J Gastrointest Surg (2013) 17:1392–1398
DOI 10.1007/s11605-013-2260-z

2012 SSAT POSTER PRESENTATION

Splenic Vein Thrombosis Is Associated with an Increase in Pancreas-Specific Complications and Reduced Survival in Patients Undergoing Distal Pancreatectomy for Pancreatic Exocrine Cancer

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Received: 9 November 2012 / Accepted: 11 June 2013 / Published online: 25 June 2013

Abstract Distal pancreatectomy and splenectomy (DPS) is the procedure of choice for the surgical treatment of pancreatic exocrine cancer localized to the body and tail of the pancreas. Splenic vein thrombosis (SVT) can occur in patients with malignant pancreatic exocrine tumors secondary to direct tumor invasion or compression of the splenic vein by mass effect. This study examines the effect of preoperative SVT on postoperative outcomes. In this retrospective cohort study, we queried our pancreatic surgery database to identify patients who underwent DPS from October 2005 to June 2011. These cases were evaluated for evidence of preoperative SVT on clinical records and cross-sectional imaging (CT, MRI, endoscopic US). Outcomes for patients with and without SVT were compared. From an overall cohort of 285 consecutive patients who underwent DPS during the study period, data were evaluated for 70 subjects who underwent surgery for pancreatic exocrine cancer (27 with SVT, 43 without SVT). The preoperative demographics and co-morbidities were similar between the groups, except the average age was higher for those without SVT ($p<0.05$). The median estimated blood loss was significantly higher in the SVT group (675 versus 250 ml, $p=<0.001$). While the overall morbidity rates were similar between the two groups (48 % SVT versus 56 % no SVT, p=NS), the group with SVT had a significantly higher rate of pancreas-specific complications, including pancreatic fistula (33 versus 7 %, $p<0.01$) and delayed gastric emptying (15 versus 0 %, $p<0.02$). Hospital readmission rates were similar between the groups (30 versus 28 %, p=NS). Patients without SVT had a trend toward longer median survival (40 versus 20.8 months), although the difference was not statistically significant ($p=0.1$). DPS for pancreatic ductal adenocarcinoma can be performed safely in patients with SVT, but with higher intraoperative blood loss, increased pancreas-specific complications, and a trend towards lower long-term survival rates. This paper was presented as a poster at the 53rd annual meeting of the Society for Surgery of the Alimentary Tract and at the 46th annual meeting of the Pancreas Club, San Diego, CA, May 2012.

Keywords Pancreatic cancer · Splenic vein thrombosis · Distal pancreatectomy

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Introduction

Pancreatic exocrine cancers are the fourth leading cause of cancer-related death in the USA, and they account for 85 % of malignant tumors of the pancreas. In 2011, there were 44,030 new pancreatic cancer diagnoses and 37,660 attributable deaths.[1] Due to the lack of effective early screening modalities, patients often present late in their disease course and at an advanced stage. At the time of diagnosis, only 15–20 % of cases are eligible for surgical resection, which is the only potentially curative treatment. Overall 5-year survival

remains low (6 %) and has only modestly improved over the past 30 years,[1,2] though it increases to 25–30 % for lymph node-negative tumors after a margin-negative pancreatic resection.[3,4]

The surgical procedure used to treat malignant pancreatic exocrine tumors depends upon the location of the lesion within the pancreas—pancreaticoduodenectomy is the operation of choice for tumors of the right side (head, neck, and uncinate process), while distal pancreatectomy and splenectomy (DPS) is utilized for tumors to the left of the superior mesenteric artery and vein (body and tail). Pancreatic exocrine cancer in the body and tail is often asymptomatic, allowing tumors time to grow and invade surrounding structures or disseminate before clinical manifestations prompt diagnosis. When symptoms do arise in these left-sided tumors, they are most commonly pain, anorexia, and weight loss.[5] As these symptoms can be somewhat vague and are relatively slow in onset, lesions in the body and tail may present at a more advanced stage when compared to their right-sided counterparts where the development of obstructive jaundice may herald diagnosis earlier in the disease course.[6]

