

Ruptured Pyogenic Liver Abscess with Pneumoperitoneum 19 Years After Pancreatoduodenectomy

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

ABCDEF 1 **Yuri N. Shiryajev**
ABCD 1 **Andrey G. Ryllo**
ABCD 1 **Sergey N. Grechukhin**
BCDF 2 **Maria O. Karpenko**
BCDF 2 **Anna I. Busheva**
BCD 1 **Elena N. Koronnova**
BCD 3,4 **Anna V. Glebova**
ABCDE 3 **Nikolay Y. Kokhanenko**

1 Fourth Department of Surgery, Saint Petersburg City Hospital #15, Saint Petersburg, Russian Federation
2 Department of Hospital Surgery #2, I.P. Pavlov First Saint Petersburg State Medical University, Saint Petersburg, Russian Federation
3 Department of Faculty Surgery named after Professor A.A. Rusanov, Saint Petersburg State Pediatric Medical University, Saint Petersburg, Russian Federation
4 Sixth Department of Surgery, Mariinsky Hospital, Saint Petersburg, Russian Federation

Corresponding Author: Yuri N. Shiryajev, e-mail: shiryajev@yandex.ru
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Patient: Male, 42
Final Diagnosis: Ruptured liver abscess • local peritonitis
Symptoms: Severe abdominal pain • fever with a body temperature of 39°C • jaundice and severe weakness
Medication: —
Clinical Procedure: Laparotomy, drainage of the liver abscess
Specialty: Surgery

Objective: Rare co-existence of disease or pathology
Background: Rupture of a pyogenic liver abscess is rare but serious complication. In patients after pancreatoduodenectomy, there are some conditions causing the development of liver abscesses (e.g., chronic reflux-cholangitis, efferent jejunal loop stasis, stenosis of the biliary anastomosis, and pancreatogenic diabetes). However, the number of published cases of liver abscess after pancreatoduodenectomy is small.

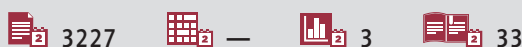
Case Report: A 42-year-old male was admitted with severe abdominal pain, fever, and jaundice. Nineteen years previously, he had undergone pancreatoduodenectomy and cholecystectomy for chronic pancreatitis with obstructive jaundice. Two years later, diabetes mellitus was diagnosed, with subsequent insulin treatment. At admission, symptoms of peritonitis were present. Plain abdominal radiography showed free gas under the right hemidiaphragm and heterogeneous liver shade with small gas-fluid levels. The rupture of a liver abscess was suspected. Laparotomy with adhesiolysis, debridement of the liver abscess cavity, and abdominal drainage were performed. The postoperative period was complicated by sepsis, right lower lobe pneumonia, and two-sided pleural effusions, on the background of insulin-dependent diabetes and malnutrition. The patient was discharged on the 40th day and the subdiaphragmatic drains were removed on the 114th day. Sixteen months after surgery, the patient's condition was satisfactory. Magnetic resonance imaging and echography showed the absence of biliary hypertension. The liver tissue had healed completely.

Conclusions: A unique case of ruptured liver abscess after pancreatoduodenectomy is presented. To the best of our knowledge, this is the first published case with such a long time interval (19 years) between pancreatoduodenectomy and the formation of a pyogenic liver abscess.

MeSH Keywords: Diabetes Complications • Liver Abscess, Pyogenic • Pancreaticoduodenectomy • Pancreatitis, Chronic

Abbreviations: PLA – pyogenic liver abscess; PD – pancreatoduodenectomy; DM – diabetes mellitus; CP – chronic pancreatitis; CT – computed tomography

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Background

Rupture of a pyogenic liver abscess (PLA) is a serious complication with a negative impact on the patient's life [1]. The rupture of a gas-containing PLA is usually accompanied by pneumoperitoneum and can mimic hollow viscous perforation [2,3]. According to the etiology, cholangiogenic, pylephlebotic, post-traumatic, postoperative, cryptogenic, and other PLAs have been described [4–6], and any type of abscess can be complicated by rupture [1].

