Pancreatic Pseudocyst in Chronic Pancreatitis: Endoscopic and Surgical Treatment

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Key Words

Endoscopy · Pancreatitis · Pseudocyst, pancreatic · Surgery

Introduction

The incidence and prevalence of chronic pancreatitis appear to be increasing [1–4]. Pancreatic pseudocyst is a common complication of chronic as well as acute pancreatitis that is unrelated to the underlying aetiology. Advances in radiological techniques have in part led to an increase in the diagnosis of pseudocyst and better characterization of associated complications. There is now a better understanding of the natural history of pseudocysts in relation to the underlying disease. The introduction of new treatment modalities has also increased the options for surgical management. Thus with better knowledge of the disease and with technical advances the indications, timing and methods to treat pancreatic pseudocysts have undergone a marked evolutionary change.

Definition

A pancreatic pseudocyst is a localised collection of pancreatic-enzyme-rich fluid, originating in or adjacent to the pancreas and enclosed in a wall of granulation and/or fibrous tissue lacking an epithelial lining [5]. The principle mechanism leading to pseudocyst formation is believed to involve disruption of the main pancreatic duct and/or peripheral ductules causing leakage and activation of pancreatic enzymes, which in turn leads to localised autodigestion and necrosis of pancreatic parenchyma. This evokes an inflammatory response with the formation of a distinct pseudocyst wall composed of granulation tissue and blood vessels that organizes with more connective tissue and fibrosis [6–11].

On-table pancreatography [12] and endoscopic retrograde cholangiopancreatography (ERCP) have demonstrated a communication between the pseudocyst and the pancreatic ductal system in up to 80% of the patients [13, 14], and peripheral or main pancreatic duct disruption is known to be an early event in acute pancreatitis [15]. Rarely disruption of a retention cyst [16] or trauma that disrupts the pancreatic ductal system may also lead to a pseudocyst [17–19].

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Pour et al. [20] showed that the histological appearance of the pseudocyst wall was independent of the cause with no discernible differences in the structure of the wall between alcoholic, gallstone and idiopathic pancreatitis. The histological findings are identical for acute pseudocysts and chronic pseudocysts [21]. The pseudocyst wall is composed of granulation and fibrous tissue without an epithelial wall in contrast to retention cysts that are lined by epithelial tissue. The wall of acute pseudocysts can be divided into four zones. The inner zone is narrow and contains haemosiderin pigment and loose connective tissue. The second zone is made up of inflammatory and capillary-rich fibrous tissue. The third area is composed of hyalinized connective tissue that is depleted of cells and the outer zone consists of capillary-rich fibrous stroma. The structure and the thickness of the enveloping fibrous capsule appear to be time dependent. Older cysts have thicker walls with increased collagen, fibronectin, and smooth muscle content. Warshaw and Rattner [22] have proposed the evaluation of the isoamylase content of pseudocysts as a predictor of pseudocyst wall maturation.

Incidence

Pseudocysts account for about two thirds of all pancreatic cystic lesions and complicate chronic pancreatitis in 20–40% of patients [23–25]. In acute pancreatitis pseudocysts arise in 10–20% of patients [26–29]. In one of our series of 102 consecutive patients with acute pancreatitis all of whom had abdominal computed tomography (CT) within 72 h of admission, repeated at 1 and 6 weeks, 14 (14%) developed a pseudocyst, that was present at 6 weeks [30].

Diagnosis

The commonest symptom is abdominal pain (76–94%), early satiety, nausea and vomiting (50%) and weight loss (20–51%). Physical examination may reveal upper abdominal tenderness and epigastric fullness rather than a mass (60%). Obstructive jaundice (up to 20%) and duodenal obstruction may result from the mass effect on adjacent viscera [31].

The key investigation is high-quality dual-phase CT performed with a specific pancreas imaging protocol but ancillary investigations include endoscopic ultrasonography (EUS), ERCP or magnetic resonance cholangiopan-

creatography and transabdominal ultrasonography. It has been argued that the risks associated with ERCP from bacterial colonization of the pseudocyst and post-procedural acute pancreatitis outweigh the potential benefits of anatomical delineation [32, 33]. Yet the information obtained by ERCP can change the operative plan in more than 50% of patients showing previously unknown biliary obstruction or pancreatic duct strictures [14, 34, 35]. Much the same information may be provided by EUS and it seems likely that magnetic resonance cholangiopancreatography will largely supplant ERCP for diagnostic uses leaving ERCP the privilege of intervention. There is general consensus that ERCP in patients with a pancreatic pseudocyst should be performed under broad-spectrum antibiotic cover whether for diagnostic or therapeutic reasons.

