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Infected Pancreatic Walled-off Necrosis Following Hypertriglyceridemia-Induced Necrotizing Pancreatitis: A Case Report

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Abstract

Infected walled-off necrosis (WON) is a serious complication of necrotizing pancreatitis that requires timely, evidence-based management. We describe the case of a 41-year-old man with a history of severe hypertriglyceridemia-associated necrotizing pancreatitis who presented with an encapsulated pancreatic/peripancreatic collection containing intralesional gas, consistent with infected WON. Contrast-enhanced computed tomography showed no evidence of new pancreatic parenchymal necrosis, and the available findings did not establish a recurrent episode of acute necrotizing pancreatitis. The patient underwent endoscopic ultrasound-guided transmural drainage followed by two endoscopic necrosectomies as part of a step-up approach, combined with antimicrobial therapy. Cultures obtained from the necrotic collection identified uncommon microorganisms, including Rothia mucilaginosa, Solobacterium moorei, and Peptostreptococcus anaerobius. During an endoscopic procedure, the patient developed narrow-complex tachycardia with transient hypotension. Subsequent electrocardiographic evaluation revealed ventricular pre-excitation consistent with Wolff-Parkinson-White syndrome, although the mechanism of the acute arrhythmia could not be definitively established. Following multidisciplinary management, the patient demonstrated favorable clinical improvement during hospitalization and was discharged. This case highlights the role of minimally invasive step-up management in appropriately selected patients, the importance of interpreting unusual microbiological findings in their clinical context, and the need to recognize cardiovascular conditions that may influence procedural safety. Further investigation is warranted to clarify the clinical significance of uncommon microorganisms isolated from infected WON.

Categories: Infectious Disease, Endocrinology/Diabetes/Metabolism, Gastroenterology
Keywords: endoscopic necrosectomy, hypertriglyceridemia, infected pancreatic necrosis, necrotizing pancreatitis, step-up approach, walled-off necrosis, wolff-parkinson-white syndrome

Introduction

Hypertriglyceridemia-induced acute pancreatitis is among the leading causes of acute pancreatitis, alongside biliary and alcohol-related etiologies, and has been associated with increased disease severity, mortality, and recurrence compared with other etiologies. Elevated triglyceride (TG) levels have also been associated with complications such as pancreatic necrosis, systemic inflammatory response syndrome, shock, and multiorgan failure [1]. In a population-based Danish cohort, 3.5% of individuals with severe hypertriglyceridemia developed acute pancreatitis, highlighting the clinical relevance of this metabolic risk factor [2]. The risk of developing pancreatitis increases markedly when TG levels exceed 1,000 mg/dL, particularly at concentrations above 2,000 mg/dL, due to mechanisms involving chylomicron accumulation and free fatty acid-mediated tissue injury [3].

Necrotizing pancreatitis secondary to severe hypertriglyceridemia represents one of the most complex forms of the disease. Among its complications, the development of walled-off necrosis (WON) is particularly significant. WON is a mature, encapsulated collection of pancreatic and/or peripancreatic necrotic tissue that typically develops four or more weeks after the onset of necrotizing pancreatitis. Infection may develop in up to 40% of cases [4]. The presence of gas within the collection, fever, a pronounced inflammatory response, or organ failure may suggest infection, a condition associated with increased mortality and that may require invasive intervention [5]. The step-up approach, which may begin with endoscopic or percutaneous drainage followed by necrosectomy when clinically indicated, is an established minimally invasive strategy for the management of infected pancreatic necrosis [6,7].

Infected WON may involve a diverse range of microorganisms, with the microbiological profile of infected pancreatic necrosis previously characterized in the literature [8]. Although enteric microorganisms are frequently implicated, the isolation of organisms more commonly associated with the oral microbiota presents additional challenges in interpreting their potential pathogenic significance. Distinguishing clinically relevant infection from possible contamination requires careful consideration of the sampling method, microbiological findings, and overall clinical context.

The clinical relevance of documenting complex cases of infected WON extends beyond the identification of unusual microorganisms. Such cases may illustrate the diagnostic uncertainty involved in distinguishing infection of a previously established necrotic collection from recurrent acute pancreatitis, the challenges of interpreting polymicrobial culture findings, and the importance of individualized multidisciplinary management when cardiovascular complications arise during treatment. Reporting these clinical challenges may contribute to a more comprehensive understanding of the diagnostic and therapeutic considerations involved in managing complicated pancreatic necrosis.

