THE JOURNAL OF SURGICAL RADIOLOGY

Journal of Surgical Radiology

Site: https://journalsurgrad.com/

ISSN Print 2156-213X| ISSN Online: 2156-4566 Volume: 05Issue: 05 Open Access

Research Article

TRACING THE TRACKS: CECT EVALUATION OF INTERNAL FISTULOUS COMPLICATIONS IN PANCREATITIS

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Article History
Received: 02.04.2026
Revised: 12.04.2026
Accepted: 06.05.2026
Published: 18.05.2026

Citations:
Sudhakar, R. C., Shirahatti, S., Savalagi, A. S., & Gowda, N. H. B. (n.d.). Tracing the tracks: CECT evaluation of internal fistulous complications in pancreatitis.J Surg Radiol, V5(5) 82-89

Abstract: ***Introduction**:* Pancreatitis is associated with a wide range of local complications, among which internal fistulous communications are rare but clinically significant. These fistulas occur due to pancreatic duct disruption with leakage of enzyme-rich secretions, leading to erosion into adjacent thoracic or abdominal structures. This retrospective descriptive case series was conducted in the Department of Radiodiagnosis, BIMS Belagavi, to evaluate the imaging spectrum of internal fistulous complications on contrast-enhanced computed tomography (CECT). CECT scans of the abdomen and thorax of six patients with pancreatitis were reviewed for type, site, and extent of fistula, associated peripancreatic collections, and secondary visceral changes, with correlation to available clinical and endoscopic findings. The spectrum included three cases of pancreatico-pleural fistula, one pancreatico-mediastinal fistula, one pancreatico-gastric fistula, and one pancreatico-colonic fistula. Characteristic imaging features included peripherally enhancing collections, linear enhancing tracts, diaphragmatic defects, air foci within collections, and adjacent visceral wall thickening. CECT proved highly effective in delineating fistulous pathways and guiding clinical management. Early recognition of these complications is essential to reduce morbidity and facilitate appropriate therapeutic planning.

Keywords: Pancreatitis, Internal pancreatic fistula, Contrast-enhanced CT, Pancreatico-pleural fistula, Walled-off necrosis

INTRODUCTION

Pancreatitis is a common inflammatory disorder of the pancreas that may manifest as acute, recurrent acute, or chronic disease, each with distinct pathological and imaging features. Acute pancreatitis ranges from interstitial edematous inflammation to necrotizing disease, whereas chronic pancreatitis is characterized by irreversible parenchymal fibrosis, ductal strictures, and calcifications. Both forms predispose to a spectrum of local and systemic complications that significantly influence morbidity and mortality [1]. Among local complications, peripancreatic fluid collections, pseudocysts, and walled-off necrosis are frequently encountered; however, internal fistulous communications remain relatively uncommon and often underrecognized. These fistulas arise primarily from disruption of the pancreatic ductal system, leading to leakage of enzyme-rich secretions into adjacent anatomical compartments. Persistent ductal disruption forms the pathological basis for internal pancreatic fistulas [2]

The extravasated pancreatic enzymes induce intense inflammatory reaction, enzymatic digestion, and pressure necrosis of adjacent tissues. Over time, these processes may culminate in erosion into nearby hollow viscera or transdiaphragmatic extension into thoracic compartments. This explains the varied anatomical spectrum of fistulous communications associated with pancreatitis [3]. Internal pancreatic fistulas may communicate directly or indirectly—via intervening collections—with the pleural cavity, mediastinum, peritoneal cavity, stomach, colon, or, rarely, the pericardium. Pancreatico-pleural and pancreatico-mediastinal fistulas typically present with respiratory symptoms, whereas gastrocolic or pancreatico-colonic fistulas may manifest with sepsis, gastrointestinal bleeding, or altered bowel habits [4]. Clinical diagnosis can be challenging because presenting symptoms often overshadow abdominal pathology. Elevated amylase levels in pleural or ascitic fluid provide important biochemical clues but lack anatomical localization. Imaging therefore plays a pivotal role in confirming the diagnosis and delineating the extent of disease [5]

Contrast-enhanced computed tomography (CECT) remains the cornerstone imaging modality in pancreatitis and its complications. It enables assessment of pancreatic morphology, ductal disruption, peripancreatic collections, and associated inflammatory changes. Multiplanar reformations further enhance visualization of subtle fistulous tracts [6]. Characteristic CT features of pancreatic fistulas include peripherally enhancing collections with internal air foci, direct visualization of linear tracts, adjacent visceral wall thickening, and secondary thoracic or abdominal fluid accumulations. Recognition of these patterns is essential for timely diagnosis [7].

