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Open Internal Stenting of the Main Pancreatic Duct as Life-Saving Surgery in a Critically Ill Patient with Chronic Frequently Relapsing Pancreatitis and Pancreatic Ductal Hypertension

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 63

Final Diagnosis: Chronic frequently relapsing alcoholic pancreatitis (relapsing phase) • pancreatic ductal hypertension • parapancreatic inflammatory mass • fermentative ascites-peritonitis • two-sided pleural effusions • sepsis • cachexia

Symptoms: Upper abdominal pain • fatigue • an increase in volume of the abdomen • loss of appetite • weight loss

Medication: —

Clinical Procedure: Open internal stenting of the pancreatic duct

Specialty: Surgery

Objective: Management of emergency care

Background: The effective and safe treatment of chronic frequently relapsing pancreatitis is challenging.

Case Report: We present the case of a 63-year-old male patient with severe complications of this variant of the disease: parapancreatitis with the formation of an inflammatory mass, fermentative ascites-peritonitis, 2-sided pleural effusions, sepsis, and cachexia. Conservative treatment was ineffective, and emergency surgery was chosen. A novel surgical procedure – open internal stenting of the main pancreatic duct via pancreatowirsungotomy and duodenotomy – was used successfully in this difficult case. The elimination of pancreatic ductal hypertension and maintenance of maximum physiological pancreatic juice outflow, achieved via surgery, led to rapid improvement in the patient's condition. He was discharged on the 26th day after surgery. The clinical outcome was good at the 2-year follow-up.

Conclusions: Open stenting of the main pancreatic duct can be recommended for treating patients similar to the patient described in this paper – having severe complications of CP against the background of a relapse, exhaustion, and being in a severely or critically ill general condition. This surgical procedure is especially important when minimally invasive methods of eliminating pancreatic hypertension are technically unsuccessful or impossible due to the lack of necessary equipment and staff.

MeSH Keywords: Pancreatic Ducts • Pancreatitis, Chronic • Stents

Abbreviations: **PDH** – pancreatic ductal hypertension; **CP** – chronic pancreatitis; **OISPD** – open internal stenting of the pancreatic duct

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/916587

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Background

Pancreatic ductal hypertension (PDH) is one of the key pathogenetic factors of chronic pancreatitis (CP) [1,2]. The main causes of PDH are fibrosis and calcification of the pancreas and surrounding fatty tissue, calculi, and strictures of the main pancreatic duct. Inadequate outflow of the pancreatic juice leads to severe pain and creates conditions for CP relapse with characteristic clinical and laboratory manifestations.

In contrast to acute necrotizing pancreatitis, in exacerbation of CP developing against the background of PDH, there is no phase of acute toxemia. It is characterized primarily by severe pain, hyperamylasemia, pancreatogenic masses and fluid collections, infiltrative duodenitis, pleural effusions and peritonitis, external and internal pancreatic fistulas, portal vein thrombosis, pylephlebitis, and sepsis [3,4]. In some cases, the disease can be most clearly and concisely defined as "chronic frequently relapsing pancreatitis". Effective treatment of this form of the disease is a challenge for surgeons and internists. None of the methods of conservative and surgical treatment have the necessary combination of qualities that combine sufficient efficacy, safety, and minimal negative impact on the patient's quality of life.

One of the authors of this article (NMZ) theoretically justified and introduced into practice a new operation in CP – open internal stenting of the pancreatic duct (OISPD). The purpose of this intervention is to restore the passage of pancreatic juice from the patent part of the pancreatic duct into the duodenal lumen by creating an artificial channel in the head of the pancreas through an area of obliteration of the ductal system, the functioning of which is supported by a rigid stent. This procedure is very effective for treatment of CP relapse developing against the background of PDH, including cases of frequently relapsing pancreatitis. From June 2010 to December 2016, 38 interventions were performed. In 18 (47.4%) cases, a relapse of CP combined with PDH was present.