As the pancreatic exocrine cancer grows, it can encroach upon or invade the surrounding visceral vasculature or adjacent organs within its local environment. The most common large blood vessel to be invaded or compressed by left-sided pancreatic exocrine cancer is the splenic vein, which courses along the dorsal aspect of the pancreas and joins the superior mesenteric vein to form the portal vein. Splenic vein thrombosis (SVT) arises in patients in whom compression, encasement, or invasion of this vein occurs by direct extension of the pancreatic exocrine tumor. The development of SVT results in an increase in left-sided venous pressures, termed sinistral hypertension. As a response to the occlusion of venous return through the splenic vein, an alternate route of venous blood flow develops from the spleen to the liver, as collateral vasculature increases in number and circuits, to allow venous blood to reach the portal circulation. These collateral vessels (termed varices) form within the retroperitoneum, lieno-colic ligament, gastrocolic ligament, and gastro-splenic ligament, and can cause an engorgement of the short gastric veins and coronary vein. These varices may rupture and cause a rare, but life-threatening gastrointestinal hemorrhage. Patients with SVT are usually identified preoperatively by cross-sectional imaging which reveals an obstructed splenic vein and the presence of isolated perigastric varices, with or without splenomegaly (Fig. 1).

Generally speaking, SVT is most commonly associated with the inflammation of acute or chronic pancreatitis. [7] To date, there have been no reports in the literature that detail the impact of SVT in the management and outcomes of patients with pancreatic exocrine cancers. In this study, we examine our institution's experience with SVT in patients with malignant disease of the pancreas and describe the implications for operative intervention, postoperative outcomes, and long-term survival.

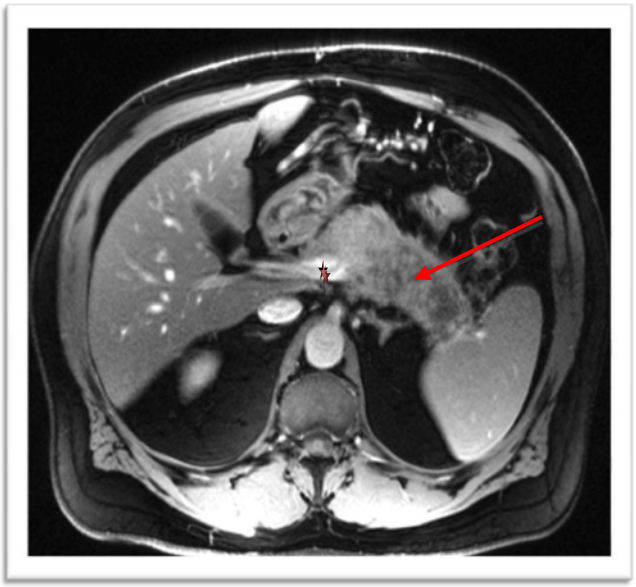

Fig. 1 SVT secondary to pancreatic exocrine cancer. (*) Superior Mesenteric Vein, (←) Pancrea4c Adenocarcinoma; involving the pancrea4c body and tail

Methods

In this retrospective study, we reviewed the pancreatic surgery database in the Department of Surgery of Thomas Jefferson University, which our Institutional Review Board has approved for data acquisition and query. To supplement this database, we also reviewed electronic medical records, preoperative imaging, operative and discharge reports, and surgical pathology reports for all patients who underwent a DPS at the Thomas Jefferson University Hospital from October 2005 to June 2011. We then restricted our focus to patients who underwent DPS for exocrine cancer of the pancreas, excluding those who underwent surgery for other pathologic entities. In addition to patients with pancreatic ductal adenocarcinoma (PDA), we also included those with adenosquamous carcinoma, acinar cell carcinoma, and undifferentiated carcinoma in the analysis. Intraductal papillary mucinous neoplasms (IPMNs) were also included in cases where an invasive component was present. The presence of preoperative SVT was judged based on review of CT, MRI, or EUS studies and operative notes detailing intraoperative findings. We looked for a direct statement of SVT or evidence of splenic vein obstruction, occlusion, encasement, or invasion and/or perigastric varices indicating SVT. The remainder of patients with SVT were identified by operative notes reporting intraoperative findings of SVT.