Accurate delineation of fistulous pathways has therapeutic implications, influencing decisions regarding conservative management, endoscopic ductal stenting, percutaneous drainage, or surgical intervention. Early imaging-based diagnosis reduces morbidity and prevents misdirected treatment [8]. Despite their clinical significance, internal fistulas are infrequently reported in literature, largely due to their rarity and variable presentation. Radiologists must maintain a high index of suspicion, especially in patients with recurrent effusions or persistent collections [9,10]. The present study aims to describe the imaging spectrum of internal fistulous complications in pancreatitis on CECT, emphasizing characteristic findings and their clinical relevance to enhance diagnostic accuracy and optimize patient management.

CASE PRESENTATION

Case 1 – Pancreatico-Pleural Fistula

A 41-year-old male with a known history of acute on chronic calcific pancreatitis presented with progressive breathlessness and right-sided chest discomfort. Contrast-enhanced CT (CECT) of the abdomen and thorax was performed to evaluate for complications. Imaging revealed an atrophic pancreas with multiple parenchymal calcifications consistent with chronic calcific pancreatitis. From the tail of the pancreas, a well-defined, peripherally enhancing fistulous tract was identified. The tract extended along the posterior aspect of the stomach and coursed superiorly toward the right hemidiaphragm. A subtle diaphragmatic defect was suspected, with subsequent communication into the right pleural cavity. This resulted in a gross right-sided pleural effusion with associated passive atelectasis of the adjacent lung parenchyma. No evidence of intrathoracic abscess was noted. Peripancreatic inflammatory changes and fat stranding were present, without active necrosis.

The diagnosis of a pancreatico-pleural fistula was further supported by pleural fluid analysis, which demonstrated markedly elevated amylase levels, consistent with pancreatic origin. The imaging findings highlighted the importance of multiplanar reconstruction in delineating the complete course of the fistulous tract. The absence of a large residual collection suggested direct ductal disruption rather than communication via a sizable pseudocyst. Recognition of this entity is crucial, as management strategies differ from those of primary pulmonary pathology. Endoscopic pancreatic duct stenting or surgical intervention may be considered depending on ductal anatomy and clinical stability. In this patient, imaging played a decisive role in identifying the pancreatic source of recurrent pleural effusion, preventing misdiagnosis and guiding appropriate gastroenterological referral.

