**Clinical cases of fatty liver disease after pancreatoduodenectomy and total pancreatectomy.**

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**Background:**

Pancreatoduodenectomy (PD) is the only possible treatment for patients with tumors of the pancreatic head and periampullary zone. Because of the malignant potential of these tumors, overall survival remains generally unsatisfactory even after PD, although some long-term survivors have been described [1,2]. It is known that some long-term surviving patients develop exocrine pancreatic insufficiency (EPI) [3-5]. One of the consequences of EPI is fatty liver disease (FLD), which has been reported to occur in 7.8% - 40.0% of patients after PD [6-14].

Possible risk factors for the development of FLD include pancreatic fistula [9], lack of insulin use [11], postoperative loss of body mass index (BMI) [11], volume of surgical blood loss [12], low remnant volume of the pancreas [7,14], postoperative diarrhea [7], infection [14], female gender [8], cancer of pancreatic head [7.10], and postoperative EPI [13], but none of these factors has been found to be fully responsible for the occurrence of FLD. In addition, no methods have been developed for the prevention and treatment of FLD after PD.

Tomimaru et. al. reported that reduced remnant pancreatic volume after PD influences the development of FLD [15].

Some studies suggest that malnutrition or malabsorption of essential nutrients caused by EPI leads to FLD [16–21]. Changes in metabolism resulting in hepatic steatosis could lead to sensitivity for hepatocyte damage, inflammation, and fibrosis [20,22]. Pancreatic enzyme administration as treatment of EPI has beneficial impact on FLD after PD, indicating that EPI could be the main cause of new onset FLD in these patients [16,21]. Other evidence for malnutrition in patients with EPI or after PD is the report of increase of taurine serum levels and decrease of methionine, tyrosine, albumin, cholinesterase, zinc, and total cholesterol serum levels [16,17,18,20,22–25]. Therefore, other nutrients or mechanism that not yet have been identified could cause the hepatic steatosis. Besides little evidence indicating that FLD could be treated with pancreatic enzyme administration, adequate treatment is not yet recognized.

The pancreas plays a central role in the absorption of essential nutrients. Its dysfunction causes various abnormalities in the body, notably in metabolic cascades in the liver. It has been previously documented that severe hepatic steatosis sometimes occurs in pancreatectomized patients [26,27]. However, the clinicopathological characteristics of this type of FLD have not been fully investigated.

FLD is characterized by two steps of liver injury: intrahepatic lipid accumulation (hepatic steatosis) and inflammatory progression to FLD (i.e., the two-hit theory). [28]. The first hit is fat accumulation in hepatocytes in the setting of obesity, type II diabetes, and hyperlipidemia, leading to development of hepatic steatosis. A second hit, including oxidative stress, inflammatory cytokines, and endotoxins, is considered to activate inflammatory cells, leading to progression of simple hepatic steatosis to FLD. This theory is well known as pathogenesis of FLD associated with obesity and metabolic syndrome.

The gold standard for diagnosis of hepatic steatosis is a liver biopsy [29,30]. Biopsies are performed only however, if the outcome significantly influences the therapeutic options and non-invasive alternatives have not yielded sufficient information. Studies have investigated the use of imaging modalities (US, CT and MRI) as non-invasive means to assess hepatic steatosis. Ultrasound is simple to employ but has limitations in both sensitivity and accuracy [29]. CT and MRI provide higher sensitivity and thus are better alternatives [29,30].

Regarding development of FLD after major pancreatectomy, there have been few papers worldwide. The previous study by Nomura et al. [31] reported in 2007 that 14 (33%) of the 42 patients who underwent PD developed hepatic steatosis (liver-to-spleen CT attenuation ratio less than 0.9), which is currently known as FLD. Kato et al. reported in 2010 [32], that 54 patients underwent PD in our department, and 20 (37%) of them developed FLD (hepatic CT value\40 HU) after surgery. The most recent study by Tanaka et al. [33] reported in 2011 that the incidence of FLD after PD (liver-to-spleen CT attenuation ratio less than 0.9) was 23% (14 of 60 patients) and liver biopsy of 8 patients showed FLD. However, FLD is a still little-known complication after PD, and the mechanism of development of FLD after PD remains unclear.

Here we describe 2 cases of rapidly progressive and fatal fatty liver disease that developed shortly after PD and total pancreatectomy without concomitant liver disease.