Differential Diagnosis

The differential diagnosis includes acute peri-pancreatic fluid collections, pancreatic necrosis, cystic pancreatic tumour (<1% of all pancreatic neoplasms, but approximately 10% of pancreatic cysts), hydatid cyst and congenital pancreatic cysts [36]. Pancreatic lesions, notably multiple cysts in conjunction with a neuroendocrine tumour, are a common feature of von Hippel-Lindau's disease. The differentiation of a pancreatic cystic tumour from a pseudocyst is often difficult. A key feature is the overall clinical presentation (notably age, history of excess alcohol consumption, history of pancreatitis, gender and location of the pseudocyst) and radiological appearance. Features that tend to favour a cystic neoplasm are an absence of a history of alcohol abuse, being relatively young, being female, location of the pseudocyst in the tail of the pancreas, the presence of central or peripheral calcification and the presence of septae or loculations. Some authors have proposed percutaneous aspiration and analysis of cystic fluid for viscosity, carcinoembryonic antigen (CEA), CA-125, and cytology. CA-125 and CEA levels are lower in pseudocysts and high in neoplastic cysts. CA 19-9 is non-discriminatory and may be even higher in pseudocysts than in neoplastic cysts. An elevated serum CA 19-9 is also common in acute and chronic pancreatitis. Amylase levels are high in pseudocysts and generally low in cystic tumours. Cytological analysis of cysts may be useful for mucinous cysts but is of limited value for serous cystadenoma. Even intraoperative biopsy of the cyst wall for frozen section histopathology is unreliable (incorrect in 20% or more of cases). Furthermore, there is also the

potential risk of tumour seeding [7, 37, 38]. Finally it is important to remember that chronic pancreatitis is a significant risk factor for pancreatic cancer [39–41], and the two conditions may coexist in 5–10% of cases.

Natural History of Pancreatic Pseudocysts

A pancreatic pseudocyst may regress spontaneously (table 1); persist with or without symptoms, or progress to produce complications [42, 43]. Many studies have been undertaken in an attempt to identify predictive factors in the outcome of pancreatic pseudocysts in order to facilitate management. Accurate interpretation of these studies is usually difficult because they frequently include a mix of cases with acute and chronic pancreatitis. In acute pancreatitis most acute fluid collections spontaneously resolve [5] although some 10–20% will evolve into pseudocysts [17, 27, 42]. Out of the 14 pseudocysts identified by serial CT in our series of 102 consecutive patients, only 5 (36%) became clinically significant [30]. The pseudocyst size indices (maximum antero-posterior $\times$ maximum transverse diameters) of the pseudocysts that were clinically apparent were significantly greater than those which were not apparent ($p < 0.001$) and only those pseudocysts with a size index greater than or equal to 15 cm² required treatment [30].

In 1981 Crass and Lawrence [17] observed that whilst some acute pancreatic pseudocysts resolved without operation over a period of 4–6 weeks, pancreatic pseudocysts that were associated with chronic pancreatitis rarely underwent spontaneous resolution. Regression rates for asymptomatic chronic pancreatic pseudocysts vary between 9 and 31% [44], with the lower figure being closer to our experience. In older series, regression rates of up to 57–67% were reported, but the criteria for distinguishing acute from chronic pancreatitis were far less stringent for inclusion.

Warshaw and Rattner [22] investigated 42 consecutive patients with pseudocysts treated over 5 years: 31 were due to alcohol, 2 were due to gallstones and 9 had idiopathic pancreatitis. Of these 42 patients, 20 had chronic pancreatitis, 14 had acute and 8 had acute on chronic pancreatitis. Spontaneous pseudocyst resolution occurred in 3 (7%) patients, all of whom had recent acute idiopathic pancreatitis and pancreatograms that showed the main pancreatic duct freely communicating with the pseudocyst. Factors associated with failure to resolve were chronic pancreatitis, persistence >6 weeks and a thick wall on imaging [22].

