Perioperative Computed Tomography Assessments of the Pancreas Predict Nonalcoholic Fatty Liver Disease After Pancreaticoduodenectomy

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Abstract: Nonalcoholic fatty liver disease (NAFLD) after pancreaticoduodenectomy (PD) has become a clinically important issue. Although pancreatic exocrine insufficiency has been reported to be a main cause of NAFLD after PD, a clinically practical examination to assess the pancreatic exocrine function has not been established. The aim of this study was to evaluate risk factors for NAFLD after PD with a focus on perioperative computed tomography (CT) assessments of the pancreas.

A retrospective review of 245 patients followed for more than 6 months after PD was conducted. We evaluated several pancreatic CT parameters, including the pancreatic parenchymal thickness, pancreatic duct-to-parenchymal ratio, pancreatic attenuation, and remnant pancreatic volume (RPV) on pre- and/or postoperative CT around 6 months after surgery. The variables, including the pancreatic CT parameters, were compared between the groups with and without NAFLD after PD.

The incidence of NAFLD after PD was 19.2%. A multivariate analysis identified 5 independent risk factors for NAFLD after PD: a female gender (odds ratio [OR] 5.66, P < 0.001), RPV < 12 mL (OR 4.73, P = 0.001), preoperative pancreatic attenuation of <30 Hounsfield units (OR 4.50, P = 0.002), dissection of the right-sided nerve plexus around the superior mesenteric artery (OR 3.02, P = 0.017) and a preoperative serum carbohydrate antigen 19–9 level of ≥70 U/mL (OR 2.58, P = 0.021).

Our results showed that 2 pancreatic CT parameters, the degree of preoperative pancreatic attenuation and RPV, significantly influence the development of NAFLD after PD. Perioperative CT assessments of the pancreas may be helpful for predicting NAFLD after PD.

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INTRODUCTION

Pancreaticoduodenectomy (PD) has been established as a standard surgical procedure to achieve curative resection for peripancreatic tumors. Recent advancements in preoperative diagnostic imaging, surgical techniques, and postoperative management have improved long-term survival of patients treated with PD.1,2 Accordingly, metabolic and nutritional consequences of an impaired pancreatic endocrine and exocrine function have become important problems in the late postoperative period after PD.3-4

Nonalcoholic fatty liver disease (NAFLD) is 1 of the late complications of PD, developing in 8% to 37% of patients treated with PD.5-10 Although the precise mechanism responsible for the development of NAFLD after PD remains unclear, some studies suggested that postoperative malnutrition caused by pancreatic exocrine insufficiency was significantly associated with NAFLD after PD.7,8,11 However, a clinically practical examination to assess the pancreatic exocrine function has not yet been established, and routine evaluations of the pancreatic exocrine function after PD are not commonly performed.

Several previous studies showed that the morphological features of the pancreas on computed tomography (CT), such as duct-to-parenchymal ratio and pancreatic parenchymal thickness, correlated with the pancreatic exocrine function after PD.12-14 and we therefore hypothesized that CT analyses of the pancreas before and after PD might be useful for predicting the development of NAFLD. The aim of this study was thus to evaluate risk factors for NAFLD after PD, with a special focus on perioperative CT assessments of the pancreas.

PATIENTS AND METHODS

Patients

A retrospective review using a prospectively collected database including 307 consecutive patients who underwent PD between July 2009 and October 2013 at Shizuoka Cancer Center was conducted. A total of 62 patients with following conditions were excluded from this study: fatty liver prior to PD (1 patient); hepatic viral infection (10 patients); steroid therapy (2 patients); death or hospital transfer within 6 months after PD (23 patients); unavailability of postoperative CT images between 3 and 12 months after PD (26 patients), and consequently 245 patients were enrolled in the present study. No
patient regularly consumed more than 150 g per week of alcohol after PD.5,6 This study was approved by the institutional review board of Shizuoka Cancer Center.