Demographics including age, gender, body mass index, diabetes mellitus status, and tobacco use were evaluated. When available, preoperative laboratory values for hemoglobin

A1C (Hb A1C), serum albumin, and the serum tumor markers carbohydrate antigen 19-9 (CA 19-9) and carcinoembryonic antigen (CEA) were also recorded. Operative notes provided information on the intraoperative findings, details of the surgery performed, and the estimated intraoperative blood loss (EBL). Surgical pathology reports provided information on tumor histology and grade, lymph node involvement, and venous and perineural/lymphatic invasion. Disease staging was consistent with the American Joint Committee on Cancer (AJCC 6th edition) guidelines.[8]

Postoperative complications were reviewed, including: pancreatic fistula (PF), delayed gastric emptying (DGE), wound infection, sepsis, urinary tract infection (UTI), chyle leak, deep vein thrombosis (DVT), pulmonary embolism (PE), cardiac complications, pulmonary complications, and hospital readmissions. PF and DGE were defined and graded according to guidelines from the International Study Group on Pancreatic Fistula and the International Study Group on Pancreatic Surgery, respectively.[9,10] Cardiac complications were defined as myocardial infarct or cardiac arrhythmia. Pulmonary complications were defined as prolonged intubation, pleural effusions requiring drainage, or respiratory tract infection. Hospital readmissions were tracked for a 30-day period and recorded based on the specific postoperative complication present. Survival data were obtained through patient records and the Social Security Death Index.

T tests or Wilcoxon rank sum tests were used to compare patients with SVT to those without SVT with respect to continuous outcomes. Fisher's exact test was used to compare SVT groups with respect to categorical outcomes. The Kaplan–Meier method was used to estimate the survival distribution by group, and the logrank test was used to test for differences in survival distribution. Statistical significance was accepted at the $p<0.05$ level. Cox proportional hazards regression was used to assess potential confounding of the association between SVT and survival. Each variable was considered individually and was included in a multivariable regression model if its inclusion changed the log hazard ratio for SVT by more than 10 %.

Results

In total, 285 consecutive patients who underwent DPS from October 2005 to June 2011 at the Thomas Jefferson University Hospital were identified. Two-hundred and fifteen patients were excluded from the analysis due to pathologic findings other than pancreatic exocrine cancer, or due to complex multiorgan resections, leaving 70 patients eligible for this study. Of these 70 patients with malignant exocrine disease of the pancreas who underwent DPS, data were evaluated on 27 patients with preoperative SVT and 43 patients without SVT. Of the 27 patients who had SVT, 21 had preoperative imaging demonstrating evidence of SVT. The remaining six were clinically diagnosed in the operating room. All patients underwent open resection of their tumors, with the exception of two patients without SVT who underwent laparoscopic surgery.

The preoperative demographics, co-morbidities, and laboratory variables were similar between the two groups, with the exception that the SVT group was younger at 62 years, as compared to 68 years for the non-SVT group ($p<0.05$) (Table 1). Pathologic findings from the study are shown in Table 2. PDA was the most prevalent pathology identified, affecting 70 % of patients with SVT and 84 % of patients without SVT. The rates of lymph node positive carcinomas were nearly equivalent between the SVT group and the group without SVT, at 52 and 56 %, respectively ($p=0.81$). There were no significant differences between the SVT and non-SVT groups when assessing for perineural invasion (81 versus 71 %, $p=0.40$) or venous/lymphatic invasion (39 versus 29 %, $p=0.57$), respectively.

