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Single Case

Severe Acute Pancreatitis Complicated by Multiple Intra-Abdominal Hemorrhages

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Keywords
Severe acute pancreatitis · Intra-abdominal hemorrhage · Digital subtraction angiography · Pseudoaneurysm · Case report

Abstract
Introduction: Intra-abdominal hemorrhage is a rare yet life-threatening complication of acute pancreatitis (AP), with a higher prevalence in cases of severe acute pancreatitis (SAP). This condition is primarily caused by vessel wall erosion and rupture of pseudoaneurysms (PSAs). However, SAP cases involving multiple sequential arterial hemorrhages are extremely rare. This condition is primarily brought on by the process of vessel wall degeneration and the development of PSAs. Nonetheless, SAP necessitating multiple episodes of arterial bleeding is very uncommon. ***Case Presentation:*** Here is the case history of a 31-year-old man already being treated for SAP. His condition was then complicated by massive, frequent intra-abdominal bleeding. The patient initially presented to the hospital with SAP. He was transferred to the intensive care unit for proper management. Massive intra-abdominal bleeds occurred on the 31st, 45th, and 60th days during hospitalization. The maximum blood loss was 1,500 mL. In each of the instances, digital subtraction angiography (DSA) embolization was carried out after the bleeding source had been verified. In order to manage SAP, continuous percutaneous drainage and staged pancreatic necrosectomy were undertaken for 6 months. No recurrence of intra-abdominal hemorrhage was detected. Infection of the abdominal cavity was properly controlled. The patient left the hospital in good condition. ***Conclusion:*** Spontaneous bleeding in the abdominal cavity is a severe and life-threatening complication of SAP. This is often caused by vessel wall erosion. In such cases, DSA plays a crucial role in diagnosis and management. Besides precisely locating the

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Case Rep Gastroenterol 2025;19:79–88
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bleeding source, one can perform a much-needed embolization immediately. Consequently, the disease is under total control, and the patient is much more likely to survive.

Introduction

Severe acute pancreatitis (SAP) represents a critical subset of acute pancreatitis (AP), characterized by systemic or local complications and a mortality rate ranging from 8% to 39% [1]. Intra-abdominal hemorrhage is a serious complication of SAP. Although it is very rare, it can occasionally be fatal. This has been reported to occur in 1%–23% of SAP patients [2–4]. This demonstrates differences in disease severity, diagnostic criteria, and treatment strategies. Factors determining the outcome of intra-abdominal hemorrhage include the CT severity index and the serum creatinine concentration [5].

The principal mechanism of intra-abdominal bleeding in SAP is damage to the integrity of the vessel walls due to erosion. It is particular a result of the aggressive proteolytic activity of pancreatic enzymes. Ultimately, this leads to the rupture of a pseudoaneurysm (PSA). Although PSA formation is often associated with the presence of a pseudocyst, it can also occur independently. Bleeding events are commonly managed with endovascular interventions, such as embolization or stent placement, which have reported success rates of 70%–90% [6, 7]. Nevertheless, the complex pathophysiology of SAP requires vascular embolization to be integrated as a component of a broader, individualized treatment strategy. Given the multifaceted etiology and management of SAP-associated hemorrhage, this case report highlights the challenges and therapeutic strategies in addressing recurrent massive intra-abdominal bleeding in a patient with SAP.

Case Presentation

Presenting Concerns

A 31-year-old man presented to the Outpatient Clinic of Tianjin Nankai Hospital (China) with a history of worsening epigastric pain over 14 h. Initially intermittent and dull, the pain became increasingly severe over time and was accompanied by nausea and vomiting. The patient had a body mass index (BMI) of 30.1, classifying him as obese, a known risk factor for AP. Notably, he had no history of gallstones, alcohol consumption, or other significant comorbidities. His overall medical history was unremarkable.

Clinical Findings

On admission, the patient exhibited laboratory and clinical findings indicative of severe disease. Blood tests revealed a serum amylase level of 2,521 U/L (normal range: 35–135 U/L), urine amylase of 10,974 U/L (47–458 U/L), and a markedly elevated triglyceride concentration of 10.9 mmol/L (0–1.7 mmol/L). Additional investigations showed a serum creatinine level of 282.0 μmol/L (44–133 μmol/L), total cholesterol of 7.35 mmol/L, and blood glucose of 21.85 mmol/L. Blood gas analysis demonstrated a pH of 7.268, potassium of 6.11 mmol/L, and calcium of 0.826 mmol/L. Clinically, the patient presented with abdominal distension, a distressed appearance, and lethargy.