Surgical Procedures
The surgical procedure was subtotal stomach preserving PD with the modified Child method in all patients. Pancreaticojejunostomy consisted of approximation of the pancreas stump and jejunal wall, followed by duct to mucosa anastomosis. All pancreaticojejunal anastomoses were stented using 4- to 7.5-Fr polyvinyl chloride tubes (Sumitomo Bakelite, Tokyo, Japan) according to diameter of the main pancreatic duct (MPD), which were guided externally through the jejunal loops. The indication for dissection of the right-sided nerve plexus around the superior mesenteric artery (SMA) was determined based on the findings of preoperative diagnostic imaging. All postoperative complications occurring within 30 days after surgery or during the hospital stay were classified according to the criteria proposed by Dindo and Clavien.15 Pancreatic fistula was defined according to the definition of the International Study Group of Pancreatic Fistula.16 Adjuvant chemotherapy was performed in 98 (40.0%) patients based on the postoperative histological diagnosis. Pancreatic enzyme supplementation was performed based on the judgment of the surgeon.

Analysis of CT Images
All patients underwent preoperative, multiphasic CT examinations (unenhanced, early arterial, late arterial, portal venous, and equilibrium phases). The raw data set was reconstructed at a thickness of 2 mm. Postoperative CT examinations, in which the assessments were biphasic (unenhanced and equilibrium phases) and reconstructed at a thickness of 5 mm, were performed between 3 and 12 months after PD. The median time interval from PD to the postoperative CT analysis was 181 days (range, 94–361 days). A radiologist (TA) blinded to the clinical course of the patients analyzed the CT images. In order to detect the development of NAFLD, liver and spleen attenuation values were measured on unenhanced CT images and presented in Hounsfield units (HU). Each region of interest (ROI) was a round area of 1.0 cm². We considered the mean value of 4 ROIs at different sectors in the liver as a marker of the degree of the liver attenuation (Figure 1). In order to measure the spleen attenuation, a single ROI was used (Figure 1). NAFLD was defined as a liver-to-spleen attenuation ratio <0.9.17

As for the pancreas, we measured several parameters that may potentially affect the postoperative pancreatic exocrine function. The degree of pancreatic parenchymal attenuation was measured in 3 ROIs at different locations in the body and tail of the pancreas on preoperative unenhanced CT images (Figure 2A). Enhanced images in the late arterial phase were reviewed side by side with unenhanced images in order to identify and exclude the MPD and major vessels (Figure 2B). Each ROI was a round area of 0.5 cm², modified according to the thickness of the pancreatic parenchyma. We considered the mean value of 3 ROIs to indicate the extent of pancreatic attenuation.18 The degree of postoperative pancreatic attenuation was not evaluated because the remnant pancreatic
The diameter of the MPD and thickness of the pancreas (including the MPD) were measured on enhanced CT images. Preoperatively, these values were measured along the line at the left edge of the superior mesenteric vein (SMV) (Figure 3A). Postoperatively, these parameters were measured at the site of the greatest length of the MPD in the remnant pancreas (Figure 3B). If the MPD was not identified, its diameter was estimated to be 1.0 mm (in 1 patient preoperatively and 33 patients postoperatively). The pancreatic parenchymal thickness was calculated by subtracting the diameter of the MPD from the thickness of the pancreas. The ratio of the diameter of MPD to the thickness of the pancreas (pancreatic duct-to-parenchymal ratio) was also calculated.

We measured the remnant pancreatic volume (RPV) on postoperative CT images using CT volumetry. A sequence of transverse CT images acquired in the equilibrium phase was obtained at 5 mm intervals. The pancreatic parenchyma was manually outlined on each slice using a free-hand ROI, and the outlined area was automatically calculated (Figure 4). Major vessels and dilated pancreatic ducts (3 mm or more) were excluded. The product of the pancreatic area and the slice thickness represented the volume of the pancreas on a single slice. The total pancreatic volume was computed by summing all slice volumes.

**Statistical Analysis**

All continuous variables are expressed as the medians with interquartile ranges and were compared using the Mann–Whitney $U$ test. The ability of each continuous variable, including the CT parameters, to predict NAFLD after PD and the best cutoff values were evaluated based on a receiver operating curve.
characteristic curve (ROC) analysis. Comparisons between categorical variables were analyzed using Fisher exact test. A P value <0.05 was considered to be statistically significant. Potentially important variables with a P value of <0.05 according to the univariate analysis were included in a multivariate analysis to identify independent risk factors for NAFLD after PD. A multivariate analysis was performed using the logistic regression method with a backward stepwise selection model. Statistical analyses were performed using JMP version 7 (SAS Institute, Cary, NC).

