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Hemorrhagic Pancreatitis Leading to Pneumoperitoneum and Septic Shock: A Rare Surgical Mimic

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Abstract

Hemorrhagic pancreatitis is an uncommon but potentially fatal complication of acute pancreatitis, as deterioration can be very rapid. The occurrence of pneumoperitoneum without evidence of hollow viscus perforation or pancreatic gas gangrene further represents exceptional diagnostic and surgical dilemmas. We report a rare case of a 70-year-old man with a history of chronic pancreatitis complicated by pancreatic duct stenosis who presented with epigastric and right hypochondrial pain, elevated pancreatic enzymes, and imaging supporting acute-on-chronic pancreatitis. The patient had progressive clinical deterioration despite normalization of serum pancreatic enzyme levels. Repeat imaging demonstrated extensive pneumoperitoneum, raising concern for perforated viscus, and the patient underwent emergency exploratory laparotomy. Surgery revealed extensive infected hemorrhagic fluid, multiple loculated collections, pyogenic membranes, and diffuse hemorrhagic pancreatitis, but no gastrointestinal (GI) perforation was identified. Despite intensive supportive management and surgical intervention, the patient developed progressive multiorgan failure and died. This case highlights the limitations of relying on pancreatic enzyme trends to assess disease progression and demonstrates that pneumoperitoneum may rarely occur in severe complicated pancreatitis without an identifiable GI perforation or definitive source of free intraperitoneal air. Clinical status, inflammatory markers, imaging findings, and hemodynamic changes should therefore be integrated rather than relying on pancreatic enzyme trends alone when evaluating patients with severe pancreatitis.

Categories: General Surgery, Internal Medicine, Gastroenterology
Keywords: acute-on-chronic pancreatitis, case report, hemorrhagic pancreatitis, infected pancreatic necrosis, pneumoperitoneum, septic shock

Introduction

Acute pancreatitis (AP) is an inflammatory condition of the pancreas that ranges in severity from a self-limiting illness to a life-threatening disease associated with pancreatic necrosis, systemic inflammation, and multiorgan failure [1,2]. Although most patients recover without major complications, approximately 20-30% develop severe disease, in which persistent organ failure and local complications contribute substantially to morbidity and mortality [2,3].

Hemorrhagic complications are uncommon but potentially fatal manifestations of pancreatitis. They may occur when inflammation and pancreatic enzymes damage adjacent blood vessels, sometimes resulting in pseudoaneurysm formation and rupture, or when extensive inflammatory and necrotic tissue damage produces diffuse bleeding [4,5]. The coexistence of hemorrhage and infection can further complicate management because patients may develop severe systemic inflammation, hemodynamic instability, and organ dysfunction.

Infected pancreatic necrosis is another serious complication of acute pancreatitis. Disruption of pancreatic tissue and the development of infected collections can contribute to persistent inflammation, sepsis, and clinical deterioration. Pneumoperitoneum, defined as free gas within the peritoneal cavity, usually raises concern for perforation of a hollow gastrointestinal viscus, particularly in patients with peritonitis or hemodynamic instability. Rare reports have nevertheless described pneumoperitoneum in severe necrotizing or emphysematous pancreatitis without an identifiable gastrointestinal (GI) perforation [3,6]. Although gas within pancreatic or peripancreatic tissues may support a diagnosis of infected necrosis in the appropriate clinical context, the presence of free intraperitoneal air alone does not establish its source or mechanism.

We report a fatal case of acute-on-chronic pancreatitis complicated by diffuse hemorrhagic findings, infected intra-abdominal collections, extensive pneumoperitoneum, and septic shock, in which exploratory laparotomy did not identify a GI perforation. The case also illustrates the discrepancy between declining serum pancreatic enzyme levels and worsening clinical status. It emphasizes the importance of integrating

clinical examination, inflammatory markers, imaging findings, and hemodynamic trends when assessing patients with complicated pancreatitis, while recognizing that the mechanism of pneumoperitoneum in this case could not be definitively established.