Table 1. Factors associated with pseudocyst resolution

Acute pancreatitis
Small pseudocyst size
Intrapancreatic pseudocyst
Pseudocyst of the head of the pancreas
Persistence <6 weeks
Thin pseudocyst wall

Gouyon et al. [43] followed up to 90 patients with alcohol-related chronic pancreatitis complicated by pseudocyst over 10 years using serial CT and/or ultrasonography to monitor the evolution of the pseudocyst. They were divided in two groups based on the pattern of evolution and the need for intervention. The conservative group comprised 25 patients with pseudocysts that regressed spontaneously and 20 patients with pseudocysts that persisted without any symptoms (group I, $n = 45$). The other group comprised patients with persisting symptoms or complications, requiring either surgical or non-surgical intervention (group II, $n = 45$). The evolution of pseudocysts was monitored by CT or abdominal ultrasonography. On univariate analysis, location of the pseudocyst in the head of the pancreas and intrapancreatic development were more frequent in group I than in group II (78 vs. 55%, $p < 0.02$; 89 vs. 60%, $p < 0.001$, respectively). The diameter of pseudocysts was smaller in group I (median: 25 mm, range 10–110 mm) than in group II (median: 40 mm, range 10–120 mm, $p < 0.0001$). No significant differences were found between any clinical or biochemical parameters. Multivariate analysis showed that the intrapancreatic development of pseudocysts and a diameter <4 cm were the only independent factors associated with spontaneous resolution. These factors accounted for 20% of the total variance. It was concluded that pseudocysts >4 cm in diameter and an extrapancreatic position were independent predictive factors of persisting symptoms and/or complications in patients with pseudocysts and alcohol-related chronic pancreatitis. Indeed most studies suggest that size is a predictive factor for pseudocyst resolution, and Nguyen et al. [45] and Yeo et al. [46] have demonstrated that there is no definable threshold.

The risk of life-threatening complications is around 10% and includes the following [16, 27, 28, 42, 47–50]: (1) biliary duct compression/stenosis; (2) duodenal compression/stenosis; (3) rupture; (4) compression or obliteration.

Table 2. Nealon and Walser [52]: ERCP classification of pseudocysts with regard to the pancreatic ductal anatomy

<i>Acute pancreatitis</i>	
Type I	normal duct/no communication
Type II	normal duct/with communication
Type III	normal duct with stricture/no communication
Type IV	normal duct with stricture/with communication
Type V	normal duct/complete obstruction
<i>Chronic pancreatitis</i>	
Type VI	abnormal duct/no communication
Type VII	abnormal duct/with communication

tion of one or more of the hepatic portal, superior mesenteric and splenic veins with the formation of collateral veins (this in turns will result in gastric varices in the first instance and later oesophageal varices); (5) pseudoaneurysm; (6) haemorrhage; (7) pancreatic ascites due to a communicating pseudocyst-peritoneal fistula, and (8) infection.

Classification

There have been many systems of pseudocyst classification (e.g. D'Egidio and Schein [51]) that have not held up as the characteristics and the natural history of the disease have become better understood. Nealon and Walser [52] have proposed a simple ERCP classification of pancreatic ductal anatomy in pancreatic pseudocysts (table 2). The following are key features that we believe must be known for optimum clinical management [53–55]. (1) The underlying cause of pancreatitis and whether there has been a history of acute pancreatitis or whether there is underlying chronic pancreatitis. (2) The duration of time that it has been present. (3) The diameter of the pseudocyst. (4) Any symptoms related to the pseudocyst and whether it is regressing, remaining stable in size or whether it is enlarging. (5) The location whether within the pancreas (head, body, or tail) or adjacent to the pancreas. (6) Whether it is distant from the pancreas such as the mediastinum, liver, and the pelvis. (7) Single or multiple pseudocysts. (8) Complex pseudocyst associated with major complications such as compression or obliteration of the hepatic portal, superior mesenteric and splenic veins with the formation of collateral veins, biliary, gastric duodenal compression, pancreato-pleural fistula and pancreatic ascites. (9) The state of the main pancreatic

