Minimally invasive retroperitoneal pancreatic necrosectomy in necrotising pancreatitis

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ABSTRACT

With the marriage of surgery and technology, applications of minimal access surgery are increasing exponentially. Pancreatic diseases are no exception. Minimally invasive retroperitoneal pancreatic necrosectomy (MIRP), or percutaneous video-assisted necrosectomy, is a new technique to debride the necrotic pancreas. We report a 51-year-old male patient who successfully underwent MIRP for infected pancreatic necrosis, and briefly review of literature.

Keywords: infected pancreatic necrosis, minimal access surgery, minimally invasive retroperitoneal pancreatic necrosectomy, necrotising pancreatitis, percutaneous video-assisted necrosectomy

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INTRODUCTION

Necrotising pancreatitis complicates nearly 20%–30% of all patients with acute pancreatitis, and has a high mortality. While surgery is contraindicated in objectively-diagnosed acute pancreatitis, it is indicated for infected pancreatic necrosis (IPN). Surgery for necrotising pancreatitis has a mortality of 20%–50%.[1,2] There are various surgical approaches for removing the necrosum. Minimally invasive retroperitoneal pancreatic necrosectomy (MIRP) is a relatively novel approach with early encouraging results and is safe in the surgical management of well-selected cases of necrotising pancreatitis.

CASE REPORT

A 51-year-old Chinese male smoker was admitted with acute pancreatitis (white cell count of 19.3, serum amylase of 3,483 U/L and blood sugar 12.6 mmol/L). On admission, contrast-enhanced computed tomography (CT) showed an oedematous pancreas with gallstones (Fig. 1). Endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy with common bile duct stone clearance was done within 24 hours. CT performed on the fifth day showed pancreatic necrosis, pancreatogenic ascites, paracolic fat stranding and a phlegmon along the greater curvature of stomach. The patient deteriorated and went into septic shock. As maximal intensive support and CT-guided drainage failed to show clinical improvement, he was operated upon.

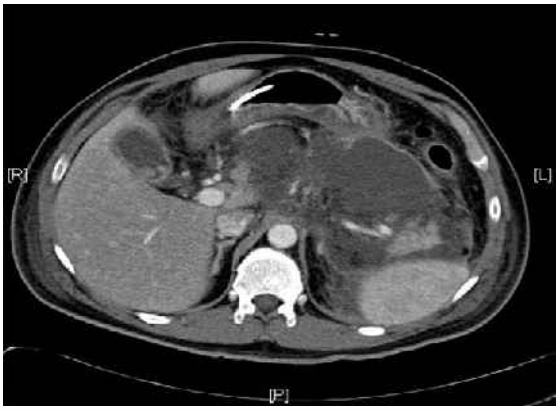

Fig. 1 Axial CT image shows pancreatic necrosis.

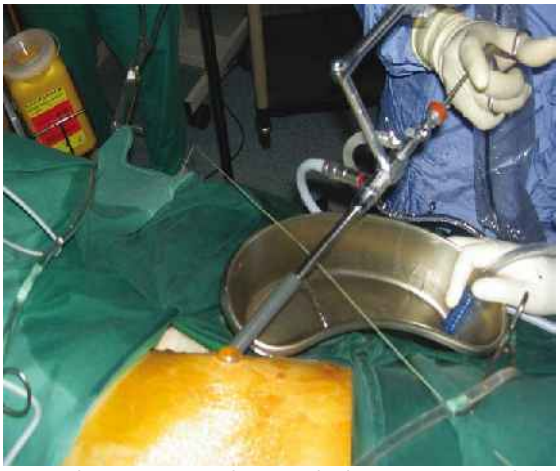

Fig. 2 Intraoperative photograph shows insertion of the Seldinger guide wire and Amplatz dilator to the necrotic area.

The Seldinger guide wire was inserted through the percutaneously sited drain (Fig. 2). The tract was dilated and the necrosum was removed under direct vision using a nephroscope (Fig. 3). Intraoperative fluid culture revealed *Pseudomonas* species and *Escherichia coli*. He received continuous postoperative lavage

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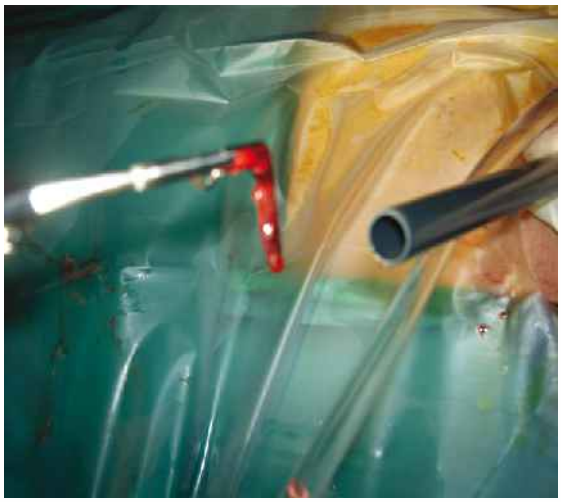