Case Presentation

Case history and initial presentation

In August 2025, a 41-year-old man with a history of severe hypertriglyceridemia-induced necrotizing pancreatitis presented to the emergency department with abdominal pain, general malaise, and elevated inflammatory markers. His previous episode had been complicated by infected pancreatic necrosis requiring antimicrobial treatment. At the current presentation, imaging demonstrated an encapsulated peripancreatic necrotic collection with intralesional gas, raising concern for infected WON. Complete resolution of the previous necrotic collection before this admission could not be established from the available clinical records.

At readmission, the patient was febrile and tachycardic, with epigastric tenderness on physical examination. Laboratory evaluation revealed leukocytosis with neutrophilia, elevated C-reactive protein (CRP) and procalcitonin (PCT) levels, and a mild increase in serum creatinine. These findings raised concern for superinfection of the previously identified pancreatic collection. Admission laboratory and arterial blood gas findings are summarized in Table *1*.

Parameter	Patient value	Reference range	Unit
White blood cell count	18.52	4.5–11.0	×10³/μL
Neutrophils	83	43.0–70.0	%
Lymphocytes	7	17.4–48.2	%
Absolute neutrophil count	16.48	2.1–7.0	×10³/μL
Absolute lymphocyte count	1.3	1.0–4.0	×10³/μL
Erythrocyte sedimentation rate	36	0–15	mm/h
C-reactive protein (CRP)	301	<8.0	mg/L
Procalcitonin (PCT)	0.491	<0.046	ng/mL
Blood urea nitrogen (BUN)	32.9	6.0–20.0	mg/dL
Creatinine	1.34	0.67–1.17	mg/dL
Total amylase	83.1	28–100	U/L
Pancreatic lipase	35.3	23–190	U/L
pH	7.47	7.40–7.50	—
PaO_2	65	83–108	mmHg
$PaCO_2$	25	35–48	mmHg
Bicarbonate (HCO_3^-)	18.2	21–28	mmol/L
Oxygen saturation	92.4	94–98	%
Base excess	-3.6	−2.0 to 3.0	mmol/L
Lactate	1.2	0.5–1.6	mmol/L
Anion gap	16	8–12	mmol/L

TABLE 1: Initial laboratory and arterial blood gas findings at admission

Initial hematologic, inflammatory, renal, pancreatic, and arterial blood gas findings obtained at admission. Reference intervals were obtained from the institutional clinical laboratory and correspond to the laboratory-specific ranges used at the time of testing. CRP: C-reactive protein; PCT: procalcitonin; BUN: blood urea nitrogen; PaO_2: arterial partial pressure of oxygen; $PaCO_2$: arterial partial pressure of carbon dioxide; HCO_3^-: bicarbonate.

Diagnostic findings

During the initial evaluation following readmission, contrast-enhanced abdominal computed tomography was performed because of recurrent abdominal pain and elevated inflammatory markers. Imaging demonstrated a heterogeneous peripancreatic collection measuring approximately 12 × 7 × 10 cm (estimated volume, 420 mL), located between the pancreas and the posterior gastric wall. The collection contained dense material and intralesional gas, raising suspicion of superinfection. The pancreatic parenchyma showed homogeneous contrast enhancement without identifiable areas of necrosis. Associated inflammatory changes involved the surrounding fat and the splenic flexure of the colon. No vascular thrombosis, stenosis, or pseudoaneurysms were identified. Although the radiological report did not describe a pancreaticocolonic fistula, the available findings did not definitively exclude this possibility. Figure *1* illustrates the peripancreatic collection, intralesional gas, and surrounding inflammatory changes.

