Natural History and Endoscopic Management of Pancreaticopleural Fistula: A Tertiary Care Center Experience

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Abstract

Background Pancreaticopleural fistula (PPF) is a rare complication associated with pancreatitis, caused by disruption of the pancreatic duct, either directly or through rupture of a peripancreatic fluid collection, resulting in leakage of pancreatic juice into the pleural space. It commonly presents as massive, relapsing pleural effusions, often on the left side with high amylase content. Nonspecific chest symptoms often predominate, making it a diagnostic challenge. There is a lack of clarity regarding the management of this rare entity.

Objectives This study aimed to review the typical presentations, pathophysiology, and current role of endoscopic therapy in patients with PPF.

Materials and Methods A retrospective analysis of the results of endoscopic treatment of patients with symptomatic PPF due to pancreatitis was done.

Results Ten patients with pancreatitis (6 males; mean age 33.6 ± 15.4 years: 6 chronic, 4 acute) with symptomatic PPF were analyzed. Endoscopic retrograde cholangiopancreatography was performed in all, with pancreatic sphincterotomy and stenting of the main pancreatic duct (passive transpapillary drainage). Technical and clinical success was achieved in 7/10(70%) and 10/10(100%) patients, respectively. Though the leak was bridged in three patients, pancreatic sphincterotomy and downstream stenting (when bridging was not possible) were successful in closing PPF. One (10%) patient needed surgery for gastric outlet obstruction. The mean duration of endotherapy was 12.1 ± 9.4 months and the time taken for leak closure was 15.3 ± 10.4 weeks. Long-term success of endoscopic treatment (median follow-up period of 48.9 ± 28.7 months) was achieved in all patients.

Conclusions Endoscopic treatment (passive trans-papillary drainage) is a safe and effective procedure for managing postinflammatory PPFs, and should be attempted in cases of failure of medical treatment.

Keywords
- pancreaticopleural fistula
- pancreatitis
- pleural effusion
- endoscopic retrograde cholangiopancreatography

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Introduction

Pancreaticopleural fistula (PPF) is a relatively rare complication following pancreatic duct disruption, characterized by an amylase-rich fluid accumulation in the pleural space.[1] PPF is usually associated with acute/chronic pancreatitis (CP), trauma, or surgery. The incidence of PPF is very low, occurring in approximately 0.4% of CP and around 1% of acute pancreatitis (AP).[2] An abnormal communication to the pleural space from posterior pancreatic duct disruption or pancreatic pseudocyst extension into the pleural cavity is often identified. Diagnostic dilemmas due to thoracic symptoms result in delayed diagnosis, as initial efforts tend to be directed toward finding a thoracic pathology.[3] Minimally invasive endoscopic intervention is usually attempted before invasive surgical management, by utilizing the endoscopic retrograde cholangiopancreatography (ERCP) technique, with endoscopic sphincterotomy and main pancreatic duct (MPD) stenting (passive transpapillary drainage) to ensure physiological outflow of pancreatic juice into the duodenum. However, data on endoscopic management for this entity is scarce, and evidence-based treatment algorithms are required. In this study, we present our experience of endoscopic management of symptomatic PPF.

Materials and Methods

Patients with PPF were identified from departmental database between 2018 and 2022. Their case records were reviewed for demographic details, clinical presentation, natural history, progression of the disease, treatment strategies, and outcome.

Cases Definitions

Diagnosis of pancreatitis, clinical and morphological categorization, and definitions of local and systemic complications were based on the 2012 revised Atlanta classification. Patients with symptomatic pleural effusion for more than 3 weeks, fluid amylase levels more than 1,000 U/L, underlying pancreatic disease, and no other causes of pleural effusion were diagnosed with PPF.[4,5] Technical success of endoscopic therapy was defined as successful deep cannulation of MPD and detection of leak. Clinical success was defined as clinical or radiological improvement of pleural effusion after endotherapy. PPF without clinical signs or those not associated with pancreatic inflammatory disease (acute or CP) were excluded from the study.

