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Original Article

Incidence and risk factors of chylous ascites after pancreatic resection

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Abstract: Chylous ascites (CA) is a rare postoperative complication. It also occurs in pancreatic surgery and can influence the patient's prognosis after pancreatic resection. There are few studies focusing on CA following pancreatic resection. We aimed to evaluate the incidence and risk factors of CA following pancreatic resection. Patients who underwent pancreatic resection from the year 2007 to 2013 were retrospectively reviewed. The diagnosis of CA was based on the presence of a non-infectious milky or creamy peritoneal fluid greater than 100 ml/day with a triglyceride concentration ≥110 mg/dl. The incidence and possible risk factors following pancreatic resection were evaluated. In this study, 1921 patients who underwent pancreatic resection were included. 49 patients developed CA. The overall incidence was 2.6 percent (49 out of 1921). The incidence following pancreaticoduodenectomy and distal pancreatectomy was much higher (35 out of 1241, 12 out of 332, respectively). A multivariable analysis demonstrated that manipulating para-aortic area and superior mesenteric artery root area; retroperitoneal invasion; focal chronic pancreatitis and early enteral feeding were the independent risk factors for CA after pancreatic surgery. In conclusion, CA is a rare complication after pancreatic resection. Some clinicopathological factors were associated with the development of CA following pancreatic resection.

Keywords: Chylous ascites, risk factor, pancreatic resection

Introduction

Complications following pancreatic resection include bleeding, anastomotic leak, pancreatic fistula, and delayed gastric emptying. Some studies have reported that chylous ascites (CA) also occurs following pancreatic surgery, and moreover, that CA is one of the factors influencing the patient's prognosis following pancreatic resection [1-3]. CA is the pathologic leakage of lymphatic fluid rich in triglycerides into the peritoneal cavity [4, 5]. It may be produced by malignancy, surgery, radiotherapy, tuberculosis, filariasis, trauma, cirrhosis or nephrotic syndrome, and congenital abnormalities of the lymphatic system [1]. CA is rich in nutrients. It may lead to malnutrition, dehydration, electrolyte imbalance and delayed wound healing. Moreover, since CA contains lymphatic fluid, which is rich in lymphocytes and immunoglobulins, severe and long-term chylous leakage may cause hypoimmunity [1]. Therefore, severe infection or even death may occur due to sepsis [6].

Chyle is transported in the lymphatic vessels, which drain the abdominal viscera and join the ascending lymphatic trunks. These trunks ascend and converge constantly and form a saccular dilatation named the cisterna chili [4]. Postoperative CA is related to a failure of the lymphatic system, which may be caused by an obstruction, rupture, or unsealing of lymph nodes or lymphatic ducts. In general, CA is a very uncommon post-operative complication. CA following a surgical procedure is commonly seen after retroperitoneal lymph node dissection, radical operations for carcinoma of the stomach, abdominal aortic aneurysm surgery; and liver transplantation [6-12]. The data on CA after pancreatic resection have been derived from limited case reports or studies with small sample sizes [1, 3, 13-20]. Most of those case reports and studies focused on the management of postoperative CA. The studies related to the incidence and risk factors of CA following pancreatic resection were rarely found out in literature.

Table 1. Incidence of CA after pancreatic resection

Type of operation	Patients (n=1921)	chylous ascites (n=49)	Incidence (percent)
Laparoscopic operation			
PD	114 (5.9)	3 (6.1)	2.6
DP	38 (2.0)	1 (2.0)	2.6
Open operation			
PD	1127 (58.7)	32 (65.3)	2.8
DP	294 (15.3)	11 (22.4)	3.7
Total pancreatectomy	113 (5.9)	0	-
Partial pancreatectomy			
DPRHP	192 (10.0)	2 (4.1)	1.0
Others	43 (2.2)	0	-

Note: Values are n (%). CA, chylous ascites; PD, pancreaticoduodenectomy; DP, distal pancreatectomy; DPRHP, duodenum preserving pancreatic head resection.

We conducted this study to investigate CA following pancreatic resection in a large patient population. Our study determined the incidence of CA following pancreatic resection and examined the possible risk factors for onset.

Materials and methods

Patients

We retrospectively reviewed patients who underwent pancreatic resection, from 2007 to 2013 at the West China Hospital of Sichuan University, Sichuan Province, China. All patients were evaluated pre-operatively with medical history; physical examination; serum laboratory tests; computed tomography or magnetic resonance imaging of the pancreas; electrocardiographic examination; and chest radiograph. All patients gained adequate postoperative drainage. The diagnosis of CA was based on the presence of a non-infectious milky or creamy peritoneal fluid ≥100 ml/day with a triglyceride concentration ≥110 mg/dl [1, 2, 21].