Pancreatoduodenectomy (PD) is one of the most technically demanding and traumatic abdominal surgeries and is accompanied by not only high morbidity [7], but also with the formation of a number of late pathological conditions, some of which (e.g., chronic reflux-cholangitis, efferent jejunal loop stasis, and stenosis of the biliary anastomosis) are responsible for the occurrence of PLA [5]. Moreover, pancreatogenic diabetes mellitus (DM) and malnutrition, which frequently develop after PD, can significantly reduce the immune defense against bacterial infection and also contribute to the formation of PLA. Finally, PLA can be the outcome of secondary infection and necrosis of the metastatic node in the liver if PD was performed for a pancreatic or periampullary malignant tumor [8].

However, the number of published cases of PLA after PD is small. Up to 2016, Virgilio et al. [9] found only 35 cases fulfilling the definition of merely post-surgical PLA [7,10–11]. We add to them a number of cases – both in earlier [5,8,12–17] and later [18,19] publications. There are only two papers analyzing the problem of post-PD liver abscesses on large clinical material [10,19]. A rather unexpected result of these two investigations is that the incidence of PLA in patients who have undergone a PD (2.6–3.4%) is much lower than could be predicted by taking into account only general considerations [10,19]. Moreover, in the vast majority of such patients, PLA does not develop in the early postoperative period, but after discharge – weeks and months after PD [W. Chen, 2019, personal communication]. In some papers, the occurrence of PLA in very late periods – 3 years [17], 3.5 years [12], 9 years [11], and 10 years [18] after PD was described. Such cases are of considerable interest from the point of view of etiological features and the optimal treatment of the developed PLA.

Here, we present our own observation of a ruptured gas-containing PLA in a patient who had undergone PD 19 years previously.

Case Report

A 42-year-old Russian male patient was urgently admitted on November 11, 2017, with complaints of severe abdominal pain,

fever with a body temperature of 39°C, jaundice, and severe weakness. He considered himself to have been sick for at least 3 weeks, and had started to notice a rise in body temperature to subfebrile ranges. Then, there was pain in the right hypochondrium, radiating to the lower back, along with chills. The patient's temperature rose to 38.5°C. He had gone to the doctor 10 days before hospitalization; despite the absence of instrumental confirmation, acute pyelonephritis was diagnosed, and antibiotic therapy was prescribed (oral ciprofloxacin). He also took analgesics (ketoprofen, nimesulide). However, the treatment had no effect. On the day of admission, the abdominal pain sharply increased and spread to the entire right half of the abdomen; at the same time, the patient noticed yellowness of the skin and sclera, as well as darkening of the urine.

From the anamnesis: in 1998, the patient had developed severe jaundice. In the infectious diseases hospital, the diagnosis of viral hepatitis was excluded. A cholecystostomy was performed, and the patient was transferred to the university surgical clinic. On the basis of cholangiography and echography, a tumor of the pancreatic head was suspected. The patient was operated upon on April 4, 1998, by Professor Nina N. Artemyeva. During the surgical revision and a study of biopsy specimens of the pancreatic head tissue, it was impossible to exclude the diagnosis of pancreatic cancer. Therefore, PD with cholecystectomy was performed. Due to the severe inflammation of the pancreas and a significant risk of pancreatic anastomosis leakage, only external drainage of the pancreatic remnant duct was performed. In the postoperative period, pancreatic drainage spontaneously migrated, but no further complications occurred. The formation of a spontaneous internal fistula between the Wirsung duct and jejunum was suspected. The patient was discharged on May 8, 1998 in a satisfactory condition. Histological examination of the specimen confirmed the diagnosis of chronic pancreatitis (CP). The final diagnosis was described as follows: "CP with pancreatic head fibrosis and multiple small pancreatic abscesses and compression of the common bile duct. Obstructive jaundice".

Was the performance of PD in this patient unavoidable? It is very difficult to discuss the features of the diagnosis of pancreatic cancer at a time when computed tomography (CT) already existed in our city, but the huge queues for the investigation made it impossible to use it in this particular case. Therefore, specifying diagnostics was limited to echography and cholangiography via cholecystostomy tube with addition of intraoperative biopsy. And last but not least: even now, when cross-sectional examinations are available in many countries of the world, the PD in CP is periodically performed in many clinics including highly specialized centers, since before the operation and intraoperatively, it is impossible to differentiate pancreatitis and cancer in some cases.