Analyses of Cross-Sectional Imaging

Computed tomography (CT) or magnetic resonance cholangiopancreatography (MRCP) images were reviewed in all patients (MRCP—6, contrast-enhanced computed tomography [CECT]–4) to determine the site of ductal obstruction, extent of fluid collections (intra-abdominal and/ or pleural), and presence of PPF.

Management

Conservative treatment of pancreatitis (nasogastric or nasojejunal feeding along with intravenous fluid therapy and analgesia) was initially done in all the patients as per international guidelines (Working Group International Association of Pancreatology (IAP)/ American Pancreatic Association (APA) Acute Pancreatitis Guidelines, 2013). Catheter drainage of pleural fluid was done in symptomatic cases and percutaneous or endoscopic ultrasound-guided (internal) drainage of intra-abdominal collections was done when indicated. The decision to use interventional treatment for PPF was based on symptomatology and cross-sectional imaging results (CECT and/or MRCP; ►**Fig. 1A–C**; ►**Fig. 2A–C**).

ERCP was performed under conscious sedation using intravenous midazolam (0.1 mg/kg) and ketamine (1 mg/kg) after obtaining informed consent. All procedures were done using carbon dioxide insufflation with duodenoscope (TJF 180V, Olympus Corporation, Tokyo, Japan). In all patients, transpapillary route was used to attempt documentation of contrast-leak site, assess morphology and integrity of MPD, opacification of upstream duct, and attempt to place stent in MPD to bridge the leak.

If MPD disruption was identified, pancreatic sphincterotomy (with sphincterotome, Fusion OMNI Sphincterotome FS-OMNI-35–480, Cook Endoscopy Inc., North Carolina, United States) was performed and a pancreatic plastic stent (5 Fr/7 Fr/10 Fr; Zimmon Pancreatic Stent, Cook, Endoscopy Inc., North Carolina, United States) was placed to bridge the leak (►**Fig. 3A–C**). MPD disruption site (head, body, tail) and diameter were taken into consideration for choosing stent

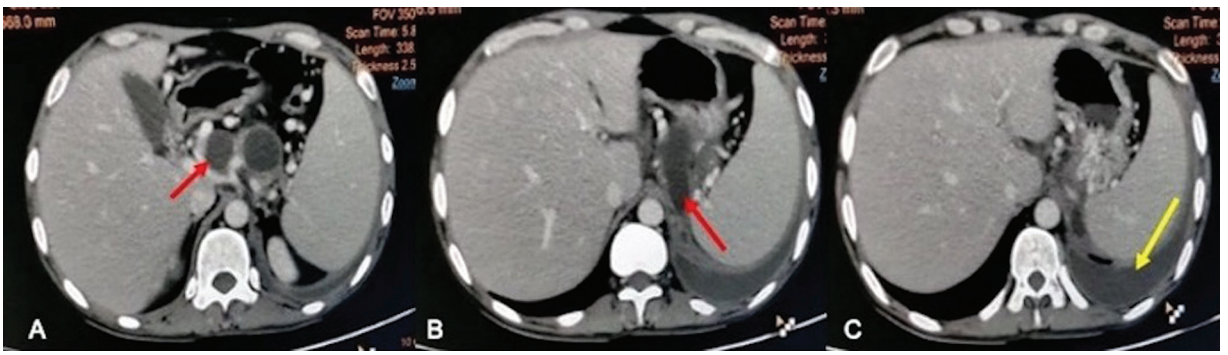

Fig. 1 Contrast-enhanced computed tomography images (**A–C**) showing the peripancreatic collection in the lesser sac extending toward the left pleural cavity. The red arrow indicates the lesser sac collection and the yellow arrow indicates the left-sided pleural effusion.