Here, we present one of our cases vividly illustrating the efficacy and safety of OISPD in complicated relapsing CP in a critically ill patient.

Case Report

A 63-year-old male patient was treated in our surgical department from 13 Feb 2014 to 2 Apr 2014 with acute onset of chronic alcoholic pancreatitis. He was a long-term drinker and tobacco smoker. The disease was complicated by pancreatic duct calculi, ductal hypertension, a parapancreatic inflammatory mass, fermentative ascites-peritonitis, 2-sided pleural effusions, sepsis, and cachexia.

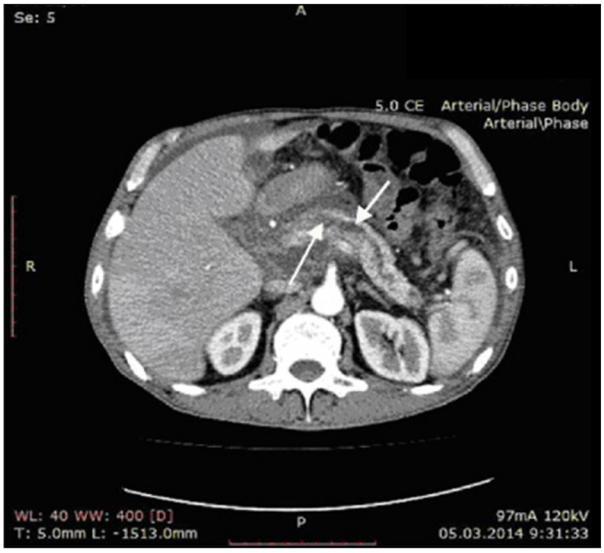

Figure 1. Contrast-enhanced computed tomogram, arterial phase. The main pancreatic duct is dilated along its length (marked by arrows). Calcifications are present in the tail of the gland.

The patient was admitted with complaints of upper abdominal pain, fatigue, an increase in volume of the abdomen, loss of appetite, and weight loss. He considered himself to have been ill since Dec 2013. He was treated in the surgical department, and then on an outpatient basis, due to the worsening of CP, and after improvement he was discharged in Jan 2014. We planned to perform OISPD 2–3 months after the inflammatory process had resolved. However, the patient's condition rapidly worsened due to the development of the next acute onset of CP. After 2 weeks of unsuccessful home treatment, he was hospitalized again on 13 February 2014. On abdominal ultrasonography, signs of acute pancreatitis, parapancreatitis, and omentobursitis were revealed. A large amount of free fluid was detected in all parts of the abdominal cavity. The blood biochemistry analysis revealed an increased amylase level (1500 IU/l). Conservative treatment was carried out (e.g., intravenous rehydration, parenteral nutrition, antibiotics, and octreotide). More than 4 liters of ascitic fluid were removed during laparocentesis, and the content of amylase in this fluid could not be measured since it was prohibitively high.

Despite ongoing treatment, the patient's condition progressively worsened. Abdominal pain persisted, exhaustion increased, and the temperature curve acquired a hectic character. The level of blood amylase increased to 2330 U/l. The patient's appetite completely disappeared, and his mental status changed. There were signs of twilight consciousness and apathy. The skin became gray and cyanotic. His weight dropped to 49 kg, with a height of 170 cm. Given the presence of ascites and pleural effusions, the patient's true weight was about 44–45 kg. A body mass index of 15.2 was accompanied by extreme exhaustion.