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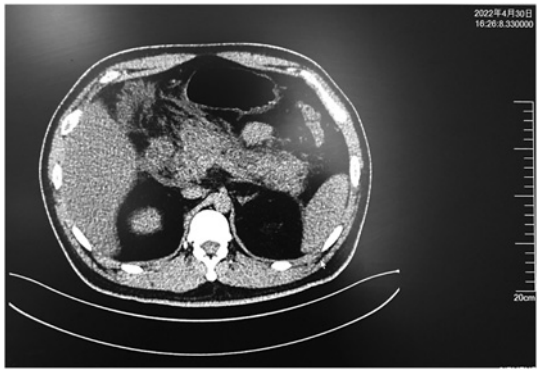

Fig. 1. The first non-contrast-enhanced CT, the pancreas was enlarged with slightly reduced density and surrounded by free fluid.

A non-contrast enhanced computed tomography (CT) scan (Fig. 1) performed during the initial outpatient visit revealed diffuse pancreatic enlargement, peripancreatic fluid collections, and reduced pancreatic density, findings consistent with AP. Although contrast-enhanced CT (CECT) would have provided more detailed imaging of vascular and pancreatic structures, it was not performed due to the patient's unstable condition. Non-contrast CT was considered sufficient for diagnosis, given its proven ability to identify pancreatic necrosis and peripancreatic fluid collections, as supported by institutional experience. After excluding biliary and alcohol-related causes, a diagnosis of hypertriglyceridemia-induced AP was established based on the patient's metabolic profile, obesity (BMI: 30.1), and clinical presentation.

The patient's condition rapidly deteriorated, with signs of acute respiratory distress syndrome, acute renal failure, and persistent abdominal infection, necessitating immediate transfer to the intensive care unit (ICU). ICU management included central venous catheterization, enteral nutrition, and tracheotomy, the latter being performed due to the need for prolonged mechanical ventilation, poor oxygenation (PaO2/FiO2 <200), and repeated airway clearance under sedation. After stabilization, the patient showed clinical improvement and was transferred to the surgical ward on day 18 post-admission. A timeline of clinical events and key interventions is summarized in Table 1.

Initially, upon returning to the surgical ward, the patient's condition appeared stable, with no signs of hemorrhage. On day 23 post-admission, the patient underwent the first staged pancreatic necrosectomy to address abdominal infection and reduce the necrotic tissue burden. This procedure was performed via three extended percutaneous catheter drainage pathways, which had been established earlier during the ICU stay.

Preoperative CT imaging revealed extensive necrosis in the pancreatic tail and surrounding regions, guiding the surgical approach and drainage strategies (Fig. 2). The left retroperitoneal abscess cavity was accessed through the original left percutaneous catheter drainage route, and a 5 cm incision was made to expose the cavity. Using video-guided cholangioscopy, necrotic tissue and purulent fluid were removed with forceps, and the cavity was irrigated with saline. Similarly, the lesser sac region was approached through the gastrocolic ligament, where extensive pancreatic and peripancreatic necrosis was debrided under cholangioscopy guidance, and two drainage tubes were placed. Finally, the right retroperitoneal abscess cavity was addressed with necrotic tissue removal, saline irrigation, and the placement of three drainage tubes.

Postoperative non-contrast CT imaging, reconstructed in 3D, provided a detailed overview of the abdominal drainage tube placement (Fig. 3). Subsequently, the patient was managed with low-pressure continuous saline irrigation of the abdominal cavities through the