RESULTS
There were 159 (64.9%) males and 86 (35.1%) females among 245 enrolled patients, with a median age of 69 years (range, 38–88 years). Of these patients, 113 (46.1%) had pancreatic cancer, 47 (19.2%) had bile duct cancer, 23 (9.4%) had ampullary cancer, 21 (8.6%) had intraductal papillary mucinous neoplasm, 16 (6.5%) had duodenal cancer, and the remaining 25 (10.2%) had other diseases. Preoperative biliary drainage was performed in 137 (55.9%) patients with obstructive jaundice, and 4 of the 245 (1.6%) patients received neoadjuvant chemoradiotherapy.

Clinical Features of the NAFLD and Non-NAFLD Groups
The incidence of NAFLD after PD was 19.2% (47 of 245 patients). Table 1 shows a comparison of the clinical features between the patients with NAFLD after PD (NAFLD group) and

| Variables                                | Present, N = 47 | Absent, N = 198 | P     |
|-------------------------------------------|----------------|----------------|-------|
| Preoperative variables                     |                |                |       |
| Age*, y                                    | 69 (62–76)     | 68 (62–74)     | 0.695 |
| Gender, male/female                        | 16/31          | 143/55         | <0.001|
| Body mass index*, kg/m²                    | 22.2 (19.9–24.9)| 21.5 (19.3–23.1)| 0.044 |
| ASA-PS, 1/2/3                              | 4/40/3         | 30/154/14      | 0.474 |
| Hypertension                              | 21 (44.7)      | 81 (40.9)      | 0.637 |
| Diabetes mellitus                         | 16 (34.0)      | 54 (27.3)      | 0.355 |
| Hyperlipidemia                            | 9 (19.1)       | 18 (9.1)       | 0.066 |
| Malignant tumor                           | 45 (95.7)      | 177 (89.4)     | 0.266 |
| Pancreatic cancer                         | 39 (83.0)      | 74 (37.4)      | <0.001|
| Neoadjuvant chemoradiotherapy             | 1 (2.0)        | 3 (1.5)        | 0.575 |
| Preoperative biliary drainage             | 25 (53.2)      | 112 (56.6)     | 0.675 |
| Preoperative laboratory data              |                |                |       |
| Albumin*, g/dL                            | 4.2 (3.8–4.5)  | 4.1 (3.7–4.4)  | 0.293 |
| AST*, U/L                                 | 27 (21–35)     | 26 (20–45)     | 0.996 |
| ALT*, U/L                                 | 27 (20–56)     | 30 (18–72)     | 0.847 |
| Total bilirubin*, mg/dL                   | 0.7 (0.5–1.7)  | 0.8 (0.5–1.9)  | 0.320 |
| Amylase*, U/L                             | 63 (50–97)     | 90 (70–128)    | <0.001|
| Total cholesterol*, mg/dL                 | 200 (164–239)  | 199 (173–234)  | 0.927 |
| Fasting glucose*, mg/dL                   | 119 (104–143)  | 106 (98–125)   | 0.007 |
| CEA*, ng/mL                               | 3.4 (2.1–5.0)  | 2.7 (1.6–4.2)  | 0.061 |
| CA19–9*, ng/mL                            | 127 (21–450)   | 27 (7–109)     | <0.001|
| Operative variables                       |                |                |       |
| Operative time*, min                      | 434 (388–534)  | 409 (352–475)  | 0.012 |
| Blood loss*, mL                           | 888 (631–1227) | 786 (514–1230) | 0.156 |
| Blood transfusion                         | 8 (17.0)       | 17 (8.6)       | 0.085 |
| Hard pancreas                             | 39 (83.0)      | 67 (33.8)      | <0.001|
| Portal vein resection                      | 25 (53.2)      | 38 (19.2)      | <0.001|
| Dissection of nerve plexus around SMA     | 35 (74.5)      | 61 (30.8)      | <0.001|
| Postoperative variables                   |                |                |       |
| Complications grade 3/4                   | 8 (17.0)       | 113 (57.1)     | <0.001|
| Pancreatic fistula grade B/C              | 8 (17.0)       | 78 (39.4)      | 0.003 |
| Hospital stay*, d                         | 18 (16–24)     | 23 (17–34)     | 0.002 |
| Adjuvant chemotherapy                     | 27 (57.4)      | 71 (35.9)      | 0.008 |
| Recurrence                                | 9 (19.1)       | 24 (12.1)      | 0.204 |
| Pancreatic enzyme supplementation         | 24 (51.1)      | 43 (21.7)      | <0.001|
| Antidiarrheal medication                  | 20 (42.6)      | 14 (7.1)       | <0.001|

ALT = alanine aminotransferase, ASA-PS = American Society of Anesthesiologists physical status, AST = aspartate aminotransferase, CA19–9 = carbohydrate antigen 19–9, CEA = carcinoembryonic antigen, NAFLD = nonalcoholic fatty liver disease, PD = pancreaticoduodenectomy, SMA = superior mesenteric artery.

