Open surgical repair of an isolated aneurysm of the arc of Riolan with celiac artery occlusion and severe superior mesenteric artery stenosis

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ABSTRACT

The arc of Riolan (AoR), a marginal vessel in the left colon, interconnects the superior and inferior mesenteric arteries. A 65-year-old woman presented with an incidental aneurysmal lesion in the left upper abdomen found on ultrasound. Computed tomography revealed a 27-mm saccular aneurysm in the AoR with occlusion of the celiac artery and severe stenosis of the superior mesenteric artery. Angiography showed that the inferior mesenteric artery provided blood to the perfusion areas of the superior mesenteric artery, celiac artery, and left colon via the AoR. We performed open surgical repair of the aneurysm and reconstruction of the inferior mesenteric artery. (J Vasc Surg Cases Innov Tech 2024;10:101435.)

Keywords: Angiography; Arc of Riolan; Computed tomography; Median arcuate ligament; Visceral aneurysm

The arc of Riolan (AoR) is a collateral artery that connects the superior mesenteric artery (SMA) with the inferior mesenteric artery (IMA). Visceral artery aneurysms are rare, with a reported incidence of 0.01% to 0.2%,[1] of which aneurysms in the AoR are rarely reported. We report a case of open surgical repair of an isolated AoR aneurysm with celiac artery (CA) occlusion and severe SMA stenosis. The patient provided written informed consent for the report of her case details and imaging findings.

CASE REPORT

A 65-year-old woman with no medical history presented to our hospital for detailed examination of an asymptomatic visceral artery aneurysm. Contrast-enhanced computed tomography (CT) showed a saccular aneurysm located on the splenic flexure of the AoR (Fig 1, *A* and *B*), accompanied by occlusion at the origin of the CA, which seemed to be due to compression of the median arcuate ligament and severe stenosis at the origin of the SMA (Fig 1, *C*). The IMA was meandering and dilated to 5 mm in diameter and was connected to the SMA through the AoR. The cause of the SMA lesions could not be identified because she had no signs or history of vasculitis, including fever, a high C-reactive protein level, or other markers. Additionally, no evidence was found of ulcerative colitis or advanced colorectal cancer on CT. Screening colonoscopy was not performed because of the increased risk of rupture.

The visceral aneurysm was saccular (28 × 27 mm) and positioned in the midportion of the AoR near the upper pole of the left kidney, without atherosclerotic calcification, rupture, dissection, or hematoma. The aneurysm presented with indications for surgical repair to avoid rupture due to its large saccular shape. Angiography was performed to evaluate the blood flow in the visceral vessels. It revealed that the SMA and CA blood flow was circulated retrogradely from the IMA via the AoR and pancreaticoduodenal arcade (Fig 2); thus, most of the blood flow to the abdominal organs was supplied only from the IMA.

Both endovascular and surgical approaches were considered. Because the IMA was the sole source of visceral perfusion and maintaining long-term IMA patency was the most important factor in treating this particular aneurysm, open surgical repair was selected.

The operation was performed with an abdominal midline incision under general anesthesia. A dilated IMA was identified in the mesentery of the colon (Fig 3, *A*). The greater omentum and the left paracolic gutter were incised and, subsequently, the splenocolic ligament was excised. The transverse mesentery was divided from the Gerota fascia to expose the aneurysm to the dorsal mesentery. The aneurysm was separated from the surrounding connective tissue, and no branches were around it (Fig 3, *B*). After a test clamp was applied for a few minutes with no signs of ischemia, aneurysm resection and arterial reconstruction by direct end-to-end anastomosis with no conduit were performed (Fig 3, *C*). The clamping time was 12 minutes. No signs or symptoms of ischemia were observed during the procedure. Finally, we confirmed that the anastomosis had no stenosis. Also, the blood flow was excellent on completion angiography.

The patient's postoperative course was uneventful, without the need for anticoagulant or antiplatelet

