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REVIEW ARTICLE

Role of Minimal Access Surgery in Management of Infective Pancreatic Necrosis

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ABSTRACT

Aim: To study various minimal access surgical techniques of pancreatic debridement for infected pancreatic necrosis (IPN).

Method: A review of literature is done using various search engines like Google, Yahoo, PubMed, etc. by using keywords: Pancreatic necrosectomy, laparoscopic, endoscopic pancreatic necrosectomy.

This article reviews various methods of minimally access pancreatic necrosectomy (MAN) can be classified by the type of scope used flexible endoscope, laparoscope, nephroscope and the route of access transperitoneal, transgastric, retroperitoneal. Each of the scopes and access routes has its advantages and disadvantages.

Result and conclusion: Only few large series of cases of MAN have been published, rest are limited to case reports. There are no comparisons of results, either with open surgery or among different minimal access surgeries but a body of evidence now suggests that acceptable outcomes can be achieved and minimal access necrosectomy is technically feasible, well tolerated and beneficial for patients when compared with open surgery.

Keywords: Infected pancreatic necrosis, Minimal access pancreatic necrosectomy.

INTRODUCTION

The gold standard for treatment of infected pancreatic necrosis (IPN) is surgical debridement. It can be achieved by open and minimal access surgical approaches. Open surgery for this condition carries a mortality rate of up to 50%,[1,2] therefore, a number of such techniques have been developed.

Pancreatic necrosis is defined as a diffuse or focal area of nonviable pancreatic parenchyma that typically is associated with peripancreatic fat necrosis.[3] Necrosis can be sterile or infected. IPN is the leading cause of death associated with severe acute pancreatitis. The incidence of acute pancreatitis varies from 10 to 40 per 100,000 population. The proportion of patients that develop pancreatic necrosis is approximately 15 to 20%. Approximately, 40% of these patients go on to develop infection of the necrosis. The overall mortality of edematous pancreatitis is 1% or less, that of sterile necrosis 5% and that of infected necrosis 10 to 20% in centers of excellence.

PATHOGENESIS OF IPN

Pancreatic necrosis occurs within the first few days of the onset of acute pancreatitis. Out of all the patients who develop pancreatic necrosis, 70% have evidence of this by 48 hours of the onset of abdominal pain and all of them by 96 hours. The premature activation of proteolytic enzymes within the acinar cells and interstitium of the lobule results in extensive necrosis of acinar cells and the substantial interstitial and intravascular accumulation and activation of leukocytes.

There are a number of factors that contribute to the failure of the pancreatic microcirculation, which is evident histologically as stasis and/or thrombosis of intrapancreatic vessels. The failure of the pancreatic microcirculation leads to ischemia, which compounds the enzymatic and inflammatory injury and leads to the full syndrome of necrotizing pancreatitis. During this first week or two, in the so-called vasoactive phase, there is the release of proinflammatory mediators that contribute to the pathogenesis of pulmonary, cardiovascular and renal insufficiency. This early systemic inflammatory response and multiorgan dysfunction are found frequently in the absence of pancreatic infection. In the later septic phase, which occurs in some patients after 3 to 4 weeks, these systemic events occur as a consequence of pancreatic infection.

There are five routes by which bacteria can infect pancreatic necrosis. These are as follows:

- Hematogenous through mesenteric vessels to the portal circulation
- Transpapillary reflux of enteric content into the pancreatic duct
- Translocation of intestinal bacteria and toxins via the mesenteric lymphatics to the thoracic duct and the systemic circulation
- Reflux of bacteriobilia via a disrupted pancreatic duct into the necrotic parenchyma and
- Transperitoneal spread.

Cultures of infected pancreatic necrosis yield monomicrobial flora in three-quarter of patients. Gram-negative aerobic bacteria usually are responsible (e.g. *Escherichia coli*, *Pseudomonas spp.*, *Proteus* and *Klebsiella spp.*), and this strongly suggests an intestinal origin, but Gram-positive bacteria (e.g. *Staphylococcus aureus*, *Streptococcus faecalis* and *Enterococcus*), anaerobes and occasionally, fungi also have

been documented. The spectrum of bacteria cultured from infected necrosis has altered with the more widespread use of prophylactic antibiotics, with a shift toward Gram-positive bacteria and fungal infections.[5]

The necrotizing process can extend widely to involve retroperitoneal fat, small and large bowel mesentery and the retrocolic and perinephric compartments.

