The Society for Vascular Surgery clinical practice guidelines on the management of visceral aneurysms

Rabih A. Chaer, MD,[a] Christopher J. Abularrage, MD,[b] Dawn M. Coleman, MD,[c] Mohammad H. Eslami, MD,[a] Vikram S. Kashyap, MD,[d] Caron Rockman, MD,[e] *and* M. Hassan Murad, MD,[f] *Pittsburgh, Pa; Baltimore, Md; Ann Arbor, Mich; Cleveland, Ohio; New York, NY; and Rochester, Minn*

ABSTRACT

These Society for Vascular Surgery Clinical Practice Guidelines describe the care of patients with aneurysms of the visceral arteries. They include evidence-based size thresholds for repair of aneurysms of the renal arteries, splenic artery, celiac artery, and hepatic artery, among others. Specific open surgical and endovascular repair strategies are also discussed. They also describe specific circumstances in which aneurysms may be repaired at smaller sizes than these size thresholds, including in women of childbearing age and false aneurysms. These Guidelines offer important recommendations for the care of patients with aneurysms of the visceral arteries and long-awaited guidance for clinicians who treat these patients. (J Vasc Surg 2020;■:1-37.)

TABLE OF CONTENTS

SUMMARY OF RECOMMENDATIONS

Renal artery aneurysm (RAA). **1.1:** In patients who are thought to have RAAs, we recommend computed tomography angiography (CTA) as the diagnostic tool of choice. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

1.2: In patients who are thought to have RAA and have increased radiation exposure risks or renal insufficiency, we recommend non-contrast-enhanced magnetic resonance angiography (MRA) to establish the diagnosis. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: C (Low)**.

From the Division of Vascular Surgery, University of Pittsburgh Medical Center, Pittsburgh[a]; the Division of Vascular Surgery, Johns Hopkins Hospital, Baltimore[b]; the Division of Vascular Surgery, University of Michigan Hospitals, Ann Arbor[c]; the Division of Vascular Surgery, University Hospitals Case Medical Center, Cleveland[d]; the Division of Vascular Surgery, New York University Langone Medicine Center, New York[e]; and the Mayo Clinic Evidence-Based Practice Center, Rochester.[f]

Author conflict of interest: none.

Independent peer review and oversight have been provided by members of the Society for Vascular Surgery Document Oversight Committee: Drs Thomas L. Forbes (Chair), Ruth Bush (Vice-Chair), Neal Barshes, Keith Calligaro, Ronald L. Dalman, Mark Davies, Yazan Duwayri, Alik Farber, Gregory Landry, Mahmoud Malas, Katherine McGinigle, J. Sheppard Mondy, Marc Schermerhorn, Cynthia Shortell.

Correspondence: Rabih A. Chaer, MD, University of Pittsburgh Medical Center, 200 Lothrop St, Pittsburgh, PA 15213 (e-mail: chaerra@upmc.edu).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214

https://doi.org/10.1016/j.jvs.2020.01.039

Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).

1.3: We recommend the use of catheter-based angiography both for preoperative planning and to better delineate distal renal artery branches that may be inadequately assessed on conventional cross-sectional imaging. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

2.1: In patients with noncomplicated RAA of acceptable operative risk, we suggest treatment for aneurysm size >3 cm. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

2.2: We recommend emergent intervention for any size RAA resulting in patient symptoms or rupture. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

2.3: In patients of childbearing potential with noncomplicated RAA of acceptable operative risk, we suggest treatment regardless of size. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

2.4: In patients with medically refractory hypertension and functionally important renal artery stenosis, we suggest treatment regardless of size. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.1: We suggest daily antiplatelet therapy (ie, aspirin, 81 mg) for patients with RAA**. Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.2: We suggest open surgical reconstructive techniques for the elective repair of most RAAs in patients with acceptable operative risk. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.3: We suggest ex vivo repair and autotransplantation for complex distal branch aneurysms over nephrectomy when it is technically feasible**. Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.4: We suggest endovascular techniques for the elective repair of anatomically appropriate RAAs to include stent graft exclusion of main RAAs in patients with poor operative risk and embolization of distal and parenchymal aneurysms**. Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.5: We suggest consideration of laparoscopic and robotic techniques as an interventional alternative based on institutional resources and surgeon experience with minimally invasive techniques. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

4.1: We suggest screening female patients of childbearing age with RAA for fibromuscular dysplasia with a focused history and one-time axial imaging study (ie, CTA or MRA) to assess for cerebrovascular, mesenteric, and iliac artery dysplasia**. Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

5.1: We suggest completion imaging after open surgical reconstruction for RAA, before hospital discharge, by way of axial imaging with CTA or MRA or arteriography in select cases, and long-term follow-up with surveillance imaging**. Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

5.2: For patients managed nonoperatively, we suggest annual surveillance imaging until two consecutive studies are stable; thereafter, surveillance imaging may be extended to every 2 to 3 years**. Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

Splenic artery aneurysm (SAA). **1.1:** We recommend computed tomography angiography as the initial diagnostic tool of choice for SAAs. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

1.2: In patients with suspected SAAs and pre-existing renal insufficiency limiting the use of iodinated contrast material, we recommend magnetic resonance angiography to establish diagnosis. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

1.3: We recommend using arteriography when noninvasive studies have not sufficiently demonstrated the status of relevant collateral blood flow and when endovascular intervention is planned. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

2.1: We recommend emergent intervention for ruptured SAAs. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High).**

2.2: We recommend treatment of nonruptured splenic artery pseudoaneurysms of any size in patients of acceptable risk because of the possibility of rupture. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

2.3: We recommend treating nonruptured splenic artery true aneurysms of any size in women of childbearing age because of the risk of rupture. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

2.4: We recommend treating nonruptured splenic artery true aneurysms >3 cm, with a demonstrable increase in size, or with associated symptoms in patients of acceptable risk because of the risk of rupture. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

2.5: We suggest observation over repair for small (<3 cm), stable asymptomatic splenic artery true aneurysms or those in patients with significant medical comorbidities or limited life expectancies. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.1: In patients with ruptured SAA discovered at laparotomy, we suggest treatment with ligation with or without splenectomy, depending on the aneurysm location. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.2: In patients with ruptured SAA diagnosed on preoperative imaging studies, we suggest treatment with open surgical or appropriate endovascular techniques based on the patient's anatomy and underlying clinical condition. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.3: We suggest elective treatment of SAA using an endovascular approach if it is anatomically feasible. However, elective treatment may appropriately involve open surgical, endovascular, or laparoscopic methods of intervention, depending on the patient's anatomy and underlying clinical condition. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.4: In treatment of SAA, we suggest that the splenic artery does not routinely require preservation or revascularization. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.5: In treatment of distal SAA adjacent to the hilum of the spleen, we suggest open surgical techniques including possible splenectomy as opposed to endovascular methods, given concern for the possibility of end-organ ischemia, including splenic infarction and pancreatitis. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.6: In pregnant women with SAA, treatment decisions should be individualized regardless of size, and the potential morbidity to both the mother and fetus should be considered. **(Ungraded best practice statement.)**

4.1: We suggest screening of patients with SAAs for other intra-abdominal, intrathoracic, intracranial, and peripheral artery aneurysms. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

5.1: In patients in whom an SAA is being observed with a nonoperative or noninterventional approach, we suggest annual surveillance with computed tomography or ultrasound to assess for growth in size. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

5.2: After endovascular intervention for SAAs, we suggest periodic surveillance with computed tomography angiography, ultrasound, or magnetic resonance angiography to assess for the possibility of endoleak or other continued aneurysm perfusion that could lead to a continued risk of aneurysm growth or rupture. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

Celiac artery aneurysm (CAA). **1.1** We suggest computed tomography angiography (CTA) as the initial diagnostic tool of choice for CAAs. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

1.2 We suggest magnetic resonance angiography in patients with suspected CAA and pre-existing renal insufficiency limiting the use of iodinated contrast material. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

1.3 We suggest arteriography when noninvasive studies have not sufficiently demonstrated the status of relevant collateral blood flow or when endovascular intervention is planned. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

2.1 We recommend emergent intervention for ruptured CAAs. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High).**

2.2 We recommend treatment of nonruptured celiac artery pseudoaneurysms of any size in patients of acceptable operative risk because of the possibility of rupture. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

2.3 We recommend treatment of nonruptured celiac artery true aneurysms >2 cm, with a demonstrable increase in size, or with associated symptoms in patients of acceptable risk because of the risk of rupture. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

2.4 We suggest observation over intervention for small (<2 cm), stable asymptomatic CAAs or those in patients with significant medical comorbidities or limited life expectancy. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.1 In patients with ruptured CAA discovered at laparotomy, we suggest ligation if sufficient collateral circulation to the liver can be documented. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low).**

3.2 In patients with ruptured CAA diagnosed on preoperative imaging studies who are stable, we recommend treatment with open surgical or appropriate endovascular methods based on the patient's anatomy and underlying clinical condition. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

3.3 For the elective treatment of CAA, we suggest using an endovascular intervention if it is anatomically feasible. However, elective treatment may appropriately involve open surgical, endovascular, or laparoscopic methods of intervention, depending on the patient's anatomy and underlying clinical condition. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

3.4 To determine the need for revascularization of the celiac artery and its branches in treating CAA, we suggest evaluating the status of the superior mesenteric artery, gastroduodenal artery, and other relevant collateral circulation, which must be carefully documented on preoperative CTA or angiography. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

4.1 We suggest screening patients with CAAs for other arterial aneurysms. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

5.1 In patients in whom a CAA is being observed with a nonoperative or noninterventional approach, we suggest annual surveillance with CTA scans to assess for growth

in size. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

5.2: After endovascular intervention for CAAs, we suggest periodic surveillance with appropriate imaging studies to assess for the possibility of endoleak or aneurysm reperfusion that could lead to a continued risk of aneurysm growth or rupture. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).**

Gastric and gastroepiploic artery aneurysms. **1.1**: In patients who are thought to have gastric or gastroepiploic artery aneurysms, we recommend computed tomography angiography (CTA) as the diagnostic tool of choice. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

1.2: In patients who are thought to have gastric and gastroepiploic artery aneurysms and have high radiation exposure risks or renal insufficiency, we recommend non-contrast-enhanced magnetic resonance angiography (MRA) for diagnosis. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).

1.3: We recommend the use of catheter-based angiography for all emergent cases presenting with rupture, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate),** and electively for preoperative planning, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

2.1: We recommend treatment of all gastric artery and gastroepiploic artery aneurysms of any size. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

3.1: We recommend endovascular embolization for first-line treatment of gastric artery and gastroepiploic artery aneurysms. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

4.1: We suggest abdominal axial imaging to screen for concomitant abdominal aneurysms. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

4.2: We suggest one-time screening CTA (or MRA) of the head, neck, and chest for those patients with segmental arterial mediolysis. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

5.1: We suggest interval surveillance (ie, every 12-24 months) with axial imaging (ie, CTA or MRA) in cases of segmental medial arteriolysis in light of reported cases of rapid arterial transformation. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

5.2: We suggest postembolization surveillance every 1 to 2 years with axial imaging to assess for vascular remodeling and evidence of aneurysm reperfusion. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

Hepatic artery aneurysm (HAA). **1.1:** In patients who are thought to have HAA, we recommend computed tomography angiography (CTA) as the diagnostic tool of choice. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

1.2: In patients with HAA who are considered for intervention, we recommend mesenteric angiography for preoperative planning. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

2.1: Given the high propensity of rupture and significant antecedent mortality, we recommend that all hepatic artery pseudoaneurysms, regardless of cause, be repaired as soon as the diagnosis is made. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High).**

2.2.a: We recommend repair of all symptomatic HAAs regardless of size. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High).**

2.2.b: In asymptomatic patients without significant comorbidity, we recommend repair if true HAA is >2 cm, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High),** or if aneurysm enlarges >0.5 cm/y, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).** In patients with significant comorbidities, we recommend repair if HAA is >5.0 cm. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate).**

2.3. We recommend repair of HAA in patients with vasculopathy or vasculitis, regardless of size, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low)**. We recommend repair in HAA patients with positive blood cultures, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

3.1: We recommend an endovascular-first approach to all HAAs if it is anatomically feasible (ie, if this approach maintains arterial circulation to the liver). **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High).**

3.2: In patients with extrahepatic aneurysms, we recommend open and endovascular techniques to maintain liver circulation. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High).**

3.3: In patients with intrahepatic aneurysms, we recommend coil embolization of the affected artery, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**. In patients with large intrahepatic aneurysms, we recommend resection of the involved lobe of liver to avoid significant liver necrosis, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low).**

4.1: We suggest abdominal axial imaging to screen for concomitant intra-abdominal aneurysms in patients who did not have CTA at the time of HAA diagnosis

Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate).