Fig. 3 Intraoperative photograph shows the necrotic pancreatic tissue being removed

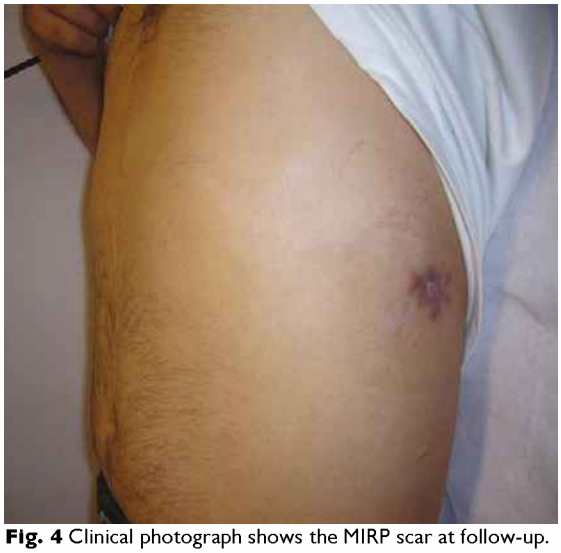

Fig. 4 Clinical photograph shows the MIRP scar at follow-up.

(125 ml/hr) through the tube drain inserted through the MIRP access site. The procedure was repeated based on appearances at the previous procedure and repeat CT findings. The irrigation was gradually stopped as the patient was well clinically (temperature and heart rate), haematologically (white cell count and C-reactive protein) and radiologically (contrast CT), and that the drainage effluent was clear with no enzyme activity. The drainage tube was gradually withdrawn 2–3cm every third day until total removal. After eight sessions of necrosectomies spanning over a period of 50 days and removing a total of approximately 200 g of necrosum, the patient's condition settled (Fig. 4). He underwent laparoscopic cholecystectomy eight weeks later and was well at follow-up.

DISCUSSION

While the morbidity and mortality of mild self-limiting acute pancreatitis are acceptably low, patients with necrotising pancreatitis can rapidly succumb despite maximal intensive care. A multitude of factors are implicated for the poorer outcome of this subset of patients; the list is headed by infection within the necrosum. Pancreatic necrosis is devitalised tissue that can be either pancreatic parenchyma or peri-pancreatic tissue.[3] Microcirculatory derangements have been implicated in the pathogenesis of acute pancreatitis and also in the progression to necrotising pancreatitis. The ischaemia-reperfusion injury due to microcirculatory derangements is implicated in the pathogenesis of acute pancreatitis. However, almost every case of severe acute pancreatitis is also associated with pancreatic duct disruption (disconnected duct syndrome), and hence the traditional concept of auto digestion of the pancreas by its own enzymes cannot be completely discounted.

In IPN, maximal optimal intensive care may not be able to halt/reverse disease progression in some patients. Oxygen debt produced, due to imbalance between pro-inflammatory (free oxygen radicals, leukotrienes, platelet activating factor, interleukins, bradykinins and endothelin) and anti-inflammatory (nitric oxide and IL-10) mediators, eventually affects remote organ systems with poor outcome. Most of the deaths occurring earlier in the course of the disease are due to multiple organ dysfunction syndrome (MODS). Infection is the super added compounding insult for the survivors.

Prediction of severity is core to the management. The Ranson and Imrie scoring systems have sensitivity of about 80% at 48 hours, and acute physiology and chronic health evaluation (APACHE) II system has a sensitivity of around 85% for score > 9 on admission. Serum biomarkers, such as C-reactive protein (> 150 mg/L at 48 hr), IL-8, IL-6, procalcitonin, IL-10 and IL-1 beta-receptor antagonist, are predictors of severity. Recently, urinary trypsin activation peptide and serum amyloid-A have also been studied as early markers for severity prediction.[4] CT severity index offers little advantage for predicting severity, and we stress that its main role should be in determining the extent of necrosis and serially monitoring the progress.

Infection in the pancreatic necrosis is not a clinical diagnosis, due to overlap of features with systematic inflammatory response syndrome; the latter would be evident. Acute infective pancreatic necrosis is an objective diagnosis following positive culture or contrast-enhanced CT showing gas pockets in/around the necrosum. Serum procalcitonin is a biomarker of infection and is a valuable tool. Bacterial isolates are of

Table I. Various methods of open necrosectomy.