Table 1. Time courses of this case

Time since SAP on	Clinical events
1 day	Transfer to ICU
	PICC
2 days	Started jejunal nutrition
6 days	Left and right retroperitoneal percutaneous drainage
	• Left side: 300 mL dark red fluid drained • Right side: 800 mL dark red fluid drained
7 days	Percutaneous tracheostomy
11 days	Pancreatic head, peripancreatic, and pancreatic tail percutaneous drainage
	• Pancreatic head: 30 mL light bloody fluid drained • Peripancreatic: 60 mL yellow-brown purulent fluid drained • Pancreatic tail: 50 mL brown purulent fluid drained
1 month	Twice slight intra-abdominal hemorrhages
	DSA examination and treatment
2 months	Severe intra-abdominal hemorrhages caused by rupture of splenic artery
	Massive blood transfusion and DSA treatment
1 month–6 months	Multiple pancreatic necrosectomy (14 times)
6 months	Hospital discharge

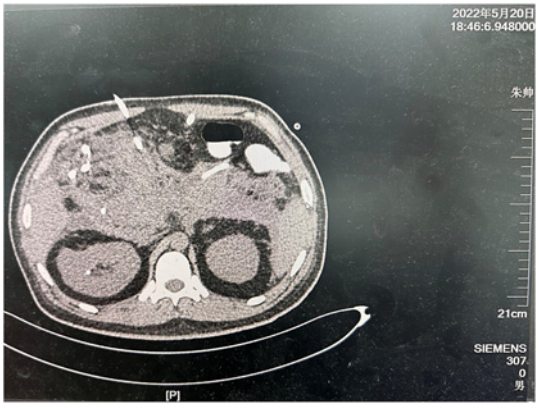

Fig. 2. Preoperative CT imaging revealed extensive necrosis in the pancreatic tail and surrounding regions, which guided the surgical approach and drainage strategies.

drainage tubes to minimize the accumulation of necrotic tissue and purulent material. Despite initial expectations for gradual improvement, the patient's clinical condition remained guarded in the week following the procedure, with persistent signs of systemic inflammation and abdominal discomfort.

Emergencies Focus and Assessment

On day 31 and day 45 post-admission, the patient experienced sudden bleeding from the abdominal incision and drainage tubes. Visible blood flow was noted, with approximately 400 mL of blood lost on both occasions. Notably, the patient did not report significant abdominal pain during these episodes. Suspecting intra-abdominal hemorrhage, emergency compression hemostasis was attempted, but it proved to be of limited efficacy. Hemostatic agents, including norepinephrine and hemocoagulase, were administered for local hemostasis.

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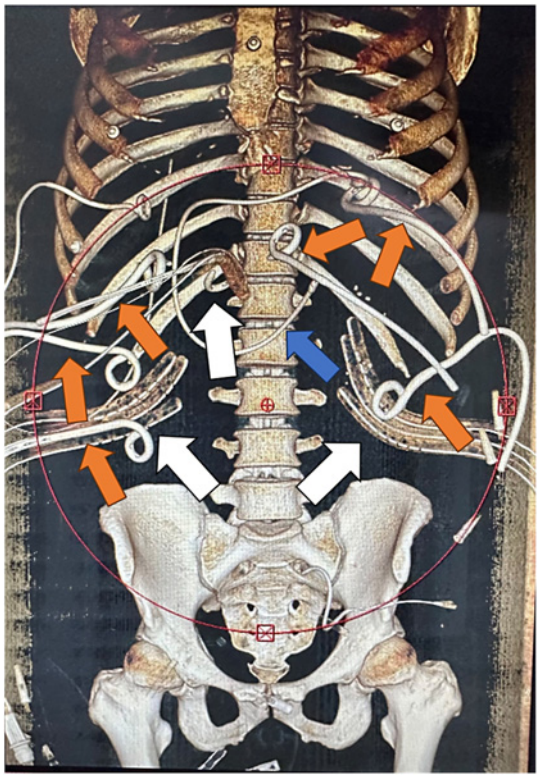

Fig. 3. Postoperative non-contrast CT imaging, 3D reconstruction. The white arrow shows the surgical placement of the drainage tube, the orange arrow shows the B-ultrasound or CT-guided abdominal puncture drainage tube, and the blue arrow shows the jejunal nutrition tube.

Subsequently, the patient underwent digital subtraction angiography (DSA) under local anesthesia for both episodes. Intraoperative findings revealed bleeding from branches of the gastroduodenal artery on the first occasion and from the gastroduodenal artery itself on the second occasion (Fig. 4). Hemostasis was successfully achieved through coil embolization in both instances.