Case Presentation

A 70-year-old male with a previous history of cigarette and alcohol use presented with a one-day history of epigastric and right hypochondrial pain. The pain increased after meals and was accompanied by nausea. The patient denied vomiting, diarrhea, fever, or any respiratory or urinary symptoms. His medical history was significant for hypertension, diabetes mellitus, and a permanent tracheostomy following a total laryngectomy for laryngeal cancer. He had also been admitted six months earlier with acute pancreatitis, during which endoscopic retrograde cholangiopancreatography (ERCP) revealed no stones but identified pancreatic duct stenosis, which was managed with sphincterotomy, drainage, and stenting.

Initial vital signs were stable, and the patient was afebrile. He was not in general distress but was experiencing pain. Abdominal examination revealed a soft, nondistended abdomen with mild tenderness localized to the epigastric and hypochondrial areas, but no rebound tenderness was present.

Upon admission, initial laboratory investigations, as shown in Table *1*, revealed an elevated white blood cell (WBC) count of 13,000/µL and a C-reactive protein (CRP) level of 8.9 mg/L. Pancreatic enzymes were markedly elevated, with amylase at 1,799 U/L and lipase at 2,562 U/L. Hemoglobin, creatinine, alanine aminotransferase, aspartate aminotransferase, and bilirubin levels were normal. An initial abdominal ultrasound indicated slightly coarse liver parenchyma, suggestive of chronic liver disease, and bowel gaseous distension that obscured the pancreas. Magnetic resonance cholangiopancreatography (MRCP) performed on the following day showed a dilated pancreatic duct with a beaded appearance suggesting chronic pancreatitis, alongside a prominent common bile duct with no stones and no signs of active inflammation (Figure *1*).

Test	Result	Reference range	Interpretation
Serum lipase	2,562 U/L	12-53 U/L	↑ (Elevated)
Serum amylase	1,799 U/L	30–118 U/L	↑ (Elevated)
WBC	13,000/µL	4,000–11,000/µL	↑ (Elevated)
CRP	8.9 mg/L	0–8 mg/L	↑ (Slightly elevated)
Hemoglobin	14 g/dL	13.0–17.0 g/dL	Normal
Creatinine	0.98 mg/dL	0.7–1.3 mg/dL	Normal
ALP	122 U/L	40–129 U/L	Normal

TABLE 1: Laboratory Results on Admission

↑ = above reference range
WBC: white blood cell; CRP: C-reactive protein; ALP: alkaline phosphatase.

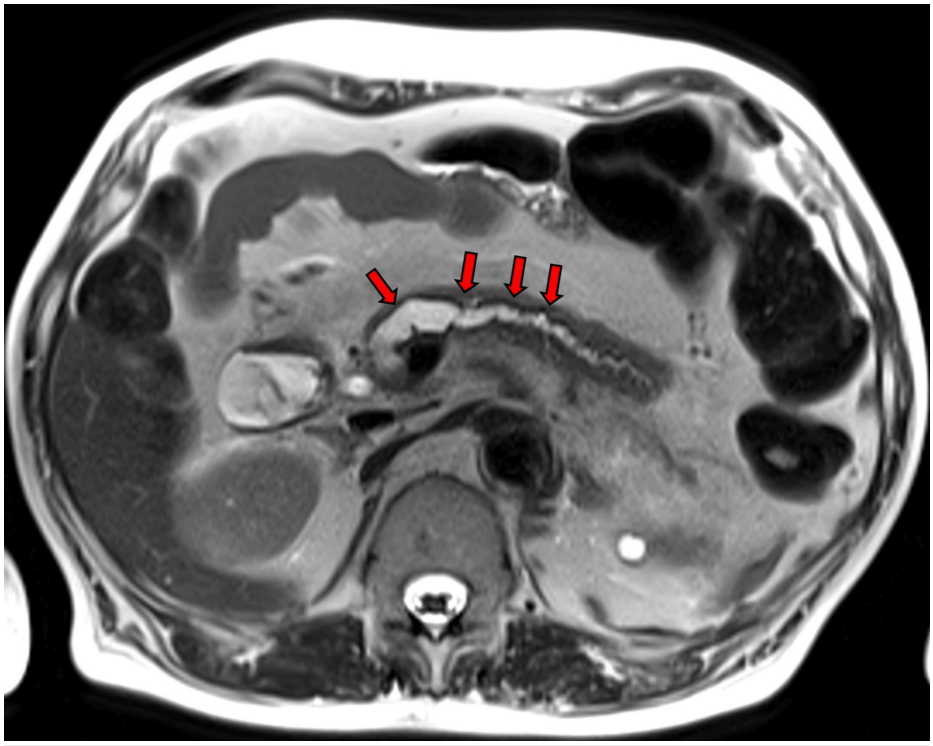


FIGURE 1: Magnetic resonance cholangiopancreatography (MRCP) showing a dilated pancreatic duct with a beaded appearance, suggesting chronic pancreatitis.