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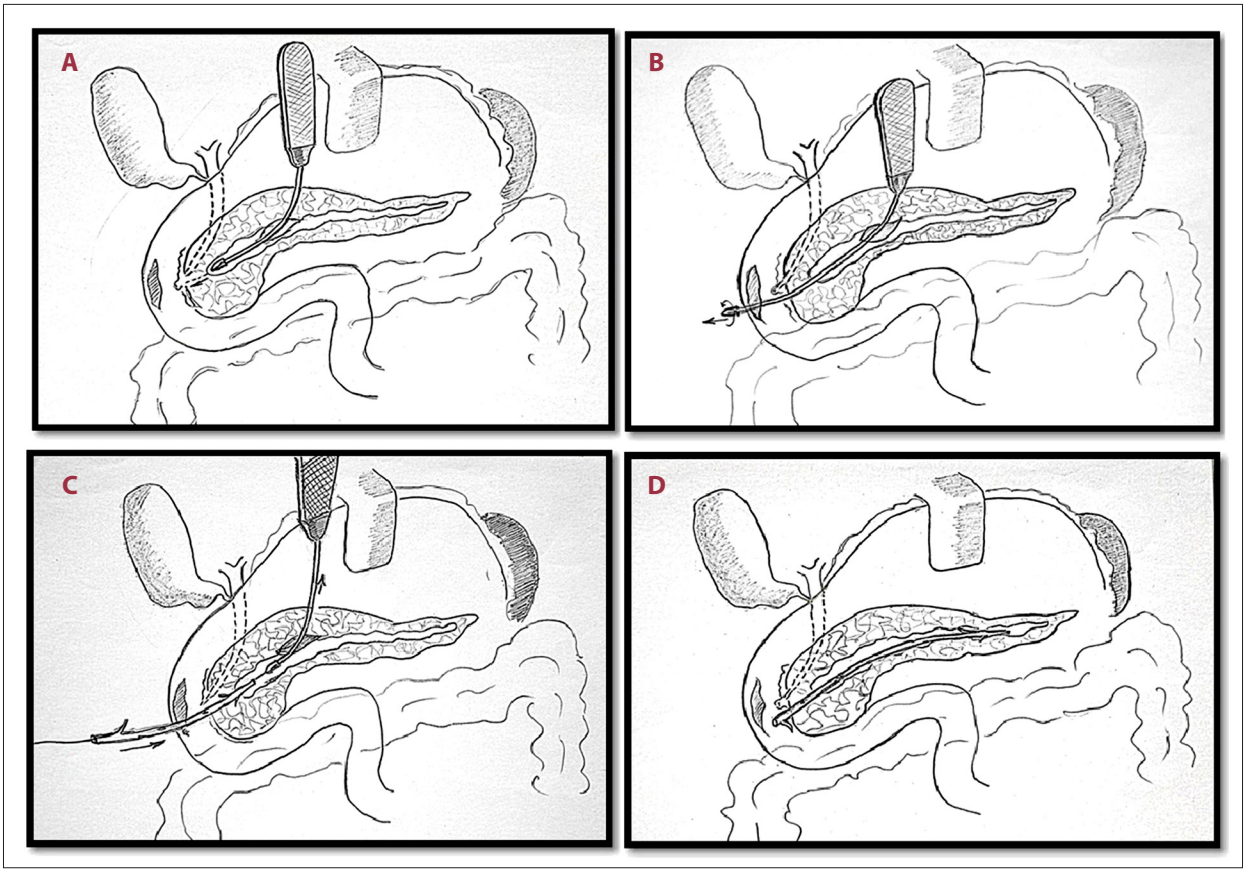

Figure 2. OISPD stages: (**A**) The tip of the bougie, inserted through the pancreatowirsungotomy into the pancreatic duct, is located close to the stenotic area; (**B**) The bougie is passed through the ductal stenosis in the pancreatic head and the medial wall of the duodenum and brought out through the duodenotomy, and then the tip of the bougie is removed (for replacement with the introducer tip); (**C**) After changing the tip of the bougie to the introducer tip, the pancreatic stent is put into an artificial channel in the pancreatic head tissue and the Wirsung duct; (**D**) Insertion of the stent is completed. The images were taken by one of the authors (NMZ).

