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Percutaneous management of necrotizing pancreatitis

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Abstract
The management algorithm surrounding necrotizing pancreatitis has altered radically in the last 15 years in response to evolving concepts, improved understanding and the development of minimally invasive techniques, including percutaneous necrosectomy, and laparoscopic or EUS-guided cystgastrostomy. This article discusses the emerging role of minimally invasive techniques, key to which is an understanding of the evolving pathology of post acute, necrosis-associated fluid collections in pancreatitis. A dynamic and multi-modal management approach is presented, the aim of intervention being the 'adequate and maintained control of sepsis': the choice of technique dependent on the anatomical position, the ratio of solid to fluid components within the collection, and in particular the degree of systemic organ dysfunction.

Key Words: *acute pancreatitis, percutaneous necrosectomy, infected necrosis, organized pancreatic necrosis, cystgastrostomy*

Introduction

The management algorithm surrounding necrotizing pancreatitis has altered radically in the last 15 years in response to evolving concepts, improved understanding and the development of alternative techniques. The median inpatient stay for a patient with infected necrosis is in excess of 2 months and no single approach is seen as universally appropriate. A multidisciplinary approach has evolved, and it is now common for several techniques to be utilized in a single patient, as the indications and clinical condition of the patient alter during that period.

Approximately 15% of patients with acute pancreatitis will have CT evidence of hypoperfusion on an early contrast-enhanced CT scan. The necrotic process is not limited to the pancreas and peri-pancreatic necrosis may occur alongside relatively minor degrees of parenchymal damage [1]. This is particularly so in the obese patient, and an excessive body mass index (BMI) is recognized to adversely affect outcome [2]. Initial extension of the pancreatic and peri-pancreatic inflammation leads to interstitial oedema, and a variable degree of devitalized tissue. The oedema within this initially essentially solid inflammatory mass subsequently coalesces into acute fluid collections, and over a period of weeks, the demarcation between viable and necrotic tissue becomes established, the collection becomes lined with granulation tissue.

The traditional approach to the management of a patient with necrotizing pancreatitis centred on the early diagnosis and particularly an aggressive approach to infection within that necrosis, as this was assumed to be key to clinical resolution. There is undoubtedly a relationship between the extent of necrosis and outcome, and the presence of infection and outcome; survival is more intimately related to the co-existence of organ failure [3,4]. Indeed significant necrosis and occasionally infection within that necrosis can occur without significant systemic upset.

In managing the patients with severe acute pancreatitis, there are two distinct phases where intervention is considered: Early (within 1–2 weeks of admission), where the main concern is minimizing the mortality from multiple organ dysfunction syndrome (MODS) and late (from 2 weeks onwards) where septic complications, particularly infected pancreatic necrosis (IPN) are the primary concern, whether or not MODS is present. The morbidity and mortality of the early phase are associated with disordered systemic homeostasis, and local peri-pancreatic complications are rare. Randomized studies of surgical [5], endoscopic [6–8] or pharmacological

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intervention [9,10] have unfortunately failed to show any advantage over optimized supportive therapy, and this has led to an early management strategy based on the principles of organ and nutritional support.

Late complications, often peri-pancreatic, may arise in those patients surviving the initial period of organ dysfunction, most commonly driven by bacterial (or fungal) colonization of the pancreatic/peri-pancreatic collection. It has also been assumed that this required immediate intervention, as the patient would not recover until complete debridement had been achieved. Surgery in patients with established sepsis-driven organ failure is associated with a poor outcome, and this led to attempts at early identification of infection by radiologically guided fine-needle aspiration (FNA) [11], with a view to almost prophylactic surgical intervention before secondary organ compromise developed. However, it is evident that not all patients with infected necrosis are unwell, and consequently the universal requirement for intervention is overstated. In most surgical conditions, sepsis-driven organ failure results from inadequate drainage of the septic focus, rather than its contents. Within the Glasgow Unit, our approach has therefore evolved to one of sepsis control rather than a preoccupation with either necrosis or bacterial contamination.