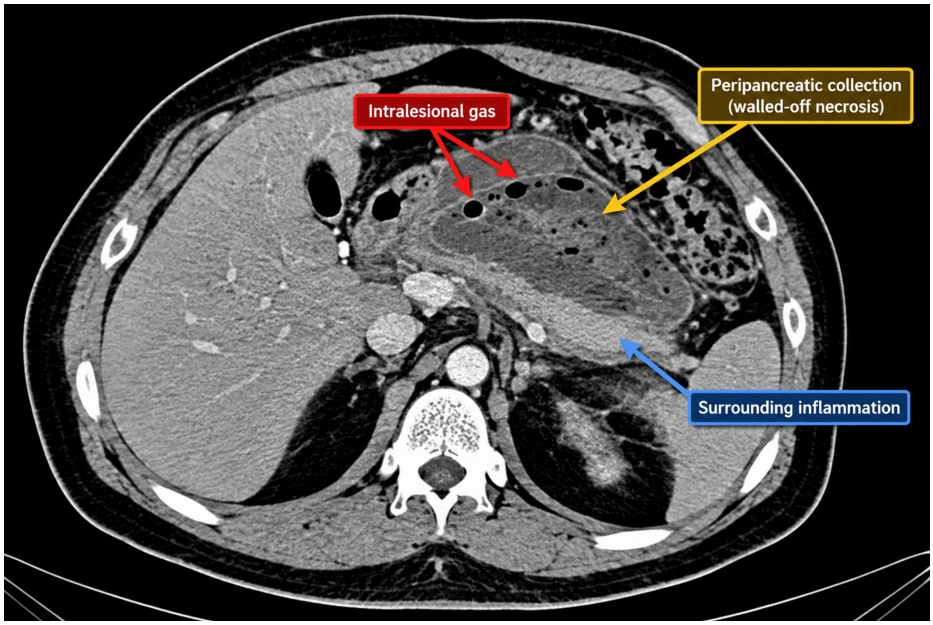


FIGURE 1: Contrast-enhanced abdominal computed tomography showing infected pancreatic walled-off necrosis

Axial contrast-enhanced computed tomography demonstrating a heterogeneous, encapsulated peripancreatic collection consistent with walled-off necrosis (yellow arrow). Intralesional gas foci (red arrows) and surrounding inflammatory changes (blue arrow) are also identified. The presence of intralesional gas supports the diagnosis of infected walled-off necrosis.

The patient had a history of severe hypertriglyceridemia associated with the preceding episode of necrotizing pancreatitis. During the current hospitalization, TG levels were monitored alongside inflammatory markers and other metabolic parameters. Figure *2* illustrates the available laboratory trends during the clinical course.

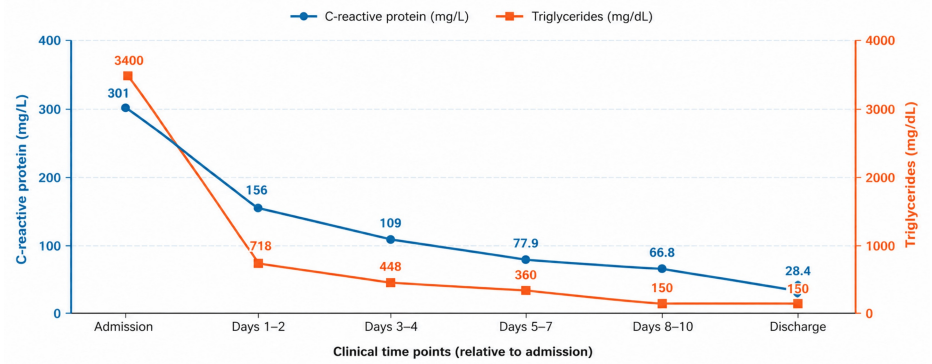


FIGURE 2: Evolution of C-reactive protein and triglyceride levels during hospitalization

Serial measurements of C-reactive protein (CRP) and triglyceride concentrations during hospitalization. CRP values are displayed on the left vertical axis (mg/L), and triglyceride values on the right vertical axis (mg/dL), using independent linear scales. The horizontal axis represents clinical assessment intervals relative to admission, without specifying calendar dates. Both parameters showed an overall decrease during hospitalization. These trends are descriptive and do not establish a causal relationship with specific therapeutic interventions.

The pancreatic parenchyma demonstrated homogeneous contrast enhancement without identifiable areas of necrosis. Together with the absence of elevated pancreatic enzymes, these findings did not establish a new episode of acute necrotizing pancreatitis. The current presentation was therefore characterized primarily by a peripancreatic collection with clinical and radiological findings suggestive of infection.

Treatment and clinical course

At readmission, intravenous metronidazole (500 mg every eight hours) was initially prescribed for suspected infection of the pancreatic collection. Following consultation with the Infectious Diseases team, metronidazole was discontinued and antimicrobial therapy was changed to intravenous ertapenem (1 g every 24 hours). Cultures obtained from the pancreatic collection subsequently identified Rothia mucilaginosa, Solobacterium moorei, and Peptostreptococcus anaerobius. Antimicrobial susceptibility testing was not performed. Given the clinical presentation and polymicrobial findings, including oral anaerobes, ertapenem was maintained as part of the antimicrobial management alongside endoscopic source control.

Endoscopic management was a key component of treatment. Following initial transgastric drainage of the pancreatic collection and collection of samples for microbiological analysis, two endoscopic necrosectomies were performed. During the first necrosectomy, most of the necrotic material was removed. The second procedure allowed further removal of residual necrotic and purulent material. A metal stent was maintained to preserve drainage through the cystogastric tract, together with an additional plastic stent. Following these interventions, the patient demonstrated clinical improvement, resolution of fever, and a progressive decrease in inflammatory markers.