On day 60 post-admission, the patient experienced a third episode of massive intra-abdominal hemorrhage. Bright red blood flowed from the abdominal drainage tubes and incision sites, amounting to approximately 1,500 mL. Vital signs indicated tachycardia with a heart rate of 120 beats per minute, and blood pressure dropped to 70/40 mm Hg. Hemoglobin levels decreased significantly from 73 g/L the previous day to 47 g/L, accompanied by coagulation dysfunction. Given the patient's critical condition, an emergency DSA was performed, revealing hemorrhage from the main splenic artery. Embolization was administered, successfully achieving hemostasis (Fig. 5). Based on the most recent non-contrast CT examination, it was inferred that necrosis from SAP had extended to the pancreatic tail region, likely serving as the primary cause of bleeding from the main splenic artery.

Follow-Up and Outcomes

Following successful embolization of the splenic artery on day 60, the patient's condition gradually stabilized. However, localized inflammation and the presence of drainage tubes required continued hospitalization to minimize the risks of fever, recurrent pancreatitis, and complications such as bleeding or intestinal fistula caused by tube friction. Regular monitoring and management within the ward provided better control of these risks compared to early discharge.

Over the following months, inflammatory markers improved, and non-contrast CT scans confirmed the gradual resolution of retroperitoneal hematomas and necrotic tissue. As the

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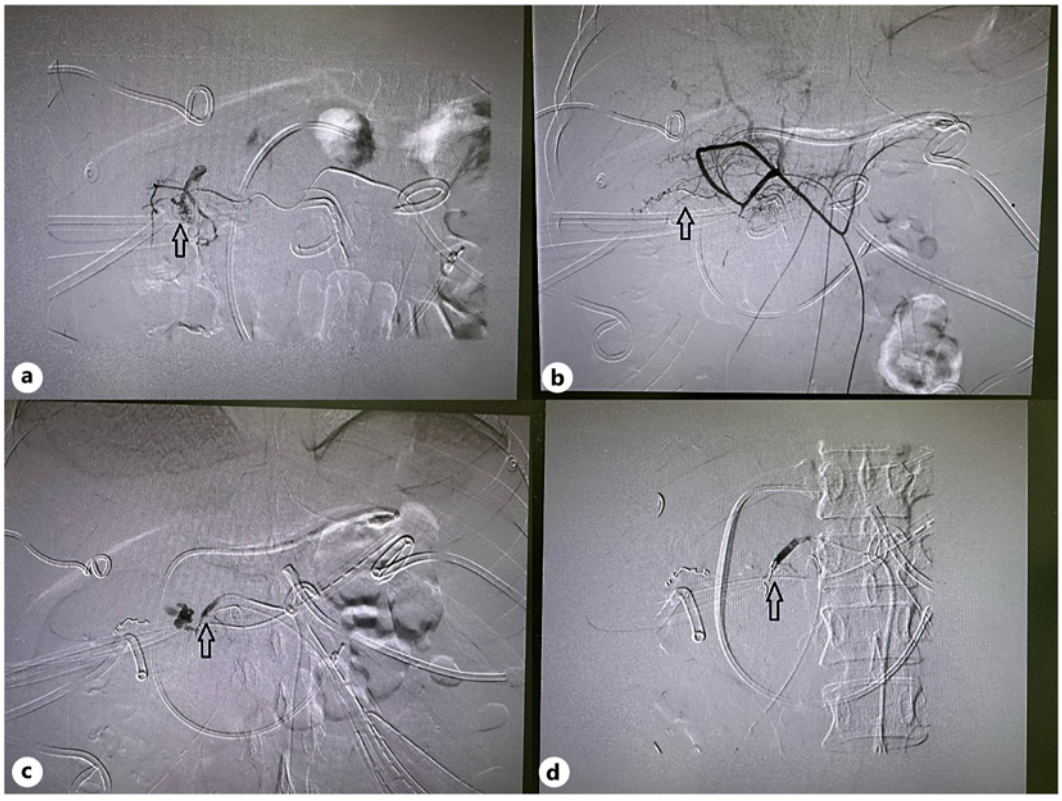

Fig. 4. Patient received DSA examination and treatment in time. **a**, **c** Diagnosis of gastroduodenal branch artery bleeding and gastroduodenal bleeding, respectively; arrows indicate the site of bleeding. **b**, **d** Embolization therapy was given under vascular intervention; arrows indicate the site of embolization.

