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ORIGINAL ARTICLE

Nonsurgical Management of Pancreaticopleural Fistula

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ABSTRACT

Context Pancreaticopleural fistula is seen in acute and chronic pancreatitis or after traumatic or surgical disruption of the pancreatic duct. Surgery leads to healing in 80-90% of cases but carries a mortality of up to 10%.

Aim Our aim was to assess the management of pancreaticopleural fistula on a specialist pancreatic Unit.

Methods Patients presenting with pancreaticopleural fistulae were identified from acute and chronic pancreatitis databases. Management and outcome were compared with previous studies identified in MEDLINE® and EMBASE®.

Results Four patients presented with dyspnoea from large unilateral pleural effusions. Three had a history of alcohol abuse and one of asymptomatic gallstones. All were treated with chest drainage, octreotide and endoscopic retrograde cholangiopancreatography plus/minus pancreatic stent. Two had a pancreatic stent in situ for 5 and 8.5 months respectively. In the third sphincterotomy was performed; in the fourth the pancreatic duct could not be cannulated. The fistula healed in all cases, with no recurrence after 12-30 months, and no deaths. There are 14 reports including 16 cases treated with endoscopic retrograde cholangiopancreatography plus/minus pancreatic stent in the literature, with no recurrence after follow up ranging 4-30 months and no deaths in these 16 cases.

Conclusions A high index of suspicion is necessary to be aware of its presence. These data suggest that endoscopic management is preferable alternative to surgery for pancreaticopleural fistula.

INTRODUCTION

Pancreaticopleural fistula is a rare entity. It is a condition in which pancreatic secretions drain directly into the pleural cavity. It occurs either as a complication in acute and chronic pancreatitis, or after traumatic or surgical disruption of the pancreatic duct [1].

It usually presents as a large recurrent pleural effusion, in either pleural space but left sided effusions are more common and are reported to account for 76% of cases [2, 3]. Pleural effusion due to pancreaticopleural fistula is extremely unusual accounting for less than 1% of cases [4]. Pancreaticopleural fistula is a rare complication of chronic pancreatitis with an incidence of 0.4-4.5% being reported [3].

We reviewed the literature in English language on the causes, investigations and treatment of pancreaticopleural fistula and compared with our own experience in the four cases managed in our Department.

CASE REPORT

Case #1

A 47-year-old female presented with a 4-week history of dyspnoea, chest pain, cough with white frothy sputum and a history of alcoholism. On examination she had features of pleural effusion that was confirmed by chest X-ray showing a massive left sided pleural effusion. Serum amylase was 221 IU/L (reference range: 0-150 IU/L). A chest drain was inserted draining 2 litres, which was removed after 6 days as it had stopped draining. No pleural fluid biochemistry was available. A gastroscopy showed external compression on body and fundus of stomach. A CT scan of chest and abdomen showed a small left pleural effusion, gallstones and atrophic pancreas with pseudocysts. A ventral pseudocyst could be traced through the diaphragmatic hiatus to the left chest. She was then referred to the surgeons. Octreotide was started at 100 μg subcutaneously (s.c.) three times a day (*tid*). An ERCP showed an irregular distal pancreatic duct and a leak from the distal pancreatic duct with a normal biliary tree. A pancreatic stent could not be inserted because of the difficult anatomy, so a sphincterotomy was performed. An abdominal CT was repeated which showed that the pseudocysts and effusion were smaller. She was discharged on octreotide. A repeat chest X-ray showed no pleural effusion and a repeat CT was requested, but the patient was lost to follow-up.

