An innovative way of managing coeliac artery stenosis during pancreaticoduodenectomy

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ABSTRACT

Coeliac artery stenosis (CAS) is rarely of consequence owing to rich collateral supply from the superior mesenteric artery via the pancreatic head. Pancreaticoduodenectomy (PD) in CAS disrupts these collaterals, and places the liver, stomach and spleen at risk of ischaemia.

A 56-year-old man presented with a 3-week history of obstructive jaundice. Computed tomography revealed an operable periaampullary tumour with CAS due to compression by the median arcuate ligament with multiple collaterals in the pancreatic head and a prominent gastroduodenal artery (GDA). Following unsuccessful coeliac axis endovascular stenting, a PD was performed. Intraoperative median arcuate ligament release failed to restore good flow in the common hepatic artery (CHA) and splenic artery (SpA).

A decision was made to use the left gastric artery (LGA) for arterial reconstruction, disconnect it from the stomach with its origin intact and anastomose it to the supraceliac aorta. Doppler ultrasonography with a GDA clamp confirmed good filling of the CHA and SpA via the LGA. The GDA was ligated and the PD completed. The patient had an uneventful recovery except for a biochemical pancreatic leak and was discharged on day 10.

CAS during PD (confirmed by a decrease in CHA flow with a GDA clamp) requires an additional procedure to restore blood flow to the liver, stomach and spleen. Anastomosing the LGA to the supraceliac aorta is a simple reconstruction technique for achieving this.

KEYWORDS

Coeliac artery stenosis – Pancreaticoduodenectomy – Arterial reconstruction methods

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Present in 7–25% of routine abdominal angiographies, coeliac artery stenosis (CAS) is rarely of clinical consequence owing to rich collateral supply from the superior mesenteric artery (SMA) via collaterals in the pancreatic head and the gastroduodenal artery (GDA) or the arc of Buchler.¹ CAS is seen in 2–11% of patients undergoing pancreaticoduodenectomy (PD).^{2,3} PD in patients with CAS puts the upper abdominal organs at risk of ischaemia owing to ligation of the GDA and disruption of these collaterals, and mandates an additional procedure to restore flow. We describe a patient with a periaampullary carcinoma and CAS who underwent PD. Blood flow was restored with an innovative technique.

Case history

A 56-year-old man presented with a 3-week history of progressive obstructive jaundice with low grade cholangitis. Computed tomography (CT) showed an operable

periaampullary tumour with CAS from median arcuate ligament (MAL) compression with multiple collaterals at the pancreatic head and a prominent GDA reformatting the common hepatic artery (CHA) and splenic artery (SpA) (Fig 1). He underwent endoscopic biliary stenting and an unsuccessful attempt at coeliac axis endovascular stenting before being listed for surgery.

Intraoperatively, the coeliac axis was exposed and MAL release was performed. A clamp on the GDA showed feeble flow in the CHA and SpA, confirmed by Doppler ultrasonography. After considering multiple options for restoring flow (such as coeliac axis reimplantation and an autologous bypass graft to the CHA), a decision was made to use the left gastric artery (LGA) as it is usually a prominent vessel with a natural communication with the CHA and SpA. The LGA stem was disconnected from the lesser curvature of the stomach, keeping its origin intact, and anastomosed to the supraceliac aorta (Fig 2). The arterial arcade along the lesser curvature was preserved. Doppler ultrasonography

Figure 1 Sagittal section of computed tomography angiography showing coeliac artery stenosis (arrow) due to compression by median arcuate ligament (A), and three-dimensional reconstruction showing extensive collaterals (arrowheads) between the superior mesenteric artery (#) and the coeliac artery branches (*) with a prominent gastroduodenal artery (arrow) (B)

A = aorta; CA = coeliac artery; CHA = common hepatic artery; LGA = left gastric artery; SpA = splenic artery

Figure 2 Intraoperative photograph showing a completed anastomosis of the LGA to the supraceliac aorta (white arrows) after taking control of all branches (arrowheads). The site from where the LGA was disconnected from the lesser curvature of the stomach can be seen (yellow arrow).

with a GDA clamp confirmed good filling of the CHA and SpA via the LGA. The GDA was ligated and a classical PD was completed.