4.2: We suggest one-time screening CTA or magnetic resonance angiography of the head, neck, and chest for those patients with nonatherosclerotic causes of HAA. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

5.1: We suggest annual follow-up with CTA or non-contrast-enhanced computed tomography to observe patients with asymptomatic HAA. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

Superior mesenteric artery aneurysm (SMAA). **1.1:** In patients with SMAAs, we recommend computed tomography angiography (CTA) as the diagnostic tool of choice. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

1.2: We recommend mesenteric angiography to delineate anatomy in preoperative planning for SMAA repair. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

2.1: We recommend repair of all true SMAAs and pseudoaneurysms as soon as the diagnosis is made regardless of size. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High)**.

2.2: We suggest careful observation of SMAA because of dissection unless refractory symptoms develop. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

3.1: We recommend an endovascular-first approach to all SMAAs if it is anatomically feasible. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

4.1: We suggest abdominal axial imaging to screen for concomitant intra-abdominal aneurysms in patients who did not have CTA at the time of diagnosis. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

5.1: We suggest annual CTA to observe postsurgical patients. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

Jejunal, ileal, and colic artery aneurysms. **1.1:** In patients who are thought to have jejunal artery, ileal artery, and colic artery aneurysms, we recommend computed tomography angiography (CTA) as the diagnostic tool of choice. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

1.2: In patients with high radiation exposure risks or renal insufficiency, we recommend non-contrast-enhanced magnetic resonance angiography (MRA) for diagnosis. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low)**.

Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).

1.3: We recommend the use of catheter-based angiography for all emergent cases presenting with rupture, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate),** and electively for preoperative planning, **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: C (Low)**.

1.4: We suggest screening all patients with jejunal, ileal, and colic artery aneurysms for vasculitis with routine inflammatory markers. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

2.1: We recommend elective intervention for jejunal and ileal artery aneurysms >2 cm in maximal diameter and for all colic artery aneurysms, any size. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

2.2: We recommend emergent intervention for any jejunal, ileal, or colic artery aneurysm, any size, resulting in patient symptoms or rupture and all mesenteric branch vessel pseudoaneurysms. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: A (High)**.

3.1: We suggest open surgical ligation or aneurysm excision for cases of jejunal, ileal, and colic artery aneurysms when laparotomy is being considered for hematoma evacuation or bowel assessment for viability. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

3.2: We suggest endovascular embolization for cases of jejunal, ileal, and colic artery aneurysm. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

3.3: We suggest medical treatment of nonruptured, asymptomatic ileal, jejunal, and colic artery aneurysms associated with polyarteritis nodosa. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

4.1: We suggest abdominal axial imaging to screen for concomitant abdominal aneurysms. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

4.2: We suggest one-time screening CTA (or MRA) of the head, neck, and chest for those patients with segmental arterial mediolysis. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

5.1: We suggest interval surveillance (ie, every 12-24 months) with axial imaging (ie, CTA or MRA) for cases of segmental medial arteriolysis in light of reported cases of rapid arterial transformation and to monitor regression in cases of polyarteritis nodosa. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

5.2: We suggest postembolization surveillance every 1 to 2 years with axial imaging to assess for vascular remodeling and evidence of aneurysm reperfusion. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

Pancreaticoduodenal artery aneurysm (PDAA) and gastroduodenal artery aneurysm (GDAA). **1.1:** In patients who are thought to have GDAA and PDAA, we recommend computed tomography angiography (CTA) as the diagnostic tool of choice. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

1.2: In patients in whom celiac stenosis is suspected, we suggest further workup with duplex ultrasound to elucidate whether the stenosis is hemodynamically significant. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

1.3: In patients with high radiation exposure risks or renal insufficiency, we suggest non-contrast-enhanced magnetic resonance angiography (MRA) for diagnosis. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).

2.1: In patients with noncomplicated GDAA and PDAA of acceptable operative risk, we recommend treatment no matter the size of the aneurysm because of the risk of rupture. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

3.1: In patients with intact and ruptured aneurysms, we recommend coil embolization as the treatment of choice. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

3.2: In patients in whom coil embolization is not feasible, we suggest covered stenting or stent-assisted coil embolization as a treatment option in select cases of GDAA and PDAA. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

3.3: In patients with appropriate anatomy, we suggest transcatheter embolization with liquid embolic agents as a treatment option for both GDAA and PDAA. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

3.4: In patients with suitable anatomy, we suggest flow-diverting, multilayered stents as a treatment option for GDAA and PDAA, although these have not been adequately studied to be recommended as a primary treatment modality. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

3.5: In patients with nonruptured aneurysms, we suggest open surgical reconstruction if needed to preserve flow. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

3.6: In patients with concomitant stenosis or occlusion, we suggest celiac artery reconstruction. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**.

4.1: In patients with median arcuate ligament syndrome, we suggest screening for GDAA or PDAA with CTA or duplex ultrasound. **Level of Recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**.

5.1: In patients status post treatment of GDAA and PDAA, we recommend follow-up imaging after endovascular treatment of GDAA and PDAA to rule out persistent flow through the aneurysm sac. **Level of Recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**.

INTRODUCTION

Aneurysms of the visceral arteries are a rare but clinically important vascular condition. Of all intra-abdominal aneurysms, only approximately 5% affect the visceral arteries. Visceral artery aneurysms include both true aneurysms and pseudoaneurysms. Many true visceral artery aneurysms are degenerative or atherosclerotic in nature, with histologic specimens demonstrating reduced smooth muscle, disruption of elastic fibers, and deficiency of the arterial media.[1] Other common causes of visceral artery aneurysms include fibromuscular dysplasia, collagen vascular diseases, inflammatory conditions, and other rare inherited illnesses, such as the Ehlers-Danlos syndrome. As such, in patients with multiple aneurysms or aneurysms in different visceral beds, genetic testing is indicated for diagnostic and prognostic purposes. In contrast to the causes of true aneurysms of the visceral vessels, visceral artery pseudoaneurysms are most commonly related to trauma, iatrogenic injury, local inflammatory processes, or infection.

The clinical significance of visceral artery aneurysms is mainly related to their potential for rupture and the extreme challenge of emergent diagnosis and treatment of these uncommon aneurysms once rupture has occurred. Nearly one-fourth of visceral artery aneurysms reported in the literature have presented with rupture, and the reported mortality rate of these diagnosed ruptures is at least 10% and is likely to be much higher.[2,3] Reported deaths after ruptured celiac artery aneurysms and ruptured splenic artery aneurysms in pregnant women approach 100%. Because of the increased use of sophisticated forms of intra-abdominal imaging, including magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), computed tomography (CT), and CT angiography (CTA), occult visceral artery aneurysms are being diagnosed with increased frequency. These detailed imaging studies are allowing an improved ability for vascular surgeons to identify asymptomatic lesions and an enhanced potential for preoperative or preprocedural planning and elective treatment of these aneurysms. Improvements in endovascular therapies have also allowed an enhanced ability for treatment of these often anatomically complex lesions with a large variety of individualized and precise catheter-based therapies.

However, the natural history of visceral aneurysms and their potential for rupture or other complications are relatively poorly defined because of their overall scarcity. The majority of the reports in the literature consist of only one or two cases. Larger institutional case series have been reported but rarely consist of more than a compilation of several dozen cases. The recent increase in the reports of visceral aneurysms in the literature is mainly related to an escalation in the use of novel and varied catheter-based techniques for their treatment. Whereas much valuable information may be gained from reading these individual reports, they may be inherently predisposed toward a representation of unusual presentations and successful outcomes. However, it is clear even from these numerous case reports that a significant proportion of visceral artery aneurysms present with rupture; therefore, an aggressive approach to their diagnosis and management certainly seems warranted.

It is somewhat difficult to precisely characterize which factors in an individual aneurysm will predispose to rupture. Splenic artery aneurysms are thought to have a particular tendency toward rupture, especially during the third trimester of pregnancy.[4] Visceral artery pseudoaneurysms certainly have a higher rupture potential than true aneurysms. Although larger size would certainly seem to imply a higher chance of rupture, small visceral aneurysms can rupture as well. There is no firm evidence that calcification in a visceral artery aneurysm protects against a risk of rupture. When it occurs, rupture of visceral artery aneurysms can occur into the peritoneal cavity, retroperitoneal space, gastrointestinal tract, or biliary tract. Free rupture into the peritoneal cavity resulting in hemoperitoneum is often termed abdominal apoplexy. Rupture of visceral artery aneurysms may also be manifested with life-threatening gastrointestinal hemorrhage as well.

Although not directed by randomized prospective trials, general principles of management of visceral artery aneurysms do exist. Because of their potential for rupture, most visceral artery pseudoaneurysms, mycotic aneurysms, and many larger true aneurysms warrant intervention. Treatment can generally be accomplished by either open surgical or endovascular approaches. The treatment goal is to prevent aneurysm expansion and potential rupture by exclusion from the arterial circulation while maintaining necessary distal or collateral bed perfusion. Depending on the location of the aneurysm, this can be accomplished in a variety of ways. In areas of the visceral circulation with an abundance of collateral flow, for example, in the splenic artery, proximal and distal ligation of the aneurysm segment is a viable surgical option. This can also be accomplished with endovascular isolation of the aneurysmal segment, either by placement of a stent graft or by coil embolization of the proximal and distal arterial segment. The preferred treatment of an individual patient and aneurysm must be carefully based on the particular anatomy and any associated clinical conditions as well as the underlying condition of the patient. The purpose of these guidelines is to inform the diagnosis, treatment options, screening, and follow-up of visceral aneurysms based on the available published literature and the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach.[5]

METHODS

Guideline framework. The committee used the GRADE approach to rate the quality of evidence (confidence in the estimates) and to grade the strength of recommendations. This system, adopted by >100 other organizations, categorizes recommendations as strong GRADE 1 or weak (also called conditional) GRADE 2 on the basis of the quality of evidence, the balance between desirable effects and undesirable ones, the patient's values and preferences, and the required resources.

GRADE 1 recommendations are meant to identify practices for which benefit clearly outweighs risk. These recommendations can be made by clinicians and accepted by patients with a high degree of confidence. GRADE 2 recommendations are made when the benefits and risks are more closely matched and are more dependent on specific clinical scenarios. In general, physician and patient preferences play a more important role in the decision-making process in these circumstances.