Case #2

A 46-year-old male presented with dyspnoea, left sided chest pain, cough with white frothy sputum and chronic alcoholism. On examination there were features of a left-sided pleural effusion, which was confirmed on a chest X-ray, showing a massive left sided pleural effusion. Serum amylase was not requested. A diagnostic pleural tap was done but a pleural fluid amylase was not requested. A chest drain was inserted draining 1.5 L of fluid. Drainage stopped after 7 days and the drain was removed. A CT scan of chest and abdomen showed a large loculated pleural effusion on left side and punctate calcifications seen mainly in the pancreatic head consistent with chronic pancreatitis. Pleural fluid amylase requested at the time of drainage was 53,040 U/L and the patient was then referred to surgeons. Octreotide 100 μg s.c. *tid* was started. An ERCP showed dilated and ectatic pancreatic duct with a 3 cm narrowed section in the head of pancreas and a fistula arising from the pancreatic tail. A 9 cm 7 Fr stent was inserted. The patient was discharged on octreotide 150 μg *bid*. A repeat ERCP showed the stent was blocked, which was removed and two 9 cm 7 Fr stents were inserted. A repeat CT scan showed calcifications in the pancreas consistent with chronic pancreatitis, no pseudocysts and no pleural effusion. Octreotide was stopped. An ERCP was done for the removal of the two stents. He was seen in the clinic in two months and was doing well.

Case #3

A 63-year-old female presented with dyspnoea, cough with sputum, and pain in her back. On examination there were features of a right-sided pleural effusion. Serum amylase was 399 IU/L (reference range: 0-150 IU/L). A pleural aspiration was done. The pleural fluid amylase was 18 IU/L. An ultrasonography (US) showed gallstones. The patient was discharged and seen in the clinic with a pleural effusion. She was admitted again for pleural aspiration. After 3rd aspiration pleural fluid amyalse was greater than 20,000 IU/L. In all aspiration was done 6 times. The patient was referred to the surgeons. An ERCP was done showing an irregular pancreatic duct and communication upwards to the chest; a 5 cm 7 Fr pancreatic stent was inserted. A CT scan showed large peripancreatic fluid collections surrounding the head and the body of pancreas, tracking superiorly and connecting with the right pleural effusion. There was some amount of pancreatic necrosis with a solitary gallstone. She was readmitted for chest drain, which

drained 2.2 L of fluid and started on octreotide 100 µg s.c. *tid*. The chest drain was removed after 24 days. Depot octreotide was started. An ERCP was repeated, which still showed the pancreatic ductal disruption. A 12 cm 7 Fr stent was inserted beyond fistula and another 5 cm 7 Fr stent was inserted not as far as fistula. ERCP was repeated in 2 months and the stents were removed. A distal pancreatic duct leak was present, so a 12 cm 7 Fr stent was inserted across the leak. An ERCP repeated in one month, showed no evidence of fistula and the stent was removed. Octreotide was stopped. A repeat CT scan showed the right pleural effusion had resolved and the fluid collections in the pancreas had almost completely resolved.

Case #4

A 54-year-old male was referred with a diagnosis of pancreatico-pleural fistula from another hospital. The symptoms were chest pain and dyspnoea. He had a history of alcoholism. A chest X-ray showed a right-sided pleural effusion. CT scan showed head of pancreas replaced by a cyst, retroperitoneal fluid tracking inferiorly to right kidney and a large right-sided pleural effusion, changes suggestive of end result of acute pancreatitis. He had pleural aspiration four times; the pleural fluid amylase requested during the last aspiration was 66,440 IU/L. He was then referred to our Department. A chest drain was inserted and octreotide was started at 50 µg s.c. *tid*. An ERCP was done but the pancreatic duct cannulation failed. A chest drain was reinserted as the first was blocked. A repeat ERCP was again unsuccessful. The patient developed acute tubular necrosis. Another ERCP was attempted, again unable to cannulate the pancreatic duct. Magnetic resonance cholangio-pancreatography (MRCP) was done, showing no significant pleural effusion, normal biliary tree and no gallstones. Fluid collections in the right pararenal region were shown tracking superiorly to the pleural cavity; the probable source of the fistula was thought to be from the pancreatic duct in the body of the gland. The chest drain fell out and the patient was discharged. Octreotide was increased to 100 µg s.c. *tid*. On follow-up he was doing well, so the octreotide was stopped after 6 months.