Table 3. Indications for intervention

<i>Complicated pseudocyst, notably of one or more of the following</i>
Causing compression of major veins (either symptomatic or on CT imaging)
Causing symptomatic compression of the stomach or duodenum
Causing compression of the main bile duct (as evident from symptoms or elevated liver function tests)
Associated with pancreatic ascites or a pancreato-pleural fistula
Infection
Haemorrhage
<i>Symptomatic pseudocyst</i>
Persistent or recurrent feeling of fullness, early satiety, nausea or vomiting despite optimum medical therapy
Severe abdominal and/or back pain not responding to optimum medical management
<i>Asymptomatic pseudocyst, with the following relative risk factors</i>
Pseudocyst >4 cm, present for >6 weeks with no evidence or regression or enlarging
Pseudocyst has a thick capsule
Background of chronic pancreatitis
No communication between the main pancreatic duct and the pseudocyst on direct ductal imaging
Pseudocyst lying outside of the pancreas
Main pancreatic duct anomaly including stones and strictures
Suspicion that the pseudocyst is actually a neoplastic cyst or is associated with a neoplastic lesion (fear of cancer – need for resection)
Patients with asymptomatic pseudocysts are carefully monitored by radiology for an increase in size and the development of any associated complications; serial tumour markers are also performed although it should again be noted that CA 19-9, probably the best serum marker for pancreatic cancer, is often elevated in patients with chronic pancreatitis

and intrapancreatic bile ducts. (10) Pseudocyst in association with pancreatic cancer (in contradistinction to a neoplastic pancreatic cyst).

Criteria for Intervention in Liverpool

On the basis of our experience and with support of the literature we have developed a useful guide to help in judging the timing for intervention when we are facing a pancreatic pseudocyst. Our views are to some extent influenced by the very aggressive chronic pancreatitis observed in the Liverpool region with a very high frequency of calcifying chronic pancreatitis and a high incidence of vascular complications. With this caveat, the indications for intervention are listed in table 3.

Endoscopic Management of Pancreatic Pseudocysts

The first successfully completed transmural drainage procedures were described as in an abstract by Khawaja and Goldman [56] in 1983 and then 1 year later in 4 patients by Kozarek et al. [57]. Initially there was a rather low success rate and a relatively high morbidity, but with increased experience and development of the endoscopic techniques there are now good results reported in two thirds or more of the patients with a low mortality and relatively few complications.

Prerequisites for the Endoscopic Treatment of Pancreatic Pseudocysts

A number of essential requirements are necessary before endoscopic drainage is undertaken that may vary according to the endoscopic technique adopted.

Transmural drainage through the stomach or the duodenum requires the following conditions [58–65]: (1) the stomach or duodenal wall must share a common wall with the pseudocyst; (2) the distance between the pseudocyst and the gastric wall must be <1 cm on preoperative investigations; (3) there must be a clear impression of the wall of the stomach or duodenum at the endoscopy; (4) the absence of varices; (5) it is imperative that the cyst structure is not a neoplasm or a pseudoaneurysm by aspiration of the cystic content.

Transpapillary drainage requires a communication between the main pancreatic ductal system and the pseudocyst. This approach may still be possible in the face of proximal duct obstruction by stones or stricture or complete disruption of the pancreatic duct. It is clear from the aforementioned that most pseudocysts located in the pancreatic tail are not suitable for endoscopic drainage.

Techniques for the Endoscopic Treatment of Pancreatic Pseudocysts

Several methods have been described for performance of endoscopic pseudocystenterostomy, but no randomised studies comparing the different techniques have been performed so that there is no standard approach. The key issues concern the localization of the puncture site, maintaining access to the cyst cavity, choice of the correct size of the endoprosthesis and the management of an infected or complex pseudocyst.

The requirements for a successful *transmural drainage* procedure include endoscopic visualization of the most prominent point of bulging into the gastric or duodenal lumen, puncture of the pseudocyst with a pre-cut knife,

cannulation of the opening with a guidewire followed by sphincterotomy to enlarge the communication, opacification of the pseudocyst cavity with contrast and finally placement of a pigtail endoprosthesis (or prostheses) of the correct size (7–12 F). Many endoscopists also now use EUS to identify the site of puncture and to avoid occasional puncture of a vessels or to perform one-step EUS-guided pseudocyst drainage [59, 62].