The Society for Vascular Surgery adapts GRADE so that the level of evidence to support the recommendation is divided into three categories: A (high quality), B (moderate quality), and C (low quality). Conclusions based on high-quality evidence are unlikely to change with further investigation, whereas those based on moderate-quality evidence are more likely to be affected by further scrutiny. Those based on low-quality evidence are the least supported by current data and the most likely to be subject to change in the future.

A GRADE 1 recommendation can occasionally be made on the basis of low-quality (C) evidence. A full explanation of the GRADE approach has been presented to the vascular surgery community.[5-7] The Committee reached consensus about all the recommendations and the level of supporting evidence. These guidelines will require periodic updates to become a "living document" that will be modified as techniques are further refined, technology develops, medical therapy improves, and new data emerge.

Evidence synthesis. The Committee commissioned a systematic review of MEDLINE, Embase, Cochrane databases, and Scopus that had wide inclusion criteria. The review included studies with 10 patients or more that reported outcomes of patients with visceral artery aneurysm treated with an open or endovascular approach. Studies were comparative or noncomparative but had to have longitudinal follow-up and to evaluate an outcome of interest (mortality, need of reintervention, myocardial infarction, stroke, end-organ ischemia, end-organ infarction, deep venous thrombosis, pulmonary embolism, postembolization syndrome, respiratory complications, gastrointestinal complications, coil migration, post-endovascular aortic repair rupture, rupture during intervention, or wound complications [eg, surgical site infection]). The systematic review eventually summarized data from 80 observational studies that were mostly noncomparative. Data were available on 2845 aneurysms (1279 renal artery; 775 splenic artery; 359 hepatic artery; 226 pancreaticoduodenal and gastroduodenal arteries; 95 superior mesenteric artery; 87 celiac artery; 15 jejunal, ileal, and colic arteries; 9 gastric and gastroepiploic arteries).[8]

A methodology group independently selected and appraised studies and subsequently collaborated with the committee to integrate evidence into recommendations. The Committee provided additional references and monitored the literature for new evidence emerging after the original search.

RENAL ARTERY ANEURYSM (RAA)

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of RAA			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	In patients who are thought to have RAA, we recommend CTA as the diagnostic tool of choice, with 1-mm-thickness sections if available.	1 (Strong)	B (Moderate)
1.2	In patients who are thought to have RAA and have increased radiation exposure risks or renal insufficiency, we recommend non-contrast-enhanced MRA to establish the diagnosis (Grade 1C).	1 (Strong)	C (Low)
	Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie pregnancy, renal insufficiency, or gadolinium contrast material allergy).		
1.3	If preoperative planning and recognition of distal renal artery branches cannot be adequately assessed on conventional cross-sectional imaging (CTA), we recommend the use of catheter-based angiography.	1 (Strong)	C (Low)

RAAs occur in approximately 0.1% of the population, although the absolute incidence is unknown.[9] Autopsy studies are likely to underestimate RAA incidence at 0.01% to 0.09%,[10,11] whereas angiographic studies are likely to overestimate this at 0.73% to 0.97%.[12,13] Overall, RAAs are most commonly identified on imaging obtained for unrelated reasons. In a multicenter study, CTA (58%) was the most commonly used modality for the diagnosis and evaluation of RAAs, followed by non-contrast-enhanced CT (24%), MRA (6%), catheter angiography (5%), and ultrasound (4%).[14] CTA with multiplanar, maximal intensity projection reconstruction, volume rendering, and three-dimensional reconstructions better assess the arterial anatomic details of the renal arteries at all branch levels, including intrapelvic and intraparenchymal locations.[15-17] Thickness sections of 1 mm, if available, should be favored for better anatomic definition. Furthermore, the three-dimensional reconstructions may give a better representation of the number and relation of all involved branches compared with two-dimensional catheter angiography or ultrasound.

In certain patients, compromised renal function puts them at increased risk of contrast-induced nephropathy. In an effort to avoid the nephrotoxic effects of iodinated contrast agents, non-contrast-enhanced MRA has been used to assess the renal arteries. MRA can be performed with a "breath-hold" steady-state free precession sequence or time-spatial labeling inversion pulse technique. Studies evaluating steady-state free precession sequences have shown excellent concordance as well as interobserver agreement with contrast-enhanced MRA.[18,19] Furthermore, time-spatial labeling inversion pulse non-contrast-enhanced MRA has also shown promise compared with contrast-enhanced CTA with 74% sensitivity, 93% specificity, and 90% accuracy.[20] Whereas contrast-enhanced ultrasound has been used in the evaluation of RAAs, this technique has not been compared with other invasive and noninvasive angiographic techniques and is operator dependent.[21] MRA may also be considered in young patients and for routine surveillance to decrease the risk of radiation-induced malignant transformation with cumulative exposure.

Whereas three-dimensional CTA is the diagnostic tool of choice for RAAs, the anatomic association of multiple renal arteries may limit the surgeon's ability to plan a successful endovascular treatment. In such cases, preoperative three-dimensional rotational catheter-based angiography may be of great benefit in planning optimal working angles.[22] Furthermore, three-dimensional rotational catheter-based angiography has been shown to be advantageous compared with two-dimensional catheter-based angiography in 75% of cases in evaluating the neck, feeding arteries, and relationship between branches of RAA.[23]

The ability of CTA to assess microaneurysms of the distal renal vasculature may also be limited even with comprehensive renal-specific CT protocols. This is due to the rapid background enhancement of the renal parenchyma of the cortex.[24] In such cases, CTA may show parenchymal infarcts or extrarenal hematomas, consistent with distal microaneurysmal disease as seen in cases of polyarteritis nodosa. However, catheter-based angiography is better to directly visualize distal microaneurysms.[25]

several series support no incidence of rupture during the surveillance of nonoperative RAAs out to 270 months.[9,14,30,35-39] Most recent estimates suggest a median annualized growth rate of 0.06 to 0.6 mm.[14,39,40] The most recent and largest multi-institutional retrospective series of nonoperative RAA surveillance found no difference in growth rate based on aneurysm morphology or calcification.[14] These authors also reported the successful surveillance of 88 aneurysms measuring 2 to 3 cm and 7 aneurysms measuring >3 cm without complication or rupture. The revised 3-cm size threshold for repair is based on limited literature, however, making this a Grade 2C conditional recommendation where variation in care is acceptable on the basis of individualized clinical decision-making.

It is unclear based on the current literature whether branch aneurysms or saccular aneurysms behave differently, but classic teaching associates such aneurysm morphology with a more aggressive risk of rupture. As such, they might merit invasive treatment regardless of size.

2. Size criteria and alternative indications for intervention

Recommendations for indications for intervention in RAA			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	In patients with noncomplicated RAA of acceptable operative risk, we suggest treatment for aneurysm size >3 cm.	2 (Weak)	C (Low)
2.2	We recommend emergent intervention for any size RAA resulting in patient symptoms or rupture.	1 (Strong)	B (Moderate)
2.3	In patients of childbearing potential with noncomplicated RAA of acceptable operative risk, we suggest treatment regardless of size.	2 (Weak)	B (Moderate)
2.4	In patients with medically refractory hypertension and functionally important renal artery stenosis, we suggest treatment regardless of size.	2 (Weak)	C (Low)

Previous guidelines have suggested the repair of most visceral artery aneurysms >2 cm in maximum diameter.[26] Whereas no prospective or randomized trial directly compares operative repair of intermediate RAAs >2 cm with surveillance, the natural history of these aneurysms appears more benign than historic rates have suggested, with lower associated risks of rupture, slow to null rates of growth, and improved survival after rupture apparent from more contemporary series.

The natural history of RAAs appears to be that of slow or no growth.[27] Contemporary reports do not support historic series that described rupture rates as high as 14% to 30% with associated mortality of 80%.[9,28,29] Modern-day rupture rates are estimated at 3% to 5%, with nongestational mortality improved to <10%.[9,30-34] Most ruptures are diagnosed at the time of presentation, and

Ruptured RAAs are associated with a mortality of approximately 10% in the general population and maternal and fetal death in 55% and 85%, respectively.[37,41] Emergent surgery is required to prevent exsanguination; aortic control may be necessary. In many cases, renal artery reconstruction and renal salvage may not be feasible, and thus nephrectomy should be considered.

Pregnancy has been associated with increased risk and rates of rupture, presumptively secondary to hemodynamic changes and increased vascular volume and flow during pregnancy, abdominal pressure changes secondary to mass effect from the gravid uterus, and hormonal changes that may disrupt the vessel wall.[27,41-43] Pregnancy-associated rupture appears to complicate the third trimester in most cases, with postpartum rupture infrequently referenced in case reports only.[44,45]

Whereas no large-scale studies detail the true incidence of gestational renal aneurysm rupture, in a series of 180,000 pregnancies brought to term, no ruptures were identified.[46] Despite this, small aneurysm size (ie, 1 cm) at rupture and high historic rates of maternal and fetal mortality that approximate 56% to 92% and 82% to 100%, respectively, support the more aggressive treatment of RAAs in women of childbearing potential.[36,41,47] Contemporary outcomes for both mother and fetus may be improving as there are reports of gestational rupture with both maternal and fetal survival.[44,48]

Two-thirds of patients with RAA also have hypertension.[9,31,36,37,39,40,49-52] Clinically relevant renal artery stenosis is present in 7% to 66% of patients with RAA across series, and although renal artery occlusive disease is not evidenced in all RAA patients with hypertension, it does remain a valid indication for intervention. Whereas the mechanism driving hypertension in RAA patients remains elusive, additional hypotheses include distal parenchymal embolization, compression or kinking of associated renal artery branches, and hemodynamic changes from turbulent blood flow within the aneurysm resulting in decreased distal renal artery perfusion pressures.[9,27,49,53,54] Whatever the etiology, most series suggest improvement or cure in the majority of hypertensive patients undergoing RAA reconstruction, particularly if renovascular hypertension is identified during the preoperative workup.[14,31,32,36-39,49,51,52,55] Martin et al[36] evaluated for renovascular hypertension preoperatively and demonstrated that 100% of those operated on with documented renovascular hypertension improved or were cured of hypertension, whereas only 60% of those with an unremarkable workup for stenosis were cured or improved. Pfeiffer et al[50] similarly noted a differential improvement in hypertension after aneurysm repair in those with documented renal artery stenosis (67%) in comparison to those without stenosis (29%).