Echography on 25 Feb 2014 showed the size of the pancreas to be 33×17×23 mm; the contours of its head were blurred and indistinct, and there was also a mass consisting of the head of the pancreas, the duodenal wall, and omentum. An area of low echogenicity measuring 27×17×16 mm was detected in the mass. The Wirsung duct was dilated to 8 mm. About 3 liters of free fluid were present in the abdominal cavity. Follow-up echography on 28 Feb 2014 showed the same findings, but dilatation of the Wirsung duct had increased to 10 mm, and the volume of free fluid had increased to about 5–6 liters. Computed tomography showed typical findings of CP relapse, with small calcifications, PDH, and ascites (Figure 1).

The persistence of PDH precluded treating the patient for the CP exacerbation, and the catabolic processes reached an almost irreversible level. We decided to perform OISPD almost as a "last chance surgery".

Surgery was performed on 7 Mar 2014 via a midline laparotomy. Four liters of clear yellowish fluid were evacuated from the abdominal cavity. In the pelvic cavity, the fluid was turbid, and there were fibrin overlaps. The stomach was slightly enlarged, and its wall was infiltrated and hyperemic. The duodenal wall was also inflamed and thickened. An inflammatory mass was palpated behind the stomach, extending down to the mesocolon and mesentery root of the small intestine and up to the hepatoduodenal ligament. The omentums and mesenteries had a few plaques of steatonecrosis. The gallbladder was not distended. The gastrocolic ligament was mobilized. The anterior surface of the pancreas was divided from the thick adhesions. Due to significant inflammation, it was almost impossible to differentiate the lower and upper margins of the pancreatic head and body.

Puncture of the Wirsung duct was performed under ultrasound guidance, followed by pancreatotomy with Wirsung duct opening. The pancreatic juice flowed freely. The bougie

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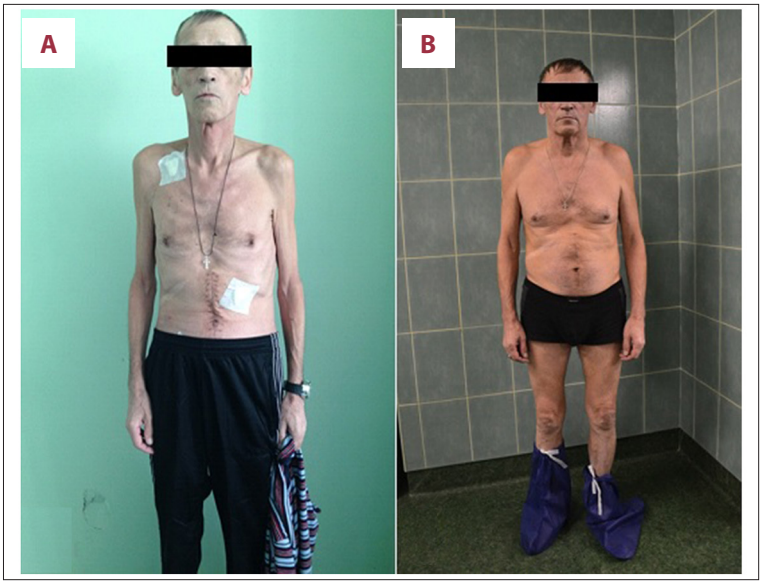

Figure 3. The patient at 2 weeks (**A**) and 10 months after surgery (**B**). Weight gain was 20 kg.

passed freely in the distal direction. During bougienage in the proximal direction, an obstruction was found about 10 mm from the duodenal wall.

Using force, the bougie was pushed through into the lumen of the duodenum, with subsequent duodenotomy. A plastic stent with a diameter of 9 Fr and a length of 9 cm (Cook, Ireland) was introduced into the main pancreatic duct. The duodenotomy and pancreatotomy incisions were sutured. The abdominal cavity and the omental bursa were washed with saline solution and drained. A diagram of the key stages of surgery is shown in Figure 2A–2D.

