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REVIEW ARTICLE

Hepatic Steatosis Following Pancreatic Surgery: A Swedish Centers Experience with Demographics, Risks and Outcome

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ABSTRACT

Objective Non-alcoholic fatty liver disease encompasses a spectrum of fatty changes in the liver ranging from simple steatosis to nonalcoholic steatohepatitis. Certain patients may develop hepatic steatosis following pancreatic resections. The aim of our study was to investigate factors possibly associated with steatosis after pancreatic resection and to see if the survival of patients was influenced by its development. **Methods** 182 consecutive patients that underwent pancreatic surgery (between 2010-2013) in a Swedish high volume pancreatic center were retrospectively reviewed from the preoperative work-up until at least 6 months postoperatively. Pre-versus postoperative values of Hounsfield Units were checked on CT scans and utilized as a proxy for development of hepatic steatosis. Demographic data and a number of possible risk factors were recorded together with survival after surgery. Results 17 percent of our patient group developed hepatic steatosis within the first year of surgery. The development of steatosis occurred significantly more frequently in females and in patients over the age of 70. Hepatic steatosis was positively associated with increased levels of alanine amino transferase (ALT) after surgery. No association was noted between grade of weight loss and steatosis. Nor were there any correlations between the incidence of steatosis and the type of operation. Post-operative hepatic steatosis did not adversely affect overall survival. **Conclusion** Our results demonstrate that certain demographic and laboratory values are associated with the development of hepatic steatosis after pancreatic resections. However, despite its development, hepatic steatosis is of no apparent clinical significance and does not affect overall survival following pancreatic surgery.

INTRODUCTION

Pancreatic resections are performed for both malignant and non-malignant disease. Resections may include not only the pancreatic parenchyma, but also the duodenum, proximal jejunum, distal part of the stomach and gallbladder [1, 2]. It is well established that resection of both exocrine and endocrine pancreatic tissues may cause metabolic disturbances. It is however unknown whether resections of the stomach and the first portion of the small intestine influence hepatic metabolism in a significant way. In contrast, it has been well described that resections of the duodenum and incretin-synthesizing cells as in gastric by-passes for obesity have profound anti-diabetic effects on glucose homeostasis [3]. Hence,pancreatic resections, either alone or in combination with resections of stomach/ intestinemay influence metabolic homeostasis. In addition, potential complications specific to pancreatic surgery such as anastomotic leakage/strictures, delayed gastric emptying and pancreatic fistula formation [1, 2, 4] may all influence hepatic metabolism.

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Hepatic steatosis, when caused by any reason other than alcohol consumption, is referred to as non-alcoholic fatty liver disease (NAFLD) [5-8]. NAFLD is an umbrella term encompassing a spectrum of fatty changes in the liver ranging from simple steatosis to nonalcoholic steatohepatitis (NASH) [6]. While simple steatosis is considered a benign condition, NASH may progress to cirrhosis, possibly leading to hepatocellular carcinoma or hepatic failure [7-9]. The initial change is due to triglyceride accumulation and has been shown to occur more frequently in certain insulin resistant states (metabolic syndrome or type 2 diabetes) and obesity [6-8, 10]. It has also been noted to occur in patients with marked weight loss and emaciation [5]. NAFLD represents the most common cause of chronic liver disease in the western world [11], with NASH being the 3rd most common indication for liver transplantation [12].

The gold standard for diagnosis of hepatic steatosis is a liver biopsy [13, 14]. 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 [13]. CT and MRI provide higher sensitivity and thus are better alternatives [13, 14].

Assessing tissue density in CT scans can be performed by assigning each pixel in the image with a numerical unit, which is expressed as a Hounsfield (HU) [15]. Air, which has a low density, appears dark and has a HU of -1000. Increasing levels of density are reflected by increases in HU with values such as 0 in water and +3000 in dense bone. A drop in HU of the liver from pre- to post-operation would signify a lower density and hence a higher triglyceride level in the hepatocytes [16].