METHODS

Our aim was to assess the management of pancreatopleural fistula on a specialist pancreatic Unit. Patients presenting with pancreatopleural fistulae from 1996 to 2003 were identified from acute and chronic pancreatitis databases. Management and outcome were recorded from the case notes and compared with previous studies identified in MEDLINE® and EMBASE®.

STATISTICS

Descriptive statistics are reported: frequency, median, and range.

RESULTS

We had four cases of pancreaticopleural fistula (Table 1); 2 men aged 54 and 46; 2 women aged 47 and 63; with a median age of 50 years. All presented with dyspnoea, chest pain and cough from large unilateral pleural effusions. Two cases had effusions on right and two on left. Three had a history of alcohol abuse and one of asymptomatic gallstones. None of the patients were admitted with pancreatitis, though one had a recent history of acute pancreatitis.

Three patients were diagnosed by pleural fluid amylase and one by CT scan. There was a CT scan evidence of pancreatitis in all four cases. ERCP showed the fistula in three cases, and in one case a pancreatogram could not be achieved. The time to diagnosis ranged from 12-49 days. There was a delay in diagnosis, as pleural fluid amylase was not requested on the first samples.

All the patients were treated with chest drainage, octreotide and ERCP plus/minus pancreatic stent. Chest drain was in place from 6-24 days. Octreotide was continued for 2.5-6 months. Two cases had it at a dose of 100 µg *tid*, one initially had it at 100 µg *tid*

Table 1. Summary of the management and outcome of the four reported cases.

	Case #1	Case #2	Case #3	Case #4
Time to diagnosis (days)	12	28	49	33
Method of diagnosis	CT scan	Pleural fluid amylase	Pleural fluid amylase	Pleural fluid amylase
Inpatient stay (days)	34	50	88	70
Endoscopic management	ERCP+ES No stent	ERCP+stent	ERCP+stent	ERCP failed
Octreotide therapy (months)	2.5	4	6	6
Complications	None	Infected chest drain site	Infected IV site, CDT diarrhoea	ATN from contrast
Time taken to healing of fistula	2 months and 20 days	3 months and 27days	4 months and 18 days	22 days

ATN: acute tubular necrosis; CDT: Clostridium difficile toxin; ERCP: endoscopic retrograde cholangiopancreatography; ES: endoscopic sphincterotomy; i.v.: intravenous

was later started on octreotide depot 4 weekly, and one was started on 50 μg *tid* but later increased to 100 μg *tid*.

Two had successful ERCP with stenting, one of whom had it done 3 times with stents changed twice and the total period the stent was in-situ was 8.5 months. The other patient had ERCP 4 times with stent put in 3 times, and the total stent in-situ time was 5 months. Both these patients subsequently had CT scan showing no pleural effusion.

Third patient had ERCP but stenting failed, and a sphincterotomy was performed. A repeat CT scan showed that the pseudocyst and pleural effusion were smaller, and a chest X-ray done in 2 months showed no pleural effusion.

Fourth patient had ERCP attempted 3 times as it was not possible to cannulate the pancreatic duct but the fistula healed with octreotide.

Complications were infected chest drain site, clostridium difficile diarrhoea, infected intravenous site and acute tubular necrosis from ERCP contrast.

The total in-hospital stay was from 34-88 days. The duration from starting the treatment to healing of the fistula, as shown on an imaging study (CT scan in two, chest x-ray in one and MRCP in one), varied from 22 days to 4 months and 18 days. The fistula healed in all cases, with no recurrence after 12-30 months, and no deaths. However the two cases with pancreatic stent did not show a decreased time to healing compared those without stent, though this is a small series.

There are 14 reports [1, 2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15] including 16 cases treated with ERCP plus pancreatic stent in the literature, some of which had aspiration/chest drainage plus/minus octreotide in addition. There was no recurrence after follow up of 4-30 months and no deaths in these 16 cases. No particular pattern was noted as regards duration of healing with or without chest drain or octreotide.