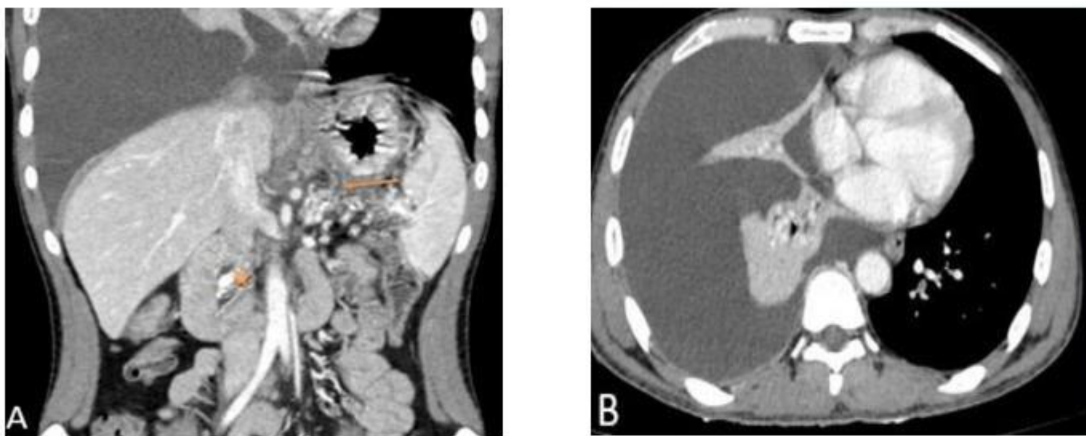

Figure 1: Contrast-enhanced CT images (A – coronal, B – axial) showing features of chronic calcific pancreatitis with an enhancing fistulous tract arising from the pancreatic tail and extending superiorly into the right pleural cavity, resulting in massive right-sided pleural effusion consistent with pancreatico-pleural fistula.

Case 2 – Pancreatico-Pleural Fistula

A 38-year-old male with sequelae of necrotizing pancreatitis presented with left-sided chest pain and dyspnea. CECT abdomen and pelvis demonstrated a well-defined peripherally enhancing collection in the region of the pancreatic tail, consistent with walled-off necrosis. The collection exhibited internal heterogeneity and measured several centimeters in maximum dimension. A slender, enhancing fistulous tract was visualized extending from the superior aspect of this collection toward the left hemidiaphragm. The tract traversed the diaphragmatic region and communicated with the left pleural cavity, resulting in moderate left-sided pleural effusion. Adjacent lung parenchyma showed compressive atelectasis without evidence of empyema.

Peripancreatic fat stranding and residual inflammatory changes were evident, indicating chronic inflammatory sequelae. Multiplanar reformatted images were instrumental in tracing the continuity between the necrotic collection and pleural space. Pleural fluid analysis revealed significantly elevated amylase levels, confirming the pancreatic origin of the effusion. The imaging appearance suggested that persistent enzymatic activity and pressure from the walled-off necrosis contributed to erosion through the diaphragm.

This case underscores the mechanism whereby pancreatic collections evolve into internal fistulas when not resolved promptly. Recognition of the enhancing wall and internal debris aided differentiation from simple fluid collections. Early radiologic identification allowed targeted management planning, including consideration for endoscopic drainage of the necrotic cavity and pancreatic duct evaluation. Accurate delineation of the fistulous tract was essential to prevent recurrent pleural effusion and to guide multidisciplinary care.

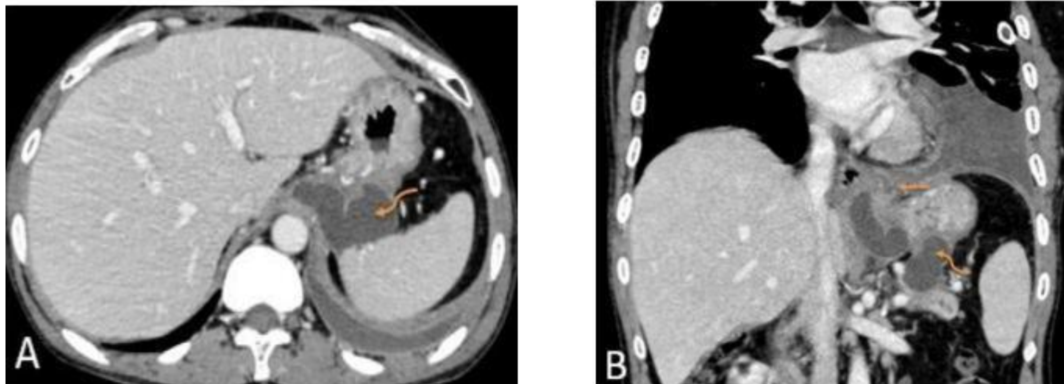

Figure 2: Contrast-enhanced CT images (A – axial, B – coronal) demonstrating a peripherally enhancing walled-off necrotic collection in the pancreatic tail region with a slender fistulous tract extending superiorly across the diaphragm into the left pleural cavity, associated with moderate left-sided pleural effusion—features consistent with pancreatico-pleural fistula.