We have thus used measurements of HU on CT as a proxy for short-time changes of steatosis after pancreatic resections as has been done in other investigations [17, 18]. By assessing hepatic steatosis with CT scans the aim of our study was (A) to determine significant increases in hepatic steatosis after pancreatic resections, (B) to find predictive factors that may be associated with hepatic steatosis after pancreatic resections, (C) to find if concomitant resection of stomach-duodenum-jejunum is of importance (left or right-sided pancreatic resections) for the development of steatosis, and (D) to evaluate if post-surgical hepatic steatosis affects the overall survival of patients.

METHODS

Subjects

A total of 354 consecutive patients that underwent elective pancreatic resections (2010-2013) at Karolinska University Hospital (Stockholm, Sweden) were retrospectively reviewed. Handling of data was carried out in adherence to the terms of the regional ethical committee's guidelines for retrospective studies without re-questioning of the patients. Exclusion criteria included patients who had been followed-up in other hospitals, patients who died within the first six months of surgery and patients who had a known recurrence of malignant disease at their first follow-up (within 6 months). Additionally, patients who did not get their intended operation due to unexpected tumor growth were excluded. The final data base included 182 patients that underwent either a pancreatoduodenectomy (PD), distal pancreatectomy (DP) or total pancreatectomy (TP).

Analysis of steatosis

All CT examinations were performed with a 64-channel MDCT scanner (LightSpeed VCT or LightSpeed VCT XTE, GE Healthcare, Milwaukee, WI). All patients underwent a preoperative CT according to standardized criteria for the multidisciplinary conference recommending surgery. The postoperative CTs used in this study were first taken between 6 months to 1 year after surgery; a time lap after surgery to allow for the possible formation of hepatic steatosis in a steady-state situation. The presence of post-operative hepatic steatosis was determined by measuring hepatic attenuation with Hounsfield units (HU). Out of 182 patients, 88 were classified on the basis of unenhanced CTs whereas 94 had the investigations performed on contrast enhanced CTs.

Hepatic attenuation was measured in Sectra (version 15.1 http://www.sectra.com/medical/) by averaging two regions of interests (15 mm diameter) in segments 2 and 8 of the liver. Splenic attenuation was measured in the periphery of the organ (**Figure 1**). Care was taken not to include vessels, calcifications and masses. The definition for steatosis on unenhanced CTs was set as a decrease of 20 HUs between the preoperative and postoperative scans. This change has been shown to indicate a 10 percent increase in the fatty content of the liver [19]. Similar methods could not be employed for analyzing contrast enhanced CTs due to varying hepatic contrast levels in the portal venous phase of the scan. Jacobs *et al*. [20] studied contrast enhanced CTs with regards to steatosis to define a liver to spleen attenuation difference of $^{-}$31 to $^{-}$10 in the portal venous phase. This definition was used for analysis of the contrast enhanced CTs. These cases were reviewed by a radiologist and were confirmed to show signs of hepatic steatosis.

All patients were checked for blood levels of bilirubin and alanine amino transferase (ALT) at the time of the evaluated CTs. The body mass index (BMI) was checked preoperatively, postoperatively and during each follow-up visit. The operative times, operative blood loss and hospital stay was noted for all patients in the study.

Statistical Analysis

Continuous data was analyzed using Welsh's two sample t-test and categorical variables were compared using the χ^2 test or Fishers exact test when appropriate. Statistical significance was set at a level of $p<0.05$. All calculations were done using the "R" program (version 3.1.1 http://www.r-project.org). Kaplan-Meier survival estimations were plotted using SPSS (version 21).