Values in parentheses are percentages unless indicated otherwise; value is *median (interquartile range).
those without (non-NAFLD group). The proportion of females was significantly higher in the NAFLD group than in the non-NAFLD group \( (P < 0.001) \). Although the proportion of patients who received hypolipidemic agent (such as statin) for hyperlipidemia seemed to be higher in the NAFLD group than in the non-NAFLD group, the difference between the 2 groups was not statistically significant \( (P = 0.066) \). The prevalence of malignant tumors was comparable between the 2 groups \( (P = 0.266) \), whereas that of pancreatic cancer was significantly higher in the NAFLD group than in the non-NAFLD group \( (P < 0.001) \). On the preoperative laboratory examinations, the serum amylase levels were significantly lower in the NAFLD group than in the non-NAFLD group \( (P < 0.001) \), and the serum fasting glucose levels and serum carbohydrate antigen 19–9 (CA 19–9) levels were significantly higher in the NAFLD group than in the non-NAFLD group \( (P = 0.007 \) and \( P < 0.001 \), respectively). As for the operative factors, the operative time was significantly longer in the NAFLD group than in the non-NAFLD group \( (P = 0.012) \). The incidence of hard pancreas was significantly higher in the NAFLD group than in the non-NAFLD group \( (P < 0.001) \). The rates of combined resection of portal vein and dissection of the nerve plexus around the SMA were significantly higher in the NAFLD group than in the non-NAFLD group \( (P < 0.001 \) and \( P < 0.001 \), respectively). The incidence of both postoperative complications and pancreatic fistula was significantly lower in the NAFLD group than in the non-NAFLD group \( (P < 0.001 \) and \( P < 0.001 \), respectively), and the postoperative hospital stay was significantly shorter in the NAFLD group than in the non-NAFLD group \( (P = 0.002) \). The proportion of patients who received adjuvant chemotherapy was significantly higher in the NAFLD group than in the non-NAFLD group \( (P = 0.008) \), whereas the rate of recurrence on postoperative CT was comparable between the 2 groups \( (P = 0.204) \). The proportion of patients who received antidiarrheal medications was significantly higher in the NAFLD group than in the non-NAFLD group \( (P < 0.001) \), and the proportion of those who received pancreatic enzyme supplementation was also significantly higher in the NAFLD group than in the non-NAFLD group \( (P < 0.001) \).

### CT Parameters of the NAFLD and Non-NAFLD Groups

Table 2 shows a comparison of the pre- and postoperative CT parameters. Both the preoperative degree of liver attenuation and liver-to-spleen attenuation ratio were comparable between the 2 groups \( (P = 0.701 \) and \( P = 0.149 \), respectively). On the preoperative analysis, there were significant differences with respect to the diameter of the MPD \( (P < 0.001) \), pancreatic parenchymal thickness \( (P < 0.001) \), pancreatic duct-to-pancreatichyal ratio \( (P < 0.001) \), and extent of pancreatic attenuation \( (P < 0.001) \) between the 2 groups. Among these 4 preoperative parameters of the pancreas, the degree of pancreatic attenuation had the largest area under the curve \( (AUC = 0.779) \) on the ROC analysis for NAFLD after PD \( (AUC = 0.779) \). On the postoperative analysis, there were significant differences with respect to the pancreatic parenchymal thickness \( (P < 0.001) \), pancreatic duct-to-pancreatic thickness ratio \( (P < 0.001) \), and extent of pancreatic attenuation \( (P < 0.001) \) between the RPV \( (P < 0.001) \). The RPV had the largest AUC among these 3 postoperative parameters \( (AUC = 0.858) \).