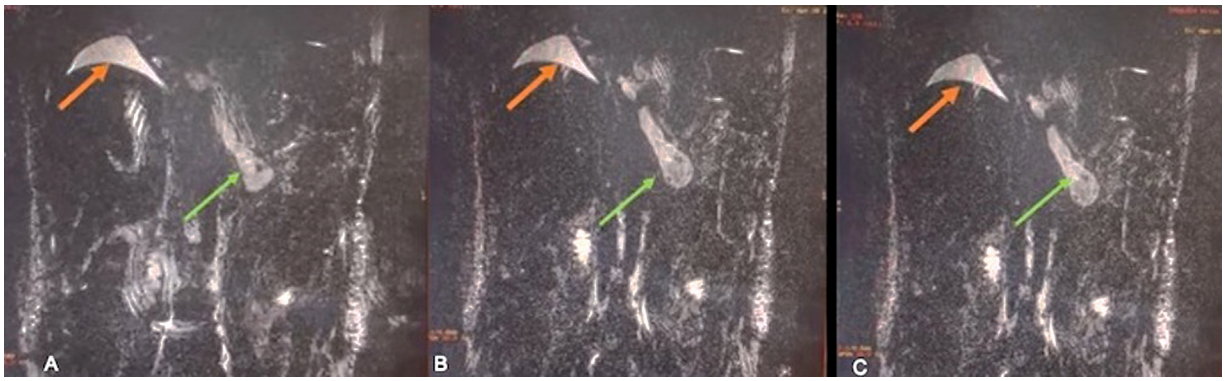

Fig. 2 Magnetic resonance imaging T2-weighted images (**A**–**C**) showing the peripancreatic collection in the lesser sac tracking toward the right pleural cavity. The green arrow indicates the lesser sac collection and the orange arrow indicates the right-sided pleural effusion.

