Saphenous vein graft aneurysm with graft-enteric fistula after renal artery bypass

Mark M. Levy, MD, William Kiang, DO, Jason M. Johnson, MD, and **Stuart I. Myers, MD,** *Richmond, Va*

A 65-year-old female presented with upper gastrointestinal hemorrhage thirty years following an aorta-to-right renal artery bypass constructed with saphenous vein. Upper endoscopy demonstrated a duodenal ulcer, and a CAT scan demonstrated aneurysmal degeneration of her renal artery bypass with duodenal impingement. Laparotomy demonstrated erosion of the aneurysm through the posterior wall of the duodenum; extra-anatomic renovascular reconstruction and primary duodenal repair was performed. Although aneurysmal degeneration of intraabdominal saphenous vein grafts is well described and rupture likewise reported, this report represents the first description of an intraabdominal autogenous vein graft aneurysm presenting with gastrointestinal erosion and fistula. (J Vasc Surg 2008;48:738-40.)

CASE REPORT

A 65-year-old white female presented with three to four days of vague abdominal pain, presyncope, and 16 hours of hematochezia. At age 35, she had undergone bilateral aortorenal arterial reconstructions for renovascular hypertension secondary to fibromuscular dysplasia. Both bypasses had been performed with reversed saphenous vein, harvested from her thigh. She had required a subsequent left nephrectomy five years later due to arteriographically demonstrated bypass thrombosis, left renal atrophy, and recurrent hypertension. Subsequently, she had remained hypertensive, with stable renal function, and a baseline creatinine of 1.5 mg/dl.

Now presenting with gastrointestinal hemorrhage requiring transfusion, she underwent emergent upper endoscopy; this demonstrated a 4 cm ulcer at the posterior junction of the duodenum's second and third portion, with fresh thrombus within the ulcer. A contrast-enhanced CAT scan of her abdomen demonstrated a 4 cm aneurysm of her aortorenal artery bypass (Fig 1, *A*), with aneurysmal impingement on the mid-duodenum. No intravenous contrast extravasated from the renal artery bypass into the duodenum, yet there was no visualized plane of tissue between the bypass aneurysm and the duodenum (Fig 1, *B*).

The patient's hematochezia continued, and following a prompt hospital transfer, she underwent urgent aortovisceral arteriography (Fig 2). Arteriography did not demonstrate visible hemorrhage from the renal artery bypass aneurysm, or from the gastroduodenal artery. The proximal aneurysmal bypass was broad based from the aorta, remained aneurysmal through the renal hilum itself, and there were no proximal or distal fixation zones appropriate for endografting. No graft or anastomotic stenosis was identified.

With persistent gastrointestinal hemorrhage, immediate laparotomy was undertaken. Upon exploration of the abdomen, a broad based 4 cm diameter aneurysmal bypass was identified and controlled from the high infrarenal abdominal aorta. Mobilization

From the Division of Vascular Surgery, Virginia Commonwealth University Medical Center.

Competition of interest: none.

Reprint requests: Mark M. Levy, MD, Division Chairman, Vascular Surgery Division, Virginia Commonwealth University Medical Center, P.O. Box 980108, Richmond, VA 23298-0108 (e-mail: mmlevy@vcu.edu).

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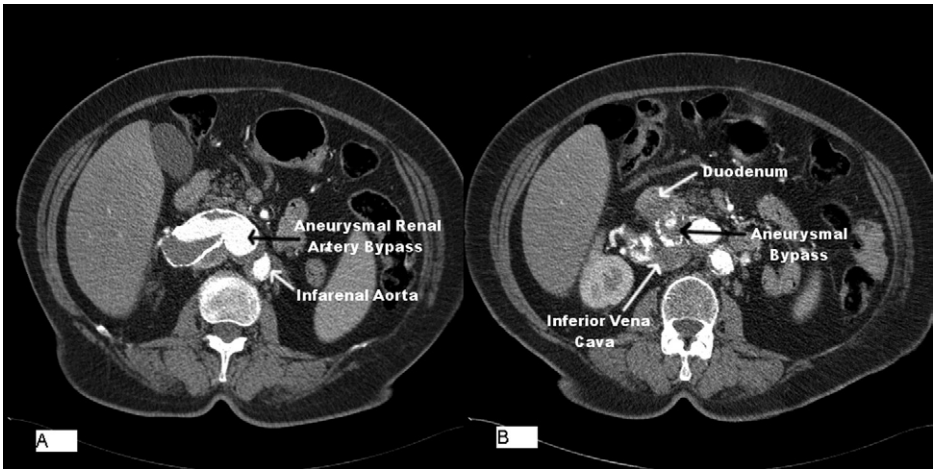

Fig 1. A, Contrast-enhanced computed tomography scan of the abdomen demonstrated aneurysmal degeneration of the aorto-to-right renal bypass immediately caudad to the duodenum; **(B)** the same scan demonstrated the absence of a distinct tissue plane between the bypass aneurysm and the duodenum.

Fig 2. Conventional contrast aortogram demonstrates the renal artery bypass as aneurysmal from its aortic origin through the distal native right renal artery.

of the duodenum demonstrated a large diffusely aneurysmal bypass that extrinsically compressed the third portion of the duodenum. Dissection of the plane between the bypass and the duodenum revealed a 1.5 cm fistula between the duodenum and the aneurysm, with direct juxtaposition of intra-aneurysmal thrombus and duodenal mucosa. No purulence was encountered. An extra-anatomic bypass was promptly fashioned between the right common iliac artery and the right renal hilum using reversed saphenous vein, and the bypass aneurysm was ligated at the aorta, and proximal to the renal hilum. Total right renal warm ischemia time was 22 minutes, and no adjunctive renal protective measures were employed. The aneurysmal sac was opened and decompressed, the duodenotomy was debrided and closed in two layers, and the surgical bed was drained.