### Risk Factors for NAFLD After PD

Table 3 shows the results of the univariate and multivariate analyses of risk factors for NAFLD after PD. The logistic regression analysis using the backward stepwise selection model identified 5 independent risk factors for NAFLD after PD: a female gender \( (OR = 5.66, 95\% \text{ confidence interval} [95\% CI] 2.39–13.40, P < 0.001) \), RPV of <12 mL \( (OR = 4.73, 95\% \text{ CI} 1.83–12.20, P = 0.001) \), preoperative pancreatic attenuation of <30 HU \( (OR = 4.50, 95\% \text{ CI} 1.73–11.70, \)
DISCUSSION

The present study investigated the risk factors for NAFLD after PD and clarified 5 independent risk factors; 3 preoperative factors: a female gender, the degree of preoperative pancreatic attenuation on CT, and the serum CA19–9 level; 1 operative factor: dissection of the nerve plexus around the SMA; and 1 postoperative factor: RPV on CT. It is noteworthy that 2 of 5 risk factors were pancreatic CT parameters which might be correlated with pancreatic exocrine function. Especially, to the best of our knowledge, there has been no report describing the association of NAFLD after PD and the degree of preoperative pancreatic attenuation on CT.

Postoperative NAFLD and malnutrition often result in a lower quality of life for patients treated with PD, thus impeding

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**TABLE 3. Univariate and Multivariate Analyses of Risk Factors for NAFLD After PD**

| Risk Factors                                      | NAFLD After PD | Univariate | Multivariate |
|--------------------------------------------------|----------------|------------|--------------|
| Present, N = 47                                  | Absent, N = 198|            |              |
| Gender                                           |                | <0.001     | <0.001       |
| Male                                             | 16             | 143        |              |
| Female                                           | 31             | 55         |              |
| Body mass index, kg/m²                           |                |            |              |
| <23.5                                            | 28             | 157        |              |
| ≥23.5                                            | 19             | 41         |              |
| Histology                                        |                | <0.001     |              |
| Pancreatic cancer                                | 39             | 74         |              |
| Others                                           | 8              | 124        |              |
| Serum amylase level, U/L                         |                | <0.001     |              |
| <80                                              | 31             | 71         |              |
| ≥80                                              | 16             | 127        |              |
| Serum FBS level, mg/dL                           |                | 0.006      |              |
| <110                                             | 16             | 113        |              |
| ≥110                                             | 31             | 85         |              |
| Serum CA19–9 level, U/mL                         |                | <0.001     | 0.029        |
| <70                                              | 18             | 137        | 1.00         |
| ≥70                                              | 29             | 61         | 2.58         |
| Operative time, min                              |                | 0.035      |              |
| <420                                             | 18             | 111        |              |
| ≥420                                             | 29             | 87         |              |
| Pancreatic texture                               |                | <0.001     |              |
| Hard                                             | 39             | 67         |              |
| Soft                                             | 8              | 131        |              |
| Portal vein resection                            |                | <0.001     |              |
| Performed                                        | 25             | 38         |              |
| Not performed                                    | 22             | 160        |              |
| Dissection of nerve plexus around SMA            |                | <0.001     | 0.017        |
| Performed                                        | 35             | 61         | 3.02         |
| Not performed                                    | 12             | 137        | 1.00         |
| Adjuvant chemotherapy                            |                | 0.008      |              |
| Performed                                        | 27             | 71         |              |
| Not performed                                    | 20             | 127        |              |
| Preoperative pancreatic duct-to-parenchymal ratio|                | <0.001     |              |
| <0.26                                            | 11             | 136        |              |
| ≥0.26                                            | 36             | 62         |              |
| Pancreatic attenuation, HU                       |                | <0.001     | 0.002        |
| <30.0                                            | 37             | 51         | 4.50         |
| ≥30.0                                            | 10             | 147        | 1.00         |
| Remnant pancreatic volume, mL                    |                | <0.001     | 0.001        |
| <12.0                                            | 38             | 48         | 4.73         |
| ≥12.0                                            | 9              | 150        | 1.00         |

CA19–9 = carbohydrate antigen 19–9, CI = confidence interval, FBS = fasting blood glucose, HU = Hounsfield units, NAFLD = nonalcoholic fatty liver disease, OR = odds ratio, PD = pancreaticoduodenectomy, SMA = superior mesenteric artery.

\( P = 0.002 \), dissection of the nerve plexus around the SMA (OR 3.02, 95% CI 1.22–7.47, \( P = 0.017 \)), and preoperative serum CA 19–9 level of \( \geq 70 \) U/mL (OR 2.58, 95% CI 1.10–6.05, \( P = 0.029 \)).
the introduction and continuation of adjuvant chemotherapy. Okamura et al. reported that NAFLD after PD might affect the long-term prognosis after pancreatectomy. Therefore, the prevention and treatment of NAFLD after PD have become clinically important issues.