DIAGNOSING OF IPN

The clinical symptoms and signs of pancreatic necrosis are indistinguishable from those of other patients presenting with acute pancreatitis. Abdominal pain, distension and guarding are associated with a low-grade fever and tachycardia. The severity of pain and the extent of hyperamylasemia do not correspond with the severity of acute pancreatitis. Patients presenting late with severe disease often will have established multiorgan dysfunction.

The classic skin signs of retroperitoneal necrosis are discoloration at umbilicus (Cullen's sign), the flanks (Grey-Turner's sign) and the inguinal region (Fox's sign), are rare and often not seen until the second or third week. The diagnosis of pancreatic necrosis requires more than clinical acumen.

The gold standard for the diagnosis of pancreatic necrosis is contrast-enhanced CT scanning demonstrating hypo-perfusion in the arterial phase. In the absence of a specific marker of pancreatic necrosis, many serum predictors have been proposed C-reactive protein (CRP) as the most widely used predictor of pancreatic necrosis and is useful as a daily monitor of disease progress. The accuracy in detecting necrosis is about 85%, but it requires 3 to 4 days to reach this level. The threshold values depend on the assay and the study used. The most commonly used threshold is greater than 120 mg/l.

Other prognostic markers, none of which has been proven to outperform CRP, include interleukin-6 (threshold > 14 pg/ml) which peaks a day earlier than polymorphonuclear elastase (threshold > 120 gm/l), which peaks early and reflects neutrophil activation and degranulation; and phospholipase A_2 type II (threshold > 15 units/l). Urinary trypsinogen-activating peptide and serum amyloid-A have also been studied as early marker for severity prediction.[4]

In practice, the indications for a CT scan to diagnose and determine the extent of pancreatic necrosis are the prediction of severe pancreatitis (usually during the second week), when a patient fails to improve with initial resuscitation and/or when the CRP has crossed the diagnostic threshold (see above). The CT scan can be used to grade the severity of acute pancreatitis [CT Severity Index (CTSI)] based on the extent of extrapancreatic changes and pancreatic necrosis.

The importance of the diagnosis of pancreatic necrosis is to initiate intensive-care management, which may necessitate transfer of the patient to a tertiary unit. The diagnosis of infected necrosis is imperative because it is an absolute indication for surgical intervention. It is more usual to suspect pancreatic infection with a secondary deterioration, often during the third and fourth weeks of admission. This is often heralded by a significant rise in CRP.

A CT scan often will confirm the presence of a tense collection with rim enhancement arising from a region of pancreatic necrosis. The presence of gas within the tissues confirms infection, with an 'air bubble' appearance, but this is present in the minority of cases. Infected necrosis usually is confirmed by fine-needle aspiration (FNA) for Gram's stain and bacterial culture. This can be guided by US or CT scan and is considered safe and reliable.

MANAGEMENT OF IPN

The goals of surgical management are to remove necrotic and infected tissue, drain pus, lavage the peritoneal cavity and avoid blood loss and injury to other organs. Few advocate only observational nonoperative intensive approach to manage IPN.[7] Preservation of vital intact pancreatic tissue is important. The choice of operation is determined by the location, extent and maturity of the necrotic material; status of the infection; the patient's condition, the degree of organ dysfunction and the preference and experience of the surgeon.

A number of different approaches have been described some of which are only of historical interest. Necrosectomy is complex, fraught with potentially life-threatening complications and should be left to the experienced surgeon. None of these surgical methods have been subjected to a randomized, controlled trial, and the minimal access approaches are still evolving. The latter are best suited to treatment of well-demarcated and localized necrosis in the later stage of the disease.