Postoperatively, the patient maintained a brisk diuresis, with a creatinine elevation to 1.7 mg/dL at 24 hours and 1.8 mg/dL at 48 hours post-procedure. Initially reliant on intra-operative inotropic support, she became progressively hypotensive and dependent on vasopressor support. Owing to this, the patient's abdomen was explored on the second postoperative day. This revealed no hemorrhage, a patent renal artery bypass, but pan-visceral ischemia (hepatic, gastric, small and large intestine) consistent with visceral arterial vasospasm. Despite supportive treatment, the patient expired in the subsequent day with progressive multisystem organ failure.

DISCUSSION

As early as 1829, Sir Astley Cooper identified fistulas between abdominal aortic aneurysms and the gastrointestinal tract as a source of gastrointestinal hemorrhage.[1] In the subsequent 175 years, such primary aortoenteric fistulae, although uncommon, have been clearly described.[2] More common have been reports of secondary aortoenteric fistulae, complicating 0.1-2% of conventional prosthetic aortic reconstructions.[3] Such secondary aortoenteric fistulae have demonstrated the propensity of prosthetic grafts, when left in direct apposition to bowel wall, to erode through the intestinal wall.

Reports of aortoduodenal fistula following prosthetic renal arterial reconstruction have been likewise described, often, but not always, in the context of graft infection.[4] In contrast however, there have been no such reports documenting aortoduodenal fistula following autogenous vein graft renal arterial reconstructions. In addition, there have been no reports documenting bypass-enteric fistula following intra-abdominal autogenous vein grafting to any other intra-abdominal viscera. This is consistent with the well-accepted perception that autogenous venous tissue may sit in apposition with intra-abdominal organs without risk of the same erosive complications that plague prosthetic grafts.

Renal artery vein graft aneurysmal degeneration was documented by Stanley et al, including their experience with 72 reoperations among 425 primary renovascular reconstructions.[5] Citing vein graft ectasia in 20% of vein grafts,[6] vein graft aneurysms in 6% of adult bypass recipients, and vein graft aneurysms in 20% of pediatric bypass recipients,[7] they considered operative repair of vein graft aneurysms primarily to avoid distal thrombus embolization.

Although historical concerns regarding rupture of such aneurysms had prompted attempts at vein graft plication, autologous vein graft aneurysmal rupture remained undocumented for decades. In 1999, however, Lavigne et al reported a 42-year-old female patient with a ruptured vein graft aneurysm 19 years following bypass;[8] one year later, Travis et al reported a 75-year-old female patient with a ruptured aneurysmal vein graft 22 years following bypass grafting.[9] Both groups recommended long-term follow-up ultrasonography to surveil well established renal artery vein graft bypasses.

This current case presentation likewise demonstrates dramatic aneurysmal degeneration of such an intra-abdominal vein graft clinically manifesting several decades following construction; moreover, it demonstrates the feasibility of erosion of such intra-abdominal aneurysmal vein grafts into the gastrointestinal tract. Together with the above two case reports, it emphasizes both the value of life-long intra-abdominal vein graft surveillance, and also the avoidance of unnecessary autogenous bypass apposition with viscera, either with omental pexy or retrocaval tunneling of right renal artery bypasses.

Although a conventional surgical repair was chosen in the patient here presented, endovascular options should likewise be discussed. The reported treatment for primary renal artery aneurysms has included both the use of coils, guidewires, thrombin and bucrylate to fill saccular aneurysms,[10,11] and stent grafts to exclude both fusiform and saccular primary renal artery aneurysms.[12] This latter technique requires proximal and distal endovascular "landing

zones" to provide circumferential graft seal against the inner arterial wall. Although this patient did not possess such congenial anatomy to address her acquired aneurysm, endograft contamination by enteric organisms would still have remained a concern. In a different but equally life-threatening context however, endograft exclusion has been documented to treat aortoenteric erosions, both for primary aortoenteric fistulae, as well as secondary fistulae;[13] despite the obvious infectious concerns, remarkable success has been described in this difficult clinical situation.[14]

In summary, the authors here present the first clearly documented clinical presentation of an aneurysmal vein graft eroding into the gastrointestinal tract, resulting in graft-to-enteric fistula. Although renal arterial reconstructions are presently performed less commonly, previously constructed renal artery bypasses are aging into their second, third, or fourth decades. With this aging, unusual long-term complications of such bypasses may emerge, such as bypass aneurysm rupture and fistulous erosion into the gastrointestinal tract. Although the patient here discussed expired postoperatively, the authors submit this unusual case presentation to raise future levels of suspicion among surgeons encountering patients with long-standing intra-abdominal vein grafts and gastrointestinal bleeding. With the knowledge that such vein graft-enteric erosions, although not commonplace, are clinically feasible, prompt workup with abdominal computed tomography scans may help to suggest or confirm this diagnosis, and guide subsequent open surgical or endovascular care. Equally important, however, this case provides continued evidence for the utility of long-term surveillance of even well established intra-abdominal bypass grafts.

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