Two pancreatic CT parameters, RPV and the degree of preoperative pancreatic atrophy, were significant risk factors for NAFLD after PD with the second and third highest ORs of 4.73 and 4.50, respectively. A reduced RPV is naturally considered to be highly associated with the impaired postoperative pancreatic function. Previous reports showed that a reduced postoperative pancreatic parenchymal thickness correlated with pancreatic exocrine insufficiency after PD. Another recent study showed that the RPV significantly influenced the development of NAFLD after PD. The results of the present study reinforce the notion that a reduced RPV is a reliable indicator of NAFLD after PD. A lower preoperative pancreatic attenuation was also significantly correlated with post-PD NAFLD. There has been no report evaluating the association between the degree of pancreatic attenuation and the pancreatic exocrine function or the development of NAFLD after PD. One recent study showed that the extent of pancreatic attenuation well correlated with the loss of pancreatic parenchyma and development of fatty infiltration. Therefore, we hypothesized that a reduction in the number of pancreatic acinar cells due to obstructive pancreatitis or fatty metamorphosis of the distal pancreas might lead to a decrease in CT attenuation of the pancreatic parenchyma. However, precise correlation between pancreatic attenuation and pathological changes has not been fully elucidated, which should be further investigated. Nevertheless, evaluation of the degree of pancreatic attenuation, based on the mean value of 3 ROIs in the pancreatic parenchyma, is an easy, noninvasive and clinically practical method to assess the risk factors for NAFLD after PD.

A female gender was found to be an independent risk factor for NAFLD after PD, with the highest OR of 5.66, in the present study. Although previous reports also showed a significant correlation between a female gender and NAFLD after PD, the etiology is still unclear. One possible reason may be that the pancreatic volume in females is smaller than that in males. This correlation of the pancreatic volume with gender likely results from anthropometric differences between males and females. The other possible explanation may be the withdrawal of estrogens in postmenopausal woman, who have a higher prevalence of NAFLD. Menopause markedly accelerates the accumulation of visceral fat, which may induce insulin resistance. One recent study reported that a female gender was a significant risk factor for intractable NAFLD even after pancreatic enzyme supplementation. This observation suggests that not only pancreatic exocrine insufficiency but also insulin resistance influence the development of NAFLD after PD in females.

Dissection of the nerve plexus around the SMA was found to be the only operative risk factor associated with NAFLD after PD, a factor that causes postoperative diarrhea and intestinal malabsorption. Several randomized controlled trials concluded that PD with extended lymphadenectomy did not have a benefit with respect to long-term survival and actually increased the incidence of severe diarrhea associated with weight loss. Kato et al. reported a significant association between postoperative diarrhea and NAFLD after PD. These reports support the result of the current study, a significant association of dissection of the nerve plexus around the SMA and NAFLD after PD.

An elevated serum CA 19–9 level was identified to be 1 of 3 preoperative risk factors for NAFLD after PD. Tanaka et al. also reported that patients with NAFLD after PD had higher preoperative CA 19–9 levels than those without NAFLD, based on a univariate analysis. CA 19–9 is a well-known tumor marker of biliary and pancreatic cancers, and has recently been reported to be associated with insulin resistance and glycemic control in diabetic patients. The characteristics of the primary tumor with an increased serum CA19–9 level might cause insulin resistance which potentially affected the development of NAFLD after PD.

The limitations of this study include the lack of histological diagnoses for NAFLD and nonalcoholic steatohepatitis. We had no histological data from liver specimens. It was not feasible to perform liver biopsies for all of the NAFLD patients due to the invasive nature of the procedure. In addition, the present study lacked a histological examination of the pancreas, which should be performed in order to elucidate the relationship between pancreatic attenuation and the quantity of pancreatic acinar cells or islet cells. This study also has retrospective nature and the presence of selection bias of the patients enrolled at a single center. Further prospective multi-institutional studies are therefore needed to objectively evaluate the usefulness of pancreatic CT parameters as predictors of the pancreatic function and incidence of NAFLD after PD.

In conclusion, 5 independent risk factors, including 2 pancreatic CT parameters, significantly influence the development of NAFLD after PD. Perioperative CT assessments of the pancreas may be helpful for predicting the development of NAFLD after PD and also may contribute to the prevention and early treatment of this late complication of PD.

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