The patient was initially kept nil by mouth, started on intravenous (IV) fluids and an empirical course of Piperacillin-tazobactam for suspected sepsis. Between the second and fourth days, the patient was maintained on a clear fluid diet, and his pancreatic enzymes improved. A follow-up contrast-enhanced computed tomography (CT) scan of the abdomen performed on the fifth day revealed a swollen, edematous pancreas with extensive peripancreatic fat stranding and multiple parenchymal calcifications, suggesting acute-on-chronic pancreatitis (Figure *2*).

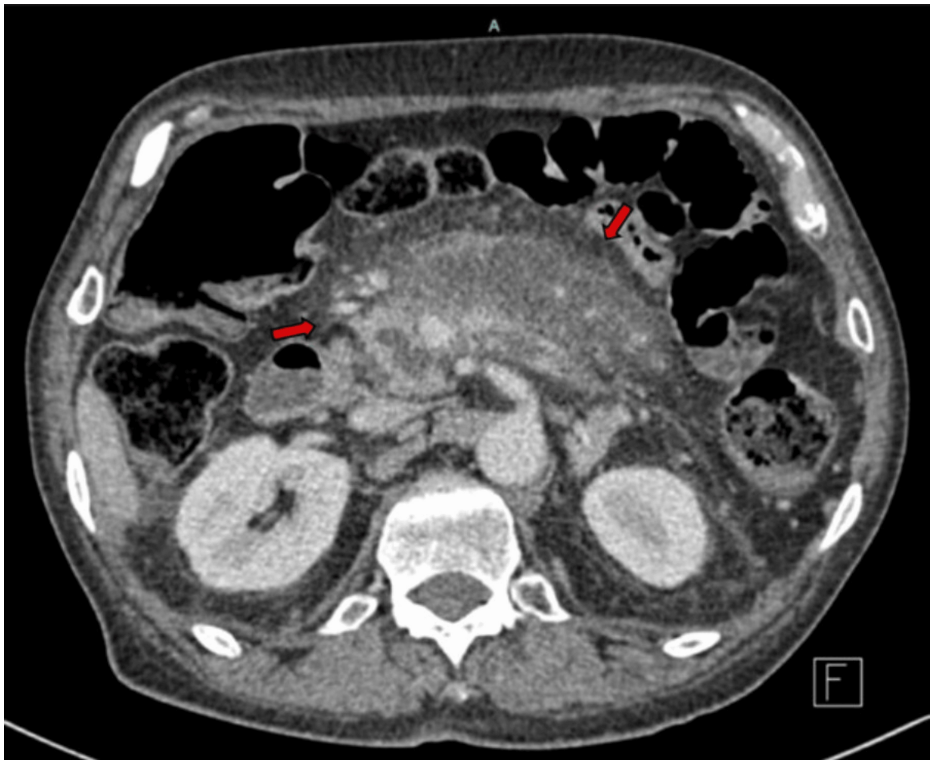


FIGURE 2: Contrast-enhanced CT of the abdomen (venous phase) showing a swollen, edematous pancreas with extensive surrounding fat stranding.

During the first week of admission, he continued to experience abdominal pain and was unable to tolerate oral intake. By the second week of admission, despite his pancreatic enzymes normalizing, he was still experiencing abdominal pain. At the end of the second week, the patient developed recurrent hypotensive episodes. Despite repeated resuscitations with IV fluids, the patient remained tachypneic, tachycardic, and hypotensive, concerning for septic shock, and was therefore shifted to the intensive care unit (ICU) for further management. In the ICU, the patient remained critically ill and developed abdominal distension with generalized tenderness and was referred for urgent surgical assessment. A repeat non-contrast CT scan of the abdomen demonstrated extensive pneumoperitoneum with free intra-abdominal air consistent with a perforated intra-abdominal viscus (Figure *3*). In addition, there was loculated abdominopelvic ascites with superimposed infection and possible abscess formation, together with edematous pancreatic parenchyma, peripancreatic fat stranding, pancreatic duct dilatation, and multiple calcifications.

