Contemporary Management of Pancreatic Pseudocysts

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A pancreatic pseudocyst is a collection of amylase-rich pancreatic juice enclosed by a wall of fibrous granulation tissue typically related to an antecedent episode of pancreatic inflammation [1]. In relation to an episode of acute pancreatitis, fluid collections do not acquire the term 'pseudocyst' until at least 4 weeks have elapsed from the onset of the episode [1]. This distinction is valid both in terms of disease biology and clinical management: early fluid collections are typical in severe acute pancreatitis and although they may go on to form pseudocysts or abscesses, they may also resolve spontaneously whereas a large postinflammatory pseudocyst persisting beyond 4–6 weeks after an episode of acute pancreatitis is likely both to lead to symptoms and to persist without intervention [2, 3]. Pseudocysts can also arise in patients with chronic pancreatitis without a clear-cut precedent episode of abdominal pain and also after abdominal trauma.

The typical clinical presentation is of abdominal pain and vomiting. Serum amylase may be elevated and the diagnosis can be established by transabdominal ultrasonography and computed tomography.

Establishment of the diagnosis of pseudocyst involves exclusion of the rare but important differential of cystic tumour of the pancreas. Although these tumours are uncommon, they are often mistaken for pseudocysts [4]. Absence of a history of an antecedent episode of acute pancreatitis, a rise in serum tumour markers such as carbohydrate antigen (CA 19–9) [5] and findings on computed tomography such as well-defined septae, irregular cyst lining or the presence of a mass lesion may suggest a cystic pathology other than a pseudocyst. The distinction between postinflammatory pseudocysts and cystic tumours may be helped by analysis of cyst fluid. Postinflammatory pseudocysts may be associated with elevated intracystic amylase values (an intracystic amylase of >5,000 U/ml had a 94% sensitivity and a 74% specificity for postinflammatory pseudocyst in the study by Hammel et al. [6]). Intracyst levels of carcinoembryonic antigen and CA 19–9 may also be of value, but the literature contains conflicting reports as to the value of these markers [7–9] and treatment may not reliably be planned on the results of cyst fluid amylase or intracystic tumour markers alone. Recent evidence also suggests that endoscopic ultrasonography is of value in distinguishing postinflammatory pseudocysts from cystic tumours. In an endoscopic ultrasonography analysis of 75 patients with pancreatic cystic lesions (58 pseudocysts and 17 cystic tumours), pseudocysts exhibited echogenic debris and parenchymal changes more often than cystic tumours whereas septa and mural nodules were found more frequently in cystic tumours (but were also present in 12% of pseudocysts) [10].

Synthesis of this evidence to form a practical management algorithm would suggest that where there is a documented episode of pancreatic inflammation followed by the development of a pseudocyst, computed tomography with or without cyst fluid aspiration (for bacteriological culture and aspirate amylase) may suffice as diagnostic tests. Where there is no antecedent episode, there must be a greater onus on the exclusion of cystic malignancy and endoscopic ultrasonography performed by a skilled operator appears invaluable in this setting.

Having established the diagnosis, what is the optimal management? The first option to consider is observation.
Smaller pseudocysts (under 6 cm in transverse diameter) may be successfully managed conservatively [11, 12]. However, much of the available data are affected by lack of clarity over disease descriptors: application of current terminology may suggest that several successfully managed ‘pseudocysts’ were in fact acute fluid collections.

Symptomatic, persistent (more than 6 weeks) and large acute pseudocysts, particularly those exceeding 10 cm in diameter, are indications for intervention as they are associated with a risk of complications such as haemorrhage or infection.

Conventionally, treatment involved the surgical creation of an internal communication between the pseudocyst and the gastrointestinal tract to allow the pseudocyst to drain. This most often took the form of a pseudocyst gastrostomy or, if there was extensive infracolic extension, a Roux-en-Y pseudocystojejunostomy.

The last decade has witnessed a revolution in the range of available therapies for the treatment of pseudocysts with the establishment of radiologic, endoscopic and laparoscopic approaches. This rapid proliferation in available treatments has led to considerable confusion in terms of the optimum therapeutic options.

In order to formulate rational management algorithms, pancreatic ductal imaging may help. Although endoscopic retrograde pancreatography carries a risk of pancreatitis and of introducing infection, there is evidence that knowledge of pancreatic ductal anatomy can help guide selection of treatment option [13]. In the modern era, magnetic resonance pancreatography can be used to obtain this information without the risks associated with endoscopic retrograde pancreatography.

A second step that may aid rational management is to categorise postinflammatory pseudocysts into two broad categories (accepting that there is a variable degree of overlap): first, ‘acute’ pseudocysts arising as a consequence of a documented episode of acute pancreatitis and second, ‘chronic’ pseudocysts presenting in patients with chronic pancreatitis.

Endoscopic internal drainage of ‘acute’ pseudocysts may be less successful in the presence of necrotic tissue within the pseudocyst cavity [14]. It is accepted that the literature contains conflicting reports as many describe evolving technology and techniques and as differentiation between postinflammatory pseudocysts and pancreatic abscess can be difficult. In a recent study using standardised disease descriptors, Baron et al. [15] reported that endoscopic resolution of pseudocysts was achieved more frequently in pseudocysts complicating chronic pancreatitis (59 of 64 (92%)) than acute pseudocysts (23 of 31 (74%)). At a median follow-up of 2.1 years recurrence developed in 9% of acute pseudocysts compared to 12% of chronic lesions. Late recurrence is a concern after endoscopic drainage and in a nonselected series, De Palma et al. [16] reported a 21% recurrence rate after a median follow-up of 26 months.

With large, persistent and/or symptomatic acute pseudocysts surgery remains a well-established therapeutic option. In this context, the question that arises is whether these pseudocysts should be treated by laparoscopic surgery [17]. Laparoscopic transgastric cystogastrostomy has the theoretical attractions of a potentially successful laparoscopic technique: it is essentially the open operation carried out by a minimally invasive approach. Although the technique is now well evolved and can be performed with a low complication rate [17], skills and familiarity with this type of surgery are probably insufficiently widely distributed to permit recommendations for general adoption.

‘Chronic’ pseudocysts, i.e. mature postinflammatory pseudocysts or pseudocysts complicating chronic pancreatitis, ought to be distinguished from cystic neoplasms of the pancreas before considering drainage. Pancreatic ductal imaging is essential in the management of this group of patients as underlying disordered main ductal pathology may be the key to the origin of the pseudocyst and thus if treated may be the route to successful resolution. In this context, Nealon and Walser [18] recently reported a cohort of patients with main pancreatic duct dilatation (>7 mm) and pseudocysts, treated by lateral pancreatico-jejunostomy alone. Pseudocyst recurrence was 1%.

Endoscopic transgastric or transduodenal drainage is currently a popular treatment option for the drainage of chronic pseudocysts but unanswered questions remain as to long-term recurrence and the risk of missed malignancy [19].

Although perhaps currently relatively unpopular, percutaneous drainage has an important clinical role: it may be potentially life-saving in the setting of an ‘infected’ pseudocyst (technically correctly termed a pancreatic abscess) [1].

To summarise, the clinical management of pancreatic pseudocyst has become complex because of the range of available treatments and the relatively limited evidence for selection of these emerging technologies. As with other disease processes, the evolution of newer treatment methods means that more precise disease descriptors are required. Optimal treatment selection should be aided by future randomised trial evidence.
References