non-enhancing areas of pancreatic or peripancreatic parenchyma on CECT is consistent with necrosis and should further be classified as sterile or infected.^{5,6} Identifying infection within necrotic tissue is critical, as it shifts management from supportive care to antibiotics and often intervention. Infection is suggested by the presence of extraluminal gas in pancreatic and/or peripancreatic tissues on CECT or confirmed by image-guided fine-needle aspiration positive for bacteria and/or fungi on Gram stain and culture.⁵

Several factors likely contributed to this patient's development of necrotizing pancreatitis, including advanced age, morbid obesity, daily alcohol use, uncontrolled diabetes, GLP-1 (Glucagon-Like Peptide-1) receptor agonist use, and a history of smoking. These are all recognized as risk factors for severe acute pancreatitis.

AP remains a major health burden; in 2012, it was the third most common gastrointestinal, liver, or pancreatic diagnosis in hospitalized patients, with 275,170 cases and a total healthcare cost exceeding \$4.8 billion.⁷ The incidence of AP continues to rise, likely due to improved diagnostic capabilities, but may also reflect a true increase driven by growing prevalence of risk factors such as alcohol consumption.^{8,9}

In this patient, necrosis involved both the pancreas and the peripancreatic tissues, which is the most common pattern of necrotizing pancreatitis.⁵ The natural history of pancreatic and peripancreatic necrosis is variable: it may remain solid or liquefy, persist or resolve, and remain sterile or become infected.⁵ When infection occurs—typically monomicrobial—it significantly increases mortality risk, as it is the leading cause of death in these cases.⁹ Common pathogens involve gram-negative rods, Enterobacter species, or gram-positive organisms such as staphylococcus.¹⁰ Due to the variability in treatment based on disease severity, early recognition and timely management is critical.

In this case, improvement in AP-related clinical signs and symptoms, absence of abdominal pain, and early normalization of lipase collectively lowered clinical suspicion for necrotizing pancreatitis. While the presentation was not overtly atypical, these factors delayed consideration of this severe form. This highlights the importance of maintaining a broad differential and high index of suspicion for complications, even when early clinical improvement is observed.

References

- Tenner S, Vege SS, Sheth SG, et al. American College of Gastroenterology Guidelines: Management of Acute Pancreatitis. *Am J Gastroenterol*. 2024;119(3):419-437. doi: 10.14309/ajg.00000000000002645
- Leonard-Murali S, Lezotte J, Kalu R, et al. Necrotizing pancreatitis: A review for the acute care surgeon. *Am J Surg*. 2021;221(5):927-934. doi:10.1016/j.amjsurg.2020.08.027
- Kota SK, Krishna SV, Lakhtakia S, Modi KD. Metabolic pancreatitis: Etiopathogenesis and management. *Indian J Endocrinol Metab*. 2013;17(5):799-805. doi:10.4103/2230-8210.117208
- Banks PA, Freeman ML; Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol*. 2006;101(10):2379-400. doi: 10.1111/j.1572-0241.2006.00856.x
- Banks PA, Bollen TL, Dervenis C, et al. Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis, 2012 revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013 Jan;62(1):102-11. doi: 10.1136/gutjnl-2012-302779
- Mao L, Qiu Y. The classification of acute pancreatitis: Current status. *Intractable Rare Dis Res*. 2012;1(3):134-137. doi:10.5582/irdr.v1.3.134
- Peery AF, Crockett SD, Barritt AS, et al. Burden of Gastrointestinal, Liver, and Pancreatic Diseases in the United States. *Gastroenterology*. 2015;149(7):1731-1741.e3. doi:10.1053/j.gastro.2015.08.045
- Kingsnorth A, O'Reilly D. Acute pancreatitis. *BMJ*. 2006;332(7549):1072-1076. doi:10.1136/bmj.332.7549.1072
- Rashid MU, Hussain I, Jehanzeb S, et al. Pancreatic necrosis: Complications and changing trend of treatment. *World J Gastrointest Surg*. 2019;11(4):198-217. doi:10.4240/wjgs.v11.i4.198
- Forsmark CE, Vege SS, Wilcox CM. Acute Pancreatitis. *N Engl J Med*. 2016;375(20):1972-1981. doi: 10.1056/NEJMr1505202