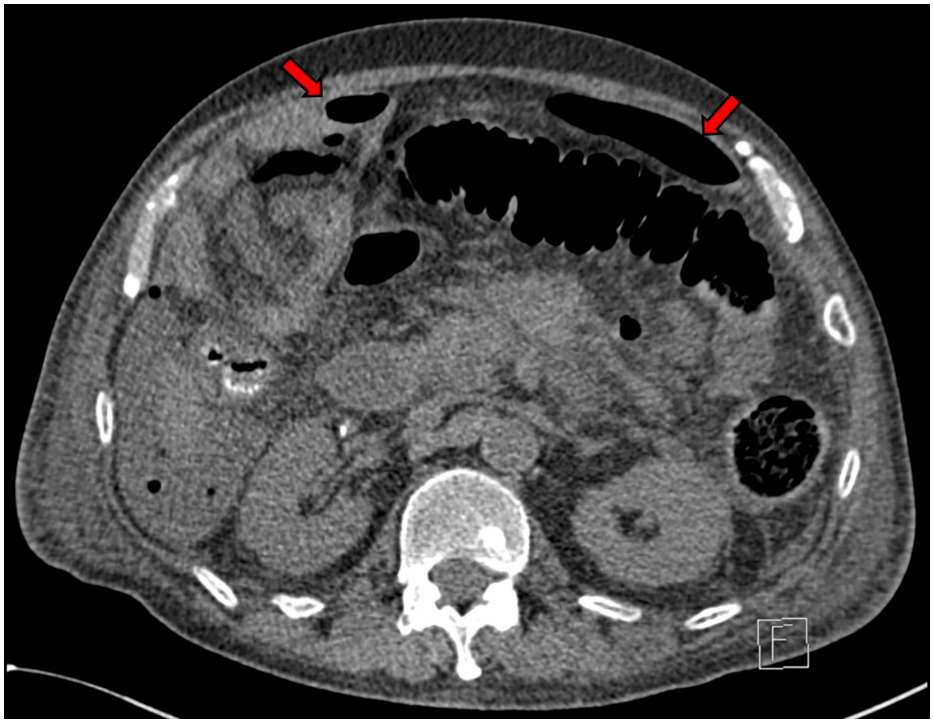


FIGURE 3: Non-contrast CT of the abdomen showing pneumoperitoneum with free intra-abdominal air.

The patient had an urgent exploratory laparotomy. Intraoperatively, a large amount of hemorrhagic, infected fluid was identified, with multiple loculations involving the subphrenic spaces, pelvis, and areas between the small intestinal loops. Extensive pyogenic membranes were noted covering the small intestinal loops, sigmoid colon, and cecum. Hemorrhagic pancreatitis was identified at the transverse mesocolon adjacent to the duodenojejunal flexure, with active oozing of hemorrhagic fluid. Despite the preoperative CT findings suggestive of perforated viscus, no bowel perforation was identified intraoperatively. Postoperatively, the patient remained critically ill and required continued hemodynamic and respiratory support. Two days later, and despite maximal supportive management, the patient remained in critical clinical condition and subsequently developed cardiac arrest. Cardiopulmonary resuscitation was initiated immediately according to advanced cardiac life support protocol, but return of spontaneous circulation was not achieved, and the patient was pronounced dead.

Discussion

Hemorrhagic pancreatitis is an uncommon but highly fatal complication of acute pancreatitis [4,7]. Reported incidence ranges from 1% to 23%, with mortality exceeding 50% once bleeding occurs [4,7,8]. In a cohort study that was done in India on 183 patients with severe acute pancreatitis, 13% of them developed a hemorrhagic complication, and this group had a considerably worse disease course as compared to those without bleeding. The likelihood of surgery and the death rate were four times greater in the hemorrhagic group (50% versus 12.6% and 41.7% versus 10.7%, respectively) [9].