After discharge, the patient felt relatively good, but approximately 2 years after surgery, he was diagnosed with DM, and insulin therapy was prescribed. At the time of the current hospitalization, he worked as a driver. He smoked for about 20 years but consumed alcohol very rarely, trying to follow the medical recommendations.

On examination: the general condition was severe; the patient was ill, and adynamic. Moderate yellowness of the skin and sclera was present. The skin was hot, and the body temperature was 38.9°C. The heart rate was 100 beats per 1 minute, and the blood pressure was 110/70 mm Hg. The abdomen was painful and moderately tender in the upper part and right half. In the same areas, the rebound tenderness was clearly present. Blood test results were as follows: hemoglobin 113 g/l, erythrocytes $4.0 \times 10^{12}/l$, leukocytes $13.9 \times 10^9/l$, neutrophils 90%, platelets $279 \times 10^9/l$, ALT 810 U/l, AST greater than 913 U/l (cannot be measured precisely), amylase 5 U/l, total bilirubin 277 $\mu\text{mol}/l$, glucose 28.0 mmol/l, creatinine 49 $\mu\text{mol}/l$, prothrombin index 65%, and INR 1.28. On a plain abdominal radiogram, free gas under the right hemidiaphragm, heterogeneity of the liver shadow with small gas-fluid levels in its tissue, and signs of bowel paresis were present (Figure 1). Ultrasonography revealed the dissimilarity of liver tissue due to multiple hyperechoic lesions, thick fluid and gas in the right subdiaphragmatic space, signs of portal hypertension (portal vein diameter 14 mm), and CP (tissue heterogeneity) in the remnant of the pancreas. No free fluid was found in other parts of the abdominal cavity. Emergency CT, which is standard for the diagnosis of PLA, could not be performed for technical reasons.

Eventually, rupture of the liver abscess complicated by peritonitis was suspected. However, it was impossible to completely exclude the perforation of the ulcer of the gastric stump or gastrojejunostomy, and emergency surgery was scheduled.

Midline laparotomy. A small amount of brownish muddy fluid in the right paracolic gutter and iliac fossa. The organs of the upper abdomen are fixed with each other forming a conglomerate. When the right lobe of the liver was separated from the parietal peritoneum, purulent-hemorrhagic fluid began to flow under pressure, resembling in color and consistency the “hot chocolate” drink, with mixed caliber fragments of liver tissue debris. After complete separation of the right liver lobe, about 1 l of exudate was totally evacuated. In the tissue of the right lobe of the liver (VII-VIII segments), there was an abscess cavity of $12 \times 10 \times 8$ cm. Intraoperative endoscopy showed no ulcer in the gastric stump and anastomosed jejunal loop. The efferent jejunal loop from the gastroenterostomy to the liver hilus was freed from adhesions. The abscess cavity and abdominal cavity were flushed with saline and drained.

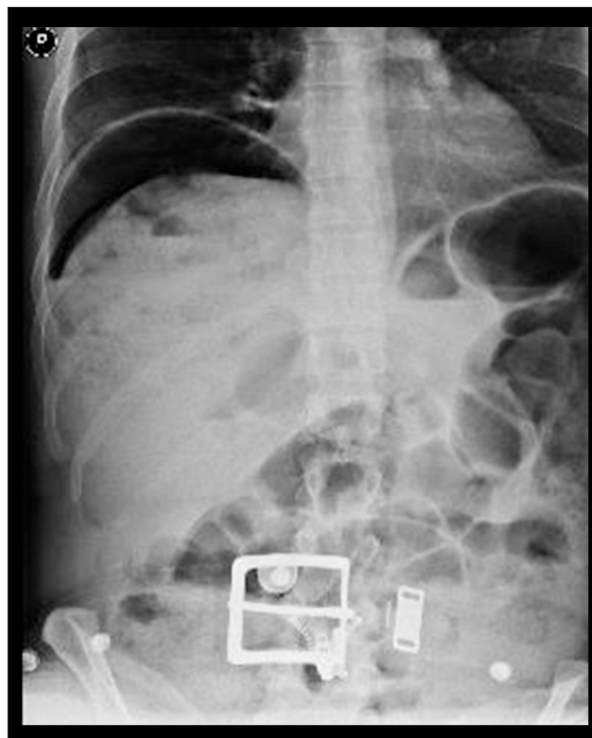


Figure 1. Plain frontal radiogram of the abdomen at admission. Free gas under the right hemidiaphragm, heterogeneity of the liver shadow with small gas-fluid levels in its tissue, and signs of bowel paresis were present.