DISCUSSION

The underlying mechanism of a pancreaticopleural fistula formation is usually a leak from an incompletely formed or a ruptured pseudocyst; a minority have direct pancreatic duct leaks. The fistulous tract passes either through the aortic or oesophageal diaphragmatic orifices or directly transdiaphragmatically. Similar patho-physiology and aetiology apply to pancreatic ascites and pancreatic pleural effusion. If the pancreatic duct disruption occurs anteriorly and is not walled off, a pancreatic-peritoneal fistula will develop that will manifest with ascites. If the disruption develops posteriorly, the pancreatic secretions will flow into the retroperitoneum and may dissect through the aortic or oesophageal hiatus into the mediastinum and form a pleural fistula or

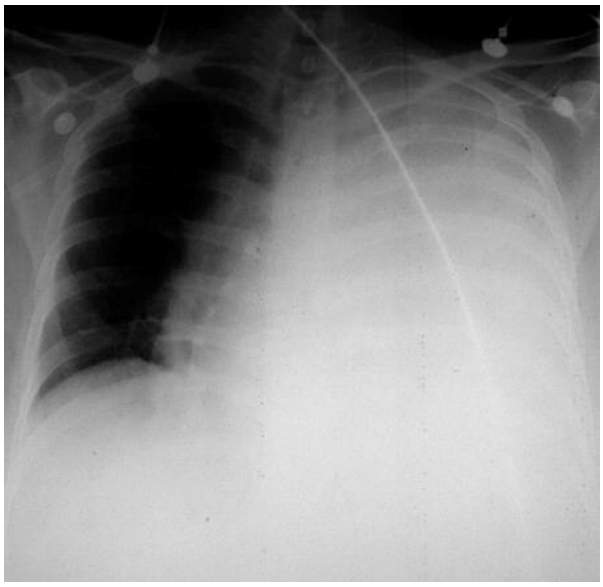

Figure 1. Chest X-ray showing large left-sided pleural effusion.

present as a mediastinal pseudocyst which in turn ruptures into the pleural cavity and forms a pleural fistula [2, 3, 16].

Majority of the cases occurred in men with chronic alcoholism aged 40-50 years [3]. Our series had 3 cases with chronic alcoholism. Half of the patients have no history of pancreatitis [2], our series had one case with history of pancreatitis. Trauma is a less common cause, having been a feature in 0 to 5% of cases [2]. Pancreatic pseudocyst was found in 69 to 77% of patients [2].

The clinical manifestations are often misleading. The average duration of symptoms is 5.6 weeks [2]. Pulmonary symptoms are more common than abdominal symptoms and are usually the presenting symptom, with dyspnoea being the most common. In our series all had pulmonary symptoms. Other symptoms could be chest pain, cough, fever and septicaemia. Abdominal symptoms like epigastric pain may be absent. Many patients go through extensive pulmonary evaluation before the pancreas is identified as the site of primary pathology [1, 2, 3, 17]. The pleural effusion is associated with ascites in 20%, none in our series of cases and pericarditis in 4% [3]. The major complication in these patients is superinfection, which results in a significant morbidity and mortality [17].

Uchiyama *et al*. [18], reviewed 113 cases in Japanese language publications, in which only 23 patients had epigastric pain and only 12% had associated pancreatic ascites. In 98% of the cases in the review from Japan, the cause was chronic relapsing pancreatitis due to a history of chronic alcoholism, 68% complained of chest symptoms and 24% had abdominal symptoms.

Delay in the diagnosis is a critical issue. It needs a high index of suspicion, in those with a history of acute pancreatitis and alcohol abuse, presenting with a pleural effusion (Figure 1), which reforms relatively rapidly after aspiration and for which there is no obvious cause. The time to diagnosis in our cases ranged from 12-49 days.

Diagnosis is usually made by a thoracentesis, after chest X-ray, (3 out of the 4 cases in our series) with a greatly elevated pleural fluid amylase and lipase levels and high albumin content. The serum amylase is usually mildly elevated [19] but not invariably and is thought to be partly secondary to resorption of amylase from the pleural surfaces [2]. It was elevated in two of our cases that had serum amylase requested.

