Endovascular Stenting for Non-Traumatic Celiac Artery Stenosis Following Pancreatoduodenectomy

Si-Yuan Yao, MD, Shintaro Yagi, PhD, and Hiroyuki Ueda, PhD

Postoperative celiac artery stenosis (CAS) rarely occurs in the absence of vascular injury or pseudoaneurysm after pancreatoduodenectomy (PD). Because of its low incidence, the optimal treatment for non-traumatic postoperative CAS is unknown. Here, we show a case of CAS possibly due to exacerbated median arcuate ligament compression after PD. The purpose of this report is to describe this rare complication and its successful endovascular treatment with review of literatures.

Keywords: pancreatoduodenectomy, celiac artery stenosis, stent

Introduction

Ischemic complication after pancreatoduodenectomy (PD) can occur because of stenosis or intraoperative injury to the celiac artery (CA) or superior mesenteric artery (SMA), and may result in anatomic dehiscence, liver failure, or bowel necrosis. All of these complications have a high rate of mortality. Although the incidence of ischemic complications has been poorly studied, a recent single-center retrospective study reported the incidence as 1.1% (6 out of 545 patients; 4 due to intraoperative hepatic artery injury, 1 due to SMA atherosclerotic stenosis and 1 due to CA fibromuscular dysplasia) with 83% mortality. While intraoperative injury is the most common cause, other latent abnormalities in a patient’s mesenteric arterial vasculature can cause stenosis, including severe atherosclerotic disease, compression by the median arcuate ligament (MAL), fibromuscular dysplasia, and mesenteric vasculitis. Because of its rare incidence and few reported cases, the optimal treatment for non-traumatic postoperative celiac artery stenosis (CAS) is unknown. Here, we present a case of CAS possibly caused by exacerbated MAL compression after PD. The purpose of this report is to describe this rare complication and its successful treatment.

Case Report

A 51-year-old Asian woman with abnormally elevated tumor markers (serum CA19-9 of 371 U/ml, SPAN-1 of 63 U/ml) was referred to our outpatient department for further evaluation. She had no significant past medical history and her physical examination was unremarkable. Abdominal ultrasound and contrast enhanced abdominal computed tomography (CT) scan revealed a 25-mm solid neoplasm in the body of the pancreas. Although there were no signs of distant metastasis, direct invasion to the portal vein was suspected. Subtotal stomach-preserving PD, including portal vein reconstruction and D2 lymphadenectomy, was performed based on the preliminary diagnosis of pancreatic cancer. No anatomical variations of the celiac trunk or mesenteric vessels and relative branches were detected during preoperative radiological imaging or during surgery. The result of preoperative CT is shown (Fig. 1). The operation was successfully performed without any vascular injury. A clamp test of the gastroduodenal artery (GDA) did not show ischemic alterations of the liver, stomach or spleen. The operative time was 449 minutes and blood loss totaled 515 ml. At definitive histological examination, a ductal pancreatic adenocarcinoma was confirmed (pT4 N1 M0).

During the postoperative course, laboratory tests showed abnormally elevated hepatic enzymes on postoperative day (POD) 2, including aspartate aminotransferase of 1160 U/L and alanine aminotransferase of 1534 U/L. Contrast enhanced abdominal CT revealed multiple liver and spleen infarctions (Figs. 2a and 2b). The reconstructed portal vein, proper hepatic artery, and splenic artery were patent. However, a stenotic celiac arterial root was evident on CT angiography, without pseudoaneurysm (Fig. 2c). Emergency celiac arteriography was performed and the stenotic celiac arterial root was confirmed (Fig. 2d). To maintain hepatic and splenic arterial flow, percutaneous endovascular stenting of the celiac arterial root was performed (Fig. 3a). The postintervention angiogram showed successful stent deployment without immediate complications (Fig. 3b).
transluminal angioplasty (PTA) was performed, followed by placement of a bare metal stent (Express™ SD, Boston Scientific, MA, USA) (Figs. 2e and 2f). A stent with 6 mm in diameter and 14 mm in length was used. No vascular adverse events were encountered during or after the procedure. The patient was discharged on POD 11 without other complications. At 30 months follow-up, CT showed a patent stent graft. The patient is currently taking aspirin for anticoagulation.

Discussion

According to past reports, CAS is found in 4% to 10.5% of patients undergoing PD.20 A preoperative multidetector CT with routine three-dimensional arterial reconstruction is now the standard method to detect arterial anatomic variants and stenosis. CT was able to characterize the etiology of CAS with 58.3% sensitivity, 98.2% specificity, and 95.6% accuracy, while a 35% failure rate was reported for
stenting for postoperative celiac artery stenosis

