Because of the increased use of pacemakers and central venous catheters for access or treatment purposes, thrombotic causes are increasing [4]. However, intravascular thrombosis leading to SVCS is still extremely rare.

We present a patient with SVCS due to unexplained thrombosis. The patient had no hypertension or diabetes and no obvious abnormal parameters in blood tests, other than a level of protein C that was lower than normal. It is entirely possible that the decreased level of protein C contributed to the pathology [5]. Another cause could have been that the patient was a smoker. Smoking is known to be a secondary risk factor for SVCS.

There are no exact guidelines for the clinical management of SVCS. The treatments include long-term anticoagulation, thrombolysis, percutaneous transluminal balloon angioplasty, stent implantation, and open surgical reconstruction. Surgical intervention has been the mainstay for treatment of benign SVCS [6]. However, Rizvi and colleagues [7] retrospectively reviewed a series of 70 patients with benign SVCS between 1983 and 2006. Of these patients, 93% were treated by open surgical reconstruction (OSR) or endovascular repair (EVR), which induced a significant relief from the symptoms [7]. These interventions are similarly effective for the short-term and midterm, whereas OSR is most effective for obtaining a long-term effect [7, 8].

To date, EVR is the first-line treatment for SVCS with a benign etiology because it is less invasive and is associated with lower morbidity by thrombosis [7]. Our patient was therefore treated by percutaneous transluminal balloon angioplasty and placement of an intravascular stent. Unfortunately, the patient presented after again 3 months later with thrombosis in the stent in the SVC. This recurrence was intervened on by surgical reconstruction. Postoperative anticoagulation with warfarin (international normalized ratio of 2.5 to 3.0) and aspirin (100 mg/d) [6] was effective for a period of 2 years of postoperative follow-up.

In conclusion, our report demonstrates that surgical management is an excellent choice for SVCS after failed EVR. EVR of benign SVCS was effective in the short-term and midterm and did not adversely affect subsequent OSR. After both EVR and OSR, long-term anticoagulation is suggested.

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References

Pylorus-Preserving Pancreaticoduodenectomy After Coronary Artery Bypass Grafting Using Right Gastroepiploic Artery
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Coronary artery bypass grafting using right gastroepiploic artery and pylorus-preserving pancreaticoduodenectomy are both well known and commonly performed procedures independently. However, pylorus-preserving pancreaticoduodenectomy after coronary artery bypass grafting using right gastroepiploic artery has not been

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reported in the literature. We report the first case with operative demonstration of pylorus-preserving pancreaticoduodenectomy in a patient who had undergone coronary artery bypass grafting using an in situ right gastroepiploic artery graft.


The right gastroepiploic artery (RGEA) has been recognized as an excellent alternative arterial graft for coronary artery bypass grafting. Pancreaticoduodenectomy and pylorus-preserving pancreaticoduodenectomy (PPPD) are also widely used procedures for the resection of benign and malignant neoplasms in the peripancreatic region. However, pancreaticoduodenectomy or PPPD in the presence of a functioning RGEA graft poses a unique surgical challenge. This case report is an operative demonstration of PPPD being performed without concomitant salvage revascularization of the RGEA graft in a patient who had previously undergone a coronary artery bypass graft using an in situ RGEA graft.

A 66-year-old woman presented with obstructive jaundice and was found to have an ampullary adenocarcinoma. There was no evidence of advanced disease or distant metastasis. Of note, her past medical history was significant for coronary artery disease, and she had undergone coronary artery bypass grafting using an in situ right gastroepiploic artery (RGEA) graft 10 years ago. Preoperative coronary and selective common hepatic angiographies demonstrated the occluded native right coronary artery and functioning RGEA conduit with excellent runoff (Fig 1). Computed tomographic scan demonstrated that the RGEA graft passed through the antegastric, antehepatic, and transdiaphragmatic route. Preoperatively, her biliary tract was adequately decompressed after a biliary stent placement.

She was taken to the operating room and underwent PPPD. A pulmonary artery catheter and transesophageal echocardiography probe were inserted. Cardiopulmonary bypass in case of major adverse cardiac events was on standby. The procedure began with exploration of the abdominal cavity through an upper median celiotomy. The RGEA conduit, which appeared to be prepared in the skeletonized method, was found at the left of the falciform ligament, reaching the pericardial cavity and coursing anterior to the lateral segment of the left liver (Fig 2A). There was no evidence of subpyloric lymph node enlargement. A clamping test of the RGEA did not cause any remarkable changes of hemodynamics, electrocardiographic monitor, or left ventricular function on transesophageal echocardiography. Dissection of the adhesions surrounding the RGEA graft and division of the duodenum was prioritized to adequately mobilize the RGEA away from the resection field. The pancreaticoduodenal arcade was also test clamped before division to assure that perfusion of the RGEA conduit was maintained after ligation of the superior mesenteric artery system. Satisfactory mobilization of the RGEA conduit was gained afterward (Fig 2C). The RGEA conduit was covered with papaverine-soaked gauze and kept on the left liver surface. The standard PPPD was achievable without difficulty while preserving the RGEA conduit. Pathologic examination showed moderately differentiated adenocarcinoma (pT2N0 stage IB). The postoperative course was uneventful. Postoperative coronary artery angiography again confirmed patency of the RGEA graft providing blood supply to the right coronary artery territory. At present, the patient remains well and free of disease 5 years after surgery.

Comment

Coronary artery bypass grafting using RGEA and PPPD are both well known and commonly performed procedures independently. However, PPPD after coronary artery bypass grafting using RGEA has not been reported in the literature. Traverso and Longmire [1, 2] in 1978 proposed preservation of the pylorus in the course of standard pancreaticoduodenectomy, which includes removal of the distal segment of the stomach, with the goals of preservation of gastric function and reduction in the incidence of postoperative anastomotic ulceration.

Fig 1. Preoperative angiography of coronary and common hepatic artery showing the right gastroepiploic artery (RGEA) conduit perfusing the posterior descending artery (PDA) of the right coronary artery (RCA). (A) Patent RGEA graft. (B) Occluded RCA. (C) Patent anastomosis to PDA with a good distal runoff.
There seems to be no difference regarding the technical difficulty in the presence of RGEA conduit between classic pancreaticoduodenectomy and PPPD. Pylorus-preserving pancreaticoduodenectomy may be recommended when the tumor does not involve the stomach and the lymph nodes along the gastric curvatures. By contrast, when PPPD is not feasible, preservation of the RGEA graft may not be oncologically achievable.

The key to the success of this procedure is to gain curative oncologic resection while preserving the RGEA conduit. Preoperative assessment of the function of the conduit and its gentle manipulation in the entirety of the procedure are crucial. Adequate oncologic curability may be achievable considering the low incidence of subpyloric nodal metastasis in early T stage ampullary adenocarcinoma. On the contrary, locally advanced adenocarcinoma arising in the head of the pancreas is associated with significantly higher nodal involvement in this territory [3, 4], and extensive resection with sacrifice of the RGEA conduit might be needed. Therefore, additional strategies for salvage revascularization of the coronary artery should be considered.

In summary, this is the first operative demonstration of RGEA graft-preserving PPPD without concomitant salvage revascularization procedure of the RGEA graft. To achieve both oncologically curative resection and safe preservation of the functioning RGEA conduit, meticulous perioperative strategies, shown in this demonstration, are crucial.

References