RESULTS

Patient Data

The study included a total of 99 men (54 %) and 83 women (46 %) with an average age of 64 years (range 21-82). Of these subjects, 85 percent were operated on with a pancreatoduodenectomy, 10 percent a distal pancreatectomy and 5 percent a total pancreatectomy with concomitant stomach, duodenum and proximal jejunum resection. Twelve percent of patients needed intraoperative resection of the portal vein (PV) and/or the superior mesenteric vein (SMV). Postoperative histology revealed the most common diagnosis to be pancreatic adenocarcinoma (45 %) followed by intraductal papillary mucinous neoplasm (10 %) and chronic pancreatitis (7 %). Other pathologies included cancer in the ampulla of Vater, duodenal cancer and cancer of the lower bile ducts (38%) **(Table 1)**.

Risk factors for steatosis

Out of 182 patients, 31 (17 %) were found to have developed postoperative hepatic steatosis 6 to 12 months after their intended operation. None of the patients had had any clinical intervention or further investigation due to the steatosis. In cases with steatosis, further CT-scans showed no tendency for an increased grade of steatosis. Decreased signs of

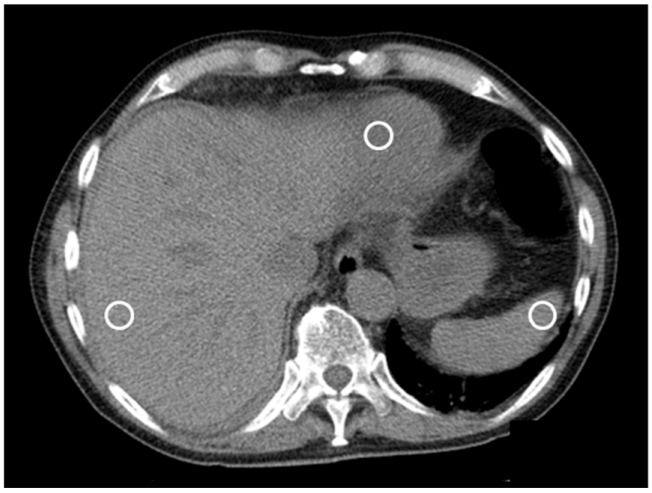

Figure 1. Hepatic Attenuation was measure in two regions of interest in the liver and in the periphery of the spleen.

steatosis were seen in only 10 percent of the 58 patients who received another CT, following the first one at 6-12 months postoperatively. Due to the natural progression of underlying diseases there were insufficient numbers of long term studies for proper statistical analysis.

Patient subsets based on age, gender, type of operation performed, the diagnosis of cancer and the presence of diabetes mellitus were analyzed for the presence of postoperative steatosis **(Table 2)**. Significant differences were noted in the age and gender of the patients. Patients over the age of 70 and women were positively associated with hepatic steatosis. In comparing the incidence of steatosis between patients that underwent a pancreatoduodenectomy with a distal pancreatic resection the percentages were 22% *vs.* 13% respectively. This finding however, was not statistically significant (P=0.33). Other investigated factors showed no association with postoperative hepatic steatosis.

The values of ALT, bilirubin and BMI were checked 1 day preoperatively, 3 days postoperatively and approximately 6 months (4-8 months) postoperatively **(Table 3)**. Patients without hepatic steatosis were seen to have a significant decrease (P=0.04) in ALT values 6 months after surgery. This decrease was not noted in patients with hepatic steatosis. In both patient groups there were significant decreases in BMI compared to the preoperative values, with no significant correlation between percentages of weight loss and steatosis. Operative factors such as time, blood loss and postoperative hospital stay were also analyzed but no significant associations to steatosis were found **(Table 4).**

Finally, overall survival was analyzed. Patients that developed hepatic steatosis and patients that did not develop steatosis were compared **(Figure 2)**. Survival was analyzed after stratification for pancreatoduodenectomies, the female gender and patients over the age of 70. No significant associations were made between hepatic steatosis and overall survival regarding all patients or the subgroups.