Distal parenchymal embolization has been described in 8% to 11% of patients with RAA and may be associated with the presence of mural thrombus within the aneurysm sac.[49,50] Duplex ultrasound may demonstrate evidence of microembolization, although elevated resistance in this setting may be difficult to differentiate from nephrosclerosis.[27]

The variable conventional in situ reconstructions available include aneurysm resection with primary angioplastic closure (with or without branch reimplantation), patch angioplasty, primary reanastomosis, interposition bypass, aortorenal bypass, splanchnic-renal bypass, and plication of small aneurysms. Surgery consistently offers low elective mortality rates that approach 0% across series.[14,32,34,37-39,49,51,56-58] Perioperative morbidity is not insignificant and is mainly reported <20% after in situ reconstruction, and secondary nephrectomy rates, when reported, are 0% to 21%.[14,31,32,34,37-39,49,51] In addition, the reported durable primary patency rates (93%-100%) are of utmost importance in light of the young age of many patients and excellent projected long-term survival that averages 90% at 10 years.[31,32,49,59]

The technical approach selected should be dictated entirely by the patient's arterial and aneurysmal anatomy. Whereas there are no prospective or randomized data comparing open surgical techniques, Henke et al[37] noted no difference in long-term event-free outcome of patients undergoing aneurysmectomy with angioplastic closure or aneurysmectomy with bypass, with mean life span calculated at 108 and 130 months, respectively. Pfeiffer et al[50] demonstrated superior patency rates for angioplastic repairs in comparison to those reconstructions requiring saphenous vein interposition (100% vs 73%). Moreover, this technique yields no recurrent aneurysmal degeneration with follow-up.[37,50] Whereas cooled (4°) renal perfusion supplemented with mannitol or prostaglandin E has been advocated by several authors either routinely or

3. Treatment options

Recommendations for treatment of RAA			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	We suggest daily antiplatelet therapy (ie, low-dose aspirin) for patients with RAA.	2 (Weak)	C (Low)
3.2	We suggest open surgical reconstructive techniques for the elective repair of most RAAs in patients with acceptable operative risk.	2 (Weak)	B (Moderate)
3.3	We suggest ex vivo repair and autotransplantation for complex distal branch aneurysms over nephrectomy when it is technically feasible.	2 (Weak)	B (Moderate)
3.4	We suggest endovascular techniques for the elective repair of anatomically appropriate RAAs to include stent graft exclusion of main RAAs in patients with poor operative risk and embolization of distal and parenchymal aneurysms.	2 (Weak)	B (Moderate)
3.5	We suggest consideration of laparoscopic and robotic techniques as an interventional alternative based on institutional resources and surgeon experience with minimally invasive techniques.	2 (Weak)	C (Low)

when >30 to 40 minutes of warm renal ischemia is anticipated to reduce the risk of acute tubular necrosis, there are no prospective or randomized data to support this practice.[37,49,50,60]

Although historically treated with nephrectomy, current data support that complex distal branch lesions are best approached with ex vivo repair and autotransplantation. The largest series of such follow. Murray et al[61] have described a 92% success rate with in situ bifurcation and ex vivo multibranch replacement with branched and unbranched internal iliac artery autograft in 12 patients without mortality or major morbidity. Gallagher et al[62] reported on seven ex vivo reconstructions after laparoscopic nephrectomy for complex aneurysmal disease to avoid incisional morbidity; these authors described excellent technical success, no mortality, no ureteral morbidity, and 28% incidence of perioperative morbidity. Chandra et al[38] compared in situ and ex vivo reconstructions for renal aneurysm across 10 patients and noted no significant difference in hospital length of stay, morbidity (20%), mortality (null), or need for reoperation at follow-up. In addition, 100% of reconstructions were patent by imaging obtained during the first year of follow-up.

Case reports and small series that suggest indications for endovascular repair have broadened with the introduction of three-dimensional detachable coils, nonadhesive liquid embolic agents (eg, Onyx [Medtronic, Irvine, Calif]), remodeling techniques (which include balloon- and stent-assisted coiling), and flow diverter stents (eg, the Cardiatis multilayer stent [Cardiatis, Isnes, Belgium]), although traditional endovascular therapies have simply used embolization techniques for distal and parenchymal aneurysms and stent graft exclusion for main renal artery lesions.[63-70] Technical success across larger series is reported as 73% to 100%, with highly variable rates of morbidity (13%-60%) that include mainly evidence of end-organ malperfusion radiographically from thromboembolism and subsequent postembolectomy syndrome.[6,34,71-75] Midterm follow-up is available out to 3 to 4 years in certain series.[72,74,76] Access-related complication, arterial dissection, and renal compromise are uncommon, and low rates of recanalization have been observed requiring reintervention (4%-13%).[6,71-75]

Prospective comparisons of open and endovascular therapies for RAA are needed, although the feasibility of such a trial is limited primarily by the low frequency of this pathologic process. To this end, there have been retrospective comparisons. A recent statewide database review identified 215 patients who underwent RAA repair between 2000 and 2006.[33] These authors noted a significant increase in the number of RAA repairs with a stable number of open repairs and an increase in the number of endovascular repairs. Analysis of in-hospital outcome events revealed similar mortality rates (1.1% endovascular vs 3.3% open) and variable patterns of perioperative morbidity; open repair was associated with more cardiac ($P = .053$) and infectious ($P = .053$) complications, whereas endovascular repair was associated with more hemorrhagic complications ($P = .08$), presumed to be access related. Importantly, a significant reduction in median hospital length of stay and need for postdischarge nursing services was identified in the endovascular cohort accompanied by a trend toward lower cost. Additional retrospective comparisons of open and endovascular procedures have reported no significant difference in mortality, perioperative morbidity, freedom from reintervention, or decline in renal function and the benefit of a shortened length of stay.[2,14,34]

Robot-assisted laparoscopy techniques for RAA repair have only more recently been described.[77-80] This approach typically requires a multidisciplinary collaborative procedural team of vascular, general, transplantation, and urology surgeons. The dexterity of the robotic arms reportedly confers a technical advantage to complex aneurysmectomy and intracorporeal vascular suturing for reconstruction. Technical success has been reported in case series with warm ischemia times between 15 and 60 minutes; however, a direct comparison to open and endovascular techniques has yet to be performed.[77,78,80]

4. Additional screening

Recommendations for treatment of RAA			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	We suggest screening female patients with RAA for fibromuscular dysplasia with a focused history and one-time axial imaging study (ie, CTA or MRA) to assess for cerebrovascular, mesenteric, and iliac artery dysplasia.	2 (Weak)	C (Low)

RAAs are associated with fibromuscular dysplasia in up to 68% of cases and concomitant arterial aneurysms affecting the aorta and visceral and iliac vessels in 7% to 30% of cases.[14,31,34,36,37,39,40,50,51,58] The most recent scientific statement from the American Heart Association recommends a focused vascular review of symptoms for all patients diagnosed with fibromuscular dysplasia, with an emphasis on quality of life-impairing symptoms

like migraine headache, tinnitus, and neck pain.[81] In addition, one-time screening for occult aortic or arterial aneurysm in these patients is recommended.

5. Follow-up and surveillance

Recommendations for treatment of RAA			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	We suggest completion imaging after open surgical reconstruction for RAA, before hospital discharge, by way of axial imaging with CTA or MRA or arteriography in select cases, and long-term follow-up with surveillance imaging.	2 (Weak)	C (Low)
5.2	For patients managed nonoperatively, we suggest annual surveillance imaging until two consecutive studies are stable; thereafter, surveillance imaging may be extended to every 2 to 3 years	2 (Weak)	B (Moderate)

Most earlier series advocate completion imaging before hospital discharge by way of arteriography or ultrasound. In the more modern era, this can probably be replaced with axial imaging with CTA or MRA, obviating the limitations of ultrasound in the postoperative period and the invasiveness of angiography and long-term follow-up with surveillance imaging.[9,31,37,49,50,61]

The natural history of RAAs appears to be that of slow to null growth. Most recent estimates suggest a median annualized growth rate of 0.06 to 0.6 mm.[14,39,40] The most recent and largest multi-institutional retrospective series of nonoperative RAA surveillance found no difference in growth rate based on aneurysm morphology or calcification.[14] Whereas short-term follow-up at 1 year remains prudent for a newly diagnosed RAA, longer intervals between surveillance imaging may be appropriate, provided the patient's compliance with follow-up can be ensured.

SPLENIC ARTERY ANEURYSM (SAA)

Although they were once thought to be uncommon, splenic and other visceral artery aneurysms are being diagnosed with increasing frequency with the use of advanced imaging techniques.[82,83] The increasing diagnosis of incidental SAAs in the United States is primarily related to the liberal use of cross-sectional imaging studies.[84] Most SAAs currently are detected incidentally during diagnostic imaging performed for other indications.[83] Whereas plain radiography, ultrasound, CT, MRA, and arteriography have identified SAAs, CTA remains the initial diagnostic and evaluative tool of choice.[83]

CTA is an important imaging modality for the vascular system and has been established as the method of choice for the diagnosis, treatment planning, and follow-up of most diseases of the abdominal arteries including the aorta and visceral vessels. Besides being able to assess the location and size of the SAA, CTA may also reveal rupture, intra-abdominal hemorrhage, and associated underlying diseases. In addition, nearly all necessary data for planning endovascular treatment can be obtained by multidimensional CTA.[83] Ultrasound examination of visceral vessels is inhibited by shadowing from bowel gas as well as by obesity; however, its sensitivity to detect SAA <3 cm is poor.[83,85] MRA may certainly play a role in patients in whom CTA is contraindicated. However, in 2006, Pilleul et al[86] compared the use of MRA and CTA for the analysis of splanchnic aneurysms and thought that the sensitivity of MRA was suboptimal in the case of small aneurysms.

Formal arteriography may rarely be necessary for preinterventional planning when noninvasive studies have not

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of SAA			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	We recommend CTA as the initial diagnostic tool of choice for SAAs, with 1-mm-thickness sections if available.	1 (Strong)	C (Low)
1.2	In patients with suspected SAAs and pre-existing renal insufficiency limiting the use of iodinated contrast material, we recommend MRA to establish diagnosis.	1 (Strong)	C (Low)
1.3	We recommend using arteriography when noninvasive studies have not sufficiently demonstrated the status of relevant collateral blood flow and when endovascular intervention is planned.	1 (Strong)	B (Moderate)

sufficiently demonstrated the status of relevant anatomy or collateral blood flow. However, it is most frequently performed at the time of a planned endovascular intervention.

2. Treatment indications, size criteria, and true vs false aneurysms

Recommended criteria for invasive intervention for SAA			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	We recommend emergent intervention for ruptured SAAs.	1 (Strong)	A (High)
2.2	We recommend treatment of nonruptured splenic artery pseudoaneurysms of any size in patients of acceptable risk because of the possibility of rupture.	1 (Strong)	B (Moderate)
2.3	We recommend treating nonruptured splenic artery true aneurysms of any size in women of childbearing age because of the risk of rupture.	1 (Strong)	B (Moderate)
2.4	We recommend treating nonruptured splenic artery true aneurysms ≥3 cm, with a demonstrable increase in size, or with associated symptoms in patients of acceptable risk because of the risk of rupture.	1 (Strong)	C (Low)
2.5	We suggest observation over repair for small (<3 cm), stable asymptomatic splenic artery true aneurysms or those in patients with significant medical comorbidities or limited life expectancies.	2 (Weak)	C (Low)

The natural history of visceral aneurysms and their potential for rupture or other complications are relatively poorly defined because of their overall scarcity. However, it is clear even from the numerous case series in the literature that a significant proportion of visceral artery aneurysms present with rupture; therefore, a relatively aggressive approach to their diagnosis and management certainly seems warranted. The overall mortality of ruptured SAAs is as high as 25%.[87]

SAAs in young women are thought to have a particular tendency toward rupture, especially during the third trimester of pregnancy.[88] Pregnancy may be associated with 20% to 50% of all ruptures.[89] Rupture of an SAA during pregnancy has devastating maternal and fetal mortality rates of 80% and 90%, respectively.[90,91]

Depending on the entirety of vessel wall involvement, SAAs can be subdivided into true aneurysms or pseudoaneurysms. Splenic artery pseudoaneurysms certainly have a higher rupture potential than true aneurysms. Furthermore, some evidence suggests that pseudoaneurysms display relatively rapid growth rates, implicating a focus on early intervention regardless of size. In the series by Tulsyan et al,[1] in which 48 visceral aneurysms were treated, 80% of 28 pseudoaneurysms were symptomatic at presentation as opposed to 30% of 20 true aneurysms. Pitton et al[92] reported a review of 233 patients with 253 visceral artery aneurysms. The rate of rupture at presentation was noted to be significantly higher in visceral pseudoaneurysms than in true aneurysms (76.3% vs 3.1%). There were 35 ruptures in their series. There was no significant difference in size between ruptured and nonruptured visceral aneurysms. The authors concluded that visceral pseudoaneurysms are at increased risk for rupture and that diameter is not necessarily a reliable predictor of rupture.[92]