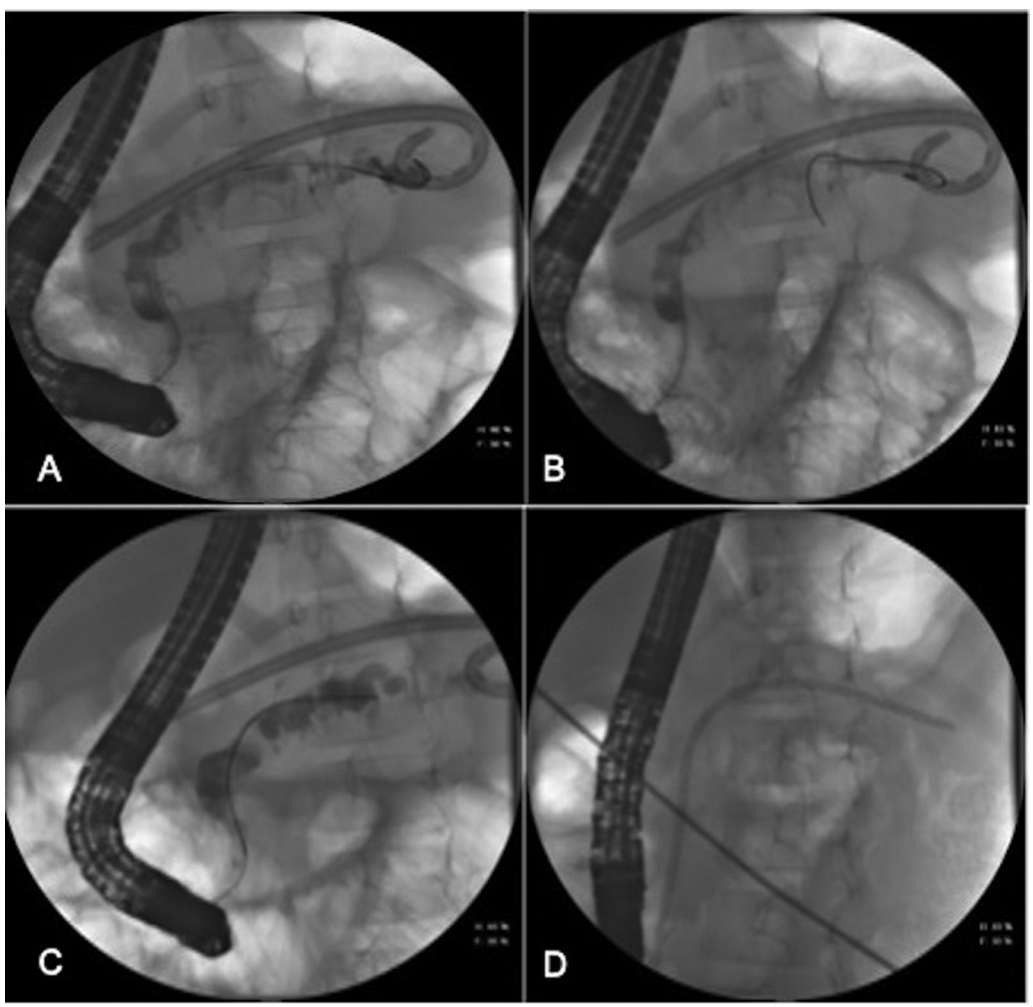

Fig. 3 Pancreatogram images (**A**–**D**) show a contrast leak from the distal body near the site of transgastric Percutaneous drainage (PCD); pancreatic duct stent was placed bridging the leak site.

length and diameter. In cases where leak was not detected, pancreatic sphincterotomy and plastic stenting of downstream duct were done. Repeat ERCP was done after 4 weeks of index procedure, to document the status of leak. Persisting leaks were managed by stent replacement after 3, 6, 12, or 24 months or until no contrast leakage was identified. In cases of CP, clearance of calculi and stricture dilatation was also done. Peripancreatic fluid collections (if present) were managed with either endoscopic ultrasound-guided or percutaneous drainage prior to ERCP.

Follow-Up

Patients were followed up with symptom and signs analysis and serial ultrasonography for any recurrence of ascites/pleural effusion after removal of the MPD stent.

Statistical Analysis

Descriptive statistics were used for presentation, clinical features, and interventions. Categorical data are analyzed as frequencies. Medians and ranges were used to analyze nonparametric continuous variables.

Results

A total of 842 (502 males) patients with pancreatitis were treated in our department between 2018 and 2023. Post-inflammatory PPF was diagnosed in 10/842 (1.2%) patients (mean age 33.6 ± 15.4 years, 6 males). The etiology of pancreatitis in the study group was alcohol-related in six and idiopathic in four patients. The mean duration of illness was 16.5 ± 21.01 weeks, while respiratory symptoms were present for 5.1 ± 2.9 weeks. The commonest presenting symptoms were abdominal pain (10/10), pleuritic pain (8/10), and dyspnea (6/10). Four patients had a history of intercostal drainage due to respiratory discomfort, while two required recurrent therapeutic pleural taps prior to admission. Fever was observed in three patients (►**Table 1**).

Majority of patients had left-sided pleural effusion (8/10), while 2/10 had only right-sided pleural effusion. Six patients had CECT features suggestive of CP. Among the CP patients, three had downstream calculi and two had downstream strictures. All patients had pleural fluid amylase levels more than 2,000 IU/mL (►**Table 2**). Endoscopic ultrasound-guided transmural drainage was done in one patient prior to transpapillary drainage, as patient had gastric outlet obstruction. Pancreatic duct leak was documented in 7/10 patients (70%); site was genu in 57.1% cases, tail 28.5%, and body 14.2%. Among 3 patients, where leak was not demonstrated, one patient had resolution of leak, while remaining two had resolution after successful treatment of downstream stricture. Pancreatic duct leak was bridged in three patients (42.8%). Pancreatic sphincterotomy and MPD stenting were done in all patients. Technical success was achieved in 70%. Successful resolution of pleural effusion (clinical success) was obtained in all the patients (►**Table 2**). The mean number of ERCP was 4.1 (range: 2–12). Two patients developed post-ERCP pancreatitis, which were mild and improved on conservative management. There was no difference in course and outcome in terms of site of ductal disruption and time of resolution of leaks between AP and CP patients.

Mean duration of endotherapy was 12.1 ± 9.4 months (10 ± 1.4 months in AP, 13.1 ± 11.4 months in CP) and mean duration for documented leak closure was 15.3 ± 10.4 weeks. Strictures (2, 22.2%) resolved on multiple stent therapy (►**Table 3**). Surgical intervention was required in one patient

Table 1 Demographics and clinical presentation

	No. of patients ($n = 10$)	Percentage (%)
Age (mean ± SD) years	33.6 ± 15.4 years	
Etiology		
Alcoholic	6	60
Idiopathic	4	40
Duration of illness (mean ± SD)	16.5 ± 21.01 months	
Duration of respiratory symptoms (mean ± SD)	5.1 ± 2.9 weeks	
Clinical presentation		
Abdominal pain	10	100
Pleuritic pain	8	80
Dyspnea	6	60
Severe dyspnea (requiring ICD)	4	40
Fever	3	30
Abdominal distension	2	20
Recurrent therapeutic pleural tap (at least 2 times/week)	2	20

Abbreviations: ICD, intercostal drainage; SD, standard deviation.