DISCUSSION

Our study indicates that hepatic steatosis is present in about one out of six patients 6-12 months after a pancreatic resection. Furthermore, the steatosis did not lead to further clinical interventions and did not influence survival, in a group of patients mainly operated upon for malignancy. The presence of steatosis after pancreatic resection is, however, not a new finding, as it was reported by us in 1991 [21] and later by others [17, 22-25]. While this has largely been considered a benign consequence of the operation, there have been selected reports of fulminant NASH and hepatic failure developing after surgery [25]. Our Kaplan-Meier estimation indicates that steatosis does not affect overall survival on a group level. This remained true when subsets like age and gender were specifically taken into account. Okamura *et al*. [17] suggested that postoperative hepatic steatosis might be associated with a decrease in recurrence free survival and overall survival but their findings were not statistically significant. Our study shows a similar trend with no statistical significance. From our study we can tell that the healthy patients (i.e. patients with no recurrent disease) who received CTs after the designated 6-12 months had no progression of their steatosis. A regression of the steatosis was seen in approximately one out of ten patients. Even though the long-term follow-up was insufficient, we believe that the steatosis is a self-limiting disease.

Table 1. Patient Demographics.

Characteristics	Value
Age; average (range)	64 (21 – 82)
Sex (M:F)	99:83
Operative Procedure; n (%)	
PD	154 (85)
DP	18 (10)
TP	10 (5)
With PV or SMV resection	21 (12)
Histopathology; n (%)	
Pancreatic Cancer	81 (45)
Ampullary Cancer	10 (6)
Duodenal Cancer	6 (3)
Lower Bile Duct Cancer	8 (4)
Neuroendocrine Tumors	9 (5)
Intraductal papillary mucinous carcinoma	11 (6)
Intraductal papillary mucinous neoplasm	18 (10)
Chronic Pancreatitis	12 (7)
Other	26 (14)

Table 2. Analysis of postoperative hepatic steatosis based on patient data.

Variable; n(%)	With Hepatic Steatosis (n=31)	Without Steatosis (n=151)	P value
Age (>70/<70)	15/16 (48/52)	46/105 (30/70)	**0.05**
Sex(M/F)	12/19 (39/61)	87/64 (58/42)	**0.05**
PD	28 (90)	126 (83)	0.33
DP	2 (6)	16 (11)	0.5
TP	1 (3)	9 (6)	0.5
PV/SMV Resection	6 (19)	15 (10)	0.2
Pancreatic Cancer	16 (52)	66 (44)	0.4
Malignancy	29 (94)	138 (91)	0.2
DM	7 (23)	28 (19)	0.6

PD pancreatoduodenectomy; DP distal pancreatectomy; TP total pancreatectomy

Table 3. Comparison of preoperative and postoperative parameters in patients with steatosis and without steatosis.

	With Hepatic Steatosis			Without Hepatic Steatosis		
Variable	**Preoperative**	**Postoperative**	**P value**	**Preoperative**	**Postoperative**	**P value**
ALAT[1]	9 (2 – 47)	15 (2 – 106)	0.1	11 (2 – 74)	14 (1– 179)	0.1
ALAT (after 6m)	9 (2 – 47)	9 (2 – 51)	0.9	11 (2 – 74)	8.5 (1 – 54)	**0.04**
Bilirubin[2]	30 (2 – 206)	13 (4 – 46)	0.08	23 (2 – 255)	17(2 – 136)	0.06
Bilirubin (after 6m)	30 (2 – 206)	14 (3 – 80)	0.1	23 (2 – 255)	17 (2 – 302)	0.2
BMI (at discharge)	25 (17 – 36)	24 (15 – 35)	0.6	25 (15 – 47)	24 (14 – 45)	0.1
BMI (after 6m)	25 (17 – 36)	22 (17 – 28)	**0.003**	25 (15 – 47)	22 (14 – 42)	**0.0000001**

Continuous Data are expressed as mean (range)
1: international units/liter
2: µmol/L

Table 4. Comparison of Operative Factors in Patients with Hepatic Steatosis and Patients without Hepatic Steatosis.