The differential diagnosis of amylase-rich pleural effusion includes acute pancreatitis, cancer of the female reproductive tract, lung carcinoma, metastatic carcinoma, pneumonia, oesophageal perforation, lymphoma, leukaemia, liver cirrhosis, hydronephrosis, and pulmonary tuberculosis [2, 5].

The diagnosis can be confirmed with an ERCP (Figure 2), although it may not always demonstrate the fistulous tract. ERCP may not demonstrate the fistula in patients in whom the site of ductal disruption exists in more distal side than the site where a ductal obstruction exists. In these cases CT may be helpful. ERCP leads to diagnosis in 80% of cases and demonstrates the fistulous tract in 59% [2, 20]. In our series 3 out of the 4 cases showed fistula on ERCP. Visualisation of the entire pancreatic ductal tree is useful in planning a rational surgical approach,

particularly in deciding between a resection and a drainage procedure [4].
A CT scan of chest and abdomen is valuable in the diagnosis, diagnosing one of the patients in our series. It may show the pleural effusion, changes of pancreatitis and identify other associated abnormalities such as pancreatic pseudocysts. Occasionally, a CT scan can demonstrate the fistulous tract, especially if obtained immediately after an ERCP [2]. Magnetic resonance cholangio-pancreatography (MRCP) can demonstrate pancreatic pathology and the fistula. It is a non-invasive alternative to ERCP and is useful where ERCP fails to give adequate information. ERCP and CT will identify the actual fistulous tract in 70% [17].
The treatment was surgical in almost all cases in the past, until there were encouraging results with somatostatin and its analogues. Measures like prohibition of oral intake, nasogastric (nasogastric) tube insertion and total parenteral nutrition (TPN), used in the past, are no longer necessary. None of our 4 patients were subjected to these measures.
A new role for ERCP emerged in the management of internal pancreatic fistulae with reports of successful treatment with insertion of stents into the pancreatic duct [2]. The first case of fistula treated with endoprostheses was reported in 1993 [3].
Complications related to non-operative therapy, including malnutrition, central venous catheter infections, deep vein thrombosis and sepsis associated with intestinal mucosal atrophy from prolonged fasting can be reduced using octreotide and ERCP with pancreatic stenting. Also prolonged hospitalisation contributes substantially to overall morbidity and cost [2, 20].
Lipsett and Cameron [21] treated 16 cases of pancreaticopleural fistula, conservatively with TPN, nasogastric suction and antisecretagogues, such as atropine and recently somatostatin. They were subsequently treated with thoracentesis and chest drain, if effusion failed to resolve. The conservative treatment failed in 50% of the patients who subsequently required surgery.

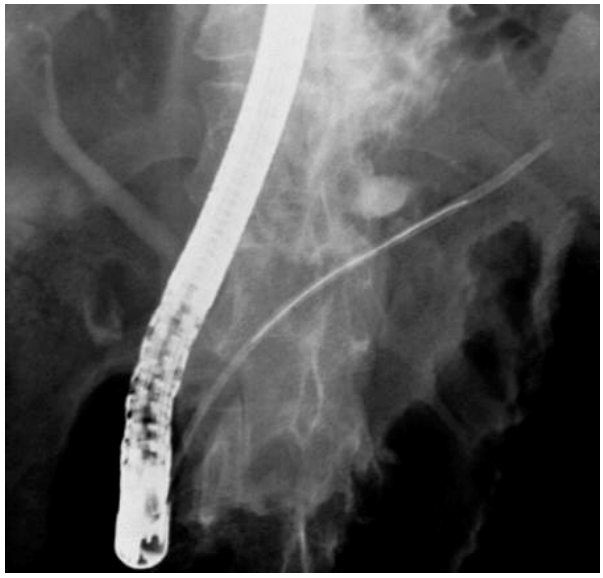

Figure 2. ERCP showing extravasation of contrast through the fistula passing upwards, and a pancreatic stent.