In the postoperative period, intravenous rehydration, antibacterial therapy, and parenteral nutrition were continued. Blood amylase level dropped to normal on the 5th day, and body temperature also normalized. The wound healed. In blood cultures, growth of *Streptococcus faecalis* was detected, but it later disappeared. About 900 ml of serous fluid was evacuated from the right pleural cavity by thoracentesis. The general condition of the patient gradually improved, including his appetite and mood. He was discharged on the 26th day after surgery.

A follow-up examination was performed in the hospital 2 months after surgery. The patient had no pain and was eating normally. His weight had reached 63 kg (weight gain of 14 kg). Blood and urine tests were within normal limits. Ultrasound findings were: chronic calculous pancreatitis, pancreas size 32×15×17 mm, and inflammatory mass completely resolved. The Wirsung duct was not dilated. There were small residual ascites, and the total volume of free fluid in the abdominal cavity was about 300 ml. There were no pleural effusions. On duodenoscopy, a patent pancreatic stent was visible.

Ten months after the operation, endoscopic replacement of the stent was performed. Upon examination of the extracted stent, it was found to have small adhesions on the walls but its lumen was patent. By this time, the patient had no complaints, and his condition was satisfactory. He had gained 20 kg in weight (Figure 3). Two years after the operation, the patient was invited for another examination. During this time, no episodes of exacerbation of CP were observed. There was no need for further replacement of the stent in this patient, so the stent was removed endoscopically. Unfortunately, the patient was then lost to follow-up. Finally, he died of acute liver failure caused by excessive alcohol consumption, 3 years and 1 month after surgery. This diagnosis was confirmed at the autopsy. There were no signs of disturbed outflow of bile and pancreatic juice, with good patency of pancreatic duct.

Discussion

Treatment of CP accompanied by PDH, especially in cases of persisting or continuously relapsing pancreatitis, is a serious problem in modern clinical medicine.

Conservative treatment, which is based on the use of analgesics, neuromodulating agents, proton pump inhibitors, octreotide, and replacement therapy with pancreatic enzymes [5,6], can only give temporary relief (and even then, not always), since it does not eliminate PDH as a key factor in the pathogenesis of CP.

Surgical treatment of CP in its relapsing stage, developing against a background of PDH, has been an unresolved problem until now. Most surgeons would not run the risk of radically operating on patients with CP relapse, preferring conservative

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therapy, with the expectation of subsiding inflammatory events. Unfortunately, the continuously relapsing form of CP is characterized by resistance to conservative treatment, and in some cases it is not possible to overcome the inflammatory process.

The standard drainage and resection-drainage procedures that are currently widely used in the treatment of CP (longitudinal pancreaticojejunostomy, Beger and Frey procedures [7]) are not applicable in exacerbation of pancreatitis. The severe inflammatory process, the presence of fluid collections, septic complications, and the poor nutritive status of the patients make the formation of pancreaticodigestive anastomoses very dangerous (or even impossible) due to the high probability of their leakage [8].

Endoscopic stenting of the pancreatic duct can help in eliminating PDH and resolving the inflammatory process, but it is not sufficiently effective due to the technical impossibility of passing the guidewire (and then the stent) into the duct [9–11], which is firmly compressed by the dense pancreas tissue in many such patients.

Another management option for PDH is the very rarely used method of open external drainage of the pancreatic duct, as advocated by Professors Nina N. Artemyeva and Nikolay Y. Kokhanenko (Saint Petersburg, Russia), and this method is almost unknown outside Russia. In this procedure, decompression of the pancreatic duct is achieved by small pancreatowirsungotomy with the introduction of 2 thin external drains in the proximal and distal direction (to the pancreatic head and tail). During surgery, stones are removed from the duct and strictures are dilated. Then, the incision in the pancreas is sutured, the drain is inserted into the omental bursa, and the integrity of the gastrocolic ligament is restored. An almost mandatory part of the intervention is jejunostomy or gastrostomy to reinfuse pancreatic juice [8].