The EBL during surgery was significantly higher in the SVT group as compared to the group without SVT (675 versus 250 ml, $p<0.001$), respectively (Table 3). There were no cases of postpancreatectomy hemorrhage in either group. Although the overall rate of postoperative complications trended lower in the SVT group (48 versus 56 %, $p=0.63$), patients in the SVT group had significantly higher rates of pancreas-specific complications, such as pancreatic fistula (33 versus 7 %, $p<0.01$) and delayed gastric emptying (15 versus 0 %, $p<0.02$). None of the other postoperative complications tabulated showed a significant difference between the two groups. The median length of postoperative hospital stay was the same for both groups (6 days), and the 30-day readmission rates were also similar between the groups (SVT, 30 %; without SVT, 28 %). Thirty-day mortality was 0 % in both groups while 90-day mortality rates were 4.6 % in the group without SVT and 3.7 % for the SVT group. Patients without SVT showed a trend favoring a longer median survival (40 versus 20.8 months, $p=0.1$), although the difference was not statistically significant. After adjustment for AJCC stage, the association between SVT and reduced survival was strengthened (adjusted HR=1.91 versus unadjusted HR=1.78), but still did not reach statistical significance ($p=0.08$) (Table 4). Figure 2 shows the Kaplan–Meier survival curve for both groups, with the SVT cohort having an inferior survival as compared to the non-SVT group ($p=0.0967$ by logrank).

Discussion

In this retrospective analysis of our pancreas resection database, we focused on the role of SVT in affecting outcomes in patients undergoing DPS for pancreatic exocrine cancer. We found that preoperative SVT resulted in a significantly

Table 1 Patient demographics and preoperative laboratory variables

Variable	SVT (*N*=27)	Without SVT (*N*=43)	Total (*N*=70)	*p* value
Age in years, mean (range)	62.2 (29–79)	68.2 (31–89)	65.9 (29–89)	0.04 (t)
BMI, mean (range)	25.0 (17.8–31)	27.0 (17–40)	26.2 (17–40)	0.1 (W)
Sex, male; *N* (%)	18 (67 %)	24 (56 %)	42 (60 %)	0.46 (F)
DM, *N* (%)	7 (26 %)	13 (30 %)	20 (29 %)	0.79 (F)
Smoking, *N* (%)	10 (37 %)	16 (40 %)	26 (39 %)	1.0 (F)
Serum lab parameters				
HgbA1C, median (range)	6.0 (4.3–11.9)	6.1 (4.6–12)	6.05 (4.3–12)	0.84 (W)
Albumin, median (range)	4.2 (2.9–5)	4.3 (2.9–5.1)	4.2 (2.9–5.1)	0.26 (W)
CA 19–9, median (range)	71 (2–2221)	54 (1–5232)	71 (1–5232)	0.39 (W)
CEA, median (range)	2.25 (0.5–17.1)	2.05 (0.6–278)	2.2 (0.5–278)	0.67 (W)

SVT Splenic vein thrombosis, *BMI* body mass index, *DM* diabetes mellitus, *HbA1C* hemoglobin A1C, *CA 19-9* carbohydrate antigen 19-9, *CEA* carcinoembryonic antigen, *F* Fisher's exact, *W* Wilcoxon nonparametric, *t t* test