One patient had a recurrence of the fistula after operation. Rockey and Cello [17] had seven cases and reviewed a further 89 patients reported in the world literature. Of the 96 patients in their report, 68 had a trial of medical therapy, which was successful in 28 (41%) and failed in 40 (59%) with two associated deaths. A total of 66 patients underwent surgical intervention, with the most frequently performed surgical procedures being distal pancreatectomy and pancreato-jejunostomy. There were eight postoperative recurrences in seven patients (11%). In the review by Uchiyama *et al*., of 113 cases in Japan, surgical therapy was performed in 61% of the patients on whom information was available [18].
The available treatment modalities are: 1) conservative/medical includes octreotide and thoracentesis; 2) ERCP plus/minus endoscopic pancreatic stent placement; and 3) surgery. The current treatment options are mentioned in Table 2.
The aim of the medical treatment is to reduce stimulation to pancreatic exocrine secretions [1]. Medical treatment constitutes thoracentesis and/or tube thoracostomy, both of which encourage apposition of serosal surfaces and symptom relief, and administration of somatostatin analogues. The

Table 2. Current treatment options for pancreatico-pleural fistula.

Treatment could be classified as:
a) First-line treatment:
• Drainage of pleural effusion: thoracentesis/ thoracostomy
• Inhibition of pancreatic secretion: octreotide
• ERCP with pancreatic duct stent
b) Second-line treatment:
• Surgery

duration of this treatment by itself varies but in the past when endoscopic stenting was not available and surgery was the next option, most suggested it should not exceed 2-4 weeks [2, 3]. Octreotide was continued for 2.5-6 months and chest drain was in place from 6-24 days in our cases.

Somatostatin is a tetradecapeptide with a wide variety of inhibitory gastrointestinal effects. It decreases gut motility and reduces splanchnic blood flow. It has a dramatic effect on pancreatic secretions with an inhibitory effect on both basal and stimulated pancreatic exocrine secretions. Octreotide acetate is a long acting synthetic octapeptide analogue of somatostatin. It has properties similar to somatostatin but has the advantage of much longer serum half-life and thus could be administered subcutaneously. An initial dose of 50 μg administered subcutaneously three times per day is used, and the dose is titrated based upon the fistula output, the maximal dose employed is 250 μg three times daily. It seems that the treatment with octreotide significantly reduces fistula output and decreases the time to fistula closure [17].

It has few significant short-term side effects mostly diarrhoea, flushing and abdominal pain. It has the potential benefit of cutting the overall cost of treatment and reducing the discomfort of patients by shortening the length of stay. There are several negative aspects of using these drugs such as no therapeutic effect on the underlying diseases, painful injection and the potential of long-term side effects [22].

Poddar *et al*. [23], Takeo and Myojo [24] and Chan [25], each had a case treated successfully with octreotide. Fistulae are likely to heal with medical measures including octreotide, if pancreatic ductal drainage is unimpaired and there is no stricture.

Failure of the medical therapy is considered failure of the pleural effusion to clear or superinfection. Although there is no systematic study evaluating medical versus surgical therapy, medical therapy and ERCP plus/minus pancreatic duct stent is usually recommended initially.

This is the optional treatment, which is invasive and carries a greater risk than octreotide therapy, but is safer than operative methods. In cases of failure of medical treatment, therapeutic ERCP is an effective management option with a low morbidity and mortality. Octreotide and pancreatic stenting could be offered as a combined treatment. ERCP facilitates papillary sphincterotomy in cases of sphincter of Oddi dysfunction, dilatation of stenoses of the main pancreatic duct (MPD) and extraction of stones from MPD with or without extra-corporeal lithotripsy [3]. Endoscopic papillotomy may precede the insertion of the stent or be used as a lesser option in those cases where stenting fails. Endoprosthesis may also be inserted into the MPD in patients with pain caused by high ductal pressure and pseudocysts that communicate with the MPD may be drained. ERCP has been used to drain fistula by insertion of naso-pancreatic drains for 1 week, followed by placement of an endoprosthesis in the pancreatic duct [1, 3, 8]. A naso-pancreatic drain in contrast to a stent allows pancreatograms to be obtained repeatedly without further invasive procedures. It also allows application of low intermittent suction, which may potentially facilitate closure of a leak or fistula. The major drawbacks are the necessity for continued hospitalisation and patient discomfort due to the presence of the tube in the nose [5].