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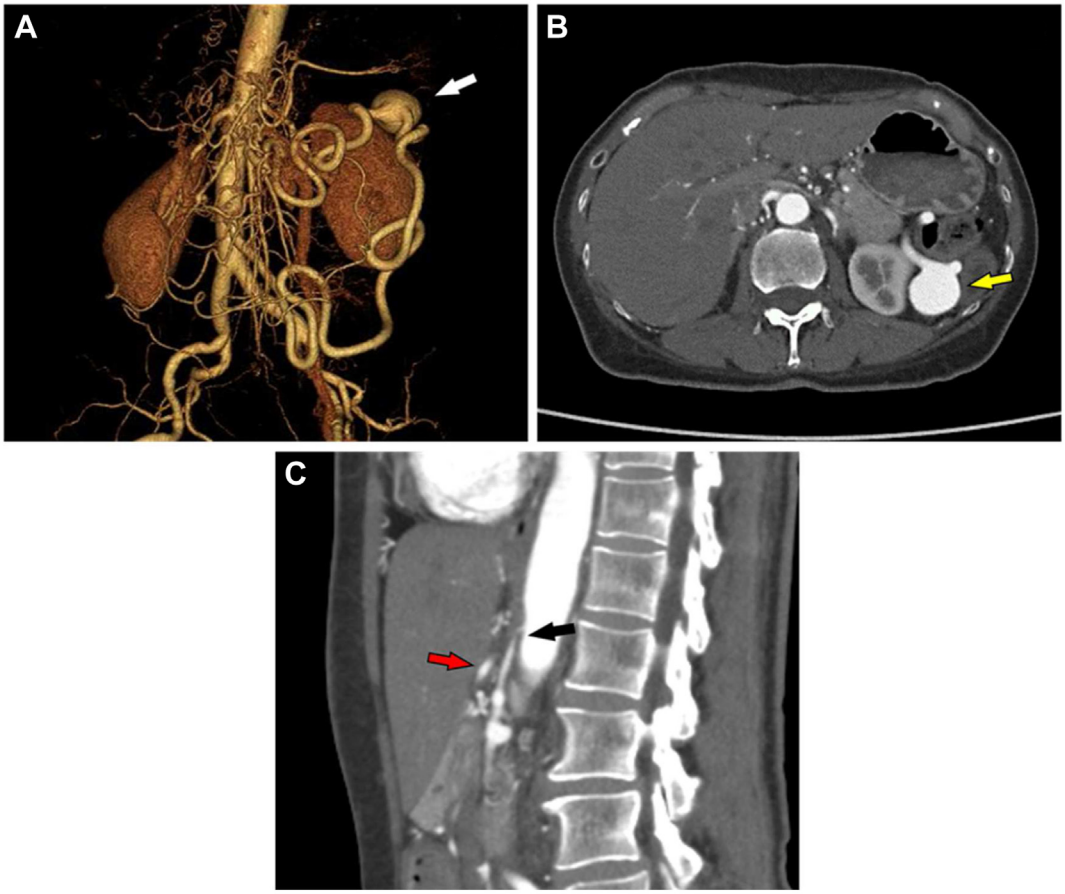

Fig 1. Preoperative computed tomography scans. **A,** Computed tomography scan revealing a dilated inferior mesenteric artery (IMA) and the aneurysm at the arc of Riolan (AoR) artery (*white arrow*). **B,** The aneurysm is seen near the upper pole of the left kidneys (*yellow arrow*). **C,** The median arcuate ligament can be seen compressing the celiac artery (CA; *red arrow*) and superior mesenteric artery (SMA; *black arrow*), and the CA is completely occluded. The SMA is highly stenotic.

agents. Histopathologic examination of the resected aneurysm revealed degeneration of the internal elastic plate (Fig 4). CT performed 4 weeks after the operation revealed no aneurysm, dissection, or hematoma at the anastomosis of the AoR (Fig 5), and the digestive organs showed good contrast enhancement.

DISCUSSION

The AoR, also known as the mesenteric meandering artery or central anastomotic mesenteric artery, is an inconspicuous artery that connects the middle colic branch of the SMA with the left colic branch of the IMA. Jean Riolan (1580–1657), a French anatomist, first reported the collateral circulation. Later, in 1743, Albrecht von Haller (1708–1777) described in detail the anatomy of the mesenteric arteries, and the "arc of Riolan" was named in honor of Jean Riolan.[2,3] Normally, the CA supplies blood to the stomach, liver, pancreas, spleen, and proximal duodenum, and the SMA provides blood to the distal duodenum, entire small intestine, and ascending to mid-transverse colon. The IMA supplies blood from the mid-transverse colon to the rectum.[4] However, in the present patient, the IMA supplied blood via the AoR to all the digestive organs that were originally supplied by the SMA and CA. Endovascular procedures such as coil embolization or sac exclusion with a stent graft are generally less invasive than open procedures for the treatment of visceral artery aneurysms.[5,6] However, because coiling was not indicated due to the wide neck, and stent graft placement would result in technical difficulties in approaching the aneurysm via the tortuous long access and the potential risk of occlusion and endoleak development, an open procedure was selected for our patient. Although stenting of the CA and SMA would be considered for revascularization, removal of external compression by ligament resection seemed necessary in our patient, because involvement with median arch ligament syndrome was suspected. However, no revascularization was performed for fear of creating new turbulence. The small intestine, which is considered relatively vulnerable to ischemia, was not considered irreversibly damaged until after 6 hours of ischemia in studies using