Variable	With Hepatic Steatosis	Without Hepatic Steatosis	P value
Operative Time (min)	355 (100 – 528)	358 (63 – 728)	0.8
Operative Blood Loss (ml)	852 (50 – 2560)	1005 (50 – 8000)	0.2
Hospital Stay (days)	18 (7 – 75)	17 (3 – 76)	0.3

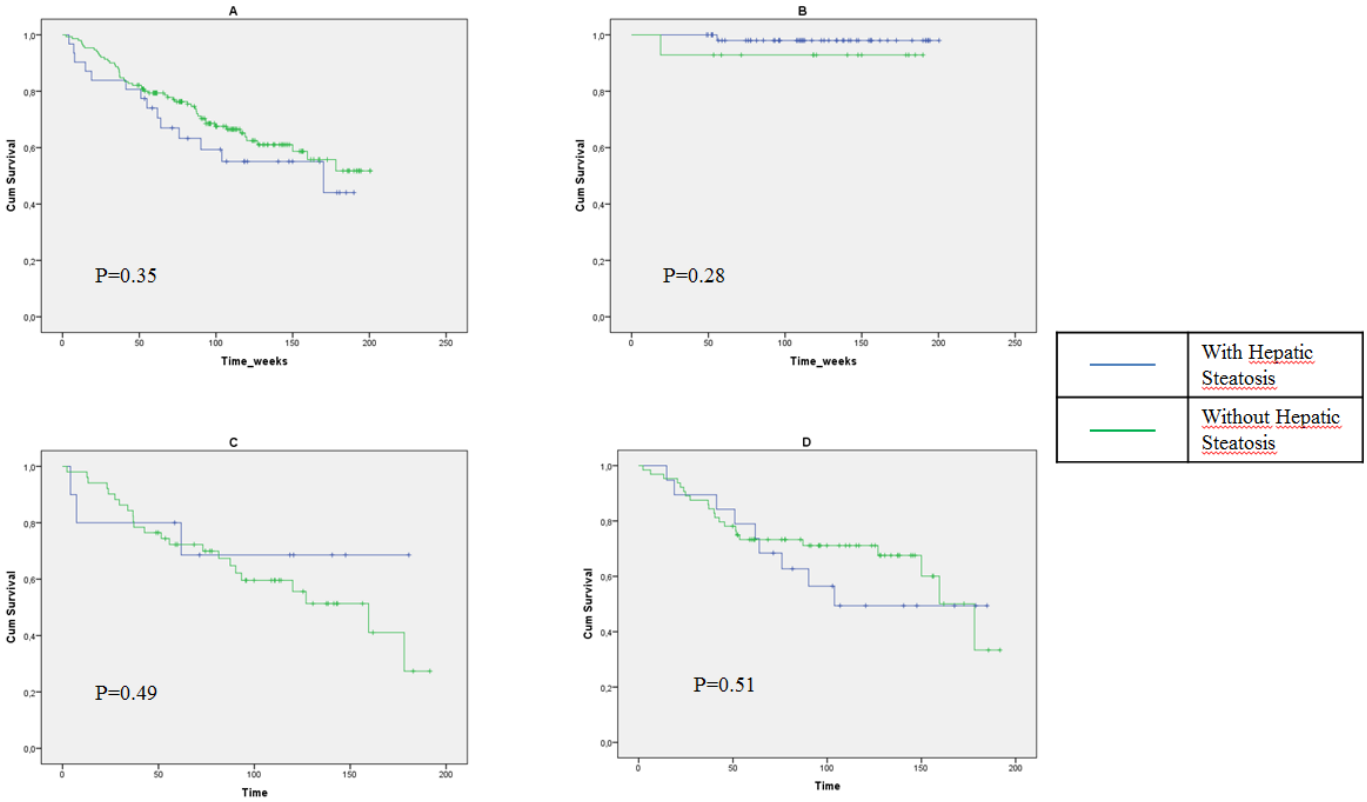

Figure 2. Overall Survival in **(a.).** all the patients, **(b.).** patients with PDAC, **(c.).** patients aged over 70 and **(d.).** all female patients.