During an endoscopic procedure, the patient developed an episode of tachycardia reaching approximately 190 beats/min, accompanied by transient hypotension. The episode was managed pharmacologically and resolved without requiring electrical cardioversion. An electrocardiographic tracing obtained during the tachycardia was unavailable, preventing definitive characterization of the arrhythmia mechanism. A subsequent electrocardiogram demonstrated a short PR interval and a delta wave, consistent with ventricular pre-excitation. Transthoracic echocardiography revealed preserved left ventricular systolic function (LVEF 68%), normal right ventricular systolic function, preserved left ventricular diastolic function, and no significant structural or valvular abnormalities. Following cardiology assessment, low-dose beta-blocker therapy was initiated. Subsequent 24-hour Holter monitoring demonstrated sinus rhythm, ventricular pre-excitation findings, and no significant tachyarrhythmias.

Metabolic complications and supportive care

During hospitalization, an episode of asymptomatic hypoglycemia was documented and treated with intravenous dextrose. Glycemic management included adjustment of insulin therapy according to glucose monitoring, with continuation of the patient's metabolic treatment as clinically indicated. The patient experienced transient hypotension associated with the endoscopic management, requiring intravenous fluid administration and close hemodynamic monitoring. Supportive care also included analgesics, antiemetics, gastroprotection, and adjustments to crystalloid fluid therapy.

Outcome

The clinical course was favorable. Following endoscopic interventions and continued antimicrobial therapy, CRP and PCT levels progressively decreased, renal function improved, and the patient achieved hemodynamic stability. At discharge, he was asymptomatic and afebrile, tolerating oral intake, with inflammatory markers continuing to decline. A total 15-day course of intravenous ertapenem (1 g every 24 hours) was planned, including six additional days after discharge. The patient was instructed to continue his lipid-lowering regimen, maintain cardiology follow-up for ventricular pre-excitation, and undergo follow-up endoscopy approximately 15 days later to assess the residual cavity and evaluate stent management.

Discussion

From a pathophysiological perspective, WON may develop following pancreatic and peripancreatic necrosis. The initial injury and necrosis of the pancreatic parenchyma and peripancreatic tissues, mediated by proteolytic enzymes, free radicals, and free fatty acids, are followed by progressive liquefaction of necrotic material. Over the following weeks, macrophages and fibroblasts contribute to the degradation of necrotic debris and the formation of granulation tissue around the collection, promoting progressive organization of its contents. During the early phase, an acute necrotic collection consists of fluid and necrotic material without a well-defined encapsulating wall. Over time, some of these collections develop a mature, well-defined wall, typically after four weeks, and are then classified as WON [9].

The development of a mature, encapsulating wall helps delineate the necrotic collection and facilitates the planning of minimally invasive interventions within the step-up approach, including transmural drainage and endoscopic necrosectomy when clinically indicated. These techniques are particularly relevant when infection occurs, a condition favored by avascular necrosis and bacterial translocation, for which the presence of intralesional gas on computed tomography may provide radiologic evidence [4,6,7].

In infected pancreatic necrosis, and particularly in the context of WON, the initiation of broad-spectrum antibiotics with adequate pancreatic penetration is a cornerstone of initial management when infection is suspected, especially when CT imaging demonstrates intralesional gas or when progressive clinical deterioration occurs. Carbapenems, quinolones combined with metronidazole, and third-generation

cephalosporins are recommended options, while the prophylactic use of antibiotics or antifungals has shown no benefit in preventing infection and is not routinely advised [7,10].

The contemporary microbiological profile of WON demonstrates a documented shift toward gram-positive cocci, including Enterococcus, coagulase-negative Staphylococcus, and Streptococcus, as well as a high frequency of Candida species, especially in patients previously exposed to antibiotics, who also have a higher risk of polymicrobial infection and treatment failure [8,11]. This pattern reflects the selective pressure generated by prior therapies and underscores the importance of obtaining targeted cultures during intervention, as pathogens isolated in WON may differ from those typically covered by traditional empiric regimens and may include unusual or opportunistic microorganisms, as in this case. Although the clinical and radiological findings, together with the recovery of microorganisms from the pancreatic collection, support the diagnosis of infected WON, the individual pathogenic contribution of each isolate cannot be definitively established. Furthermore, antimicrobial susceptibility testing was unavailable, limiting the microbiological assessment of the definitive antimicrobial regimen [11].