Transpapillary pseudocyst drainage is technically more demanding than transmural drainage. At the ERCP the guidewire is advanced into the main pancreatic duct and then into the pseudocyst cavity. A pancreatic duct sphincterotomy may be necessary and the stent is placed in situ. Unfortunately it is usually only possible to place a stent that is of rather small calibre, so it is important that the pseudocyst fluid should be clear otherwise the stent will rapidly become occluded by viscid fluid or thick necrotic debris. If a stricture of the main pancreatic duct is found, dilatation should be attempted before stent placement [66–69].

Results of Endoscopic Treatment of Pancreatic Pseudocysts

The results for endoscopic drainage are generally good, with a technical success rate between 80 and 90% for transmural pseudocystgastrostomy and pseudocystoduodenostomy and almost 85% for transpapillary methods (table 4). The long-term resolution rate for both methods is of the order of 65–75% with a recurrences rate of up to 30% of patients and stent migration. These complications occur in up to 30% of patients and may require emergency surgery; thus an experienced pancreatic surgical team and expert interventional radiologist should always be available at short notice when these techniques are being undertaken. The published mortality rate is now less than 1% but appears to be biased in favour of experienced endoscopic teams and highly selected cases [56–59, 62–83].

Surgical Treatment

The treatment of pancreatic pseudocysts has traditionally been surgical. The advantages are that associated underlying pathology may be dealt with and drainage of the pseudocyst itself may be regarded as definitive. The surgeon may also navigate around varices in occlusive venous disease considered a major contraindication for radiological or endoscopic intervention [84]. Jedlicka [85] performed the first pseudocystgastrostomy in 1921.

Table 4. Results of the endoscopic treatment of pancreatic pseudocysts

References	Year	Patients	Trans-mural	Trans-papillary	Trans-mural and trans-papillary	Morbidity	Failed or needed surgery	Mortality	Recur-rence
Kozarek et al. [57]	1985	4	4	0	0	1 (25%)	2 (50%)	1 (25%)	0
Cremer et al. [73]	1989	33	33	0	0	3 (9%)	5 (15%)	0	4 (12%)
Sahel et al. [63]	1991	37	37	0	0	5 (14%)	5 (14%)	0	2 (5%)
Kozarek et al. [69]	1991	14	0	14	0	3 (21%)	3 (21%)	0	2 (21%)
Bejamin et al. [72]	1993	26	26	0	0	4 (15%)	7 (27%)	0	4 (15%)
Funnel et al. [76]	1994	5	5	0	0	0	0	0	0
Deviere et al. [74]	1995	12	5	3	4	2 (13%)	2 (13%)	0	0
Barthet et al. [26]	1995	30	0	20	10	4 (13%)	7 (23%)	0	3 (10%)
Binmoeller et al. [67]	1995	53	16	33	4	6 (11%)	6 (11%)	0	11 (22%)
Smits et al. [64]	1995	37	18	12	7	7 (19%)	13 (35%)	0	3 (8%)
Vitale et al. [49]	1999	36	27	9	0	1 (2.8%)	5 (13.9%)	0	5 (14%)
White et al. [83]	2000	20	20	0	0	2 (10%)	2 (10%)	0	0
Libera et al. [79]	2000	25	25	0	0	6 (28%)	4 (16%)	0	1 (4%)
Giovannini et al. [77]	2001	15	10	5	0	1 (6.6%)	0	0	1 (6.6%)
Norton et al. [80]	2001	17	14	0	0	1 (5.9%)	3 (17.6%)	0	1 (7.1%)
Sharma et al. [81]	2002	38	33	5	0	5 (13%)	1 (3%)	0	7 (16%)
Baron et al. [70]	2002	64	54	10	–	11 (17%)	–	0	7 (12%)
Total		466	327/466 (70.2%)	111/466 (23.8%)	25/466 (5.4%)	62 (13.3%)	72 (15.4%)	1 (0.2%)	50 (10.7%)

Since then many reports have been published with excellent operative results, low morbidity, mortality and recurrences rates. Despite the recent introduction of minimally invasive techniques for dealing with pancreatic pseudocysts, there remains a clear preference for the open surgical treatment of established pseudocysts. With this viewpoint the indications for surgical treatment are as follows [22, 25, 28, 86–93]: (1) contraindication or failure of endoscopic and radiological methods; (2) pseudocysts with complex or multiple main pancreatic duct strictures; (3) associated complex pathology such as an inflammatory mass in the head of the pancreas; (4) pseudocysts with a main bile duct stricture; (5) venous occlusive disease; (6) multiple pseudocysts; (7) most pseudocysts of the pancreatic tail; (8) haemorrhage not adequately controlled by angiographic transcatheter embolization, and (9) suspicion of a neoplastic cyst.