One possible benefit of this approach is a reduction in the number of patients who need intensive-care support. The minimal access surgical approaches to pancreatic necrosectomy can be classified according to the type of optical system (flexible endoscope, laparoscope or operating nephroscope) and the route used (via the stomach, peritoneum or retroperitoneum).

Open and Minimal Access Approaches to the Treatment of Pancreatic Necrosis

As per review of literature,

- Open approaches:
  - Pancreatic resection
  - Necrosectomy + wide tube drainage[8]
  - Necrosectomy + staged laparotomy (reexploration)
  - Necrosectomy + drainage + relaparotomy
  - Necrosectomy + laparotomy + open packing[10]
  - Necrosectomy + drainage + closed continuous lavage[9]
  - Retroperitoneal routed necrosectomy[11,12]
- Minimal access approaches:
  - Laparoscopic necrosectomy
  - Laparoscopic intracavitary necrosectomy
  - Laparoscopic-assisted percutaneous drainage
  - Laparoscopic transgastric necrosectomy

  - Laparoscopic transmesocolic necrosectomy
  - Laparoscopic transgastrocolic necrosectomy
  - Endoscopic transgastric necrosectomy
  - Endoscopic transduodenal necrosectomy
  - Percutaneous necrosectomy and sinus tract endoscopy[15]
  - Translumbar retroperitoneal endoscopic necrosectomy[13]
- Radio-guided surgical approaches:
  - MRI-assisted necrosectomy[6]
  - Video-assisted retroperitoneoscopic debridement[17]
  - Nephroscopic retroperitoneal[16]
  - Endoscopic transgastric necrosectomy
  - Endoscopic transduodenal necrosectomy
  - Endoscopic transpapillary necrosectomy
  - Endoscopic transmural necrosectomy
  - Combined method
  - EUS-guided drainage.

TIMING OF SURGERY

There has been a change in the treatment standard for necrotizing pancreatitis from an aggressive policy favoring early surgical intervention to a more conservative strategy of delayed and less invasive intervention.[7] Early surgery was advocated in order to remove the focus of infection and terminate the inflammatory process.

However, the inflammatory cascades are not easily switched off and are compounded by the surgery itself. Early surgery is more difficult because necrotic tissue is immature and not easily separated from viable tissue, resulting in a significant risk of bleeding. Additionally, early surgery may infect sterile necrosis. Delayed surgery may allow time for stabilization of the patient and the more easy removal of well-demarcated necrosum.

There is a balance between operating too early and leaving it too late and the decision needs to be individualized. The decision is aided by close surveillance of the patients' clinical trajectory with frequent clinical review and daily CRP measurements.

From a review of published studies, the lowest mortality is associated with surgery after 3 to 4 weeks. However, the clinical picture (severity and evolution) should be the primary determinant of the timing of intervention.

BASIC PRINCIPLES OF PANCREATIC NECROSECTOMY BY OPEN TECHNIQUE

Pancreatic resection is a historical approach that has been associated with unacceptable complication and mortality rates. Pancreatic necrosectomy involves removing the devitalized pancreatic and peripancreatic tissue and drainage of associated pus. The usual approach to the pancreas is through the gastrocolic omentum into the lesser sac.

Sometimes, it is easy to enter the region through the transverse mesocolon from the greater sac and to the left of the DJ flexure. At the same time, it is useful to take down both colonic flexures, providing better exposure and reducing the risk of subsequent injury to the colon from tube drains.

The head of the pancreas then can be approached anteriorly and posteriorly (after Kocherization). If the abdomen is opened though a bilateral subcostal incision, inline with the opening to the lesser sac, subsequent laparotomies do not need to disturb the greater peritoneal sac or the upper abdomen.

It is not necessarily a one-stage procedure, especially if an early necrosectomy is embarked on. Necrosectomy is a careful process, best accomplished by an educated finger. The extent of the cavity can be explored and the gentle separation of necrotic material accomplished. Necrotic extensions from the primary cavity need to be explored, often into the root of the small bowel mesentery and down the retrocolic gutters.