Table 2 Imaging findings and endotherapy results

CT findings	No. of patients	Percentage (%)
Pleural effusion (right/left)	2/8	
Features of CP	6	60
MPD stricture/calculi	2/3	
Pleural fluid amylase (>2,000)	10	100
ERCP details		
Endotherapy done	10	100
Leak detected	7	70
Site of leak (identified in 7 patients)		
AP	2/3	66.6
CP	5/6	83.3
- Body (CP-1)	1	14.2
- Genu (AP-2, CP-2))	4	57.1
- Tail (CP-2)	2	28.5
Leak bridged	3	42.8
AP	1/3	33.3
CP	2/6	33.3
Sphincterotomy performed	10	100
PD stent placed	10	100
Technical success (detection of leak)	7	70
Clinical success (clinical improvement/ resolution of effusion)	10	100

Abbreviations: AP, acute pancreatitis; CP, chronic pancreatitis; CT, computed tomography; ERCP, endoscopic retrograde cholangiopancreatography; MPD, main pancreatic duct.

Table 3 Follow-up of Patients after Endotherapy.

Follow-up and outcome (of 10 patients)	Duration
Median follow-up	39.3 ± 13.4 months
Chronic pancreatitis	13.1 ± 11.4 months
Acute pancreatitis	10 ± 1.4 months
Mean duration of endotherapy	12.1 ± 9.4 months
Mean duration for leak closure	15.3 ± 10.4 weeks
Association with local collection	10/10
Surgical intervention	1/10 (for GOO)
Recurrence rate	Nil
Mortality	Nil

Abbreviation: GOO, Gastric outlet obstruction.

(despite ERCP documentation of leak and successful MPD stenting) after resolution of PPF as patient developed groove pancreatitis requiring laparotomy, gastrotomy, drainage of retro gastric phlegmon, and loop gastrojejunostomy.

Long-term success of treatment with a median follow-up of 39.3 ± 13.4 months was seen in all 10 cases.

Discussion

The current literature lacks clear guidelines defining an algorithm for performing diagnostic and therapeutic procedures in patients with PPFs. Most of the available data are in the form of individual case reports or case series. PPF represents both a diagnostic and a therapeutic challenge. This is an uncommon complication associated with AP, CP, and trauma to the pancreas.[6] The incidence of PPF is extremely low, occurring in approximately 0.4% of CP patients, around 1% in AP. In our study, the incidence rate of PPF in patients with pancreatitis was 1.2%. However, this may not reflect the true incidence, as our facility is a tertiary referral center. Disruption of MPD occurs in over 80% patients with postinflammatory pancreatic and peripancreatic fluid (PPF) collections during acute or CP. Typically, PPF occurs following MPD disruption, when the pancreatic duct opens into the pleura, or when a pseudocyst forms and communicates with the pleural cavity. The pancreatic fluid, rich in proteolytic enzymes, disrupts fascial planes and flows through the retroperitoneum, usually through the esophageal hiatus, into the pleural cavity. Occasionally transdiaphragmatic communication may also be the route of fluid movement.[7,8] Internal pancreatic fistula forms following MPD disruption, and ERCP is considered the gold standard method for diagnosing PD disruption, defined as extravasation of contrast medium from the pancreatic ductal system.[4,9,10] On pancreatogram, partial disruption is recognized when PD opacification is seen upstream to the point of disruption, or complete disruption when no PD can be seen upstream to the point of disruption.[9,11,12]

There are no typical clinical features of postinflammatory PPF, making its diagnosis difficult. A patient with history compatible with pancreatitis along with demonstration of pancreatic ductal disruption with pleural effusion on imaging, and pleural exudate showing high amylase levels is considered diagnostic of PPF.[4,5,7,8] Majority of patients present with dyspnea (65–76%) followed by abdominal pain, cough, chest pain, and fever.[3,4] Predominance of chest symptoms may lead to delay in diagnosis and treatment. According to Uchiyama et al, 68% of PPF present with dyspnea, abdominal pain, cough, and chest pain.[13] In our study, chest symptoms (dyspnea, pleuritic pain) were present in 80% cases. Abdominal pain was observed in all the cases.