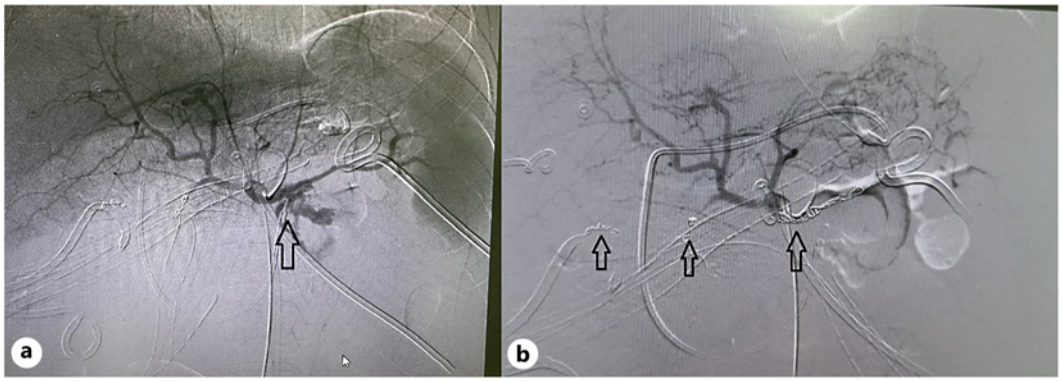

Fig. 5. DSA was urgently performed by the interventional vascular team after a sudden abdominal hemorrhage. The black arrow indicates the site of bleeding, suggesting the source of the main splenic artery, and hemostasis was given, showing the coil shadow of three embolization hemostasis.

recovery continued, the drains were gradually removed, and the patient went over to oral feeding with better nutritional status. On the 180th day from hospital admission, the patient was discharged. The patient's condition was stable. During hospitalization, he did not have any further bleeding or infections. Long-term follow-up was planned to identify potential complications, including exocrine deficiency and diabetes mellitus.

Case Rep Gastroenterol 2025;19:79–88
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Discussion

Mechanisms of Intra-Abdominal Hemorrhage

AP is a disease prone to localized complications. These include fluid collections around the pancreas, pancreatic and peripancreatic necrosis (sterile or infected), and walled-off necrosis (also sterile or infected) [8]. Arterial bleeding in SAP occurs more frequently than in other types of pancreatitis. In SAP, there is a tremendous inflammatory response and diffusion of proteolytic enzymes in the retroperitoneal tissue, which contribute to the digestion of both pancreatic tissue and vascular walls, including the relatively resistant arterial walls. This process causes acute erosion, disruption, or weakening of the vessel wall, ultimately leading to PSA formation, which might eventually rupture and cause bleeding [2, 3, 9].

Once a PSA vessel ruptures, blood enters the digestive tract, abdominal cavity, retroperitoneal space, and other organs, manifesting clinically as severe complications such as gastrointestinal or abdominal bleeding [10, 11]. PSA primarily originates from the celiac artery, the superior mesenteric artery, and their branch artery. The peripancreatic pseudoaneurysm is the most common [12], frequently involving the splenic artery [13], but also including gastroduodenal artery, pancreaticoduodenal artery, distal gastric artery, and hepatic artery.

Another potential mechanism of intra-abdominal hemorrhage in SAP is intracystic hemorrhage, which occurs when ruptured vessels bleed into pseudocysts or walled-off necrotic cavities. This phenomenon is often identified through imaging findings, such as hyperattenuating fluid or irregular cyst wall enhancement on CECT [14]. However, in our case, non-contrast CT performed during each episode of hemorrhage did not reveal such findings, and the absence of pseudocysts further reduced the likelihood of this mechanism. The possibility of drainage tube-associated trauma must also be considered, particularly in patients requiring prolonged drainage. Mechanical irritation from drainage tubes can exacerbate inflammation and erode vascular structures, potentially contributing to arterial bleeding [15]. In this case, bleeding episodes coincided with blood-stained fluid draining from retroperitoneal tubes, raising concerns about tube-related vascular injury. However, DSA findings during the third episode confirmed bleeding from a splenic artery PSA, suggesting that while drainage tubes may have exacerbated the bleeding, they were not the primary cause. Persistent pancreatic inflammation and enzyme-induced vascular erosion likely played a more significant role in the hemorrhage's development.