Most specialist centres have addressed pancreatic sepsis by way of a single treatment approach, despite the diversity of presentation and clinical condition of the patient. We look on the diversity of described techniques as complementary rather than exclusive. A variety of approaches may be appropriate depending on the anatomical location of the collection, the duration from presentation and the clinical well-being of the patient at that particular time.

Our approach is to avoid intervention unless we suspect that the patient's clinical condition is being compromised by an undrained, presumably infected, collection. The initiator for sequential imaging, usually by way of CT, is therefore a secondary deterioration in organ failure scores or serial biochemistry, rather than routine interval CT scanning or a protocol-driven repetitive FNA approach. In those patients in whom clinical sepsis is suspected, contrast-enhanced CT is performed with a view to probable intervention, the nature of which is determined by the clinical condition of the patient and the time course from initial presentation.

As a general principle, the mortality associated with secondary infection of a pancreatic collection lessens as the collection matures, being in excess of 25% for true pancreatic necrosis and almost zero for a pancreatic abscess. Mortality is also associated with collections with a high ratio of solid components, and careful radiological assessment is essential before treatment. The aim is therefore to avoid intervention whilst any necrosis-associated collection is in the maturation phase; however, intervention may become necessary owing to clinical deterioration. When early intervention is required we try to minimize the surgical insult even if this means a requirement for interval procedures once sepsis is controlled, rather than adopt the traditional open necrosectomy approach.

Key to this policy is an understanding of the evolving pathology of post acute, necrosis-associated collections in pancreatitis. The aim of intervention is the 'adequate and maintained control of sepsis'. The success of various approaches is dependent on the anatomical position and particularly the ratio of solid to fluid components within the collection. The development of secondary infection may be the likely initiator that demands intervention, but the timescale and patho-radiological appearance will influence the optimum intervention. The process of maturation or 'organization', with separation and partial liquefaction of the solid components within a collection, takes in excess of 12 weeks to complete, during which four stages can be recognized. (1) True pancreatic necrosis – minimal separation of devitalized tissue with a high solid/fluid ratio. (2) Transitional pancreatic necrosis. (3) Organized pancreatic necrosis (OPN) – good separation of devitalized tissue within a fluid-filled cavity, and formation of a fibrous wall lined with granulation tissue. (4) Pseudocyst – almost complete resolution of any solid component and a well formed fibrous wall lined with granulation tissue.

Management of infected pancreatic collections

True pancreatic necrosis

The late peak in the mortality curve associated with acute pancreatitis results from secondary infection of the devitalized tissue and sepsis-driven secondary organ dysfunction. This peak occurs early in the recovery phase (2–4 weeks), when organization of the peri-pancreatic collection is incomplete, and radiological targets are poorly defined. Aggressive open surgical exploration encounters semi-adherent devitalized tissue, which results in bleeding if removed. Staged open approaches of open laparostomy, closed packing or closed lavage are all attempts at controlling sepsis in the presence of incomplete debridement. For some time we have argued that complete debridement is in any case unnecessary, and provided that adequate control of sepsis is maintained, organ failure will recover and removal of necrosis may be achieved in a delayed fashion.

Mortality is highest in patients with established organ dysfunction requiring intervention during the early 'true necrosis' phase. Following open surgical debridement, despite maximal supportive measures, terminal postoperative decline is not uncommon. In these patients, a staged approach with initial radiological drainage to downstage the septic process will often result in a short period during which organ dysfunction may improve. Delaying definitive

intervention/drainage to coincide with this therapeutic window may be appropriate.