The postoperative period was not uneventful. During the first 1.5 days, prolonged mechanical ventilation and inotropic support with epinephrine were necessary. Subsequently, the patient was diagnosed with sepsis – the concentration of blood procalcitonin was 5.1 ng/ml, *Staphylococcus haemolyticus* was grown in the blood, and *Klebsiella pneumoniae* and *Escherichia coli* in the urine cultures. *E. coli* and *S. haemolyticus* were detected in the pus from the PLA, and later *Klebsiella* and *Acinetobacter* were grown repeatedly from the drained peritoneal exudate. The patient received vancomycin and meropenem and later vancomycin and amikacin in standard doses for a long time. The postoperative course was also complicated by right-sided pneumonia and bilateral pleural effusions. In this regard, both pleural cavities were subsequently drained on the 10th and 11th days. On the 14th day, the patient was transferred from the intensive care unit to the surgical ward.

Then, gradual improvement in the patient’s condition followed. Echography and CT revealed a gradual reduction of the liver lesion (Figure 2). The patient was discharged on the 40th day after surgery with two subphrenic drains. The draining fluid was initially serous-purulent with little bile staining, then serous. The drains were removed during an outpatient visit on the 114th day post-surgically.



Figure 2. CT scan of the abdomen (delay phase) on 20th day after PLA surgical drainage. In the right lobe of the liver, residual lesion is visualized (arrows). Drainage tubes are also seen (arrowheads).

Sixteen months after surgery, the patient's condition is satisfactory. He works occasionally as a driver at piecework. He constantly receives insulin and enzyme replacement therapy. At follow-up examination in March 2019, according to the echography, the liver tissue had healed completely, with no signs of biliary hypertension. Magnetic resonance imaging showed non-dilated bile ducts and a very small (22×36 mm), almost completely atrophic pancreatic remnant with non-dilated Wirsung duct (Figure 3). Excluding mild hyperglycemia and low serum amylase, the other parameters of blood tests, biochemistry and coagulograms are in the normal ranges.

Discussion

PLA, complicated by rupture with the development of pneumoperitoneum, was first described quite recently – in 1982 [20]. With the accumulation of clinical material, it became evident that this complication dramatically increases mortality compared with uncomplicated AP [1], and that it is often necessary to differentiate PLA rupture from perforation of the gastrointestinal hollow organs [1–3,21].

In the 1990s, some publications analyzing the risk of developing PLA after PD to varying degrees were published [7,12,13]. In 2014, the first paper on PLA after PD, based on a very large volume of clinical material, was presented. Here, 839 patients who underwent PD at the University of Indiana Hospital (USA) were analyzed. The incidence of PLA was 2.6%. The authors considered postoperative biliary fistula and the need for reoperation in the early postoperative period to be the main risk factors for PLA [10]. A similar design study was published in 2019 by Chinese authors [19]. Their study group was smaller (n=326), and the incidence of AP was greater (3.4%) than in the previous study. The paper focuses on the fact that DM