Table 2 Pathology results

Variable		SVT (*N*=27)	Without SVT (*N*=43)	Total (*N*=70)	*p* value
Histologic type	*N* (%)				
Pancreatic ductal adenocarcinoma		19 (70 %)	36 (84 %)	55 (79 %)	NS
Adenosquamous carcinoma		3 (11 %)	2 (5 %)	5 (7 %)	NS
IPMN—with invasive component		2 (7 %)	2 (5 %)	4 (6 %)	NS
Intraductal oncocytic papillary tumor		1 (4 %)	1 (2 %)	2 (3 %)	NS
Acinar cell carcinoma		0 (0 %)	2 (5 %)	2 (3 %)	NS
Mixed ductal–endocrine carcinoma		1 (4 %)	0 (0 %)	1 (1 %)	NS
Undifferentiated carcinoma		1 (4 %)	0 (0 %)	1 (1 %)	NS
Histologic grade[a]	*N* (%)	(*n*=23)	(*n*=40)	(*n*=63)	
Poor		9 (38 %)	2 (5 %)	11 (24 %)	NS
Moderate		13 (54 %)	34 (85 %)	47 (69 %)	NS
Well		1 (4 %)	4 (10 %)	5 (7 %)	NS
T stage[a]	*n*/*N* (%)	(*n*=26)	(*n*=39)	(*n*=65)	
T1		0 (0 %)	6 (15 %)	6 (9 %)	0.11 (F)
T2		8 (31 %)	8 (21 %)	16 (25 %)	NS
T3–T4		18 (69 %)	25 (64 %)	43 (66 %)	NS
Lymph node positive	*N* (%)	14 (52 %)	24 (56 %)	38 (54 %)	0.81 (F)
Number positive lymph nodes	Mean (range)	1.59 (0–11)	1.79 (0–14)	1.71 (0–14)	1.0 (W)
Percent positive nodes	Mean (range)	13.1 (0–75)	9.0 (0–60)	10.6 (0–75)	0.70 (W)
Perineural invasion	(*n*/*N*, %)	21/26 (81 %)	29/41 (71 %)	50/67 (75 %)	0.40 (F)
Venous/lymphatic invasion	(*n*/*N*, %)	9/23 (39 %)	11/38 (29 %)	20/61 (33 %)	0.57 (F)
AJCC stage:[b]					
IA		0 (0 %)	5 (13 %)	5 (8 %)	0.29 (F)
IB		5 (19 %)	7 (18 %)	12 (18 %)	NS
IIA		7 (27 %)	6 (15 %)	13 (20 %)	NS
IIB		14 (54 %)	20 (51 %)	34 (52 %)	NS
III		0 (0 %)	1 (3 %)	1 (2 %)	NS
Collapsed:					
IA/IB, IIA		12 (46 %)	18 (46 %)	30 (46 %)	1.0 (F)
IIB/III		14 (54 %)	21 (54 %)	35 (54 %)	NS

SVT splenic vein thrombosis, *IPMN* intraductal papillary mucinous neoplasm, *F* Fisher's exact, *W* Wilcoxon nonparametric

[a] Patient data not available for all patients. Total number of available patients with data listed above

[b] American Joint Committee on Cancer (6th edition)

Table 3 Complications following DPS

Variable		SVT (*N*=27)	Without SVT (*N*=43)	Total (*N*=70)	*p* value
EBL[a]	Median (range)	675 (150–2,600)	250 (50–1,200)	400 (50–2,600)	<0.001 (W)
Total complications[b]	*N* (%)	13 (48 %)	24 (56 %)	37 (53 %)	0.63 (F)
P. fistula		9 (33 %)	3 (7 %)	12 (17 %)	<0.01 (F)
DGE		4 (15 %)	0 (0 %)	4 (6 %)	0.02 (F)
Intra-abdominal abscess		2 (7 %)	2 (5 %)	4 (6 %)	0.64 (F)
Cardiac		1 (4 %)	5 (12 %)	6 (9 %)	0.39 (F)
UTI		4 (15 %)	1 (2 %)	5 (7 %)	0.07 (F)
Wound infection		4 (15 %)	7 (16 %)	11 (16 %)	1.0 (F)
Sepsis		2 (7 %)	1 (2 %)	3 (4 %)	0.55 (F)
DVT/PE		2 (7 %)	1 (2 %)	3 (4 %)	0.55 (F)
Pulmonary		1 (4 %)	5 (12 %)	6 (9 %)	0.39 (F)
Length of stay (days)	Median (range)	6 (4–19)	6 (4–21)	6 (4–21)	0.12 (W)
Readmission	*N* (%)	8 (30 %)	12 (28 %)	20 (29 %)	1.0 (F)
1-year survival rate[c]	%	59 %	76 %	69 %	0.08 (F)
Median survival (months)	Median (95 % CI)	20.8 (8.9, ∞)	40.0 (22.7, ∞)	28.4 (15.6, ∞)	<0.1 (logrank)

EBL estimated blood loss, *SVT* splenic vein thrombosis, *P. fistula* pancreatic fistula, *UTI* urinary tract infection, *DGE* delayed gastric emptying, *DVT/PE* deep vein thrombosis/pulmonary embolism, *F* Fisher's exact, *W* Wilcoxon nonparametric

[a] EBL data not available for four patients. Total numbers for these variables exclude these patients

[b] Number of patients with one or more complications

[c] Only includes patients with date of surgery from Oct. 2005–Apr. 2011

greater amount of intraoperative blood loss, as well as higher pancreas-specific complications, and a trend towards reduced long-term survival. These findings suggest that SVT is a negative prognostic indicator in malignant exocrine tumors of the pancreatic body and tail.