Postoperative hepatic steatosis appeared to occur more frequently in females and older patients. Ye *et al*. found similar significance regarding female patients [25] while Okamura *et al*. [19] refuted such an association. Nakagawa *et al*. [17] also refuted this gender association although their data tended towards statistical significance (P=0.053). With regards to age, a significant association was found between patients over 70 and hepatic steatosis. A possible explanation for the different results could be differences in population demographics. Our patient group mainly came from Sweden, whereas the other studies were performed on East Asian populations with possibly other liver statuses preoperatively. An alternate explanation could be that most studies have had limited number of patients with hepatic steatosis, which may not detect smaller differences.

The days of postoperative stay was utilized as a proxy for postoperative complications as it has been shown that unexpectedly long hospital stays are associated with greater incidences of postoperative complications [26]. There were too few severe complications in our cohort to investigate the relation of these to steatosis. However, the average hospital stay was similar in patients with hepatic steatosis and patients without steatosis; 18 and 17 days, respectively. Intra-operative factors such as blood loss, PV/SMV resection and the overall time for surgery were also

similar between both patient groups. Thus, perioperative factors were not found to be significantly associated with hepatic steatosis.

Although the mechanism of postoperative hepatic steatosis remains to be further elucidated there have been some proposed theories. Recently, there have been suggestions that the pancreatic exocrine insufficiency that develops after pancreatoduodenectomies, with its concomitant malabsorption not only of lipids but also carbohydrates and proteins, may be a main factor in the development of NAFLD [19]. It is also possible thatelderly patients and women have less preoperative reserves predisposing them to postoperative hepatic steatosis. As a result, this has led to the rational for an intensification of pancreatic enzyme replacement which may prove to be useful in management of this entity [18, 24]. There have been speculations regarding the role of protein energy malnutrition due to rapid postoperative weight loss. [27]. This is reflected in our study as patients with hepatic steatosis underwent a significant weight loss following surgery (**Table 3**). However, patients without hepatic steatosis also underwent significant weight loss making it a general occurrence following pancreatic surgery.

These findings should be viewed with a number of limitations in mind. This was a retrospective study and is susceptible to bias from the clinical management of individual patients. Analysis of distal and total pancreatectomies was limited due to a small number of patients in those subsets. Comparison with a control group was not optimal due to the heterogeneity of the patients undergoing pancreatic surgery (**Table 1**). Yoo DG *et al*. [28] compared de novo hepatic steotosis following a pancreatoduodenectomy with a control group of bile duct resections. Significant differences were found in the occurrence of postoperative hepatic steatosis; 26% following pancreatoduodenectomy versus 4% following bile duct resections. Unique aspects of our study include a large number of patients, all of whom were operated in a high volume center. This entailed that all patient received standardized pre- and postoperative management with proper documentation.

To conclude, the presence of hepatic steatosis is an established entity that may occur following pancreatic surgery. However, it is still not known with certainty why only about one sixth of our patients developed steatosis. While certain preoperative factors may be linked to hepatic steatosis, its presence is not related to intraoperative and postoperative complications. On the other hand, it remains to be elucidated whether there is an increased risk of after resection of the stomach-duodenum-upper jejunum. It is also not understood why patients that underwent tail resections of the pancreas may develop liver steatosis as they generally do not have pancreatic insufficiency. Our patients with steatosis all experienced weight loss after the resection, but they were not malnourished and there were no indications that those with greater weight loss or more rapid initial weight loss had a higher risk of steatosis. Of clinical importance is, however, that the postoperative hepatic steatosis in this selected group of patients is not associated with a decrease in overall survival and therefore requires no additional investigations or therapy per se.

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Conflict of interest

The authors have no conflict of interest to declare

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