Procedure	Main proposer	Comments
Necrosectomy + conventional drainage	Altemeier & Alexander[8]	As the necrotising process is ongoing, multiple second laparotomies are often necessary. Necrotic contents are thick and hence there are high chances for drainage tube blockage. Mortality is high.
Necrosectomy + closed lavage	Beger et al[9]	Removal of both "toxic" and "septic" substances is best achieved with continuous lavage. Re-explorations may be needed. Drains can be removed when the effluent is clear and enzyme activity is low.
Laparostomy + open/semi-open packing + planned reoperations +/- zipper	Bradley[10]	The scheduled reoperations are done under sedation in intensive care unit (ICU) and hence a longer ICU stay is anticipated. Laparostomy is a "damage control" measure in a moribund patient. The "zipper" reduces operative time and minimises evaporative fluid loss. High incidence of enteric fistulas and haemorrhage are seen due to repeated debridement, adhesions and pressure necrosis.
Retroperitoneal approach	Fagniez et al[11] and Villazon et al[12]	This approach does not contaminate the peritoneal cavity and serves the purpose of removing the necrosum with continuous lavage. There is a high risk of left sided colonic fistulas and access to biliary tree is not possible.

endogenous origin from the gut and suggests mucosal barrier disruption. In various trials, imipenem has shown to reduce incidence of infection to the necrosis; however, routine use has not been shown to reduce mortality or need for surgery, and on the contrary, may give rise to drug resistance or fungal infections with increased mortality.[5]

Sterile necrosis can either resolve, form peri-pancreatic fluid collections, pseudocyst or can become infected. While every tenth patient with sterile necrosis has the potential to die, every other patient can succumb to IPN. Pancreatic abscess is a delayed complication of fluid collection secondary to infection after 3–4 weeks of the acute attack, and should not be confused with pancreatic necrosis. Patients with necrotising pancreatitis should be managed intensively as they have a potential for developing MODS. The demarcation of necrotic tissue takes at least one week after the acute attack, and hence, surgery should be delayed until at least the second week of the attack, when possible. Removing the necrotic tissue removes the toxic inflammatory mediators that can gain systemic access via portal circulation or retroperitoneal lymphatics. Various studies have been supportive of initial conservative management followed by surgery when indicated.[6,7]

The current consensus is for the removal of necrosum and preservation of viable pancreas along with maximal physiological support. In the past, surgical management consisted of tissue sparing procedures to total pancreatectomy. Removal of the necrosum has been done by various open procedures (Table I), until recently, when laparoscopy has entangled necrosectomy into its claws. The endoscope can be introduced through the mature drainage tube sinus tract after formal open necrosectomy. This "sinus tract endoscopy" can remove both the residual necrosum and the ongoing developing necrosis. In a series of 11 consecutive patients managed by translumbar retroperitoneal endoscopy, Castellanos et al concluded that this procedure had no added morbidity/mortality, facilitated lavage, minimised the need for subsequent surgeries, and decreased the need for repeated CT.[13]

If acute pancreatitis is a model of sepsis, then conventional surgery with its high complication rates is the second hit,[14] which could in part be accountable for high mortality. This concept, compounded by the fact that minimal access surgery has less activation of inflammatory response than equivalent open surgery,[15] paved the way for MIRP. MIRP demands technical expertise and the availability of skilled interventionist. MIRP is unlikely to be successful when there is a "horseshoe" necrosis extending into both paracolic gutters, and when the extent of necrosis is multifocal and discontinuous. MIRP is also not suitable for necrosis of head/uncinate process of pancreas due to difficult access. Lack of access route for guide wire insertion is also an indication for standard open necrosectomy. We employed the technique proposed by Carter et al.[15] There are series/reports of laparoscopic management of infected necrosis via percutaneous and transgastric routes.

Percutaneous drainage of pancreatic necrosis is minimal access procedure for drainage of abscess and has a success rate of less than 50%. MIRP requires multiple sessions, as it is difficult to remove necrosum completely in a single sitting. With MIRP, it is not possible to deal with gallstones. Our case demonstrates that ERCP can help clear the duct in emergencies and the gallbladder can safely be removed at a later date when the patient has recovered. The early data on MIRP shows it to be beneficial over conventional

Table II. Reported case series on MIRP.

Series	No. of patients	No. of sessions	Median hospital stay (days)	Mortality (%)	Complications/open procedure
Carter et al[15]	10	1–4	42	20	One bleeding
Connor et al[16]	21	1–8	51	25	Three sepsis, two bleeding
Risse et al[17]	6	1–4	75	17	One peritonitis, one pyrexia, one colocutaneous fistula, one flank hernia

surgery; however no significant statistical data is available as yet.[15-17] (Table II). The management of necrotising pancreatitis demands a high level of surgical intensive care and resources. MIRP is a new technique, technically feasible and needs multiple sessions in open surgery. It is limited by the fact that it cannot be applied universally. However, when it is applicable, there are benefits of reducing the physiological insult in the gravely-ill patient. It is also suggested that MIRP reduces the need for intensive unit care. We believe that MIRP should be considered in all patients with severe acute necrotising pancreatitis when necrosectomy is indicated, and it is clinically feasible by minimal access route.

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