Several clinical trials have marked a decisive shift in the management of necrotizing pancreatitis by demonstrating the benefits of minimally invasive approaches in appropriately selected patients [12-14]. The magnitude of these findings is clinically significant: PANTER reported a reduction in major complications or death (40% vs. 69%) [12], while TENSION demonstrated a lower incidence of pancreaticocutaneous fistulas (5% vs. 32%) [13]. Similarly, MISER reported lower rates of pancreaticocutaneous fistulas (0% vs. 28%) and major complications (12% vs. 41%) with endoscopic management [14]. Long-term follow-up studies of PANTER and TENSION have further contributed to the understanding of the sustained outcomes associated with these treatment strategies [15,16]. Collectively, these findings support the use of minimally invasive approaches in appropriately selected patients with infected pancreatic necrosis, although the comparative benefits depend on the intervention and outcomes evaluated. In the present case, clinical improvement following endoscopic drainage, two necrosectomies, and antimicrobial treatment was consistent with the established role of the step-up approach, without establishing the independent contribution of each intervention.

The incidental identification of ventricular pre-excitation consistent with Wolff-Parkinson-White syndrome added complexity to the patient's clinical management. During an endoscopic procedure, the patient developed narrow-complex tachycardia accompanied by transient hypotension. Although subsequent electrocardiographic findings demonstrated ventricular pre-excitation, the absence of an electrocardiographic tracing during the acute episode prevented definitive identification of the arrhythmia mechanism. This finding highlights the importance of cardiovascular monitoring and individualized assessment during invasive procedures in patients with suspected accessory conduction pathways [17].

Importantly, according to current arrhythmia guidelines, several medications commonly used to treat supraventricular tachyarrhythmias can be dangerous in patients with accessory pathways, particularly during episodes of pre-excited atrial fibrillation. Agents that block atrioventricular (AV) nodal conduction-such as verapamil, diltiazem, adenosine, digoxin, and beta-blockers-as well as intravenous amiodarone, are contraindicated in this specific setting because they may promote rapid conduction through the accessory pathway and precipitate ventricular fibrillation. This consideration is especially relevant during endoscopic procedures or periods of physiological stress, when an arrhythmia may arise unexpectedly. In this patient, low-dose beta-blocker therapy was initiated following cardiology assessment. However, the contraindication of AV nodal blocking agents in pre-excited atrial fibrillation should not be generalized to all supraventricular tachyarrhythmias associated with ventricular pre-excitation; treatment decisions depend on the specific arrhythmia and clinical circumstances [18].

Several limitations should be acknowledged. First, although the patient had a documented history of hypertriglyceridemia-associated necrotizing pancreatitis, the available findings did not conclusively establish a new episode of acute necrotizing pancreatitis, and complete resolution of the previous collection could not be confirmed. Second, antimicrobial susceptibility testing was unavailable, limiting the interpretation of the microbiological findings and the assessment of antimicrobial activity against individual isolates. Third, the absence of an electrocardiographic tracing during the tachycardia prevented definitive characterization of its mechanism. Finally, the documented favorable outcome was limited to the hospitalization and discharge period, without sufficient long-term follow-up to establish sustained recovery. Further investigation is warranted to clarify the clinical significance of uncommon microorganisms isolated from infected pancreatic collections and their implications for antimicrobial management.

Taken together, this case illustrates the inherent clinical complexity of infected pancreatic WON and highlights the importance of integrating pathophysiological understanding, rational antimicrobial management, an evidence-based step-up approach, and multidisciplinary care. The patient's favorable in-hospital clinical evolution following transmural drainage, serial necrosectomies, and antimicrobial treatment is consistent with the established role of minimally invasive management as an effective therapeutic option for infected WON in appropriately selected patients. Furthermore, this case emphasizes the importance of carefully interpreting microbiological findings and assessing cardiovascular conditions that may influence procedural safety and therapeutic decisions.

Conclusions

This case highlights the favorable in-hospital clinical outcome of a patient with infected pancreatic WON managed through a multidisciplinary approach combining antimicrobial therapy, endoscopic drainage, and sequential necrosectomies. The findings are consistent with the established role of minimally invasive step-up management in appropriately selected patients. Although the isolation of uncommon microorganisms raises important microbiological considerations, their individual pathogenic significance remains uncertain. Given the limitations of a single case report and the absence of long-term follow-up, further investigation is needed to clarify the clinical relevance of these organisms and the long-term outcomes of similar patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Esteban Zavaleta-Monestel, Daniel Odio Cortes, Kevin Cruz Mora, Jeaustin Mora-Jiménez

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Drafting of the manuscript: Kevin Cruz Mora, Jeaustin Mora-Jiménez

Disclosures

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