Hemorrhagic complications secondary to pancreatitis might result from damage to the nearby vascular structures through ongoing inflammation and enzymatic action. Local infection, activated pancreatic enzymes, and necrosis can erode blood vessel walls, producing pseudoaneurysms or causing diffuse hemorrhage from multiple arteries [4,5,7]. Infected necrosis appears to contribute greatly to this condition by accelerating the rate of vascular wall degradation, which makes it the strongest independent predictor of hemorrhage in previous research [9].

Our patient had several features associated with a complicated disease course, including advanced age, diabetes mellitus, and documented chronic pancreatitis with ductal stenosis that had been treated with sphincterotomy and stenting six months earlier. The current presentation therefore represented an acute exacerbation of established chronic pancreatitis. The Revised Atlanta Classification stratifies severity by the presence and duration of organ failure and by local or systemic complications into mild (neither), moderately severe (transient organ failure lasting less than 48 hours and/or local or systemic complications), and severe (persistent organ failure beyond 48 hours) [10]. Our patient met the criteria for severe acute pancreatitis, with cardiovascular and respiratory failure persisting for more than 48 hours, together with local complications in the form of infected, hemorrhagic peritoneal and peripancreatic collections.

An important feature of this case was the discordance between the decline in serum pancreatic enzyme levels and the patient's worsening clinical condition. Serum amylase decreased from 1,799 U/L on admission to 121 U/L on hospital day 13, while lipase decreased from 2,562 U/L to 24 U/L. However, CRP increased from 8.9 mg/L at admission to 371.3 mg/L on day 7 and remained markedly elevated at 340 mg/L on day 13 (Table *2*). These trends occurred in the context of persistent abdominal pain, poor oral intake, subsequent hypotension, and extensive infected hemorrhagic collections identified at surgery.

Parameter	Day 1	Day 4	Day 7	Day 13	Reference Range
Serum amylase	1,799	294	476	121	30–118 U/L
Serum lipase	2,562	226	212	24	12–53 U/L
CRP	8.9	298	371.3	340	0–8 mg/L

TABLE 2: Serial trend of pancreatic enzymes and C-reactive protein (CRP) during hospitalization.

Note: Serum amylase and lipase levels decreased substantially by hospital day 13, whereas CRP remained markedly elevated. The decline in pancreatic enzyme levels did not correspond to clinical improvement, as the patient continued to experience abdominal pain and subsequently developed hemodynamic instability.

Serum amylase and lipase are useful in establishing the diagnosis of acute pancreatitis, but their concentrations do not reliably reflect disease severity or the extent of pancreatic tissue injury. A decline in these enzymes therefore cannot be interpreted as evidence that the inflammatory process or local complications have resolved. Conversely, persistent CRP elevation indicates ongoing systemic inflammation but cannot independently establish infection or pancreatic necrosis. In this case, the combination of persistent symptoms, worsening hemodynamic status, markedly elevated CRP, abnormal repeat imaging, and operative findings was more clinically informative than the pancreatic enzyme trajectory alone. This case reinforces the importance of repeated clinical assessment and timely reassessment of imaging when patients with pancreatitis fail to improve as expected [1,2]. In this patient, the markedly elevated CRP was consistent with ongoing systemic inflammation; however, CRP elevation alone does not establish infected pancreatic necrosis.

The severity of the disease became particularly apparent at laparotomy. Operative findings demonstrated large-volume infected hemorrhagic fluid, multiple loculated collections, pyogenic membranes, and diffuse hemorrhagic oozing from the pancreatic bed. No discrete pseudoaneurysm or actively bleeding vessel was identified. This finding is clinically relevant because pseudoaneurysms and focal arterial bleeding may provide a target for endovascular embolization, whereas diffuse hemorrhage associated with extensive necrosis and infection may not have a clearly identifiable vascular target for either embolization or surgical control [4,9]. In this patient, the diffuse nature of the hemorrhage may have been related to extensive inflammatory and necrotic tissue destruction and may have contributed to the difficulty in achieving definitive control of the bleeding despite operative intervention.