The retrospective design of this study has inherent limitations. Using a historic database of patient information was problematic, as records in some instances were found to be incomplete. We also did not have information on postoperative adjuvant treatment regimens. The method by which we ascertained the presence or absence of SVT was by direct review of CT or MRI scans, but some preoperative scans that were completed at outside institutions were not available, and in these instances, we had to rely solely on imaging and operative reports. The relatively small number of patients in each cohort may have contributed to the inability to reach statistical significance for a number of the described differences, especially long-term survival. All of these factors limited our ability to fully and definitively elucidate the role of SVT in pancreatic exocrine cancer.

Table 4 Survival in pancreatic exocrine cancer

Variable	No. of patients alive at 1 year	Hazard ratio (95 % CI)	*p* value[a]
SVT	26 (SVT), 39 (without SVT)	1.78 (0.87, 3.64)	0.116
LN involvement[a]	30 (LN negative), 35 (LN positive)	1.84 (0.87, 3.88)	0.110
SVT adjusted for LN involvement	26 (SVT), 39 (without SVT)	1.91 (0.92, 3.94)	0.080

LN lymph node, *SVT* splenic vein thrombosis

[a] Cox regression, *N*=65

DPS has an inherently high overall rate of morbidity (roughly 50 %), so any effort to identify subsets of patients at an increased risk of surgical complication is important. SVT, which can arise in either inflammatory or neoplastic diseases of the pancreas, results in the development of numerous venous collaterals, including dilation and tortuosity of the short gastric vessels, retroperitoneal veins, the coronary vein, and veins in the lineo-colic and gastrocolic ligaments. [11] These venous collaterals are often thin walled and easily rupture during surgical dissection, accounting for the higher rates of blood loss seen in patients with SVT in our study. For these patients, surgical management at our institution has incorporated the concept of attempted early ligation of the splenic artery to control arterial inflow to the spleen, prior to any attempts to ligate or divide venous collaterals. Approaching and dividing the dilated veins first (prior to the ligation of the splenic artery) can lead to increased venous hypertension in the remaining veins and a greater risk of vein rupture and bleeding or splenic capsular bleeding as the operation proceeds. The presence of SVT may be an important indicator of

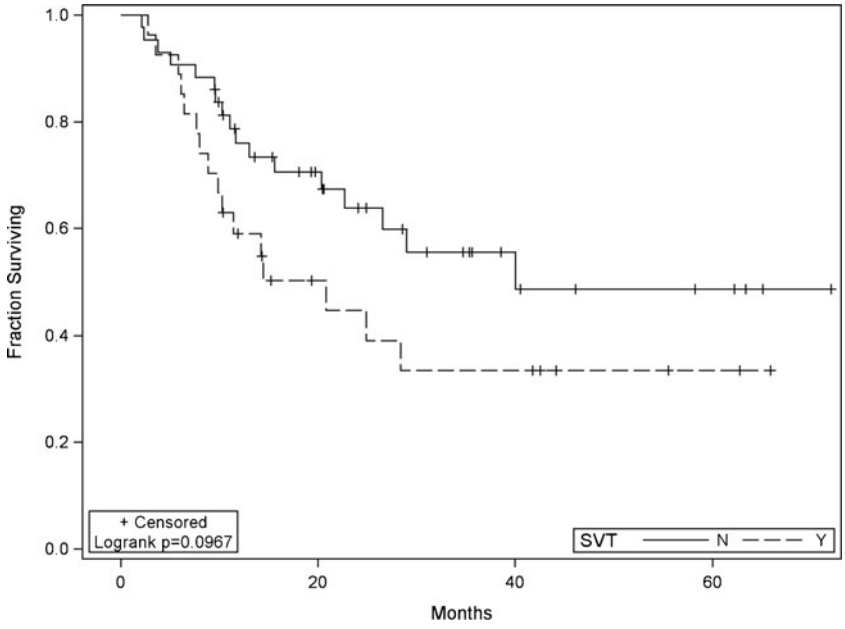

Fig. 2 Kaplan–Meier curves demonstrating a trend towards reduced survival in patients with SVT

the increased complexity and risk involved in a given DPS procedure, thus allowing the surgeon to better inform the patient of the risks of surgery and to be better prepared to handle those complexities during the operation.