After documentation of pleural effusion on chest radiography, thoracocentesis is performed, and elevated levels of amylase (> 2000 IU/mL) in the pleural fluid are considered diagnostic of PPF.[4,14–16] PPF usually results in left-sided pleural effusion; however, right-sided and bilateral effusions can occur in 19 and 14% of patients, respectively. In our study, 80% patients had left-sided pleural effusion. Cross-sectional imaging (CECT/MRCP) identifies PPF in approximately 70 to 80% of cases.[4,13,16–18]

Initial treatment includes conservative measures, that is, nil per oral, parenteral nutrition, thoracocentesis, and octreotide infusion (used to decrease pancreatic fistula output and closing time.[19–21] Even though conservative treatment can resolve the condition in 20 to 50% of cases, prolonged treatment time, infection risk, and long duration hospitalization are limiting factors.[15,19]

A recent addition in the management of PPF is endotherapy with either transpapillary nasopancreatic drainage or MPD stenting.[5,10,22,23] Goals of therapy are to restore normal anatomy and attempt to close the leak. Pancreatic sphincterotomy along with MPD stenting to bridge the disruption is determinant of successful outcome when partial PD disruption is present.[12] ERCP confirms the diagnosis of PPF in 80% of cases and reveals a fistulous pathway in approximately 59%.[12,24,25]

In the largest study on endotherapy of postinflammatory PPFs, 22 patients were treated endoscopically,[26,27] and technical success was achieved in all cases. Clinical success was achieved in 21 (95.45%), and long-term success of endoscopic treatment was noted in 19 (86.36%) patients. In our study, technical success (successful deep cannulation of MPD and detection of leak) was 70%, and clinical and long-term success was achieved in all (100%) patients. Though leak was bridged in only three patients, pancreatic sphincterotomy and downstream stenting (when bridging was not possible) were successful in closing the PPF. This is possibly due to reduction in downstream pressure gradient, facilitating flow of pancreatic juice into duodenum. Although endotherapy is more effective in cases of partial PD disruption (compared with total disruption), our study demonstrated that pancreatic sphincterotomy with MPD stenting is beneficial even in cases of complete PD disruption. However, it is important to highlight that patients with complete MPD disruption often require transmural drainage, in addition to passive transpapillary drainage, especially if there is pancreatic fragmentation (disconnected duct syndrome).

The strength of our study is the step-wise algorithm used for managing PPF patients, which highlights the role of minimally invasive endoscopic therapy as a useful therapeutic strategy after failure of medical therapy. Moreover, we have a long follow-up demonstrating long-term efficacy of endoscopic treatment. We propose a step-wise algorithm for