The presence of pneumoperitoneum on CT scan prompted urgent surgical evaluation. In a patient with severe pancreatitis who was developing refractory septic shock, pneumoperitoneum raises significant concern for perforation of a hollow viscus, particularly when accompanied by diffuse peritonitis. Imaging alone cannot reliably exclude GI perforation in an unstable patient. The World Society of Emergency Surgery (WSES) recommendations support immediate operative intervention in patients with pneumoperitoneum, diffuse peritonitis, and septic shock when perforated hollow viscus is suspected [11]. Similarly, ERAS Society emergency laparotomy guidelines emphasize the importance of timely surgical intervention in patients with sepsis or septic shock, as delays may be associated with poor outcomes and increased mortality [12]. In our patient, the combination of pneumoperitoneum, diffuse peritonitis, hypotension, vasopressor-dependent septic shock, and concern for a perforated viscus therefore provided a strong indication for emergency exploratory laparotomy.

Despite careful intraoperative inspection of the stomach, small bowel, and colon, no GI perforation was identified. The operation instead revealed extensive infected hemorrhagic fluid, multiple loculated collections, pyogenic membranes, and hemorrhagic changes adjacent to the pancreas. These findings provided a plausible explanation for the patient's severe intra-abdominal inflammatory condition but did not establish the source of the pneumoperitoneum. Although the discrepancy between preoperative imaging and operative findings is uncommon, a similar phenomenon has been reported previously. Stockinger and Corsetti described a patient with fulminant pancreatic necrosis, extensive gas within the pancreas, and pneumoperitoneum who underwent emergency laparotomy for presumed hollow-viscus perforation; no perforation was identified, and the free intraperitoneal air was ultimately attributed to emphysematous pancreatitis caused by Clostridium perfringens [6]. Our case differs from that report because there was no operative evidence of pancreatic gas gangrene and no gas-forming organism was identified. Consequently,

the precise mechanism responsible for the pneumoperitoneum in our patient remains uncertain. Potential explanations include an unconfirmed gas-forming infection, rupture of an infected pancreatic or peripancreatic collection, or extension of gas from the retroperitoneum into the peritoneal cavity through disrupted inflammatory tissue planes. None of these mechanisms could be established definitively, and the source of the free intraperitoneal air therefore remains speculative.

Management of hemorrhagic pancreatitis depends on the hemodynamic status of the patient and the suspected source of bleeding. CT angiography can identify arterial bleeding or pseudoaneurysms and facilitate endovascular embolization in appropriately selected, hemodynamically stable patients. In contrast, operative management may be required in patients with hemodynamic instability, suspected GI perforation, or a need for urgent surgical source control [2,13]. In our patient, emergency laparotomy allowed assessment for a perforated viscus and drainage and lavage of the extensive infected collections. Despite operative source control and intensive treatment with broad-spectrum antibiotics, the patient subsequently developed multiorgan failure. Organ failure is the main factor that defines severe acute pancreatitis and is highly correlated with death, especially if there is involvement of several organ systems. According to the American College of Gastroenterology guideline, factors that determine severity include advanced age, comorbidities, Systemic Inflammatory Response Syndrome (SIRS), pancreatic necrosis, and signs of organ dysfunction [2]. The development of multiorgan failure in this instance was likely related to the cumulative effects of extensive pancreatic necrosis, hemorrhage, infection, septic shock, and systemic inflammation, ultimately resulting in a fatal disease course.

Conclusions

Hemorrhagic necrotizing pancreatitis is a life-threatening complication of acute pancreatitis. This case highlights that normalization of serum pancreatic enzyme levels does not necessarily indicate clinical improvement and that persistent or worsening symptoms warrant careful reassessment and consideration of repeat imaging. Pneumoperitoneum was observed in the setting of suspected infected pancreatic necrosis without an identifiable GI perforation, illustrating a rare presentation that may mimic a perforated viscus. However, the underlying mechanism of pneumoperitoneum remains uncertain, and the diagnosis of infected pancreatic necrosis should be interpreted in light of the available microbiological and histopathological evidence. As this report describes a single case, these observations cannot establish causality or be generalized to all patients with severe pancreatitis. Nevertheless, the case emphasizes the importance of integrating clinical findings, inflammatory markers, imaging results, and hemodynamic status rather than relying solely on pancreatic enzyme levels. Early recognition of atypical presentations and timely escalation of management are essential in the care of patients with severe, complicated pancreatitis.

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: Ameen A. Al Borom, Thamer Samha, Haneen R. Alhaj Younes, Shahd F. Alqudah, Yahia Alassi

Supervision: Thamer Samha

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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