The pancreatic stent should be placed so as to bridge the site of duct disruption, if possible. Most fistulae appear to arise from head or body of the pancreas and are thus amenable to bridging with a pancreatic stent. Bridging may not be possible in patients in whom the

fistula arises from the tail of the pancreas and the stent may have to be placed close to the point of duct disruption [7].

Bridging pancreatic stent helps to close the fistula rapidly by not only decreasing the ductal pressure and abolition of pancreatic pressure gradient, but also by mechanically blocking the fistula lumen. Bridging the origin of the fistula does not seem to be mandatory though, as shown in some case reports. It is also unclear whether sphincterotomy alone might be sufficient. One of our cases had sphincterotomy, after unsuccessful attempts at stent insertion, with good result. Stent decreases intraductal pressure by bypassing either the sphincter of Oddi or any stricture in the ducts. Stents used are either 5 Fr or 7 Fr [1, 2, 6]. Fistulae from a pseudocyst, which is no longer in direct communication with the pancreatic ductal system, may heal spontaneously [4].

The aim of the treatment is short-term drainage of ducts with fistulae, and long-term drainage (for 2-12 months) of stenoses of the pancreatic duct [1]. The optimum duration of drainage for fistulae is unknown at present. Some have proposed 6 weeks while others from 4-12 weeks [2, 3]. One approach would be to repeat ERCP at 6 weekly intervals and test whether dye passes up into the chest. Stent itself causes ductal changes that do not always regress after removal [15].

Data are lacking on the long-term consequences of pancreatic duct stent placement [2, 20]. A significant proportion of these patients may still require surgery, particularly for persistent, recurrent fluid collections secondary to stenosis or disruption of the main pancreatic duct [26]. These patients may require prolonged follow up. In one study [5], 7 of 18 cases ultimately required surgery. However, 16 of 18 cases had initial resolution of pancreatic duct disruption and 12 of 14 fluid collections resolved. The issue of how long to continue with endoscopic treatment, is largely unresolved.

There are multiple reports of endoscopic treatment of pancreatic pleural effusion. Safadi and Marks [2], Hastier *et al*. [3], Neher *et al*. [5], Shah *et al*. [6], Macduff *et al*. [7], Kin *et al*. [8], Nagai *et al*. [9], and Griesshammer and Strobel [15], have each published case reports of successful treatment with pancreatic stents.

The main indications for surgery are failure of other treatments, obstruction of the pancreatic duct and a symptomatic fit patient. Surgical treatment includes either some form of a pancreatic resection or entero-pancreatic anastomosis, to the site of pancreatic duct leakage or to the pseudocyst [4]. If there is an obstruction of the main pancreatic duct proximal to the fistula, surgical treatment is necessary to decompress the obstructed duct with or without excision of the involved portion of the obstructed pancreas [4].

In summary, one should be aware of this entity, and request pleural fluid amylase, particularly where there is no obvious cause for a large recurrent pleural effusion, and a history of pancreatitis and alcohol abuse. Current evidence is based on case reports and small series of cases. ERCP is a standard investigation and CT scan is complementary. Medical measures like nil by mouth, nasogastric tube suction, parenteral nutrition and protease inhibitors are no longer required. The first-line of treatment should be a chest drain, octreotide therapy and ERCP with attempt at pancreatic stent insertion. Endoscopic pancreatic stenting is an effective therapeutic option associated with minimal morbidity and mortality, combined with somatostatin analogues it can shorten the duration of hospital stay. Surgical intervention is the second-line of treatment with an appreciable morbidity and mortality.

Octreotide combined with endoscopic placement of stent gives reasonably good results. The fistula healed in all our cases, with no recurrence after 12-30 months, and no deaths though long-term follow up and larger series is needed. Further the issues that need to be clarified are, how long to persist with endoscopic treatment, when would surgery be appropriate and those cases where there is no stenosis or obstruction of the main pancreatic duct would octreotide by itself be enough.

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