It is in these patients that we most commonly utilize the percutaneous necrosectomy approach [12], the aim of the procedure being to control sepsis rather than achieve debridement. In this, within 24–36 hours of initial CT-guided puncture and drainage, the patient undergoes tract dilatation, cavity irrigation and partial debridement. Under general anaesthesia the patient is placed supine with sand-bags used to optimize access to the drain site, which is ideally utilizing a left flank approach via the lieno-colic window, to promote dependent drainage. A guidewire is used to exchange the drain for a 30FG balloon dilator (Cook Ltd, Herts, UK) followed by a graduated dilator to 34FG to allow insertion of an Amplatz sheath. Cavity lavage (normal saline at body temperature is infused via a rapid infuser) and suction using a modified nephrostomy rigid rod lens system until the fluid within the cavity allows adequate visualization. At the initial procedure the aim is to achieve adequate and sustainable sepsis control, rather than debridement, and so while piecemeal removal of any loose necrotic material is performed, prolonged attempts at debridement during the initial procedure are avoided as this may result in worsening sepsis and systemic compromise. An 8 Fr umbilical catheter is sutured to a 32 Fr Portex chest drain (SIMS Portex Ltd, Kent, UK) in two positions and this is advanced into the cavity. Closed lavage at 250 ml/h is commenced and continued postoperatively. A median of three, secondary interval procedures are usually required over the coming weeks, hopefully in a patient with improving organ dysfunction and controlled sepsis.

Organized pancreatic necrosis (OPN)

These patients are in general in reasonable health, having been nursed through the initial 10–12 weeks of the illness. Pressure symptoms, pain, non-resolution of a large collection/abdominal mass or occasionally infection are the common indications for intervention. Previous reports recommending intervention for non-resolving collections before this time have little clinical basis. Significant organ dysfunction or sepsis are rare and our approach is toward managing the collection as a single intervention. Our preference is for a transgastric necrosectomy, allowing adequate surgical drainage and removal of any separated necrotic material at the same time. This procedure may be performed either by open surgery or by a laparoscopic, intra-visceral approach (although this procedure is still within an evaluation phase and would not be considered standard management). Both have the advantage that a simultaneous cholecystectomy (with cholangiogram) can be performed. The open approach has been described extensively before. The laparoscopic approach involves insertion of a subumbilical blunt port using a cut-down technique. Intra-abdominal inflammatory adhesions are not uncommon and a Veress needle approach is not recommended. An endoscope is passed perorally, and 100 ml of saline is instilled into the duodenum to act as a sump. The stomach is inflated using the endoscope and utilizing dual imaging (endogastric and intraperitoneal), three 'Step ports' (Tyco Healthcare Ltd, Hampshire, UK) (2×12 mm and 1×5 mm) are then inserted through the abdominal wall and into the stomach. These are then dilated to allow intragastric insertion of the laparoscope. The OPN collection is identified on laparoscopic ultrasound, allowing diathermy-assisted puncture of the cavity. A cystgastrostomy (10–12 cm in length) is then created using three firings of the Endo GIA stapler. Any necrosis can be removed and placed in the fundus. The ports are removed and the gastrotomy puncture sites are closed by intracorporeal suture.

In some patients, large OPN collections require drainage but the patients are either frail, morbidly obese or significant co-morbidity makes an operative approach unattractive. In these patients we utilize an aggressive EUS-guided cystgastrostomy approach [13]. EUS-guided cystgastrostomy is modified to allow dilatation of the cystgastrostomy tract using a 15 mm balloon at the time of initial puncture. Two double pigtail stents maintain tract patency in addition to cavity lavage using a naso-cystic catheter, irrigating the cavity using warmed dialysis fluid at 100 ml/h. Secondary endoscopic procedures to allow tract dilatation (20 ml balloon), occasionally combined with intracavity endoscopy and piecemeal debridement, are usually required prior to resolution.

Transitional pancreatic necrosis

Patients falling between the two extremes described above often present the greatest management challenge. Timing and choice of intervention can be difficult and the wrong choice has the potential to worsen the clinical situation. Between 3 and 10 weeks after illness onset, patients with infected pancreatic necrosis may be managed by a variety of approaches. As a general principle we would prefer to delay intervention to allow organization to occur, and we would consider giving antibiotics if the patient's clinical condition allowed. We have occasionally observed complete resolution in some patients with antibiotic therapy alone but in most cases some form of definitive drainage is necessary.

In those patients who have ongoing sepsis and MOF, our approach is similar to that in patients with early IPN, with percutaneous drainage and necrosectomy. Patients with no organ dysfunction but demonstrable infected necrosis may be managed by laparoscopic transgastric drainage if sufficiently late in the course of the illness, but this technique is less suitable for patients <8–10 weeks from onset.