Endoscopic techniques are available for treating biliary strictures, but long-term success is unlikely in the presence of severe underlying chronic pancreatitis in the head of the pancreas. Radiological techniques are also available to relieve partial occlusion of the porto-splenic-mesenteric system but are probably best reserved as an initial procedure prior to definitive surgery. For these reasons we

believe that the criteria we use in Liverpool (outlined above) reflect the optimum surgical approach.

The three main surgical operations available for the treatment of pancreatic pseudocysts are (a) internal drainage, (b) pancreatic resections and (c) external drainage.

Internal Drainage

In 1921 Jedlicka [85] sutured the posterior wall of the stomach to a pancreatic pseudocyst, performing the first reported pseudocystgastrostomy. Since then two other types of anastomosis have been introduced, the pseudocystduodenostomy and the pseudocystjejunostomy. The classical approach requires a midline or sub-costal incision, exposure of the lesser sac, biopsy of the pseudocyst wall, aspiration of pseudocyst fluid, breakdown of any multilocules and finally anastomosis of the pseudocyst with the stomach, duodenum or jejunum according to the preference of the surgeon and anatomical situation. In patients with multiple pseudocysts, multiple anastomoses may be fashioned. Whilst the lesser sac approach is preferable, the anastomosis in some cases may be more safely performed using an approach through the transverse mesocolon, especially on the left (space of Riolan). It is important to underline that in all cases a non-epithelial-

Table 5. Results of internal drainage

Author	Year	Type of operation	Patients	Success rate	Morbidity	Mortality	Recurrence
Sankaran and Walt [96]	1975	pseudocystduodenostomy	8	8 (100%)	NR	0	0
		pseudocystgastrostomy	25	23 (92%)		2 (8%)	2 (8%)
		pseudocystjejunostomy	23	19 (83%)		1 (4%)	4 (17%)
Altimari et al. [94]	1986	pseudocystduodenostomy	11	11 (100%)	4 (36%)	0	0
Nealon et al. [14]	1989	pseudocystduodenostomy	1	1 (100%)	3 (9%)	0	NR
		pseudocystgastrostomy	11	11 (100%)		0	NR
		pseudocystjejunostomy	22	22 (100%)		0	NR
Newell et al. [95]	1990	pseudocystgastrostomy	39	35 (90%)	13 (13%)	2 (5%)	4 (10%)
		pseudocystjejunostomy	59	55 (93%)		2 (3%)	4 (7%)
Vitas and Sarr [50]	1992	pseudocystduodenostomy	2	2 (100%)	5 (36%)	0	0
		pseudocystgastrostomy	8	7 (90%)		0	1 (10%)
		pseudocystjejunostomy	4	4 (100%)		0	0
Spivack et al. [97]	1998	pseudocystgastrostomy	14	13 (93%)	5 (24%)	0	NR
		pseudocystjejunostomy	7	6 (86%)		0	NR
Heider et al. [29]	1999	pseudocystduodenostomy	4	4 (100%)	(21%) ¹	0	0
		pseudocystgastrostomy	10	7 (70%)		0	3 (30%)
		pseudocystjejunostomy	28	24 (86%)		0	4 (14%)
Usatoff et al. [25]	2000	pseudocystduodenostomy	2	2 (100%)	(28%) ¹	0	0
		pseudocystjejunostomy	43	42 (98%)		1 (2%)	1 (2%)
					total morbidity		
	total	pseudocystduodenostomy	28	28 (100%)	25/157 (16%) ²	8/321 (2.5%)	0
	total	pseudocystgastrostomy	107	96 (90%)			10/82 (12%) ³
	total	pseudocystjejunostomy	186	172 (92%)			13/153 (8.5%) ³

NR = Not reported.