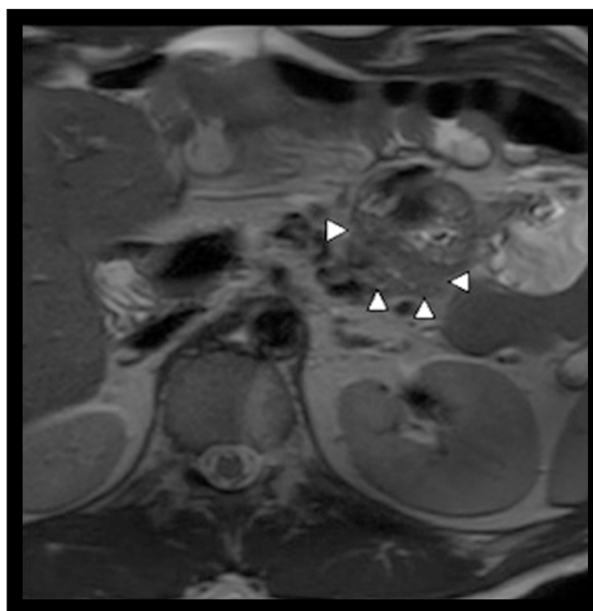


Figure 3. Magnetic resonance imaging 16 months after surgery for PLA. The pancreas remnant (arrowheads) is very small (22×36 mm), with non-dilated Wirsung duct.

(including impaired fasting plasma glucose) is a significant risk factor for the development of PLA after PD.

The risk factors for the formation of PLA after PD are substantially greater in number. In the recent review on PLA, the French authors distinguish PLA after the PD in a special subgroup and described them in three paragraphs [5]. Etiopathogenetic factors are slightly different for PLA, developing in the **early** postoperative period of the PD and in the long-term. For “early” abscesses, the initial presence of cholangitis, caused both by the biliary obstruction itself and by procedures aimed at its resolution (endoscopic transpapillary interventions and percutaneous drainage/stenting), plays an important role. A certain immunodeficiency due to preoperative chemotherapy may be considered. Postoperative septic complications (partial necrosis of the pancreatic remnant, intra-abdominal abscesses, and anastomotic leaks) also increase the risk of developing liver abscesses. Specific to PD are situations of accidental (unintentional) or deliberate ligation of the branches of the hepatic artery (e.g., for stopping bleeding). In such cases, the risk of developing an infarction of part of the liver (sometimes a whole lobe) with the outcome of PLA is very high. A rare cause of PLA after PD is liver ischemia following stenosis of the celiac trunk that was not recognized in the perioperative period. Finally, a similar mechanism of liver ischemia (an inflow block via an artery of large caliber) with an outcome in the PLA occurs during embolization of the hepatic artery or gastroduodenal artery stump for stopping the erosive bleeding in the early postoperative period [5,16].

Chronic reflux-cholangitis, accompanied by chronic contamination of the bile ducts with intestinal flora, plays an important role in the etiopathogenesis of PLA, which develops in the **long-term period** of PD. It can develop both in cases of stenosis of the biliary anastomosis, and during its normal patency [5]. Biliary hypertension after PD may be a consequence of hypertension in the efferent jejunal loop. Its causes are different – technical errors of the reconstructive phase of the operation (too short jejunal loop), adhesions with the formation of intestinal twists and kinks, stenosis of the jejuno-jejunal anastomosis, and compression of the bowel by metastatic nodes (most often in carcinomatosis). This situation is much more difficult in terms of providing adequate care to the patient. If the biliary anastomosis stricture can be successfully treated by transhepatic minimally invasive intervention (balloon dilatation and/or stenting), and in recent years with double balloon enteroscopy, the efferent loop syndrome is always a relative or absolute indication for reconstructive surgery. Two clinical cases of this kind were presented by Japanese authors. In one case, it was necessary to convert the reconstructive scheme from the Imanagi to the Child method 13 years after the PD [18] with a good immediate result. In the second patient, recurrent reflux-cholangitis began to appear soon after PD. Nine years after the PD the PLA occurred and was drained. The symptoms of cholangitis progressed further, and bile duct stones were identified. Their removal using the Dormia basket, carried out during double balloon enteroscopy, was successful. The diagnosis of efferent loop syndrome has been proven with hepatobiliary scintigraphy (long-term retention of the radiopharmaceutical media in the efferent loop) [11]. The patient is under dynamic observation (which is caused, apparently, by the elderly patient's age and the high risk of reconstructive surgery, as well as a good effect of lithoextraction, followed-up over 2 years).