Patients showing signs of clinical deterioration while on antibiotic therapy will undergo percutaneous necrosectomy if <8 weeks from illness onset.

A percutaneous necrosectomy approach will result in a prolonged hospital stay, and inevitably serial interventions compared with a single transgastric intervention. Our general aim is therefore to manage these patients by conservative means if possible to allow maturation to occur. If demanded by the presence of sepsis or symptoms, the type of intervention will be governed by the degree of associated organ failure and the suitability for a trans-gastric approach, our preference being for intervention along a one-stop OPN algorithm, reserving a percutaneous approach for those with significant co-morbidity or organ dysfunction in whom a single intervention may be considered excessive.

Pseudocyst

By definition a pseudocyst contains minimal necrosis, and its management – and that of pancreatic abscess (infected pseudocyst with minimal necrosis) – in patients following an attack of acute pancreatitis, is usually by trans-gastric drainage. Following acute pancreatitis, many patients with apparent 'pseudocysts' will have significant necrosis which may not be obvious on CT but which is easily identified on MRI or at EUS. Transpapillary drainage, as favoured for simple pseudocysts, runs the risk of infecting these necrotic collections and we therefore favour transgastric drainage under EUS guidance. Where there is minimal necrosis and no infection, two pigtail stents are left in place without the use of a naso-cyst lavage catheter but where there is significant necrosis, these patients are managed as with OPN by post-procedural lavage. Follow-up trans-abdominal ultrasound is carried out within 48 h to ensure that the cyst has been adequately drained, and if not, further endoscopic dilatation of the cystgastrostomy track is carried out.

Rarely, patients may have persistent pseudocysts despite endoscopic drainage and this is usually due to disconnected duct syndrome. This occurs when central pancreatic necrosis results in complete disconnection of the pancreatic tail, which remains functional. These patients usually need distal pancreatectomy, although in patients with comorbidity prolonged relief can be obtained by EUS-guided drainage, leaving the stents *in situ* for an indefinite period.

Secondary haemorrhage and enteric fistulation are the two most common surgical complications within the recovery phase. Bleeding into the retroperitoneum is evident from the presence of fresh blood in a lavage catheter. Gastrointestinal bleeding may also be seen but is usually associated with a retroperitoneal source and fistulation into the GI tract. In either situation, the preferred management is mesenteric angiography and embolization. The usual bleeding site is the splenic artery or, less commonly, the gastroduodenal artery, but other sites may be involved, particularly where there is extensive necrosis of the pancreatic head. A small 'herald' bleed is common and ward staff must be alert to this complication so that urgent arrangements for angiography are made before the inevitable massive bleeding ensues. Failure of mesenteric embolization obviously necessitates surgical intervention, but in these cases prognosis is very poor.

Gastrointestinal fistulae are commonly seen in the later stages of management of infected pancreatic necrosis. Most cases are due to focal colonic necrosis, and are managed by simple defunctioning ileostomy, which we perform through a small trephine incision. More extensive colonic necrosis presents as catastrophic worsening of MOF but in our experience is very rare unless open necrosectomy has been carried out.

Duodenal and gastric fistulae can be managed conservatively, although a period of TPN may be required. Pancreatic fistulae are expected following percutaneous necrosectomy. Patients may be sent home with a soft catheter in place and this can be removed at the outpatient clinic when drainage stops, usually within 3–4 weeks. Persistent fistulae may necessitate pancreatic duct stenting or rarely distal pancreatectomy in cases of disconnected duct syndrome.

Conclusion

Management strategies for patients presenting with acute pancreatitis associated with significant pancreatic and peri-pancreatic necrosis have changed radically in the last 10 years. Previously held dogma and uncompromising surgical strategies have matured into a complex and dynamic multi-modal management strategy. Central to this is the timing of intervention and a flexibility of approach. Clinical organ dysfunction is now recognized as being more significant in terms of outcome than either the presence of necrosis or infection. Inadequate drainage of sepsis is the key to that organ failure and therefore ultimately mortality. However, general principles remain: to avoid any major procedure in a patient with organ dysfunction and where possible to delay intervention, allowing organization of the necrosis, until a phase when morbidity and mortality are minimal.

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