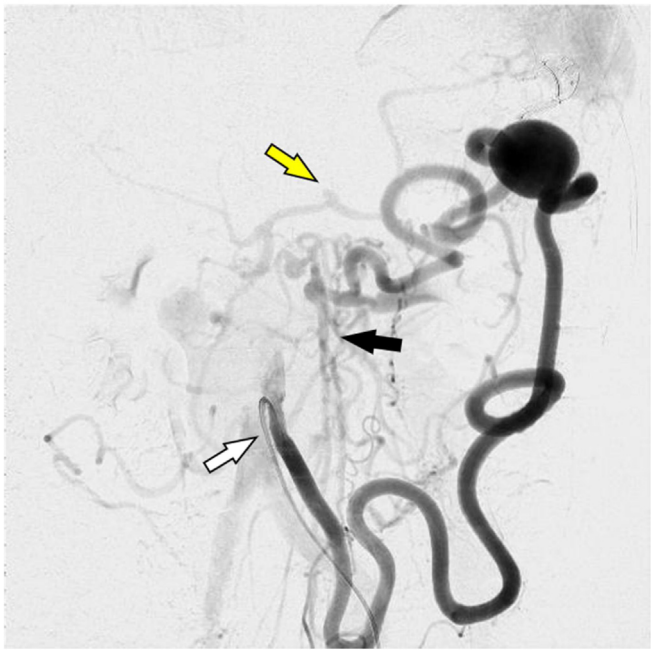

Fig 2. Intraoperative angiography showing the hemodynamics of the inferior mesenteric artery (IMA; *white arrow*) and its flow into the superior mesenteric artery (SMA; *black arrow*). From the dilated arc of Riolan (AoR) and the celiac artery (CA; *yellow arrow*), the blood circulates retrogradely via the pancreaticoduodenal arcade.

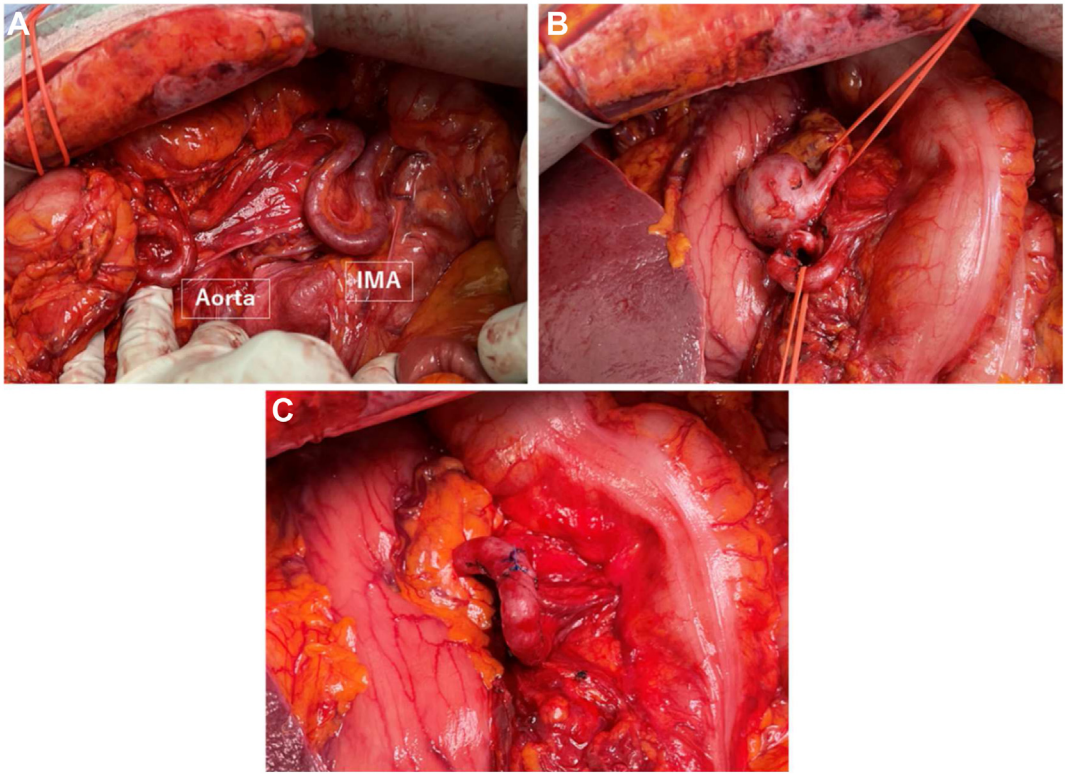

Fig 3. Surgical findings. **A,** The dilated inferior mesenteric artery (IMA) on the colon mesentery meanders and flows into the arc of Riolan (AoR) artery. **B,** The aneurysm is located on the dorsal colon mesentery, with no problems regarding the properties of the parental vessels for anastomotic reconstruction. **C,** The reconstruction is performed by an end-to-end anastomosis.