Once CAS is diagnosed, the decision to intervene should be made. It is generally accepted that asymptomatic or incidentally discovered CAS does not warrant intervention. Smith et al. reported that stenosis of <60%, as seen on CT, was not associated with an increased risk of postoperative or perioperative complications in patients undergoing PD. The extreme variability of pancreatic and peripancreatic collateral vascularization and the common existence of collateral pathways from the dorsal pancreatic artery or from a replaced right hepatic artery may explain why hepatic blood flow can be maintained even after divisions of the GDA in cases of CA stenosis or occlusion. Ouaissi et al. mentioned that decompression procedures were not necessary for patients with arterial stenosis of <50% before or during PD, while stenosis >50% needed treatment by revascularization surgery and/or endovascular stenting. In addition to the degree of stenosis, post-stenotic dilation, vessel collateralization, gastroduodenal artery dilation, and pancreaticoduodenal artery aneurysm formation might indicate consideration for surgical management, even if asymptomatic. Celiac artery stenosis can also be suspected and diagnosed during PD. When ischemic disorders to the supramesocolic organs appear after temporary closure of the GDA, the surgeon should suspect the presence of CAS. Because compression by the MAL is the most common cause of severe CAS (along with atherosclerosis accounting for nearly 90% of cases), CAS with positive GDA clamping test would be an indication for MAL division during PD. The median arcuate ligament is a connective tissue ligament that runs transversely anterior to the L1 vertebral body and ventral to the abdominal aorta, proximal to the origin of the celiac trunk. In 10% to 24% of normal asymptomatic individuals, the MAL may cause compression and downward angulation of the CA. Median arcuate ligament syndrome (MALS) is defined as ischemia in the gastrointestinal system due to compression of the CA by the MAL. As there have been no accepted classifications of CAS caused by MAL compression, patient selection remains a challenge.

In our case, the patient was a previously healthy middle-aged woman. The existence of severe atherosclerotic disease was ruled out and only a slight “fish hook” appearance was observed in retrospective review of the preoperative CT, suggesting mild compression by the MAL (Fig. 1b). The degree of stenosis was apparently <50% and there were no signs of adverse hemodynamics during the arterial reconstruction, like acquired pancreaticoduodenal collateral pathways or post-stenotic dilation. In addition, the intraoperative GDA clamping test was negative. According to previous reports, surgical intervention was not deemed necessary before or during PD in this case. However, postoperative CAS complicated by organ infarctions did occur (Fig. 2c). Not only diagnosis, but also the judgment on

<table>
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<th>No</th>
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<th>Sex</th>
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<th>Intraoperative decompression</th>
<th>Preoperative diagnosis of CAS</th>
<th>Organ infarction interaction</th>
<th>Date of diagnosis of CA stenosis</th>
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PD: pancreaticoduodenectomy; CAS: celiac artery stenosis; CA: celiac artery; AdenoCa: adenocarcinoma; POD: postoperative day; MAL: median arcuate ligament; SMA: superior mesenteric artery.
whether the decompression procedure is necessary before or during operation is still difficult.

While there are many reports on the evaluation and management of CAS before PD, few exist regarding managing postoperative CAS. A literature search of the PubMed database between 1950 and 2015 retrieved only three other cases of postoperative CAS after PD (Table 1). Although liver infarction was detected in all cases, massive spleen infarction is reported here for the first time. Among the reported cases, two patients underwent inadequate MAL division for preoperatively diagnosed MALS and had re-stenosis, followed by complications with poor outcomes.1,5) Only one case was identified in which the patient had symptomatic CAS after PD caused by undiagnosed MAL compression. Conservative treatment was chosen, including total parenteral nutrition, antibiotic therapy, and non-steroidal anti-inflammatory drugs, without surgical or endovascular intervention, and the patient recovered well.3) The author concluded that lymphadenectomy around the CA or the prolonged bent back position of the patient during surgery may have led to temporary MALS with exacerbation of a non-significant CAS. If the stenosis is due to a temporary edema or compression, conservative treatment may be effective. However, the possibility of further infarctions or organ dysfunction should be considered when acute stenosis without collateral pathway development occurs. Because of the paucity of previous reported cases, there was no evidence to suggest conservative treatment for this rare condition, leading us to select endovascular stenting to maintain arterial flow. As manipulation of the CA might exacerbate stenosis and inadequate MAL division could result in re-stenosis, surgical intervention was thought to be too invasive and insecure in this case. Fortunately, the clinical course was uneventful and the stent patency has been maintained for more than 2 years. To the best of our knowledge, this is the first reported case of successful stent placement performed on a patient with CAS secondary to undiagnosed MAL compression following PD.

The utility of endovascular stenting for treatment of CAS has been reported. Immediate success rate of 81% to 96% was reported, with a low incidence of complications and a 6-month patency rate of 92%.3) This minimally invasive procedure is thought to be an alternative especially for ostial stenosis secondary to arteriosclerosis. As for the treatment of MALS, the adaptation of stenting is still controversial. Sullivan et al. described PTA as traumatic for the vessel and claimed that it would render the vessel wall more susceptible to collapse by intense extrinsic pressure, resulting in exaggeration of MAL.5) Delis et al. argued that long-term stent patency is compromised by slippage and mechanical stress, and suggested that stent insertion into the vessel must only be considered as a temporary treatment, unless primary arcuate release is done first.10) In our case, as the severe stenotic lesion was located in the root of CA with short distance, a balloon expandable stent was selected in anticipation for its stronger expanding force.

Conclusions
Postoperative CAS following PD occurs very rarely, even with prudent preoperative evaluation and surgical procedure. To the best of our knowledge, this is the first reported case successfully treated by endovascular stenting. Although the application is still controversial, prompt management with endovascular stent placement can be a safe and effective procedure.

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Author Contributions
Study conception: SY
Data collection and analysis: SY
Investigation: SY, HU
Writing: SY
Critical review and revision: all authors
Final approval of the article: all authors
Accountability for all aspect of the work: all authors

References