Methods

All data were retrospectively collected in a database at the West China Hospital of Sichuan University. The incidence of CA following pancreatic resection was evaluated according to the operative method, such as pancreaticoduodenectomy (PD) or distal pancreatectomy (DP). To identify the risk factors of CA following pancreatic resection, the patients were divided into two groups: with or without CA. The risk factors were evaluated throughout the perioperative period (pre-operation, intra-operation and post-operation). Additionally, the pathological findings were considered during the evaluation of possible risk-factors. In our study, the term 'manipulation of the para-aortic area' or 'superior mesenteric artery root area' indicates the exposure of the aortic or root of the superior mesenteric artery wall, such as during para-aortic lymph node dissection, coeliac axis resection or para-superior mesenteric artery lymph node dissection [1, 22]. Those procedures were performed to achieve a margin free from the carcinoma [1, 22].

Statistical analysis

The statistical analyses were performed using the SPSS software version 16.0 (SPSS Inc., Chicago, IL, U S A). We used the the Chi-square test or Fisher's exact test (to compare frequencies of categorical variables among groups) and the Mann-Whitney U test (to compare continuous variables). The prognostic factors related to CA were evaluated by a univariate logistic regression, and a multivariable analysis determined the independent risk factors. Specific statistical analysis methods will be indicated in the tables and figures below. A *p*-value of $P \leq 0.05$ was considered to be significant.

Results

During the year 2007 to 2013, 1921 patients underwent pancreatic resection (laparoscopic operation, 152; open operation, 1769). In total, 49 patients developed CA following pancreatic resection (**Table 1**). The overall incidence of postoperative CA was 2.6 percent (49 out of 1921 patients). The incidence was dependent upon different pancreatic resections. Overall, the incidence of developing postoperative CA after an open operation or a laparoscopic operation was not significantly different (2.5 percent (4 out of 152) vs. 2.6 percent (45 out of 1769)). The incidence of CA following PD was 2.8 percent (32 out of 1127) with an open operation and 2.6 percent (3 out of 114) with a laparoscopic operation. There was a higher incidence of postoperative CA following a DP surgery (3.7 percent (11 out of 294) with an open operation and 2.6 percent (1 out of 38) with a laparoscopic operation). Postoperative CA has not yet been observed following a total pancreatectomy.

Table 2. Risk factors of CA following pancreatic surgery

	CA (n=49)	No CA (n=1872)	*P* value§
Age (years)	59 (35-79)	58 (17-84)	0.977†
Sex (Male: Female)	32:17	1186:686	0.881
Pre-operative factors			
Primary diagnosis (cancer or not)	48:1	1678:194	0.055
Intra-operative factors			
Surgical management			
Operation method (Laparoscopic: Open)	4:45	162:1710	1.0
Operation type			
PD	32 (65.3)	1060 (56.6)	0.245
Pylorus-preserving PD	3 (6.1)	146 (7.8)	1.0
DP	12 (24.5)	320 (17.1)	0.181
DPRHP	2 (4.1)	190 (10.1)	0.226
Manipulation area			
Para-aortic area	30 (61.2)	487 (26.0)	<0.001
Superior mesenteric artery root area	20 (40.8)	428 (22.9)	0.006
NO. of lymph nodes harvested	10 (0-23)	10 (0-24)	0.249†
Vascular resection	21 (42.9)	523 (27.9)	0.035
Pathological findings			
Tumor size (diameter, mm)	30 (10-40)	30 (10-50)	0.228†
Malignancy as pathological diagnosis	47 (95.9)	1575 (84.1)	0.026
Lymph node metastases	30 (61.2)	844 (45.1)	0.029
No. of positive nodes	2 (1-9)	2 (1-10)	0.390†
Retroperitoneal invasion	33 (67.3)	845 (45.1)	0.002
Focal chronic pancreatitis	11 (22.4)	209 (11.2)	0.022
Postoperative factors			
Somatostatin	40 (81.6)	1478 (79.0)	0.726
Enteral feeding time (days)	3 (3-9)	4 (3-17)	<0.001
Operation day 3	36 (73.5)	779 (41.6)	<0.001
Before operation day 4	44 (89.8)	1307 (69.8)	0.001
Before operation day 5	47 (95.9)	1682 (89.9)	0.226

Note: Values are n (%) or median (ranges). CA, chylous ascites; PD, pancreaticoduodenectomy; DP, distal pancreatectomy; DPRHP, duodenum peserving resection of head of pancreas; §, Chi-square test or Fisher's exact test; †, Mann-Whitney U test.