Our (YNS) observation of patients who underwent this procedure in the clinic headed by Prof. Kokhanenko allow us to consider it safe and effective. There were no technical complications or mortality, and PDH was relieved in all cases. A very unexpected phenomenon was the significant increase in outflow rate of the pancreatic juice 2–5 weeks after surgery. It would seem that a pancreas with severe pathologic changes can produce only a small amount of juice. However, in some patients the daily volume of juice reaches 700–800 and even 1000 ml. Reinfusion of such a large volume of juice via a jejunostomy is much more convenient and comfortable for a patient than peroral administration. These data perfectly demonstrate the importance of the therapeutic role of eliminating PDH in CP surgery. Pancreatic drains should be left in place for a long time, usually several months. In some (30–50%) patients, with the subsidence of the CP relapse, the patency of the Wirsung duct in the pancreatic head is restored, and natural passage of the pancreatic juice recovers. In such cases, drains should be removed. With compliance with the indicated diet, cessation of alcohol intake, and adequate maintenance therapy, CP patients can have long-term remission. In the remaining patients in whom PDH persists, radical surgery (usually pancreaticojejunostomy) is indicated after their condition improves, the exacerbation of CP is relieved, and their nutritional status is restored [8].

Thus, neither extensive nor less-invasive surgeries, nor conservative treatment in an isolated variant, can be considered as a universal method for the management of persisting CP.

The OISPD approach developed by us combines the positive features of both minimally invasive and “big” surgeries currently used for the treatment of CP. At the same time, it is free of many disadvantages that are common in the alternative surgical interventions.

After pancreatowirsungotomy, stones can be removed from the pancreatic duct and/or strictures can be dilated, thereby improving pancreatic juice outflow. Due to severe fibrosis of the pancreatic head, forced instrumental tunnel formation in the gland tissue and medial duodenal wall is much safer than it might seem at first glance. We did not observe any significant bleeding or damage to adjacent anatomical structures. The pancreatic juice flows through the stent into the duodenum but not into the jejunum (as after pancreaticojejunostomy), which is physiologically better. Unlike procedures involving external drainage of the pancreatic duct (both less-invasive and open), our OISPD approach allows direct outflow of the pancreatic juice into the duodenum and does not require its reinfusion. During this single procedure (unlike endoscopic stenting), we can correct the other presenting complications of CP (e.g., obstructive jaundice, duodenal stenosis, and large pseudocysts) by simultaneous interventions. All these arguments emphasize the special value of the described procedure as a universal method of treating patients similar to those described in this paper (ie, those with severe complications of CP against a background of its exacerbation, exhaustion, and a severely or critically ill general condition).

Our first OISPD procedures were performed under such circumstances, when endoscopic (ERCP-based) and endoscopic ultrasound-guided interventions were impossible due to technical and organizational reasons. Subsequently, after being impressed by the good immediate results, we began to use this procedure as the method of choice in complicated CP, and also in the described case.

The most serious drawback of OISPD is its invasiveness, as it requires a laparotomic approach. However, OISPD is much

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less time-consuming than pancreaticojejunostomy and especially Frey and Beger procedures. The second disadvantage of this surgery is the need for regular endoscopic replacement of stents. Nevertheless, the need for periodic visits to the doctor and repeated endoscopic interventions disciplines patients and encourages them to follow the necessary diet and proper lifestyle.

In the future, the technical details of the procedure can be improved. When creating a tunnel in the pancreatic tissue, elements of coagulation can be used to reduce the mechanical force at this stage of the operation. Such a technique will, in some cases, make it possible to completely avoid duodenotomy in favor of endoscopic assistance while placing the stent into the formed tunnel.

Conclusions

The novel OISPD procedure can be effectively and safely used in chronic frequently relapsing pancreatitis with PDH, even in seriously or critically ill patients.

Conflict of interest

None.

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