Care must be taken to remove only what easily separates and to avoid injury to major vessels. The removal of necrotic material may be assisted by irrigation, pulsatile irrigation, gauze and sponge forceps. When contained by a mature wall, it is advisable to avoid opening up the area too widely. The next step is placement of large-bore, soft, dependent drains to cover all the regions of what is often a complex area.

Continuous lavage with peritoneal dialysis fluid, at flow rates of 300 to 1000 ml/h, may reduce the need to reoperate and often is continued for 2 to 3 weeks. The most common postoperative complications are hemorrhage and fistulization (pancreas, small and large intestine). The use of packing is lifesaving for major hemorrhage that occurs at the time of necrosectomy, but when used routinely, it is associated with a higher incidence of enteric fistulas.[3]

NEPHROSCOPIC RETROPERITONEAL PANCREATIC NECROSECTOMY[16]

Under CECT guidance, access to the necrotic cavity is obtained via the predetermined approach. Under local anesthetic (in the absence of mechanical ventilation), an Accustick set is used to access the area of necrosis. This is subsequently exchanged (with the use of a guidewire) for a percutaneous drainage catheter. The patient is transferred to the operating suite.

Depending on the patient's condition, the following procedure can be performed under either general anesthetic or local anesthetic infiltration with IV sedation (anesthetist controlled). The patient is placed supine and a sandbag can be used under the site of catheter entry to improve access to the tract with the operating nephroscope. The entry site is prepared in a sterile fashion using a waterproof drape with a catch all as used for urological procedures as large amounts of irrigation are required. Under fluoroscopic control, the previously placed percutaneous catheter is exchanged for a guidewire.

Using a Seldinger technique the tract can then be dilated to 30 French using a renal dilatation set. It is important to reinforce the guidewire with the supplied plastic tapered sheath to prevent buckling and misplacement of the wire. A three-dimensional concept of the surrounding structures as shown by the CE-CT

is crucial to avoid inadvertent injury to surrounding vessels and viscera.

There should be very little resistance to dilatation and any resistance encountered should lead to reevaluation to the line of dilatation. The exception to this is during introduction of the dilators through the skin, subcutaneous tissues and rib space and if this creates a problem increasing the size of the wound and dissecting down to the entry site may aid insertion.

With the tract dilated, an Amplatz sheath is placed over the dilator and a rigid operating nephroscope can be introduced into the cavity. The scope requires both an irrigation and biopsy channel. With continuous irrigation (warm sterile 0.9 % saline, 10–20 liters) under direct vision the necrosis can be removed piecemeal. It is vitally important that granulating tissue, visible vessels (aorta, superior mesenteric artery, splenic artery) or adherent tissue is not biopsied as it may result in catastrophic bleeding.

Often at the first procedure, minimal necrosis can be removed and it is prudent to be conservative with this attempt. The procedure should be repeated on a weekly basis until the cavity appears clear and all visible necrosis is removed. At the end of each procedure, an irrigating system is constructed using a 28 French chest drain with extra side holes (cut to shape) sutured to a 10 French nasogastric tube. This is passed along the established tract until resistance is met and then secured with a suture to the skin. Post-operatively, this can be irrigated with 0.9% saline via the nasogastric tube at a rate of 50 to 250 ml/hour depending on the degree of contamination.

PERCUTANEOUS NECROSECTOMY AND SINUS TRACT ENDOSCOPY IN THE MANAGEMENT OF INFECTED PANCREATIC NECROSIS[15]

Percutaneous Drainage

Percutaneous drains placed by the interventional radiologist in the treatment of infected necrosis should be used cautiously. The catheter size will not cope with the solid necrotic tissue. It achieves drainage and not necrosectomy. There are two settings in which percutaneous drainage is useful. The first is in an unstable septic patient with evidence of a tense rim-enhanced collection (pancreatic abscess) with a significant fluid component on CT scanning.

Percutaneous drainage in this setting may take the 'heat out of the fire', allow stabilization of the patient and 'buy time' until necrosis is more amenable to surgical removal. The second setting in which percutaneous drainage is important is to establish the optimal route for dilatation and subsequent percutaneous necrosectomy, should this be appropriate. This will require careful discussion between the radiologist and surgeon. It usually involves a left-flank puncture and a route along the axis of the body/tail of the pancreas.