Case 3 – Pancreatico-Pleural Fistula

A 15-year-old male with a history of necrotizing pancreatitis presented with persistent respiratory symptoms despite prior intercostal drainage (ICD). CECT abdomen revealed a peripherally enhancing collection in the gastrosplenic region. The collection demonstrated features consistent with organized walled-off necrosis. On axial and sagittal reconstructions, the superior margin of the collection was seen abutting the left crus of the diaphragm. A focal defect was identified in the left hemidiaphragm, through which inflammatory extension into the thoracic cavity was evident.

The communication resulted in a loculated, mild left-sided pleural collection. An ICD tube was noted in situ, indicating prior therapeutic intervention. The fistulous tract appeared short and direct, suggesting erosion of the diaphragm due to enzymatic digestion and pressure

effects from the adjacent pancreatic collection. No mediastinal extension was identified in this case. Associated findings included mild ascites and peripancreatic fat stranding.

This case highlights the importance of careful evaluation of the diaphragmatic contour and integrity in patients with upper abdominal collections. In pediatric and adolescent patients, such complications, though rare, can occur following severe necrotizing pancreatitis. The loculated nature of the pleural fluid suggested chronicity and incomplete drainage. CECT provided comprehensive assessment of both abdominal and thoracic components in a single examination. Identification of the diaphragmatic defect was crucial for definitive diagnosis, as persistent pleural effusion despite drainage should prompt evaluation for an abdominal source. Imaging findings guided further management decisions, including consideration for drainage of the abdominal collection and evaluation of pancreatic duct integrity.

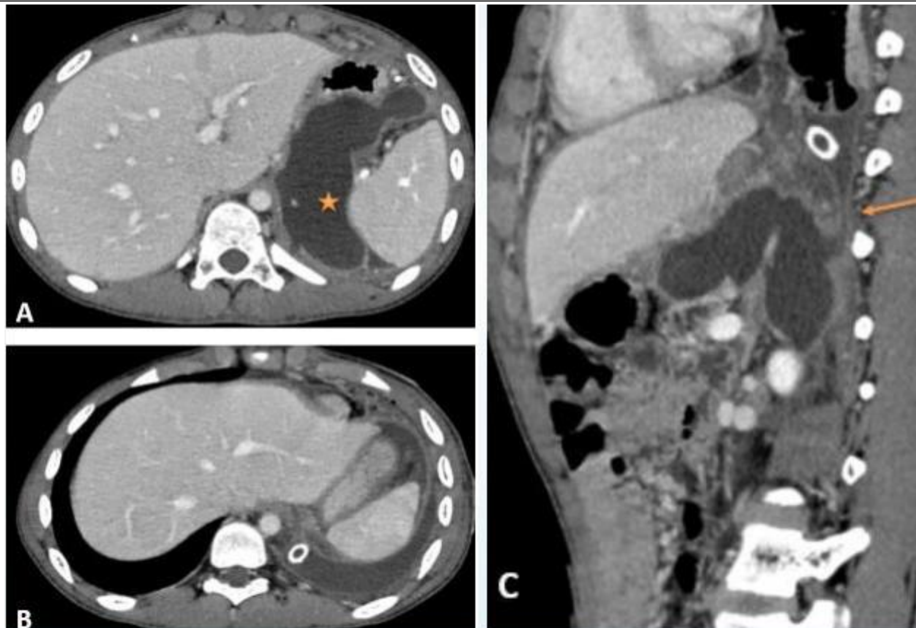

Figure 3: Contrast-enhanced CT images (A, B – axial; C – sagittal) demonstrating a peripherally enhancing collection in the gastrosplenic region abutting the left hemidiaphragm (star), with a focal diaphragmatic defect and superior extension into the left pleural cavity (arrow), resulting in loculated left-sided pleural effusion—features consistent with pancreatico-pleural fistula.

Case 4 – Pancreatico-Mediastinal Fistula

A 31-year-old patient with prior necrotizing pancreatitis presented with chest discomfort and dysphagia. CECT abdomen and thorax revealed a peripherally enhancing collection near the head of the pancreas. The collection extended superiorly in an elongated

configuration along the esophageal hiatus. Multiplanar images demonstrated upward tracking of inflammatory fluid into the posterior mediastinum, forming a continuous enhancing tract.