Pancreas-specific complications related to DPS include PF and DGE.[12,13] Patients in our study with SVT had higher rates of both of these complications as compared to patients without SVT. It is unclear why patients with SVT would have higher rates of PF, as one would expect pancreatic gland texture in the remnant gland to be similar to that seen in patients without SVT. One could theorize that perhaps the increased rate of intraoperative blood loss and the potential for increased operative time and fluid administration in the SVT cases accounted for an increase in pancreatic gland swelling, leading to a greater friability when placing and tying sutures. The method of pancreas gland transection and remnant closure varied between cases and by surgeon, and included: (a) electrocautery with direct pancreas duct ligation and mattress suturing of the cut gland, (b) stapled pancreas transection with and without prosthetic staple line reinforcement, or (c) the addition of fibrin glue and a falciform patch (as part of a dual institution randomized controlled trial).[14] The association between PF and DGE in this study is not surprising, as DGE is often a secondary complication related to PF or intra-abdominal abscess.

We found in this study, a trend towards reduced long-term survival in the SVT cohort, despite the similarity in the pathologic findings between the SVT and non-SVT groups in terms of the rate of lymph node involvement and venous/lymphatic/perineural invasion. The two groups also had similar 90-day mortality rates, suggesting that differences in postoperative complications do not likely account for the difference on long-term survival rates. Although the reason for the apparent survival difference is not entirely clear, one may theorize that it may be related to direct tumor invasion into the splenic vein, leading to increased rates of hematogenous spread and the development of local or distant micrometastatic disease not identifiable at surgery. In addition, more aggressive cancers may be more apt to locally invade the splenic vein, and therefore, this finding may merely be a marker of aggressive tumor biology. Pathologic analysis of the tumor samples to document splenic vein invasion as opposed to encasement and cause-specific mortality information with local and distant recurrence pattern mapping would aid in answering this question. We unfortunately did not have this information available to us.

The ability to perform a margin-negative surgical resection of exocrine cancer involving the pancreas body or tail is determined by the absence of local invasion of vascular structures such as the celiac axis, superior mesenteric artery, and superior mesenteric vein (SMV).[15,16] Recent data have suggested that resection can be performed safely in some cases where there is only focal tumor infiltration of the SMV/portal vein axis or hepatic artery.[17] These tumors are termed borderline resectable. Such cases are regarded as being higher risk operations, and preoperative treatment with neoadjuvant chemotherapy and/or chemo-radiation therapy in this setting has been shown to be a sound approach.[18] It remains to be determined whether a neoadjuvent approach is beneficial for patients with resectable left-sided cancers that are associated with splenic vein thrombosis. Importantly, survival in this group of patients (median=20.8 months) is comparable to previous large series of patients with resected pancreatic cancers (left or right sided).[2,19] Therefore, SVT

should not be considered a contraindication to surgery, and medically fit patients with otherwise resectable cancers should be offered surgical management.

Conclusions

Based on our findings, we believe that SVT is an important factor in the preoperative evaluation of patients undergoing DPS for malignant exocrine disease of the pancreas. Although surgical resection can be performed safely in this setting, we found it to be associated with higher rates of intraoperative blood loss, pancreas-specific surgical complications, and a trend towards a reduced long-term survival rate. We look forward to future analyses that evaluate the significance of SVT both as a factor increasing pancreas-specific complications and in reducing postresection survival. If our data prove to be corroborated by others, then the presence of preoperative SVT would have implications for surgical planning, patient education, and possibly the introduction of neaoadjuvant treatment.

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