Nevertheless, several large case series of SAA management have included an observation cohort with acceptable results.[93-95] General guidelines state that true SAAs <3 cm, asymptomatic, and showing little or no growth can be safely observed and monitored with serial imaging studies. As indicated before, splenic artery pseudoaneurysms and SAAs in women who are either pregnant or of childbearing age should be treated regardless of size.[90,96] There is no firm evidence to show that aneurysm calcification protects against growth or rupture, but calcified SAAs may be associated with smaller size at initial diagnosis.[94]

A large review was reported from the Mayo Clinic involving 217 patients with SAAs. Of these patients, 168 underwent nonoperative management for a mean period of 75 months.[95] The mean size in the nonoperative group was 2.1 cm with a range from 0.8 to 5 cm in diameter. Approximately half of these aneurysms were monitored with serial imaging, of which only 10% were noted to have growth averaging 0.06 cm/y. No rupture or other complications related to the SAAs occurred, and only 3 of the original 168 required eventual intervention because of aneurysm growth. Similar results were reported by Lakin et al[94] in review of the Cleveland Clinic experience of 128 SAAs managed during a 15-year period. This observational cohort of 66 SAAs had a mean size of 1.7 cm at presentation with a range from 0.8 to 4.2 cm. Serial imaging was available for 94% of the aneurysms, and these patients received an average of 2.5 CT scans during 4.6 years of follow-up. The average growth rate was a nominal 0.2 mm/y during 3.1 years of follow-up. There were again no ruptures or other complications attributed to the aneurysms in the observed group.

Observation of small, <3-cm, true aneurysms therefore seems an appropriate approach except in women of childbearing age.

Additional suggested indications for intervention in SAAs include patients with portal hypertension, patients who may require liver transplantation, patients whose aneurysm has a nonatherosclerotic or nondegenerative cause, and patients whose aneurysm demonstrates interval growth >0.5 cm/y.[97]

3. Treatment options

Recommendations for treatment of SAA			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	In patients with ruptured SAA discovered at laparotomy, we suggest treatment with ligation with or without splenectomy, depending on the aneurysm location.	2 (Weak)	B (Moderate)
3.2	In patients with ruptured SAA diagnosed on preoperative imaging studies, we suggest treatment with open surgical or appropriate endovascular techniques based on the patient's anatomy and underlying clinical condition.	2 (Weak)	B (Moderate)
3.3	We suggest elective treatment of SAA using an endovascular approach if it is anatomically feasible. However, elective treatment may appropriately involve open surgical, endovascular, or laparoscopic methods of intervention, depending on the patient's anatomy and underlying clinical condition.	2 (Weak)	B (Moderate)
3.4	In treatment of SAA, we suggest that the splenic artery does not routinely require preservation or revascularization.	2 (Weak)	C (Low)
3.5	In treatment of distal SAA adjacent to the hilum of the spleen, we suggest open surgical techniques including possible splenectomy as opposed to endovascular methods, given concern for the possibility of end-organ ischemia, including splenic infarction and pancreatitis.	2 (Weak)	C (Low)
3.6	In pregnant women with SAA, treatment decisions should be individualized regardless of size, and the potential morbidity to both the mother and fetus should be considered.	Ungraded best practice statement	

Treatment of SAA can generally be accomplished by either open surgical or endovascular approaches. In areas of the visceral circulation with an abundance of collateral flow, as in the splenic artery, proximal and distal ligation of the aneurysm segment is a viable surgical option. This can also be accomplished with endovascular isolation of the aneurysmal segment, either by placement of a stent graft or by coil embolization of the proximal and distal arterial segment. The preferred treatment of an individual patient and aneurysm must be carefully based on the particular anatomy and any associated clinical conditions as well as the underlying condition of the patient.

The traditional surgical management of SAA includes proximal and distal ligation and aneurysmectomy for lesions in the proximal or middle portion of the splenic artery. Revascularization of the distal splenic artery is generally not warranted because collateral flow to the spleen is maintained by the short gastric arteries. For more distal lesions adjacent to the splenic hilum and for mycotic aneurysms, excision with or without splenectomy has traditionally been the most commonly performed operation. This is clearly required when the aneurysm involves intrasplenic branches within the splenic parenchyma. Distal pancreatectomy may occasionally be warranted in treatment of these distal lesions as well.[98,99] Laparoscopic repair of SAA by clipping or exclusion has been reported; intraoperative ultrasound is believed to be an important adjunct to this procedure.[100] Combined laparoscopic occlusion and coil embolization has been proposed as a treatment method for aberrant SAA located in the retropancreatic position, in which traditional surgical exposure would be exceedingly difficult.[101]

Endovascular approaches to managing visceral artery aneurysms offer the benefit of low procedural morbidity and mortality and are generally considered to be the preferred initial approach to most anatomically suitable visceral aneurysms considered appropriate for intervention.[1] Endoluminal ablation of SAAs has been shown in multiple reported series to be highly technically successful, but there is some concern of end-organ malperfusion and aneurysm reperfusion during follow-up.[1,102]

Endovascular treatment options include coil embolization of the splenic artery both proximal and distal to the aneurysm itself, effectively "trapping" the lesion. Other options for a saccular-type aneurysm include embolization of the aneurysm sac itself with coils or cyanoacrylate glue and occlusion of the lesion with percutaneous or open thrombin injection.[103] In addition, stent grafting has been performed, particularly for saccular lesions of the mid splenic artery. There has been concern about splenic infarction or pancreatitis when embolization of

very distal splenic artery lesions has been performed.[104,105] In a review of 48 endovascular procedures for visceral artery pseudoaneurysms, 20 interventions on the splenic artery were performed.[1] Six end-organ infarcts in this series were identified; all were within the splenic bed. Two additional patients displayed splenic atrophy on CT scanning after prior embolization of the splenic artery, without obvious clinical evidence for initial splenic infarction. In another report, one episode of splenic infarction associated with severe pancreatitis was noted after embolization of a distal splenic artery lesion.[105] However, other authors have noted splenic infarction after embolization of even more proximal SAAs as well.[7]

Ruptured SAAs are challenging and represent a true surgical emergency. Patients should be expeditiously transferred to the operating room for exploratory laparotomy in the setting of hemodynamic collapse. Ligation of the splenic artery proximally and distally is required. Patients presenting with ruptured SAA are most often treated with concomitant splenectomy without vascular reconstruction.

superior to open surgery in this model. However, the authors concluded that elderly patients should be considered for conservative therapy, given the small incremental benefit and high cost. An open surgical approach may remain appropriate in cases of rupture and pregnancy-related SAAs[107] as well as in the setting of mycotic aneurysms.

Patients undergoing urgent ligation of SAA or splenectomy should be vaccinated on or after postoperative day 14 to decrease the risk of overwhelming postsplenectomy sepsis due to organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae* type B, and *Neisseria meningitidis*. Patients undergoing elective repair of SAA who are at risk of splenic loss if the splenic artery cannot be preserved with planned embolization or with known preoperative anatomic challenges to stent grafting that could lead to fallback embolization should be considered for vaccination at least 14 days BEFORE intervention.

4. Screening

Recommendations for screening of patients with SAA			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	We suggest screening of patients with SAAs for other intra-abdominal, intrathoracic, intracranial, and peripheral artery aneurysms.	2 (Weak)	B (Moderate)

The immediate benefits associated with endovascular intervention include local anesthesia, shorter hospital stay, and faster recovery. Drawbacks to endovascular therapy include end-organ embolization and a relatively higher rate of failure compared with open surgery. Last, patients with splenic artery ablation for SAA can develop postembolization syndrome with ongoing pain, fevers, and other systemic symptoms. Complete exclusion of flow within the aneurysm sac occurred in 97% of interventions with follow-up imaging in the Cleveland Clinic experience. In the Mayo Clinic experience, initial intervention was successful 98% of the time. Coiling was used alone in 75% of the cases and in combination with at least one other technique in 11% of cases.

The risk of end-organ ischemia appears to be an especially salient concern pertaining to endovascular repair. Some authors have concluded that patients with aneurysmal disease at the splenic hilum may be better managed with open repair and splenectomy.[105]

A study using a Markov decision model compared open surgery vs endovascular repair vs conservative therapy for a patient with SAA.[106] Endovascular repair was found to be associated with highest quality of life and to be the most cost-effective strategy for most groups, and it was

SAAs may be multiple and may be found in association with other visceral and nonvisceral aneurysms. In a review of 212 cases of SAAs seen during a two-decade period at a single institution, 3.3% of patients were found to have concomitant visceral aneurysms, with the most common location being extrahepatic (2.3%) and additionally including aneurysms in the celiac, superior mesenteric, gastric, and pancreaticoduodenal territories.[108] In addition, 14.3% of patients were found to have concomitant nonvisceral aneurysms, with the most common location being renal (7.4%) and abdominal aortic (3.7%) and additionally including aneurysms in the carotid, intracerebral, thoracic aortic, and popliteal territories.[108]

SAAs selected for conservative management with observation require surveillance to assess for progression and size growth. The mode of assessment and the appropriate imaging technique depend on the visibility of the aneurysm to the various imaging techniques available.[84] CT or ultrasound should be performed every 12 months; CT is generally preferred because of the detailed cross-sectional imaging provided, and it can be done with or without contrast enhancement.[84]

This strategy appears to be safe on the basis of the literature described earlier from the Mayo Clinic[94] and the

5. Surveillance and follow-up

Recommendations for surveillance and follow-up of SAA patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	In patients in whom an SAA is being observed with a nonoperative or noninterventional approach, we suggest annual surveillance with CT or ultrasound to assess for growth in size.	2 (Weak)	B (Moderate)
5.2	After endovascular intervention for SAAs, we suggest periodic surveillance with CTA, ultrasound, or MRA to assess for the possibility of endoleak or aneurysm reperfusion that could lead to a continued risk of aneurysm growth or rupture.	2 (Weak)	B (Moderate)

Cleveland Clinic.[93] There were again no ruptures or other complications attributed to the aneurysms in the observed group.

In the study of Saltzberg et al,[105] a total of 38 visceral aneurysms were observed during a mean follow-up period of 31 months, including 5 celiac aneurysms and 25 splenic aneurysms. The mean diameter in the observation group was 2 cm. No known ruptures or interventions occurred in the observation group, but complete follow-up was available in only 50% of the patients.