The mediastinal component appeared as a tubular, peripherally enhancing collection paralleling the esophagus. Bilateral mild-to-moderate pleural effusions were also noted. No evidence of esophageal perforation was identified. The imaging features were consistent with a pancreatico-mediastinal fistula, a rare but recognized complication of ductal disruption in pancreatitis. The path of extension likely followed fascial planes through the diaphragmatic hiatus.

Ancillary findings included mild ascites and residual peripancreatic inflammation. Recognition of the elongated configuration and anatomical continuity from pancreas to mediastinum was essential to differentiate this entity from primary mediastinal pathology. The absence of mediastinal lymphadenopathy or mass effect supported a secondary inflammatory process. This case illustrates the ability of pancreatic fluid to traverse retroperitoneal and diaphragmatic planes, leading to thoracic manifestations. Timely identification on CECT enabled appropriate referral for endoscopic and surgical consultation. Understanding this unusual pathway prevents misinterpretation as isolated mediastinal abscess or neoplasm and underscores the critical role of cross-sectional imaging in comprehensive evaluation.

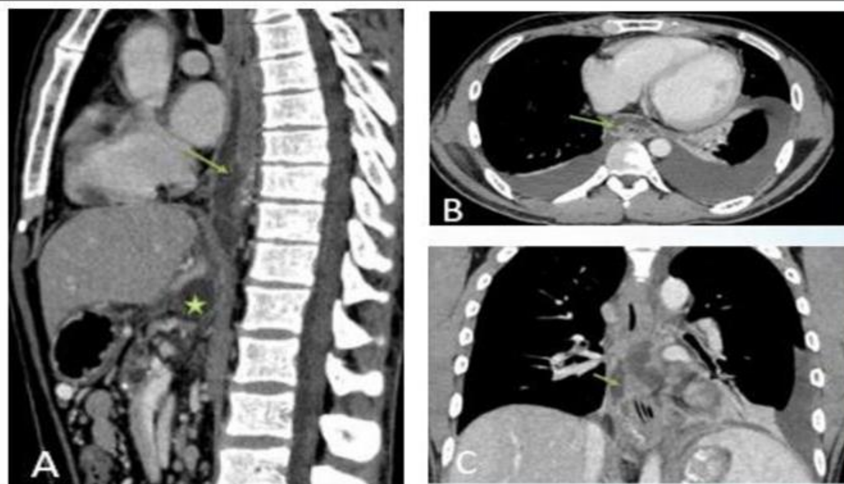

Figure 4: Contrast-enhanced CT images (A – sagittal, B – axial, C – coronal) showing a peripherally enhancing pancreatic collection (star) with superior extension along the esophageal hiatus into the posterior mediastinum (arrows), consistent with pancreatico-mediastinal fistula, associated with bilateral pleural effusions.

Case 5 – Pancreatico-Gastric Fistula

A 37-year-old male with sequelae of necrotizing pancreatitis underwent CECT evaluation for persistent abdominal discomfort. Imaging demonstrated a well-defined walled-off necrosis in the pancreatic bed. The collection showed peripheral enhancement and internal heterogeneous attenuation. Along the posterior wall of the stomach, there was loss of normal delineation of the muscularis propria and serosal layers. A band-like hypodense thickening of the submucosa was evident, suggesting inflammatory infiltration.

Small enhancing tracts were visualized extending from the walled-off necrosis into the gastric wall. On sagittal reformatted images, a focal mucosal defect along the posterior gastric wall was identified. Subsequent endoscopy confirmed the presence of a communication, establishing the diagnosis of a pancreatico-gastric fistula. No active contrast extravasation was observed at the time of imaging.

The presence of gastric wall thickening and focal mucosal breach distinguished this from simple inflammatory adhesion. Air foci within the collection further supported communication with the gastrointestinal tract. Recognition of this fistula is clinically significant, as it may allow spontaneous internal drainage of the necrotic collection, potentially reducing the need for external intervention. However, it may also predispose to infection and bleeding. CECT provided precise delineation of the tract and assessment of surrounding structures, guiding therapeutic planning. This case highlights the importance of evaluating adjacent hollow viscera when pancreatic collections persist.