This case underscores the multifactorial mechanisms of intra-abdominal hemorrhage in SAP. Recognizing these mechanisms, particularly PSA formation and drainage tube-related risks, is critical for timely and effective management.

Surgical Management of Intra-Abdominal Hemorrhage

Timely surgical or endovascular intervention is crucial for managing active intra-abdominal hemorrhage and the formation of PSAs. PSAs, in particular, can develop silently, and once formed, they pose a life-threatening "sword of Damocles," with the potential risk of rupture at any moment. While a definitive PSA was not identified in this case, the patient came perilously close to death due to a third massive abdominal hemorrhage.

Currently, common diagnostic methods for PSA include abdominal color Doppler ultrasound, CECT, and DSA. Embolization of the bleeding artery source is considered a safe and effective nonsurgical treatment for intra-abdominal bleeding, providing either temporary or permanent control of acute bleeding episodes. DSA, in particular, has a high positive rate during dynamic artery rupture bleeding assessments, with sensitivity ranging from 80% to 90%. It often serves as the preferred diagnostic method for the combined treatment of active bleeding [16].

When the patient is hemodynamically unstable, unable to undergo DSA, experiences rebleeding after DSA treatment, or develops complications such as infection or external compression

Case Rep Gastroenterol 2025;19:79–88
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secondary to bleeding, surgical intervention becomes necessary. Surgical approaches may involve ligation of the proximal or distal part of the bleeding vessel, and, if needed, pancreatectomy. If the source of bleeding remains unidentified during DSA, it may be attributed to intermittent arteriovenous bleeding or diffuse bleeding from large capillaries. In such cases, surgical tamponade becomes the only viable option, particularly in instances of diffuse venous bleeding.

In our experience, vascular embolization serves as a critical and urgent rescue measure for patients with SAP and recurrent postoperative bleeding. However, the key to effective treatment remains the drainage and removal of pancreatic necrotic tissue to control infection. This was highlighted during the case discussion when iterative pancreatic necrosectomy produced the needed results. However, intra-abdominal bleeding and coagulation dysfunction often aggravate each other, forming a vicious cycle. Therefore, a comprehensive and objective understanding of the cause, nature, and location of bleeding is crucial. In order to control the problem and improve survival, it is of paramount importance to closely monitor coagulation parameters.

Conclusion

Intra-abdominal hemorrhage is one of the severe complications of SAP. As previously mentioned, this is the consequence of vessel destruction and PSA occurring. Early detection of this development and immediate intervention are the cornerstones for ensuring a safe recovery. DSA should be the first choice for diagnosing and treating emergency intra-abdominal hemorrhage. This is due to its low invasiveness and great efficacy.

To reduce recurrences to a minimum, active invasive interventions such as pancreatic necrosectomy and percutaneous drainage are indispensable. Apart from controlling infection, these interventions also reduce inflammation and vascular complications. Ongoing imaging and careful monitoring of the patient's vascular status are crucial in high-risk patients. Another key point is the inclusion of the active preventive methods in a comprehensive care plan. This includes, on the one hand, vascular surgery and, on the other, adequate intensive postoperative care with a view to preventing similar disastrous complications in SAP.

Statement of Ethics

Ethical approval is not required for this study in accordance with local guidelines. Informed written consent was obtained from the patient for publication of this report and any accompanying images. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000543626).

Conflict of Interest Statement

The authors declare no conflict of interest.

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Author Contributions

C.-Y.W. and X.L., F.H., X.-L.F., and G.Z. contributed to manuscript writing and editing, and data collection. Y.G., R.-P.Y. revised the manuscript critically for important intellectual content. Y.-F.C. contributed to conceptualization and supervision; all authors have read and approved the final manuscript.

Data Availability Statement

The laboratory test data and imaging results involved in this article are available and can be obtained from the HIS medical record system of Tianjin Nankai Hospital. The data that support the findings of this study are openly available in CDMS digital medical record system at https://172.17.100.171/cdms/home/Index. Further inquiries can be directed to the corresponding author.

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