Postoperatively, the patient had a biochemical pancreatic leak and high drain output, managed with diuretics. Apart from this, he had an uneventful recovery with normal liver

enzymes and was discharged on day 10. The histopathology results showed a moderately differentiated ampullary adenocarcinoma with positive lymph nodes (2/7) and extra-nodal deposits. The patient is undergoing adjuvant chemotherapy.

Discussion

CAS is an important condition to be looked for when evaluating patients for PD. The aetiology of CAS can be extrinsic or intrinsic, the most common being MAL compression and arteriosclerosis, contributing to approximately 90% of cases.⁴ PD in these patients usually disrupts the collateral pathways from the SMA, leaving the upper abdominal organs such as the liver, spleen and stomach at a significant risk of ischaemia after surgery. This leads to a high incidence of postoperative ischaemic complications (eg liver necrosis/abscess, gastric necrosis, lesser curvature perforation and disruption of multiple anastomoses [hepaticojejunal or gastrojejunal in addition to pancreaticojejun]),^{2,5} with a prolonged hospital stay, reoperation and a mortality rate of up to 40%.⁵

Nowadays, the diagnosis of CAS is made by multidetector CT performed for the routine preoperative evaluation and confirmed by CT angiography with sagittal reconstruction of the visceral arteries. Typical findings include narrowing at the coeliac axis origin with its hooked appearance, thickened crura and MAL, calcification at the coeliac origin, prominent collaterals at the pancreatic head and a prominent GDA, inferior pancreaticoduodenal artery or dorsal pancreatic artery. Mesenteric angiography demonstrates retrograde filling of the CHA via the GDA. Intraoperatively, a decreased flow in the CHA with a GDA clamp confirms significant CAS.

PD in patients with CAS mandates careful planning to avoid the above mentioned ischaemic complications. Preoperative endovascular stenting may be successful in a few but carries the risk of stent crushing due to persistent external compression and has an inferior long-term patency compared with arterial reconstruction.⁴ In MAL compression, MAL release can restore good CHA flow in 60–70% cases.^{3–5} However, when this fails to restore blood flow (as in our case) or in arteriosclerosis with severe/complete occlusion, arterial reconstruction is the only way to restore adequate blood flow to the upper abdominal organs. PD with preservation of the GDA or collateral pathways has been described but most centres have limited experience of this technique and fear compromise in oncological principles when performed in cases other than benign or small malignant lesions.⁵ Consequently, planning for PD in patients with CAS should always involve preparation for a possible arterial reconstruction.

Arterial reconstruction may be carried out either as an artery-to-artery anastomosis (eg inferior pancreaticoduodenal artery to GDA stump, middle colic artery to GDA stump, middle colic artery to CHA, coeliac axis to aorta reimplantation) or as an arterial bypass procedure (aorta to CHA/coeliac axis, SpA to iliac artery) using autologous or synthetic grafts. Autologous grafts are usually preferred owing to a

potentially contaminated operative field (bile, gastric contents etc) or a possible postoperative pancreatic anastomotic failure with infected collections in the vicinity. The most commonly used grafts are the saphenous vein, inferior mesenteric vein and internal jugular vein.

We performed arterial reconstruction by anastomosing the LGA to the supraceliac aorta. This restores flow to both the CHA and SpA as the LGA is naturally connected to these arteries. After LGA disconnection, the gastric blood supply is through the left gastroepiploic artery, which feeds the arcade along the greater curvature of the stomach. In order to prevent ischaemia to the lesser curvature, the arterial arcade was preserved here. This method does not involve any additional dissection as the supraceliac aorta already lies exposed after MAL release, it involves only a single anastomosis and it does not require venous graft harvesting. As a result, it decreases the operative time and complexity of the procedure. Furthermore, it does not compromise oncological clearance in PD. The technique is applicable in both classical and pylorus preserving PD.

Conclusions

Surgeons must be wary of CAS when performing PD, confirmed by decreased CHA flow with a GDA clamp. Ligation of the GDA in such cases causes significant ischaemia to the

liver, stomach and spleen. If MAL release does not restore flow, arterial reconstruction is needed. Anastomosing the LGA to the supraceliac aorta is a simple technique for restoring blood flow to these organs.

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