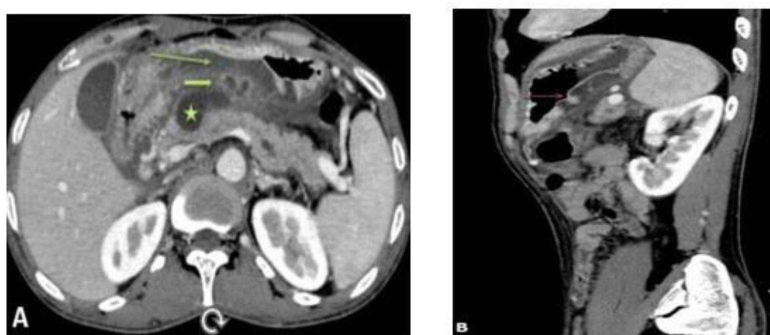

Figure 5: Contrast-enhanced CT images (A – axial, B – sagittal) demonstrating a walled-off necrotic collection in the pancreatic bed (star) with focal discontinuity of the posterior gastric wall and enhancing fistulous tracts communicating with the stomach (arrows), consistent with pancreatico-gastric fistula.

Case 6 – Pancreatico-Colonic Fistula

A 27-year-old female with necrotizing pancreatitis and coexisting abdominal tuberculosis presented with abdominal pain and signs of systemic inflammation. CECT abdomen revealed a fistulous communication between the posterior wall of the mid-segment transverse colon and the pancreatic fossa. The tract appeared as a linear hypodense

channel containing small air foci, extending from the colonic wall toward the inflamed pancreatic bed. Adjacent to the fistulous tract, multiple conglomerated peripherally enhancing necrotic lymph nodes were identified in the aortocaval, pre-aortic, and para-aortic regions, consistent with tuberculous lymphadenitis. The pancreatic region showed residual inflammatory changes without a large organized collection. Colonic wall thickening was noted at the site of communication, suggesting inflammatory involvement.

Air within the pancreatic bed was a key imaging clue indicating enteric communication. Differentiation from infected necrosis was achieved by tracing the direct continuity between colon and pancreatic fossa. The coexistence of abdominal tuberculosis likely contributed to local tissue vulnerability and inflammatory adhesions, facilitating fistula formation. Pancreatico-colonic fistulas are associated with higher morbidity due to risk of sepsis and fecal contamination. Prompt radiologic recognition is critical for surgical planning. CECT provided comprehensive evaluation of both pancreatic pathology and associated lymphadenopathy, allowing holistic assessment of disease burden. This case emphasizes the importance of considering secondary infectious etiologies in complex fistulous complications.

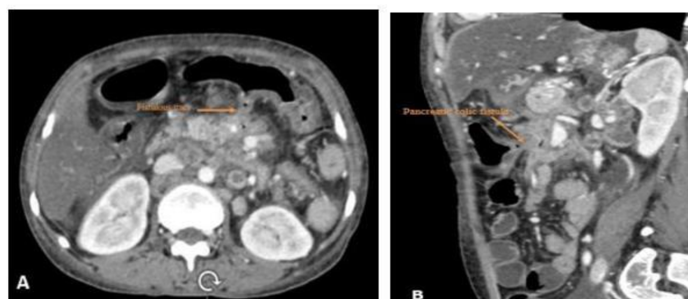

Figure 6: Contrast-enhanced CT images (A – axial, B – sagittal) showing a linear fistulous tract containing air foci extending from the pancreatic bed to the posterior wall of the transverse colon (arrows), consistent with pancreatico-colonic fistula, with associated peripancreatic inflammatory changes.

DISCUSSION

Internal pancreatic fistulas represent a rare yet clinically significant group of complications arising from pancreatic ductal disruption, commonly secondary to chronic or necrotizing pancreatitis. The cases presented highlight diverse fistulous pathways—pleural, mediastinal, gastric, and colonic—each demonstrating distinct imaging features and clinical implications. The role of contrast-enhanced computed tomography (CECT) is crucial for tracing the fistulous tracts, assessing the integrity of surrounding structures, and guiding management.