Among the 1921 patients who underwent pancreatic resection, 1575 resections were performed due to malignancies, and pancreatic cancer was the primary malignancy (878 cases). The other malignancies were bile duct cancer (288), carcinoma of the ampulla of vater (137), and other (272). Among the 49 patients who developed CA following pancreatic resection, 47 underwent resection due to malignancy (pancreatic cancer, 30; bile duct cancer, 11; carcinoma of the ampulla of vater, 3; intraductal papillary mucinous carcinoma, 3). Two of the 215 patients without malignancies developed postoperative CA (intraductal papillary mucinous adenoma, 1; neuroendocrine tumor, 1).

We evaluated the general condition and clinical and pathological characteristics of the patients who did or did not develop CA after pancreatic resection (**Table 2**). The frequency of CA was significantly different when manipulating certain areas, such as the para-aortic area or superior mesenteric artery root area. In addition, vascular resection, malignancy as pathological diagnosis, lymph node metastases, retroperitoneal invasion, focal chronic pancreatitis and the time to start enteral feeding were significantly different between patients with or without CA.

The initiation of enteral feeding was significantly earlier in the patients with CA (3 days in median, range from 3 to 9 days) compared to the patients without CA (4 days in median, range from 3 to 17 days, P<0.001). The incidence of CA was significantly increased in the early enteral feeding patients compared to the later enteral feeding patients (3.3 percent vs. 0.9 percent, respectively, P=0.001, Fisher's exact test). In the present study, early enteral feeding indicates that the initiation of enteral feeding was on postoperative day 3 or 4 (**Table 2**).

A multivariable analysis demonstrated that manipulating para-aortic area and superior mesenteric artery root area; retroperitoneal invasion; focal chronic pancreatitis and early enteral feeding were independent risk factors for CA after pancreatic resection (**Table 3**).

Table 3. Logistic regression analysis of prognostic factors related to CA following pancreatic resection

Prognostic factor	Univariable analysis		Multivariable analysis	
	OR (95% CI)	P	OR (95% CI)	P
Manipulating para-aortic area	4.49 (2.51-8.05)	<0.001	4.54 (2.41-8.52)	<0.001
Manipulating superior mesenteric artery root area	2.33 (1.30-4.16)	0.004	2.75 (1.47-5.16)	0.002
Vascular resection	1.94 (1.09-3.44)	0.024	1.62 (0.88-2.99)	0.125
Malignancy as pathological diagnosis	4.43 (1.07-18.34)	0.040	0.67 (0.13-3.52)	0.636
Lymph node metastases	1.92 (1.08-3.44)	0.028	1.70 (0.91-3.12)	0.097
Retroperitoneal invasion	2.51 (1.37-4.60)	0.003	1.98 (1.03-3.83)	0.041
Focal chronic pancreatitis	2.30 (1.16-4.58)	0.017	2.59 (1.25-5.37)	0.011
Enteral feeding time	0.39 (0.25-0.61)	<0.001	0.39 (0.25-0.61)	<0.001

Note: CA, chylous ascites; OR, odds ratio; CI, confidence interval.

Discussion

Since CA was first reported in 1694, it has been studied in many case reports and reviews [4]. In general, it is a rare postoperative complication. However, the incidence is increasing following abdominal surgery as a consequence of more aggressive abdominal surgeries and prolonged survival times of patients with cancer [8]. Following hepatopancreatobiliary surgery, approximately 1.0 percent patients develop CA [1]. The incidence of CA following pancreatic resection is largely unknown but has been reported to be 1.3 to 6.7 percent [1-3, 23]. In the present study, the overall incidence is 2.6 percent (49 out of 1921). The incidence of CA after PD was 2.8 percent in our study, which is in accord with others studies (range from 1.8-11.0 percent) [1-3, 18, 21]. The present study also found that the incidence of CA was the highest following DP (3.6 percent, 12 out of 332 patients). The incidence of CA after DP in other reports ranges from 3.4 to 12.0 percent [1, 2, 19, 21], which is similar to the present study.

Aalami OO [4] and colleagues concluded that the causes of CA are as follows: (1) obstruction: lymph vessels are obstructed at the base of the mesentery or the cisterna chili cause dilation of the lymphatics on the walls of the bowel or in the mesentery and cause the rupture of chyle; (2) traumatic injury: surgical injury of the cisterna chyli or its major lymphatic tributaries results in direct leakage of chyle through a fistula of lymphatic vessels; (3) exudation: extensive lymph node dissection leads to contracture of the surrounding tissue, which causes the lymphatic return to get blocked and results in the exudation of chyle through the lymphatic endings. The mechanism of the formation of postoperative CA is related to a failure of the lymphatic system, which may be caused by an obstruction, rupture, or unsealing of the lymph nodes or lymphatic ducts. The reasons for developing CA following pancreatic resection have not yet been clearly identified. In our study, manipulating the para-aortic area and superior mesenteric artery root area; retroperitoneal invasion; focal chronic pancreatitis. Early enteral feeding were the independent risk factors for developing CA after pancreatic resection.