**Clinical case**

**Case 1.** Patient I., 67 years old, was admitted to our clinic as planned "National Scientific Center of Surgery named after A.N. Syzganov", Department of hepatopancreatobiliary surgery and liver transplantation with a diagnosis of a tumor of the pancreatic head. It is known from the medical history of the disease that the patient has been ill since July 2022, when she began to complain of pain in the epigastrium and right hypochondrium, general weakness, dry mouth, and nausea. After 1 week, the patient's general condition worsened, pain in the epigastric region increased, complaints of jaundice of the skin and sclera, and acholic stools joined.

She was urgently admitted to a clinic for obstructive jaundice in one of the clinics in Almaty. At that time, the total bilirubin level in the analyzes was 269 mmol/l. The patient underwent surgical treatment in the following volume: percutaneous transhepatic biliary drainage by ultrasound control. The dynamics of total bilirubin decreased to 135 mmol/l. After that, the patient was sent to our clinic for examination and radical surgical treatment. Upon admission, the condition was stable. During the examination, a contrast-enhanced CT scan revealed a tumor emanating from the head of the pancreas.

After preparation, laparotomy and standard PD were performed under general anesthesia. Pancreatojejunostomy was performed using a modified Blumgart method using the duct-to-mucosa method. The histopathological diagnosis is a morphological picture of a moderately differentiated ductal adenocarcinoma of the pancreatic head with negative resection margins. On the 2nd day, water intake was started, and 4 days after the operation, a fat-restricted diet was started. Pancreatic fistula was not observed. Large losses of ascites fluid were noted through drainage tubes from the abdominal cavity, and albuminotherapy was performed. After the operation, the biochemical parameters of liver function returned to normal. However, on the 18th day after the operation, biochemical parameters of liver function, white blood cells in the blood began to increase, and coagulopathy was observed. The patient's general condition worsened, she was prone to hypotension (arterial pressure – 60/40 mmHg), and hemodynamics was unstable. After that, the patient was transferred and received treatment in the department of anesthesiology and intensive care.

On the 19th day after surgery, the patient underwent contrast-enhanced computed tomography, where there was a diffuse decrease in liver parenchyma density, the presence of ascites and signs of FLD. The patient's general condition gradually worsened. The subsequent clinical course of liver dysfunction worsened, and the patient developed severe liver failure with increasing hyperbilirubinemia. She died on the 22nd day after surgery by FLD. Histological examination revealed pronounced fatty tissue with microdrops and large droplets; signs of fibrosis, balloon degeneration of hepatocytes, fatty deposits indicating hepatitis or FLD.

**Case 2.** Patient N., 62 years old, was hospitalized at the National Scientific Center of Surgery named after A.N. Syzganov in the Department of General Surgery for the diagnosis of IPMN of main duct of pancreas. The patient also has type 2 diabetes mellitus. The disease began in September 2021, when pain appeared in the epigastrium and right hypochondrium, general weakness and itching of the skin. Since October 2021, symptoms of jaundice and a 15 kg decrease in body weight have appeared. In October 2021, the patient was treated in a hospital, where the total bilirubin level was 223 mmol/l, and the level of the cancer marker CA 19-9 was 199 U/ml. She underwent percutaneous transhepatic biliary drainage by ultrasound control, after which the bilirubin level decreased to 78 mmol/l.

According to the results of contrast-enhanced computed tomography, the diagnosis of IPMN of the main duct of the pancreas was established, with an increase in the size of the tumor compared to the previous examination. After preparation, the patient underwent laparotomy and total pancreatoduodenectomy. Histological examination revealed invasive intraductal mucinous papillary carcinoma of the pancreas. After the operation, the patient recovered without complications and was discharged after 14 days with an improvement in her condition.

In March 2022, the patient developed symptoms: abdominal enlargement, swelling on her legs, weakness, vomiting and diarrhea. The patient was re-admitted to the clinic with a diagnosis of malabsorption syndrome and protein-energy deficiency, as well as signs of ascites. Blood tests revealed hypoalbuminemia and marked electrolyte abnormalities. The patient underwent paracentesis and was prescribed albuminotherapy.

On the 48th day after surgery, the tests showed signs of severe coagulopathy and the level of total and direct bilirubin in the blood began to rise. The patient's general condition worsened. After that, the patient was transferred and received treatment in the department of anesthesiology and intensive care.

Contrast-enhanced computed tomography of the abdominal cavity revealed liver atrophy, accompanied by marked fat accumulation and massive ascites (Fig. 3). Subsequently, the patient's clinical condition continued to worsened, and severe liver failure with progressive hyperbilirubinemia developed. She died on the 69th day after surgery by FLD.