Treatment failure including persistent perfusion, recanalization, and coil migration has been observed. The potential for early or late failure, such as growth in sac size or leak that would require reintervention, requires early and serial imaging follow-up.[93,97] Because artifact due to the radiopaque materials used may make sensitive CT evaluation difficult, some authors recommend contrast-enhanced ultrasound or MRI as follow-up imaging techniques.[6,97]

CELIAC ARTERY ANEURYSM (CAA)

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of CAA			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	We suggest CTA as the initial diagnostic tool of choice for CAAs.	2 (Weak)	B (Moderate)
1.2	We suggest MRA in patients with suspected CAA and pre-existing renal insufficiency limiting the use of iodinated contrast material.	2 (Weak)	B (Moderate)
1.3	We suggest arteriography when noninvasive studies have not sufficiently demonstrated the status of relevant collateral blood flow or when endovascular intervention is planned.	2 (Weak)	C (Low)

Continued surveillance, even after secondary technical success, is imperative as the natural history of visceral artery aneurysms after endovascular treatment remains unclear. This is especially true of saccular aneurysms treated with coil or thrombin embolization; unlike formal exclusion with a covered stent, these aneurysms are not technically "excluded" from arterial circulation. Indeed, sac thrombosis may not protect the aneurysm sac from pressure transmitted through thrombus, and eventual sac growth or rupture may still occur.[109,110] Reports of reperfusion and even rupture after "successful" embolization of visceral aneurysms support the notion that a thrombosed aneurysm may not represent the definitive treatment in all cases.[111,112] Other authors also noted that regular follow-up with duplex ultrasound or CT is necessary for patients with visceral aneurysms treated by embolization.[113] The recanalization rates after endovascular intervention were 18% and 30%, respectively, in two reported series.[111,114]

Although they were once thought to be uncommon, CAAs are being diagnosed with increasing frequency with the use of advanced imaging techniques.[82,83] The increasing diagnosis of incidental visceral aneurysms in the United States is primarily related to the liberal use of cross-sectional imaging studies.[84] Most visceral aneurysms currently are detected incidentally during diagnostic imaging performed for other indications.[83] Whereas plain radiography, ultrasound, CT, MRA, and arteriography have identified CAAs, CTA remains the initial diagnostic and evaluative tool of choice.[83]

Besides being able to assess the location and size of the CAA, CTA may also reveal rupture, intra-abdominal hemorrhage, and associated underlying diseases as well as celiac axis stenosis, which should be systematically evaluated on axial imaging of patients with CAAs. In addition, nearly all necessary data for planning endovascular treatment can be obtained by multidimensional CTA.[83]

Ultrasound examination of visceral vessels is inhibited by shadowing from bowel gas as well as by obesity; however, its sensitivity to detect CAA of <3 cm is poor.[83,85] MRA may certainly play a role in patients in whom CTA is contraindicated. However, in 2006, Pilleul et al[86] compared the use of MRA and CTA for the analysis of splanchnic aneurysms and believed that the sensitivity of MRA was suboptimal in the case of small aneurysms.

Formal arteriography may rarely be necessary for preinterventional planning when noninvasive studies have not sufficiently demonstrated the status of relevant anatomy or collateral blood flow. However, it is most frequently performed at the time of a planned endovascular intervention.

that CAAs after spontaneous dissection behave differently, and no distinct recommendation for this subgroup can be made. Similarly, patients with post-stenotic dilation of the celiac artery due to compression by the median arcuate ligament have a different pathologic process and require individualized decision-making. They should be treated conservatively unless they become symptomatic or are determined to have true aneurysmal degeneration.

Like other visceral aneurysms, pseudoaneurysms of the celiac artery appear more prone to rupture than true aneurysms, and most authors recommend treatment of all visceral pseudoaneurysms in any anatomic territory. In the series by Tulsyan et al[1] in which 48 visceral aneurysms were treated, 80% of 28 pseudoaneurysms were symp-

2. Treatment indications, size criteria, and true vs false aneurysms

Recommended intervention criteria for CAA			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	We recommend emergent intervention for ruptured CAAs.	1 (Strong)	A (High)
2.2	We recommend treatment of nonruptured celiac artery pseudoaneurysms of any size in patients of acceptable operative risk because of the possibility of rupture.	1 (Strong)	B (Moderate)
2.3	We recommend treatment of nonruptured celiac artery true aneurysms >2 cm, with a demonstrable increase in size, or with associated symptoms in patients of acceptable risk because of the risk of rupture.	1 (Strong)	C (Low)
2.4	We suggest observation over intervention for small (<2 cm), stable asymptomatic CAAs or those in patients with significant medical comorbidities or limited life expectancy.	2 (Weak)	C (Low)

Numerous case reports and series in the literature have shown that a significant proportion of CAAs present with rupture. Therefore, an aggressive approach to their diagnosis and management certainly seems warranted. Reported mortality after ruptured CAA approaches 100%.

CAAs appear to have a strong tendency to rupture with a resultant high mortality rate; it was reported that 33 of 34 patients with CAA diagnosed in 1943 died of rupture.[115] The overall reported risk for rupture appears to range from 10% to 20%.[116,117] The majority of patients with CAA have been described as symptomatic at presentation, although occult asymptomatic aneurysms are more likely to be diagnosed radiologically in the current era. Rupture has been reported to occur in approximately 5% of celiac trunk aneurysms ranging from 15 to 22 mm in diameter and in 50% to 70% of those that exceed 32 mm in diameter.[118,119]

There are no absolute size criteria with which to direct the indication for treatment; however, treatment of lesions >2 cm seems appropriate. Despite this, in a reported series of 18 CAAs in which nonoperative management was performed in 8 asymptomatic patients, only one subsequent rupture was noted to occur.[117] The other observed CAAs had no evidence of enlargement or rupture during a mean 91-month follow-up period.[117] In patients with spontaneous dissection of the visceral arteries, aneurysmal degeneration is rare.[120,121] There is no evidence to suggest

tomatic at presentation as opposed to 30% of 20 true aneurysms. Pitton et al[92] reported a review of 233 patients with 253 visceral artery aneurysms. The rate of rupture at presentation was noted to be significantly higher in visceral pseudoaneurysms than in true aneurysms (76.3% vs 3.1%). There were 35 ruptures in their series. There was no significant difference in size between ruptured and nonruptured visceral aneurysms. The authors concluded that visceral pseudoaneurysms are at increased risk for rupture and that diameter is not necessarily a reliable predictor of rupture.[92]

In an additional series of 155 patients with visceral aneurysms, Guo et al[122] found no significant difference of the diameters between symptomatic and asymptomatic patients (36.9 vs 33.6 mm). Pseudoaneurysms were more common in the patients who presented with rupture. Symptomatic patients had a significantly higher 30-day mortality.[122] Celiac aneurysms were more likely to be in the symptomatic group, whereas splenic aneurysms were more likely to be in the asymptomatic group. Most of the ruptures were in pseudoaneurysms (80%).[122] Of a total of eight celiac lesions, five were symptomatic. Tetreau et al[123] reviewed 112 patients with splanchnic aneurysms, including 44 splenic aneurysms and 6 celiac aneurysms. Of the celiac aneurysms, one was ruptured at a diameter of 17 mm.

In the appropriate clinical scenario, specifically in patients with pancreatitis or infected pancreatic pseudocysts, coexisting CAAs should be suspected to be mycotic in nature. Although the literature is sparse in this subgroup, they should be excised regardless of size, given the high risk of progression to rupture.

Aneurysmorrhaphy for isolated saccular lesions of the celiac artery has also been reported.[87]

Endovascular approaches to managing CAAs offer the potential benefit of low procedural morbidity and mortality and are generally considered to be the preferred initial approach to most anatomically suitable visceral an-

3. Treatment options

Recommendations for treatment of CAA			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	In patients with ruptured CAA discovered at laparotomy, we suggest ligation if sufficient collateral circulation to the liver can be documented.	2 (Weak)	C (Low)
3.2	In patients with ruptured CAA diagnosed on preoperative imaging studies who are stable, we recommend treatment with open surgical or appropriate endovascular methods based on the patient's anatomy and underlying clinical condition.	1 (Strong)	B (Moderate)
3.3	For the elective treatment of CAA, we suggest using an endovascular intervention if it is anatomically feasible. However, elective treatment may appropriately involve open surgical, endovascular, or laparoscopic methods of intervention, depending on the patient's anatomy and underlying clinical condition.	2 (Weak)	B (Moderate)
3.4	To determine the need for revascularization of the celiac artery and its branches in treating CAA, we suggest evaluating the status of the superior mesenteric artery, gastroduodenal artery, and other relevant collateral circulation, which must be carefully documented on preoperative CTA or angiography.	2 (Weak)	B (Moderate)

Historically, surgical treatment of CAAs was the only feasible option for management. Open surgical options included aneurysmectomy, aneurysmorrhaphy, aortoceliac or aortohepatic bypass, and ligation.[98,116] The necessity of celiac or celiac branch revascularization depends on several factors, including the location of the aneurysm and the nature of the collateral mesenteric circulation. Simple ligation of the celiac artery is a viable option in a meaningful proportion of cases and has been undertaken in as much as 35% of reported surgically treated cases in the literature.[116] Ligation of the celiac artery is reportedly well tolerated in most cases but may be problematic in patients with underlying hepatic disease. Ligation can be used for emergency treatment of celiac trunk aneurysms with a relatively low risk of hepatic ischemia.[113] When the celiac artery is ligated, collateral flow is provided by the superior mesenteric, pancreaticoduodenal, and gastroduodenal arteries. The standard surgical approach involving revascularization is celiac aneurysmectomy with aortoceliac bypass grafting, most commonly using prosthetic materials.[117] In one reported series of nine patients undergoing elective open surgical repair, revascularization was performed in 89%.[117]

eurysms considered appropriate for intervention.[1] Reported successful endovascular techniques have included coil or glue embolization, percutaneous or open thrombin injection, and endovascular stent grafting.[3,124-129] One report of five cases of endovascular occlusion of CAA revealed no ischemic sequelae and uniformly good technical results.[130] Late coil migration into the stomach with development of a fatal aortogastric fistula has been reported after coil embolization of a CAA.[131] However, there are significantly fewer reports in the literature of CAAs being treated by endovascular techniques compared with SAAs. Because CAAs typically involve the proximal portion of the celiac trunk, absence of a proximal landing zone may limit endovascular treatment with coils.[99,107] This can be potentially achieved with embolization using endovascular plugs, which can allow a more controlled and precise occlusion of the celiac axis.

Roberts et al[132] reported on a series of emergently treated hemorrhaging celiac or mesenteric artery aneurysms using an initial endovascular approach to all patients in whom the diagnosis was made on preoperative imaging studies. Ultimately, open surgical treatment was required in <5% of cases.

4. Screening

Recommendations for screening of patients with CAA			
	Recommendation	Strength of Recommendation	Quality of Evidence
4.1	We suggest screening patients with CAAs for other arterial aneurysms.	2 (Weak)	B (Moderate)

CAAs are associated with other visceral artery aneurysms in 40% of cases and with aortic aneurysms in 20% of cases.[133,134] Of patients with a CAA, 18% to 20% will have an aortic aneurysm, and 18% to 38% will have an additional visceral artery aneurysm.[135] Peripheral artery aneurysms are seen in 18% to 67% of patients.[117]

5. Follow-up and surveillance

Recommendations for follow-up and surveillance of CAA patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	In patients in whom a CAA is being observed with a nonoperative or noninterventional approach, we suggest annual surveillance with CTA scans to assess for growth in size.	2 (Weak)	B (Moderate)
5.2.	After endovascular intervention for CAAs, we suggest periodic surveillance with appropriate imaging studies to assess for the possibility of endoleak or other continued aneurysm perfusion that could lead to a continued risk of aneurysm growth or rupture.	2 (Weak)	B (Moderate)

In a reported series of 18 CAAs in which nonoperative management was performed in 8 asymptomatic patients, only one subsequent rupture was noted to occur.[117] The other observed CAAs had no evidence of enlargement or rupture during a mean 91-month follow-up period.[117] Because rupture of CAAs has been reported to occur at diameters <20 mm, close radiologic follow-up of those selected for observation with yearly CT scans is appropriate.