Traumatic injury and obstruction of the lymphatic system are the primary causes of postoperative chylous leakage [7, 8]. CA following pancreatic resection likely results from both causes. Because the cistern chili is located anterior to the first and second vertebrae, which is at the same level as the pancreas, injury is likely to occur during pancreatic resection [24]. Extensive lymph node dissection or dissection of the retroperitoneum increases the risk of surgical damage of the cistern chyli or its major tributaries, and consequently, the occurrence of CA [2, 7, 18]. Thus, the manipulation of specific areas during operation and extensive lymph node dissection are possible risk factors for its onset. Kuboki S [1] and colleagues found that manipulating the para-aortic area and retroperitoneal invasion of tumor were the two independent risk factors for developing CA after pancreatic resection, which agrees with our study. In addition, we found that manipulation of the superior mesenteric artery root area was an independent risk factor. No study has reported this result before. Multiple studies have reported that extended lymph node dissection during various surger-

ies, such as neck dissection, esophagectomy and gastrectomy were related to an increased risk of chylous leakage [25-27]. Yol et al., [27] found that a D3 extended lymphadenectomy during gastric carcinoma surgery led to a higher incidence of chylous leakage. One of the possible reasons for this increase is that the extensive loss of lymph nodes following resection prohibits adequate drainage causing congestion of the upstream lymph ducts. Assumpcao [2] and colleagues found that harvesting a high number of lymph nodes at the time of pancreatic resection was associated with a higher risk of chyle leak. However, in the present study, no significant difference was observed related to this factor. They also reported that significantly more patients with postoperative CA underwent vascular resection and had lymph node metastases and dissection. But lymph node metastases and vascular resection was not the independent risk factors of CA following pancreatic resection in present study.

Niels A van der Gaag [18] found that focal chronic pancreatitis was independently associated with the development of isolated CA. They suggested that the association between CA and chronic pancreatitis might be due to the extensive surgery required by these patients (increasing the surrounding infiltration or inflammation), which may cause the risk of chyle leak highly increased. Another hypothesis is that a long-term inflammatory process results in the congestion of lymph fluid, which subsequently causes lymph duct enlargement, which is more likely to be damaged during operations. The present study also found that a pathologic diagnosis of focal chronic pancreatitis was an independent risk factor of CA following pancreatic resection, which supports their hypothesis.

The abdominal lymph fluid flow increases quickly after an oral diet with fat. If there is a traumatic injury or an obstruction of the lymphatic system, lymph fluid will leak out of the lymph vessels, and the volume of lymph fluid leakage will increase following the initiation of enteral feeding. Shortening the fasting period following the operation may increase the risk of chylous leakage. Noji T [19] and colleagues found that early enteral feeding may contribute to chyle leakage following PD. The study of Kuboki et al [1], also revealed that the early introduction of enteral feeding is closely related to postoperative CA. Our study also found early enteral feeding to be an independent risk factor. Malik et al [3], suggested that the mechanism of early enteral feeding causing chyle leakage may be due to the lipid content of the food, which may keep the visceral lymphatic channels open that have been divided as part of the standard resection leading to the persistent chyle leak. Noji et al [19]. suggested that the visceral lymphatic channels may remain open for at least 4 days postoperatively. The present study supports their viewpoint. We found that enteral feeding initiated at postoperative day 3 or 4 increased the risk of CA. There is little doubt that enteral nutrition has advantages over total parenteral nutritional support [19]. Because early enteral feeding enhances body immunity and reduces the risk of infection, it should be introduced after pancreatic surgery [28, 29]. However, it should be noted that early enteral feeding may significantly increase the incidence of CA. Therefore, more studies are needed to establish the appropriate time to initiate enteral feeding or to find a better diet for early enteral feeding following pancreatic surgery. The long chain triglycerides are absorbed through the lymphatic vessels of the gastrointestinal tract; and the medium chain triglycerides (MCT) are absorbed straight into the intestinal cells and transported directly to the liver via the portal vein in the form of free fatty acids and glycerol [30-32]. Therefore, feeding with a MCT diet other than ordinary diet could be used for early enteral feeding in patients who underwent pancreatic resection to avoid the increasing risk of CA. Still, it needs further research.

In conclusion, CA is a rare complication (2.6 percent) after pancreatic resection. The incidence of CA was the highest following a DP (3.6 percent). The independent risk factors for developing CA included manipulating the paraaortic area and superior mesenteric artery root area; retroperitoneal invasion; focal chronic pancreatitis; and early enteral feeding. More studies are needed to establish an appropriate time to initiate enteral feeding or to identify a better diet (a MCT diet may be possible) for early enteral feeding following pancreatic surgery.

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Disclosure of conflict of interest

None.

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