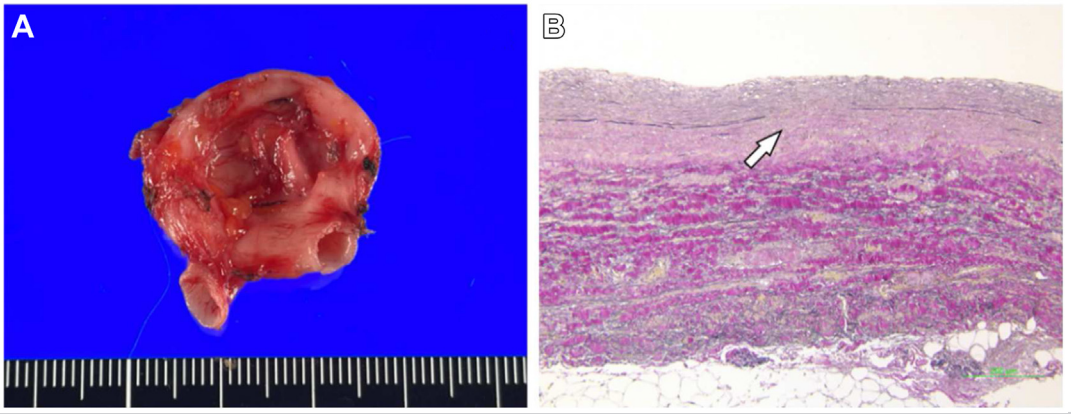

Fig 4. Surgical and pathologic specimens. **A,** Macroscopic photograph of the specimen, without aneurysm rupture, atherosclerotic calcification, dissection, or hematoma. **B,** Elastica Van Gieson stain showing a disrupted internal elastic lamina (*white arrow*).

Fig 5. Postoperative computed tomography scan showing no aneurysm at the arc of Riolan (AoR) artery, and the superior mesenteric artery (SMA) and celiac artery (CA) are well enhanced.

porcine organs,[7] during an open procedure. Thus, for these specific anatomic reasons, special attention was required regarding the duration of temporary vascular occlusion.

The AoR is a minute artery and inconspicuous on imaging studies; however, its expansion for collateral mesenteric vessels is infrequently present if severe stenosis or occlusion of either the SMA or IMA has occurred.[3,8,9] There are also reports that some patients with ulcerative colitis or advanced colon cancer will have an expanded AoR.[10] It has been suggested that an expanded AoR often develops as a compensatory response to reduced blood flow or increased demand. Several similar aneurysm formations derived from visceral arteries due to hyperdynamic flow are known. Sutton and Lawton[11] suggested that CA occlusion or stenosis would cause an increase in blood flow in the collateral vessels for revascularization of the CA, leading to the formation of an aneurysm. According to the literature, pancreaticoduodenal aneurysms are sometimes caused by median arcuate ligament syndrome.[12,13] Also, splenic aneurysm rupture in pregnant women is frequent, suggesting aneurysm formation involves hemodynamic changes due to increased circulating plasma volume.[14] Similarly, medial degeneration due to hyperdynamic flow has been implicated in cases of AoR aneurysms,[15] and the findings from the present case support this opinion. In our patient, the digestive organs received cardiac output almost exclusively from the IMA via the AoR, suggesting that the aneurysm was caused by compensatory increased blood flow and its turbulence. Thus, additional aneurysm formation could occur in the remaining AoR, inferior pancreaticoduodenal artery, or gastroduodenal artery as long as the root cause remains. However, we focused solely on treating the aneurysm due to concerns that dual revascularization might introduce new turbulence in the highly developed collateral vessels and because whether revascularization prevents the formation of new aneurysms remains controversial.[16,17] We will follow-up our patient lifelong and provide additional treatment in the event of new aneurysm formation.

CONCLUSIONS

We performed open surgery to treat an AoR aneurysm in a patient with CA occlusion and severe SMA stenosis with no comorbidities. The findings from this case suggest that excessive blood flow and turbulence stimulate aneurysm formation. Treatment of the stenotic lesion, which is the root cause of the hyperdynamic flow, is also required.

DISCLOSURES

None.

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