Continued surveillance, even after secondary technical success, is imperative as the natural history of visceral artery aneurysms after endovascular treatment remains unclear. This is especially true of saccular aneurysms treated with coil or thrombin embolization; unlike formal exclusion with a covered stent, these aneurysms are not technically "excluded" from arterial circulation. Indeed, sac thrombosis may not protect the aneurysm sac from pressure transmitted through thrombus, and eventual sac growth or rupture may still occur.[109,110] Reports of reperfusion and even rupture after "successful" embolization of visceral aneurysms support the notion that a thrombosed aneurysm may not represent the definitive treatment in all cases.[111,112] Regular follow-up with duplex ultrasound or CT is necessary for patients with visceral aneurysms treated by embolization because of the risk of recanalization.[111-114]

Treatment failures including persistent perfusion, recanalization, and coil migration have been observed. The potential for early or late failure, such as growth in sac size or leak that would require reintervention, requires early and serial imaging follow-up.[93,97] Because artifact due to the radiopaque materials used may make sensitive CT evaluation difficult, some authors recommend contrast-enhanced ultrasound or MRI as follow-up imaging techniques.[6,97]

GASTRIC AND GASTROEPIPLOIC ARTERY ANEURYSMS

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of gastric and gastroepiploic artery aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	In patients who are thought to have gastric or gastroepiploic artery aneurysms, we recommend CTA as the diagnostic tool of choice	1 (Strong)	B (Moderate)
1.2	In patients who are thought to have gastric or gastroepiploic artery aneurysms and have high radiation exposure risks or renal insufficiency, we recommend non-contrast-enhanced MRA for diagnosis.	1 (Strong)	C (Low)
	Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).		
1.3	We recommend the use of catheter-based angiography for all emergent cases presenting with rupture (Grade 1B) and electively for preoperative planning (Grade 1C).	1 (Strong)	B (Moderate) C (Low)

Gastric and gastroepiploic artery aneurysms account for approximately 4% to 5% of all visceral aneurysms, affecting men more frequently than women (three to one).[87] Etiologic risk factors include primarily arterial dysplasia with segmental arterial mediolysis and periarterial inflammation like pancreatitis and vasculitis; atherosclerosis, when present, is believed to be a secondary process.[87,111,136] Axial imaging, ideally with CTA, remains the diagnostic study of choice as it detects incidental and asymptomatic aneurysms and guides surgical and endovascular planning.[137,138]

supports catheter-based embolization of gastric and gastroepiploic artery aneurysms as the new standard of care, with multiple case reports and small series documenting successful aneurysm occlusion with coils and thrombin injection.[1,2,141-143] Embolization for gastric and gastroepiploic artery aneurysms, even ruptured, offers >90% technical success and low morbidity and mortality. Stent grafting has been described but remains anecdotal.[1,144]

Axial imaging as recommended will screen for concomitant nonaortic intra-abdominal arterial aneurysms, which are common.[136,139,145]

2. Size criteria for invasive intervention

Recommended criteria for invasive intervention in gastric and gastroepiploic aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	We recommend treatment of all gastric artery and gastroepiploic artery aneurysms of any size.	1 (Strong)	B (Moderate)

Gastric artery aneurysms are 10 times more common than gastroepiploic artery aneurysms. Although patients may present with abdominal pain, up to 90% of patients have historically presented acutely ruptured, with evidence of gastrointestinal bleeding more common than intraperitoneal rupture (1/3).[87,111] Treatment is recommended for all gastric and gastroepiploic artery aneurysms, regardless of size, in light of this rupture risk at relatively small aneurysm sizes.[139]

We recommend screening for cerebrovascular and coronary artery aneurysm when the diagnosis of segmental arterial mediolysis is suspected, given the systemic nature of this arterial dysplasia, with one-time screening CTA or MRA of the head, neck, and chest.[136,146]

We recommend routine interval surveillance (ie, every 12 months) with axial imaging (ie, CTA or MRA) in cases of segmental medial arteriolysis in light of reported cases of rapid arterial transformation.[147]

3. Treatment options

Recommended criteria for invasive intervention in gastric and gastroepiploic aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	We recommend endovascular embolization for first-line treatment of gastric artery and gastroepiploic artery aneurysms	1 (Strong)	B (Moderate)

Surgical management has historically relied on simple arterial ligation or excision without reconstruction, whereas intramural aneurysms required a wedge excision of involved gastric wall.[2,56,87,140] Contemporary literature

We recommend postembolization surveillance every 1 to 2 years with axial imaging to assess for vascular remodeling and evidence of aneurysm reperfusion.[1,148,149]

4. Screening for concomitant aneurysms

Recommendations for screening of gastric and gastroepiploic artery aneurysm patients			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	We recommend abdominal axial imaging to screen for concomitant abdominal aneurysms.	2 (Weak)	B (Moderate)
4.2	We recommend one-time screening CTA (or MRA) of the head, neck, and chest for those patients with segmental arterial mediolysis.	2 (Weak)	C (Weak)

5. Follow-up and surveillance

Recommendations for follow-up and surveillance of gastric and gastroepiploic artery aneurysm patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	We suggest interval surveillance (ie, every 12-24 months) with axial imaging (ie, CTA or MRA) in cases of segmental medial arteriolysis in light of reported cases of rapid arterial transformation.	2 (Weak)	B (Moderate)
5.2	We suggest postembolization surveillance every 1 to 2 years with axial imaging to assess for vascular remodeling and evidence of aneurysm reperfusion.	2 (Weak)	C (Moderate)

HEPATIC ARTERY ANEURYSM (HAA)

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of HAA			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	In patients who are thought to have HAA, we recommend CTA as the diagnostic tool of choice.	1 (Strong)	B (Moderate)
1.2	In patients with HAA who are considered for intervention, we recommend mesenteric angiography for preoperative planning.	1 (Strong)	B (Moderate)

HAA is the second most common type of visceral artery aneurysm reported.[56,98,134] The actual incidence of HAA is unknown; the commonly used incidence figures are derived from small case series, autopsy, and anecdotal evidence. In a large series reported from the Mayo Clinic, the incidence of HAA was noted to be 0.002% among the 2,091,965 patients seen at the Mayo Clinic between 1980 and 1986.[150] Most HAAs are diagnosed incidentally on CT performed for unrelated issues and are most commonly observed during the sixth decade of life with a 3:2 male predominance.[150]

Given the numerous collaterals from the gastroduodenal artery and right gastric branches, both open surgical ligation and endovascular embolization of these aneurysms are reported. To evaluate the collaterals, selective angiography and high-resolution CTA are recommended.

2. Size criteria for invasive intervention

Recommendations for diagnosis and evaluation of HAA			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	Given the high propensity of rupture and significant antecedent mortality, we recommend that all hepatic artery pseudoaneurysms, regardless of cause, be repaired as soon as the diagnosis is made.	1 (Strong)	A (High)
2.2.a	We recommend repair of all *symptomatic* HAAs regardless of size.	1 (Strong)	A (High)
2.2.b	In *asymptomatic* patients without significant comorbidity, we recommend repair if true HAA is >2 cm (Grade 1A) or if aneurysm enlarges >0.5 cm/y (Grade 1C). In patients with significant comorbidities, we recommend open repair if HAA is >5.0 cm (Grade 1B).	1 (Strong)	A (High)
			B (Moderate)
			C (Low)
2.3	We recommend repair of HAA in patients with vasculopathy or vasculitis, regardless of size (Grade 1C). We recommend repair in HAA patients with positive blood cultures (Grade 1C)	1 (Strong)	C (Low)

Hepatic artery pseudoaneurysm. False aneurysm of the hepatic artery accounts for 25%[93] to 80%[148] of reported cases and often occurs after iatrogenic injury or penetrating or blunt liver trauma, leading to symptomatic presentation of these aneurysms. These antecedent clinical events along with specific imaging distinguish false aneurysms from true aneurysms. Imaging findings, which include focal arterial disruption in the setting of otherwise normal arteries and inflammatory changes around an irregular aneurysm sac, are reported.[1] The majority of pseudoaneurysms are symptomatic at presentation, thereby differing from true aneurysms, with gastrointestinal bleeding or hemobilia.[1,148]

True HAA. The true natural history of HAA is unknown because of the rarity of these aneurysms, making any recommendation for repair of asymptomatic HAA controversial. In the series published by the Mayo Clinic, these aneurysms appeared to be relatively benign with a slow rate of enlargement and relatively uncommon rate of rupture.[150] Retrospective series of ruptured aneurysms suggest that the majority of these lesions rupture when they are >2 cm in diameter.[93] In one series, when patients were observed nonoperatively for 68 months, only 27% of patients showed enlargement of the aneurysm without any complication.[150] Given these findings, Abbas et al[150] recommended careful observation for aneurysms <5 cm among high-risk patients and repair for lesions >2 cm only among low-risk patients unless these aneurysms enlarge or become symptomatic. Given the significant high rate of morbidity and mortality after HAA rupture (30% mortality rate in one series[150]) and overall low rate of morbidity and mortality after elective HAA repair (0% mortality in the same series[150]), the current recommendation is for repair of aneurysms >2.0 cm in diameter in low-risk patients and >5.0 cm among high-risk patients if open repair is planned. In these patients, endovascular therapy with stent or coil embolization of the HAA can be contemplated at the same size criteria as in low-risk patients, provided there is adequate collateralization, given the low rate of comorbidities in the presence of collaterals.[151,152]

There is an association between HAA and the patients with fibromuscular dysplasia, vasculitis, systemic lupus erythematosus or polyarteritis nodosa, Takayasu arteritis, and Wegener granulomatosis.[153-158] Congenital causes of HAA, such as Marfan syndrome, Ehlers-Danlos syndrome, and Osler-Weber-Rendu syndrome, are reported.[159] Patients with fibromuscular dysplasia and polyarteritis nodosa are at significant risk of HAA rupture, accounting for 50% of ruptured HAA in one series.[150] Bacterial endocarditis was the main cause of HAA before the adequate treatment of endocarditis with antibiotics was widespread.[153] Aneurysm rupture is reported with a wide range of 14%[150] to 80%,[159] but given the retrospective nature of these studies, the true risk of rupture is unknown. Nonatherosclerotic aneurysms, however, are at significantly higher risk of rupture as they often present as multiple aneurysms.[150,153-159]

3. Treatment options

Recommendations for treatment of HAA			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	We recommend an endovascular-first approach to all HAAs if it is anatomically feasible (ie, if this approach maintains arterial circulation to the liver).	1 (Strong)	A (High)
3.2	In patients with extrahepatic aneurysms, we recommend open and endovascular techniques to maintain liver circulation.	1 (Strong)	A (High)
3.3	In patients with intrahepatic aneurysms, we recommend coil embolization of the affected artery (Grade 1B). In patients with large intrahepatic HAA, we recommend resection of the involved lobe of liver to avoid significant liver necrosis (Grade 1C).	1 (Strong)	B (Moderate)
			C (Low)

Both open and endovascular options exist for HAA repair. All retrospective case series have shown that the outcome for visceral artery aneurysms after open or endovascular repair yielded similar long-term results, but morbidity is significantly worse with open repair than with the endovascular approach.[2,7] With the improvement of endovascular techniques and relative low morbidity associated with endovascular repair, endovascular techniques should be preferentially offered in anatomically suitable candidates.