Pancreatico-pleural fistula (PPF) is among the most frequently reported internal fistulous complications. It occurs due to posterior ductal rupture or pseudocyst erosion through the diaphragm into the pleural cavity. The presented cases correspond with literature findings showing that PPF often manifests as massive pleural effusions—predominantly left-sided, though right or bilateral effusions may also occur [11]. Similar to the current series, most patients in past studies presented with respiratory complaints rather than abdominal symptoms, leading to delayed diagnosis [12]. Elevated pleural fluid amylase remains the key diagnostic indicator across studies. Radiologically, CECT and magnetic resonance cholangiopancreatography (MRCP) help delineate the fistulous tract, though endoscopic retrograde cholangiopancreatography (ERCP) provides direct ductal visualization and therapeutic potential [13]. The literature emphasizes a stepwise management approach—initial conservative therapy with somatostatin analogs, followed by endoscopic duct stenting when necessary [14]. In refractory cases, surgery may be warranted. Our presented cases corroborate these patterns, underscoring CECT's critical role in early recognition and anatomic mapping.

Mediastinal extension of pancreatic collections is an uncommon but serious event, typically following posterior ductal rupture. In our case, CECT delineated a tract ascending via the esophageal hiatus into the posterior mediastinum, paralleling findings from prior MR imaging reports [15]. Recent case series have documented mediastinal pseudocysts and fistulas associated with both pleural effusion and esophageal compression [16]. These correlate with our observation of posterior mediastinal inflammatory tracking and the absence of primary thoracic pathology. CECT provides cross-sectional clarity for distinguishing such entities from mediastinal abscesses or neoplasms.

Fistulization into the gastrointestinal tract represents another internal decompressive phenomenon, often arising from walled-off necrosis. The current cases align with literature documenting pancreatico-gastric fistulas as rare sequelae of necrotizing pancreatitis [17]. These fistulas may spontaneously decompress pancreatic collections, reducing pressure but introducing infection risks. Pancreatico-colonic fistulas, though rarer, are clinically severe due to potential for sepsis and fecal contamination. Consistent with the present findings, CECT typically reveals air within the pancreatic bed or direct tract continuity to the colon. The literature notes similar imaging cues in the presence of associated inflammatory bowel or tuberculous processes [18].

CECT remains the cornerstone for diagnosis and characterization of internal fistulas in pancreatitis. In a large retrospective analysis of 129 patients, CECT detected pancreatico-pleural fistulas in 2.3% and pseudocyst-related extensions in varied extra-pancreatic locations [19]. Multiplanar reconstructions are particularly vital for tracing small tracts, as demonstrated in our cases. While MRCP offers superior ductal visualization, CECT better assesses necrotic debris, vascular complications, and diaphragmatic or visceral wall defects.

Clinical Implications and Management

The management consensus across studies advocates a multidisciplinary approach—radiologic, endoscopic, and surgical. Endoscopic pancreatic duct stenting is favored for partial ductal disruptions, while complete disruptions or multiple fistulas may necessitate surgical intervention [20]. In alignment with global data, our case series reinforces that timely recognition via CECT not only avoids misdiagnosis as primary thoracic or gastrointestinal disease but also optimizes therapeutic strategy.

Conclusion

Internal fistulous complications of pancreatitis represent uncommon yet clinically significant sequelae of pancreatic duct disruption. These fistulas may extend into thoracic or abdominal compartments, producing diverse and sometimes misleading clinical manifestations. Contrast-enhanced computed tomography plays a central role in early detection by accurately demonstrating ductal disruption, peripancreatic collections, diaphragmatic defects, and direct fistulous tracts. Multiplanar reconstructions further enhance delineation of the anatomical pathway and associated secondary changes in adjacent viscera. Prompt radiologic recognition is crucial for guiding appropriate management, whether conservative, endoscopic, percutaneous, or surgical. Awareness of the varied imaging spectrum ensures timely diagnosis, reduces morbidity, prevents recurrent complications, and significantly improves overall patient outcomes in complicated pancreatitis cases.

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