¹ Including pancreatic resections and external drainage.² Result obtained considering [14, 50, 94, 95] only.³ Result obtained considering [25, 29, 50, 94–96] only.

lined structure (pseudocyst) is sutured to epithelial tissue (stomach or small intestine). Cooperman [88] showed that these anastomoses will soon stricture and finally obliterate with the disappearance of the pseudocyst.

Pseudocystduodenostomy is indicated for small pseudocysts (<4 cm) in the head of the pancreas and uncinate process that impinge on the duodenal wall. The results of this operation have been consistently very good (table 5) with a reported surgical failure rate of up to 5% and a low morbidity and mortality, but for anatomical and clinical reasons only a few patients are suitable for this operation [14, 25, 29, 50, 94–97].

Controversy continues to animate discussion as to whether pseudocystgastrostomy should be preferred to pseudocystjejunostomy. There is no definitive answer from the review of the literature and in particular there is a lack of randomised controlled clinical trials. The propo-

nents of pseudocystgastrostomy argue that it is a relatively simple and quick procedure with a low infection rate [95]. On the other hand, there are a number of drawbacks. These include the risk of post-operative life-threatening upper gastrointestinal bleeding (from the anastomosis between the pseudocyst and the posterior wall of the stomach, erosion of the pseudocyst wall by gastric acid, or development and rupture of a pseudoaneurysm), and from the problems encountered in draining pseudocysts not adherent to the stomach or contained outside of the lesser sac [87]. Cooperman [88] argued that the risk of reflux contamination of the cavity by gastric contents is small because peristalsis governs emptying of the stomach and the union between the stomach and pseudocyst is short-lived.

The results of major series of pseudocystgastrostomy and pseudocystjejunostomy are shown in table 4. The use

of pseudocystjejunostomy seems to be more popular and perhaps has somewhat better results than pseudocystgastrostomy. Our approach along with most leading specialist pancreas centres is always to use a Roux-en-Y pseudocystjejunostomy. Indeed the same loop may be used to drain an obstructed bile duct and/or a pancreatic duct dilatation via pancreaticojejunostomy [98, 99]. Traditionally only non-infected pseudocysts were suitable for internal drainage, although recently Boerna et al. [100] reported good results with this technique even for the treatment of infected pancreatic pseudocysts.

Resection

Pancreatic resection consists of partial left pancreatectomy preferably preserving the spleen if possible, right-sided partial pancreatectomy (pylorus-preserving pancreatoduodenectomy, Beger's operation or Frey's procedure) [47, 101–103]. Resections are more frequently done for multiple small pseudocysts, biliary and pancreatic duct obstruction, duodenal obstruction, haemorrhage and underlying extensive chronic pancreatitis with severe symptoms.

External Drainage

External drainage is used mainly for infected pseudocysts and thus hardly ever applies to patients with chronic pancreatitis unless they have developed a superimposed attack of necrotizing pancreatitis. In fact this situation is quite unusual and most reports mistakenly refer to the external drainage of sterile or infected necrosis (with a liquid component) from acute pancreatitis per se or the usually unnecessary drainage of acute fluid collections.

Conclusions

A pancreatic pseudocyst associated with chronic pancreatitis represents one aspect of a complex disease process with multiple clinical presentations. There are widely differing degrees of morbidity that range from simple asymptomatic pseudocyst that can resolve without treatment, to multiple pseudocysts associated with biliary and pancreatic duct obstruction that necessitate surgery. The management of pseudocysts that complicate acute pancreatitis is quite different from those that affect chronic pancreatitis, in that once the attack has resolved the background pathology essentially returns to normal, that is to say a normal pancreatic parenchyma with little or no associated damage to adjacent viscera or vessels. Thus the distinction between acute and chronic pseudocysts is paramount for a successful treatment strategy. Endoscopic and surgical treatments of pancreatic pseudocysts have a high success rate in expert hands, although there are still surprisingly few studies that can be directly compared.

An integrated multi-disciplinary team approach that involves pancreatic specialist surgeons, gastroenterologists and interventional radiologists is essential to identify the best treatment for each individual patient. Once internal drainage has been chosen our preference is for pseudocystjejunostomy as this has many advantages in expert hands.

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