Percutaneous Necrosectomy

Under computed tomography guidance, an 8 French pigtail nephrostomy catheter is inserted into the infected cavity, the surgeon having carefully selected a path that will allow subsequent dilatation. Correct route is to enter the area of infected necrosis between the lower pole of the spleen and the splenic flexure. In predominately right-sided pancreatic head necrosis, a route through the gastrocolic omentum, anterior to the duodenum is selected.

However, this results in a more technically difficult necrosectomy and prevents dependent postoperative drainage. The catheter is secured and the patient transferred to the operating room. With the patient under general anesthesia, access to the abscess cavity is maintained using a guidewire, over which the catheter tract is then dilated to 30 French using graduated dilators and radiologic guidance.[12] This allows a 30 French Amplatz sheath to be inserted. An operating nephroscope that allows intermittent irrigation and suction, with a 4 mm working channel, is then passed along the Amplatz sheath into the abscess cavity. Piecemeal removal of solid material is then performed using soft grasping forceps through the working channel by repeatedly passing the instrument into the cavity until all loose necrotic tissue is removed.

Finally, an 8 French umbilical catheter sutured to a 28 French tube drain is then passed over a 12 French stiffener to the distal end of the cavity to allow continuous postoperative lavage (500 ml/hr) through the umbilical catheter. Because of the high-volume lavage, we use a fluid normally used for peritoneal lavage to minimize the potential of electrolyte imbalance. The lavage is continued at this rate until the lavage fluid clears or until a further procedure.

SINUS TRACT ENDOSCOPY

In patients with a previous primary debridement, either at open laparotomy or after the above technique, in whom residual sepsis is suspected, a second computed tomogram is obtained and, provided there are no satellite collections, secondary sinus tract endoscopy is performed. In the operating room and under general anesthesia, the previously sited drain or drains are removed. Either a flexible or a rigid endoscopic system is used, depending on the suspected amount of residual necrosis.

Sinus tract endoscopy using a flexible endoscope is tedious and only small fragments of necrotic tissue can be removed with each pass of the endoscope. As a result, we have moved to using the operating nephroscope as described above for most primary explorations. The major alteration in the technique is that the Amplatz sheath is not required. Access to pockets of necrosis is occasionally limited by the rigidity of the system, and flexible endoscopy remains useful to check the tract before drain removal if residual necrosis is not suspected.

For flexible endoscopy, each tract is dilated to 45 French using a balloon dilator. A twin-channel endoscope is then passed through the skin opening. Further endoscopic antegrade dilatation of the tract is then performed until the entire length of the drain tract can be visualized. Jet irrigation using a heater probe and suction allows fluid collections to be cleared, and residual solid necrotic tissue or adherent slough can be teased away using a variety of endoscopic instruments (e.g. snares, stent retrieval forceps). A guidewire is then passed through the endoscope and an 8 French umbilical catheter sutured to a 28 French tube drain is placed in the cavity, after which lavage begins again.

Method of EUS-Guided Endoscopic Transgastric Pancreatic Necrosectomy[18]

Pancreatic pseudocyst drainage was the first therapeutic application of EUS. The cyst is punctured under ultrasound guidance, contrast injected and a guidewire inserted. Initial dilation to 8 mm is performed over the wire. The EUS scope is then exchanged over the wire for a forward viewing endoscope. A second dilation to 18 mm is performed. This enables entry of the endoscope into the cyst perform cystoscopy, debridement if necessary and insertion of multiple large bore double pigtail stents.

We report on the use of the prototype forward viewing echoendoscope in six consecutive patients who were referred for pancreatic cyst drainage. Here, you see endoscopic view indistinguishable from that of a gastroscope showing a bulge where the cyst impinges against the posterior gastric wall. Power Doppler is switched on and highlights multiple vessels interposed in the wall. This allows selection of a safe vessel-free window for a cyst puncture. A 19 G needle is advanced into the cyst lumen. A sample of contents is aspirated for fluid analysis.