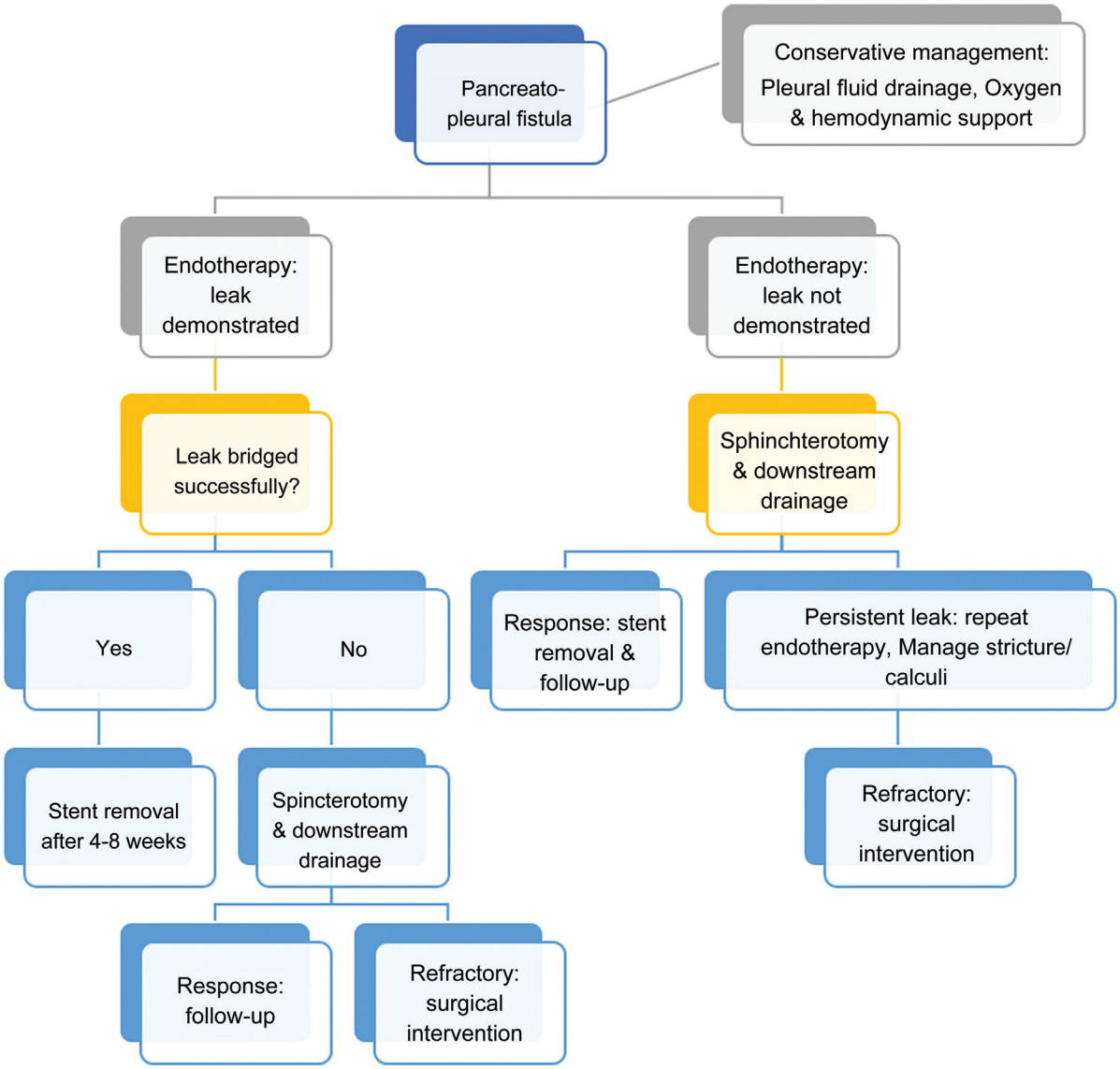

Fig. 4 Stepwise algorithm describing the approach to patients with pancreaticopleural fistula.

endoscopic management of PPFs based on our experience (►**Fig. 4**).

The main limitations are the relatively small number of patients, lack of randomization, and single-center experience. Further studies with larger number of patients are needed to formulate guidelines for the management of PPF.

Conclusion

PPFs create a diagnostic dilemma, as their symptoms mimic thoracic emergencies. Patients with a history of pancreatitis or abdominal trauma with chest symptoms and pleural effusion require a high index of suspicion for PPF. Cross-sectional imaging is essential as it identifies the anatomy of duct disruption and fistula track in most cases. Early restoration of ductal continuity with MPD stenting is very effective, if conservative management fails.

Authors' Contributions

S.M. and V.A.S were involved in conceptualization, methodology, investigation, validation, resources, writing (original draft, review and editing), visualization, and supervision. P.D. and S.R.K. contributed to methodology, validation, writing (review and editing), and visualization. S.M. helped in methodology, investigation, data collection, validation, resources, and writing (original draft, review, and editing). D.T. and N.K. contributed to resources, visualization, and writing (reviewing and editing). K.B. helped in format analysis, reviewing, and editing. G.P. contributed to resources, visualization, and supervision.

Informed Consent

Informed consent was obtained from all individual participants included in the study.

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Conflict of Interest
None declared.

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