The next important risk factor for the formation of PLA after PD is DM. It is well known that DM develops in a number of post-PD patients, and that in some people who have diabetes before the operation, its course worsens [7], which requires an increase in the dosage of insulin or peroral anti-diabetic drugs. However, apparently, in our case, pancreatogenic diabetes was not so much a result of the operation itself, but rather a result of the progression of CP with the loss of not only acinar, but also islet pancreatic cells. In the overall group of PLA patients, the proportion of people with DM is very high and amounts to 36.6–48.3% [22–24], reaching in specific variants of PLA such as gas-forming and ruptured, 83.5–85.5% [25,26] and 60.9% [1], respectively. A specific biochemical mechanism has been described to explain the formation of gas in the PLA cavity during hyperglycemia [23,27]: under conditions of ischemia (tissue hypoperfusion), the tissue pH decreases, and glucose metabolism switches to another pathway – microorganisms secrete an enzyme, hydrogenlyase, which catalyzes the catabolism of formic acid (intermediate product of

metabolism) to carbon dioxide and hydrogen. Excessive plasma glucose is associated with the rapid and massive appearance of gas in the PLA cavity.

Finally, in some cases, PLA can develop into liver metastasis. A metastatic node, with necrosis and the addition of a secondary infection, can spontaneously evolve into PLA [28,29]. Still, the formation of PLA after therapeutic procedures for metastases is more usual – most often in this aspect, transarterial chemoembolization of the branches of the hepatic artery and radiofrequency ablation of metastatic nodes are reported [5,8]. In clinics specializing in surgical and interventional oncology, hepatobiliary surgery, the proportion of such observations can be quite high. Thus, in the material of Mezhir et al. [8], in a group of 58 patients with PLA, 51 (88%) had a proven oncological diagnosis, 14 underwent PD, and a liver abscess developed in 13 (22%) after embolization of the hepatic artery or radiofrequency ablation.

In our case, it is difficult to highlight a single etiological factor that led to the development of PLA. The patient did not have stenosis of the biliary anastomosis and biliary hypertension, as proven by the results of both repeated echographies in the perioperative period and MRCP 16 months after surgery. The efferent jejunal loop was not short, which was defined intraoperatively. However, intra-intestinal hypertension could well have occurred, since the efferent loop was deformed and represented by areas with twists and kinks, forming a hard fibrous conglomerate. In our opinion, the most significant etiological factor of PLA in our patient can be considered to be the prolonged hyperglycemia due to the loss of glycemic control and, in particular, the insulin administration regimen. The incorrect management of the patient at the pre-hospital stage, when without proper justification and in the absence of at least an echographic examination, the diagnosis of pyelonephritis was established, led to a serious complication – rupture of the PLA with the subsequent development of sepsis. If the diagnosis of PLA was made before its rupture, it could have been drained in a less-invasive fashion. Then, the post-operative course would have been incomparably easier, and the risk of complications would have been significantly lower. Due to the presence of pneumoperitoneum and the clinical picture of peritonitis, when before the operation it was impossible to completely exclude the perforation of the hollow organ, the indications for laparotomy were indisputable.

Rupture of PLA dramatically worsens the prognosis for the patient. Thus, in a group of 62 patients with gas-containing PLA, no patients with pneumoperitoneum (n=5) survived [30]. In the classical work of Taiwanese authors, it was reported that mortality in the ruptured PLA was 43.5%, whereas in the absence of rupture it was 15.5% [1]. In recent publications, lower mortality rates for PLA rupture are given, of up to 4.3% [31]; however, this complication remains very dangerous to date.

Over the past 20 years, the approach to the treatment of ruptured PLA has changed. A quarter of a century ago it was believed that surgery was the only possible treatment for this complication [1]. With the development of new imaging techniques and interventional technologies, it was found that, in the absence of diffuse peritonitis, percutaneous drainage of localized purulent collections forming subdiaphragmatic or perihepatic/subhepatic abscesses can be performed successfully [31], in the same way as laparoscopic drainage. However, in cases similar to the one described here, laparotomy with debridement and drainage of the abscess cavity (and sometimes liver resection [32,33]) remains an indispensable option of surgical treatment.

Conclusions

The authors presented an exclusively rare case of ruptured PLA in a patient with prior PD, with the development of

pneumoperitoneum. To the best of our knowledge, this is the first published case with such a long time interval (19 years) between PD and the formation of PLA.

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Conflict of interests

None.

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