A guidewire under ultrasound guidance is inserted into the cyst. An 18 mm balloon is coaxially thread over the wire and advanced across the cyst wall. Note that resistance is encountered, but the forward transfer of force overcome this. The dilation is performed under forward viewing endoscopic and ultrasound guidance. As the balloon is maximally inflated we see the cystogastrostomy open up. The balloon is then deflated while simultaneously advancing the scope into the cyst cavity.

Cystoscopy is now performed showing the cyst contents to be filled with pasty wall-adherent necroses. Pulsed power Doppler is switched on, we can see and hear arterial flow vessels within the wall of the cyst. This identifies sensitive areas at bleeding risk when performing debridement. In this case, vigorous water jet irrigation is performed through an accessory water irrigation channel built into the echoendoscope.

This issued to clear nonadherent debris. Our experience has shown that it is not necessary to actively remove wall-adherent debris using extraction tools as such Dormia or Roth net basket to achieve cyst resolution. Three large bore 10 French double pigtail stents are now inserted into the cyst under direct endoscopic guidance. The first stent is delivered over a guide catheter then the second stent and lastly the third stent. All three stents are deployed. Finally, a nasocystic catheter is inserted for maintenance of irrigation.

DISCUSSION

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 accountable for high mortality.

In IPN, maximal optimal intensive care may not be able to halt/reverse disease progression in some patients. Most of the deaths occurring earlier in the course of the disease are due to multiple organ dysfunction syndromes (MODS). Infection is the superadded 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.

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.

Sterile necrosis can either resolve from peripancreatic fluid collections, pseudocyst or can become infected. 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. 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.

Minimal access pancreatic necrosectomy has its own limitations. Each of the scopes and access routes has its advantages and disadvantages. The scopes can be compared for field of view, working channel for instruments and irrigation, external diameter, length, flexibility and angulation.

The routes can be compared for ease of access, risk of collateral injury and unnecessary contamination, and the ability to deal with multiple and complex collections. The two

approaches that have risen to favor are the endoscopic transgastric and nephroscopic retroperitoneal routes, probably because they are based on conventional operations.

The former is an adaptation of an open surgical approach, developed to treat retrogastric pseudocysts, that has been extended to include endoluminal ultrasonographically-guided transgastric puncture of the lesion, balloon dilatation of the track, insertion of multiple stents, direct basket extraction of necrosum and transpapillary stenting of the pancreatic duct. These technically demanding endoscopic maneuvers are likely to become more widespread and supercede the laparoscopic transgastric operation.

The endoscopic transgastric procedure avoids peritoneal contamination and external pancreatic fistula formation, but it may not be possible if there is no abutment of the lesion against the stomach or duodenal wall. The nephroscopic retroperitoneal procedure has been advocated by the Glasgow group and appears now to be the most popular MAN approach. It is an adaptation of the open lumbotomy technique to left sided organized pancreatic necrosis.

MAN has now passsed the stage of feasibility testing and it can be done. What is now needed is evidence to guide the decision about which technique should be selected for which patient and about the timing of its application.

It appears to be associated with a reduction in duration of stay in the intensive care unit. Another challenge to progress is technical and involves the extraction of necrosum. With MAN, the 'educated finger' cannot be deployed for digital debridement. The small forceps and baskets currently in use mean tedious, piecemeal extraction.

Now, with the advent of robotic surgery even within few years it will be possible to perform the IPN surgery with maximum accuracy. However, a prospective double-blind study is required for the same over a span of at least 5 years with meticulous follow-up and data recording.

CONCLUSION

Pancreatic necrosectomy by minimal access surgery is feasible and on the available evidence there is no doubt that it has a major role to play in reducing both systemic insult and the subsequent mortality, but it demands technical expertise and availability of skilled interventionist. It requires multiple sessions as it is difficult to remove necrosum in a single sitting.

Currently, majority of patients are suitable for minimal access surgery and with the development of better instruments and increasing experience this number is likely to increase, although it is unlikely to completely replace open necrosectomy.

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