**Discussion**

These clinical cases represent a significant problem in the treatment of patients who have undergone pancreatic surgery, in particular, associated with the development of life-threatening FLD, which can develop in just a short time after surgery. One of the characteristic features of this case was a significant loss of body weight due to malnutrition caused by surgery.

The most common patients with FLD have a number of risk factors, such as obesity, type 2 diabetes mellitus, dyslipidemia, and hypertension, which makes FLD a manifestation of metabolic syndrome [34-36]. However, in recent years, it has been noted that pancreatic surgery may also play a role in the development of FLD [37-42]. According to the latest data, the prevalence of this type of liver disease ranges from 23 to 37% among such patients [37,39,40]. Tanaka and his colleagues reported that the main risk factor for developing FLD in such patients is cancer of the pancreatic head [40]. At the same time, Kato et. al. identified five risk factors that may contribute to the development of FLD after pancreatic surgery: pancreatic adenocarcinoma, cancer in the resection area, pancreatic consistency, postoperative diarrhea, and eating disorders [39].

The exact mechanisms leading to the development of FLD after PD have not yet been fully studied. Unlike the classical form of the disease, this version is not accompanied by obesity, hyperlipidemia or insulin resistance [40]. EPI caused by its reduction in size after surgery, as well as dietary disorders leading to impaired absorption of essential amino acids, fats, and fat-soluble vitamins (for example, choline), play a key role in the pathogenesis. This is due to impaired synthesis of low-density lipoproteins in the liver and deficiency of methionine and choline [38,40,43]. The use of high doses of pancreatic enzyme replacement therapy in patients with FLD after PD has shown a positive effect, which supports the proposed hypothesis about the pathogenesis of the disease [39-41].

One possible mechanism is that a lack of nutrition caused by a decrease in the exocrine function of the pancreas may contribute to the conversion of carbohydrates into fats in the liver. The key factors are neurogenic diarrhea associated with dissection of the nerve plexus around the superior mesenteric artery, impaired fat absorption and steatorrhea caused by a decrease in the exocrine activity of the pancreas. It is believed that this impaired fat absorption contributes to an increase in the conversion of carbohydrates into fats in the liver [44].

Another possible mechanism suggests that endotoxins may cause liver damage. A decrease in the intestinal exocrine function can lead to the movement of bacteria due to atrophy of the intestinal mucosa. This, in turn, can promote the entry of endotoxins into the liver through the portal vein, which activates Kupffer cells and causes fatty deposits in the liver [45]. It has also been hypothesized that this mechanism may be related to zinc deficiency. In cases after extensive resection of the pancreas, there is a significant decrease in the level of zinc in the blood and its content in the pancreatic tissues. Zinc is absorbed mainly in the duodenum and proximal jejunum with the help of zinc-binding proteins contained in pancreatic juice, and plays an important role in the regeneration and maintenance of the epithelial structure of the intestinal mucosa. In addition to restoring part of the gastrointestinal tract after PD, the amount of zinc ingested from food also decreases due to a decrease in overall exocrine function. In addition, the hypofunction of insulin after PD increases the excretion of zinc in the urine, which leads to a deficiency of this trace element. It is believed that this reduces the protective role of zinc on the intestinal mucosa, which increases intestinal permeability and promotes the penetration of endotoxins through the mucous membrane [46].

In these clinical cases, we did not measure the serum zinc levels in these patients, as they began receiving parenteral nutrition immediately after surgery, which contained zinc and magnesium. Fatty liver hepatosis, which has developed against the background of protein and energy deficiency with severe liver failure, can in some cases lead to death.

Future recommendations should focus on the study of specific methods of perioperative nutrition, including caloric calculation and the use of dietary supplements, taking into account the dynamics of weight changes and progress or improvement in liver steatosis. Our clinical cases indicate that following dietary recommendations in the postoperative period, together with insulin therapy, can play an important role in preventing a decrease in BMI in patients undergoing pancreatectomy.

**Conclusion**

This clinical case demonstrates that after PD and total pancreatectomy, a life-threatening FLD with severe fibrosis can occur. When treating patients who have undergone pancreatic head resection or total pancreatectomy, doctors should consider the likelihood of developing fatty liver, as well as the importance of assessing nutritional status. It is necessary to use preventive drugs with pancreatic enzymes and supplements.

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