The majority of these aneurysms are extrahepatic (75%-80%).[150,153,159] Most are solitary aneurysms, with multiple HAAs reported in only 8% of these series.[150] The ideal procedure of choice should allow the exclusion of aneurysm while maintaining the liver circulation, which can be achieved by either resection of the aneurysm with interposition graft or by placement of a stent graft and endovascular exclusion of the aneurysm. Given the possibility of central liver necrosis despite adequate collateral flow by endovascular exclusion,[93] in low-risk patients,

open surgical revascularization using autologous vein conduit is recommended if endovascular stent graft exclusion is not possible.[151] Temporary occlusion of the hepatic artery during reconstruction may guide revascularization or ligation of the HAA.[151] More distal extrahepatic HAA branches are often associated with biliary inflammation, making these repairs challenging. Endovascular repair of extrahepatic HAA depends on the collaterals and location of the HAA, similar to open repair. Given that maintenance of distal organ perfusion is important, in patients with proper hepatic artery, endovascular repair requires covered stent exclusion of the aneurysm rather than coil embolization.

The Table represents the summary of treatment recommendations for all extrahepatic aneurysms.

Intrahepatic aneurysms will require resection of the lobe in which the aneurysm is located. Given the significant comorbidities associated with liver resection, endovascular interventions have become the primary treatment modality for these intrahepatic lesions when feasible. Complications of embolization include hepatic ischemia, abscess, cholecystitis, and possible recanalization.[151,161,162] Coil embolization is discouraged in patients with large parenchymal lesions or if large segments of liver are at risk of ischemia. In these patients, liver lobe resection should be considered.

As noted, most patients with HAA are diagnosed with CT performed for other reasons.[150] Given the sensitivity and specificity of CTA for diagnosis of other intra-abdominal aneurysms, we recommend CTA to diagnose other intra-abdominal aneurysms. In thin patients, abdominal duplex ultrasound is sensitive and specific in detecting abdominal or iliac artery aneurysms, but this study needs to be performed on individual arteries and therefore may miss other noninvestigated vessels.

There is an association between HAA and the patients with fibromuscular dysplasia, vasculitis, systemic lupus erythematosus or polyarteritis nodosa, Takayasu arteritis, and Wegener granulomatosis.[153-158] This screening study may detect other pathologic processes that will require attention. In addition, congenital causes of HAA, such as Marfan syndrome, Ehlers-Danlos syndrome, and Osler-Weber-Rendu syndrome, are reported.[159] Axial CTA or MRA may detect thoracic or intracerebral aneurysms.

Given that these are slow-growing aneurysms,[150] annual follow-up is adequate. CTA with or without contrast enhancement provides the best modality to observe these aneurysms. Abdominal duplex ultrasound can also be used in certain patients to observe these aneurysms.

4. Screening for concomitant aneurysm and vascular disease

Recommendations for screening of patients with HAA			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	We suggest abdominal axial imaging to screen for concomitant intra-abdominal aneurysms in patients who did not have CTA at the time of HAA diagnosis.	2 (Weak)	B (Moderate)
4.2	We suggest one-time screening CTA or MRA of the head, neck, and chest for those patients with nonatherosclerotic causes of HAA.	2 (Weak)	B (Moderate)

Table. Summary of treatment recommendations for extrahepatic aneurysms

Location of extrahepatic HAA	Indication	Treatment[1,2,7,148,160]
Common hepatic artery	Ruptured	Open surgical ligation
	Symptomatic	Endovascular embolization
	Asymptomatic (>2 cm)	Resection/reconstruction
	Asymptomatic in patients with fibromuscular dysplasia or polyarteritis nodosa	Aneurysmorrhaphy
		Endovascular
		Covered stent
		Coil embolization
Proper hepatic	Same as above	Resection with arterial reconstruction
		Endovascular stent graft
Proximal right or left hepatic branches	Same as above	Resection with arterial reconstruction
		Endovascular stent graft

5. Follow-up and surveillance

Recommendations for follow-up and surveillance of HAA patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	We suggest annual follow-up with CTA or non-contrast-enhanced CT to observe patients with asymptomatic HAA.	2 (Weak)	B (Moderate)

SUPERIOR MESENTERIC ARTERY ANEURYSM (SMAA)

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of SMAA			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	In patients with SMAA, we recommend CTA as the diagnostic tool of choice.	1 (Strong)	B (Moderate)
1.2	We recommend mesenteric angiography to delineate anatomy in preoperative planning for SMAA repair.	1 (Strong)	B (Moderate)

Symptomatic SMAA presents with similar symptoms as other acute abdominal emergencies, such as perforated viscus, making the diagnosis difficult without an appropriate level of suspicion.[163] Although many SMAAs show a rim of calcification on plain kidney, ureter, and bladder radiography[164] and duplex ultrasound can be helpful in diagnosis,[165] CTA is the most expeditious and reliable diagnostic tool.[166] Isolated superior mesenteric artery dissection has been diagnosed more routinely because of an increase in use of CTA for patients who present with abdominal pain.[167,168]

2. Size criteria for invasive intervention (true and false aneurysms)

Recommended intervention criteria for SMAA			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	We recommend repair of all SMAAs and pseudoaneurysms as soon as the diagnosis is made regardless of size.	1 (Strong)	A (High)
2.2	We suggest careful observation of SMAA because of dissection unless refractory symptoms develop.	2 (Weak)	B (Moderate)

SMAA represents 3.5% to 8% of visceral artery aneurysm cases,[1,98] and autopsy results suggest that these aneurysms constitute 1 in every 12,000 to 19,000 autopsies.[169] Although the etiology of these aneurysms is diverse, SMAA most commonly results from an infectious cause or dissection,[166] with the superior mesenteric artery the most common site of infection outside of the aorta.[166] The most common reason for mycotic aneurysm of the superior mesenteric artery is subacute bacterial endocarditis by nonhemolytic streptococcus.[166,169] Atherosclerosis accounts for 25% of cases of SMAA, whereas inflammatory conditions such as pancreatitis and trauma are the other causes of SMAA.[169] Mycotic SMAAs usually present in younger patients (<50 years of age), whereas nonmycotic SMAAs are seen in older patients.[169] Unlike other visceral aneurysms, 70% to 90% of SMAAs are symptomatic at the time of presentation, with abdominal pain the most common symptom, followed by abdominal mass, fever, nausea, and gastrointestinal bleeding.[166,170] The natural history of SMAA appears to be one of expansion and rupture,[92,166] with 38% to 50% of patients presenting with ruptured aneurysm[92,170]; the mortality rate is 30% to 90%.[92] At the same time, the overall mortality from elective repair of SMAA is <15%,[171] and the mortality is even better when endovascular procedures are used for elective SMAA repair.[1] Given the significant incidence of rupture and the high mortality after repair of ruptured SMAA, once SMAAs are diagnosed, they should be treated.

Inferior mesenteric artery aneurysms are rare and are sparsely reported in the literature[172-174]; they have been described in patients with collagen vascular disease or

vasculitis. Given the rarity of this condition, no specific guidelines were generated, and the management of those patients should be individualized.

Some authors have pointed to the possible association of dissection and aneurysmal degeneration of the dissected artery.[175] Yun et al[168] classified these isolated dissections into three groups based on angiographic findings. In patients followed up by Dong et al[167] or Yun et al,[168] no aneurysmal degenerations were observed. Initial treatment of these entities is often antiplatelet therapy unless symptoms recur or are refractory to conservative management.[168] The presence of superior mesenteric artery dissection is not an indication for repair as the majority are treated conservatively without any need for intervention.[167,168]

CTA of abdomen and pelvis has significant specificity and sensitivity for detecting concomitant visceral artery aneurysm and has proved to be the best modality in detecting visceral artery aneurysm.[166,177]

Annual CTA scans are specific and sensitive in following up the repair and assess other vascular beds.[166,177]

JEJUNAL, ILEAL, AND COLIC ARTERY ANEURYSMS

Aneurysms of the jejunal, ileal, and colic arteries account for <3% of all visceral aneurysms, affecting men and women equally beyond the sixth decade of life.[87,163] Most of the literature on these aneurysms is limited to case reviews and small case numbers within the context of larger series on visceral aneurysms. We recommend CTA as the diagnostic modality of choice for these aneurysms; often in asymptomatic patients, this modality identifies a visceral branch aneurysm incidentally.[99,178,179] MRA may also be considered. Axial imaging of the abdomen will facilitate the assessment of other abdominal aneurysms, which are common as referenced before. Arteriography is invaluable for aneurysm identification preoperatively and in operative planning, and it is mandatory for patients with polyarteritis nodosa to assess for additional aneurysms.[180]

Aneurysms of the jejunal, ileal, and colic arteries are associated with medial degeneration, infection, inflammation, various autoimmune diseases (ie, polyarteritis nodosa and Behçet disease), and connective tissue disorders. Multiple aneurysms are identified in approximately 10% of cases.[87,111,136,146,181-184] Atherosclerosis, when present, is thought to be a secondary process. We recommend confirmation of the diagnosis of polyarteritis nodosa in any patient with a history of fever, arthralgia, weakness, abdominal pain, or pleuritic chest pain that accompanies mesenteric branch aneurysm.[107]

Whereas patients may present with abdominal pain, most jejunal and ileal artery aneurysms are

3. Treatment options

Recommendations for treatment of SMAA			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	We recommend an endovascular-first approach to all SMAAs if it is anatomically feasible.	1 (Strong)	B (High)

Treatment must be individualized on the basis of anatomy and characteristics of the SMAA. Angiography is critical to delineate superior mesenteric artery anatomy and collaterals. Both open and endovascular techniques must exclude the aneurysm and maintain patency of the vessels. Endovascular procedures are significantly less morbid than open procedures and should be preferentially offered if it is anatomically feasible. Endovascular interventions include coil embolization and use of covered stents,[176] with good results.

The significant issue with endovascular repair is that SMAA beyond the proximal few centimeters will include major tributaries that must be preserved. Sacrifice of these vessels during an endovascular approach will lead to significant morbidity. In such cases, when an endovascular approach leads to significant loss of collaterals, strong consideration should be given to open repair. Open repair options include open surgical procedures such as simple ligation of aneurysm, aneurysmorrhaphy in case of saccular aneurysms, and repair with an interposition graft. Open resection may require intestinal resection, particularly in symptomatic patients. Close observation for occurrence of peritoneal symptoms is essential after either type of repair.

4. Screening for concomitant aneurysm

Recommendations for screening of patients with SMAA			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	We suggest abdominal axial imaging to screen for concomitant intra-abdominal aneurysms in patients who did not have CTA at the time of diagnosis.	2 (Weak)	B (Moderate)

5. Follow-up and surveillance

Recommendations for follow-up and surveillance of SMAA patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	We suggest annual CTA to observe postsurgical patients.	2 (Weak)	B (Moderate)

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of jejunal, ileal, and colic artery aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	In patients who are thought to have jejunal artery, ileal artery, and colic artery aneurysms, we recommend CTA as the diagnostic tool of choice.	1 (Strong)	B (Moderate)
1.2	In patients with high radiation exposure risks or renal insufficiency, we recommend non-contrast-enhanced MRA for diagnosis.	1 (Strong)	C (Moderate)
	Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).		
1.3	We recommend the use of catheter-based angiography for all emergent cases presenting with rupture (Grade 1B) and electively for preoperative planning (Grade 1C).	1 (Strong)	B (Moderate)
			C (Moderate)
1.4	We suggest screening all patients with jejunal, ileal, and colic artery aneurysms for vasculitis with routine inflammatory markers.	2 (Weak)	C (Moderate)

2. Size criteria for invasive intervention (true aneurysms vs pseudoaneurysms)

Recommended intervention criteria for jejunal, ileal and colic artery aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	We recommend elective intervention for jejunal and ileal artery aneurysms >2 cm in maximal diameter and for all colic artery aneurysms, any size.	1 (Strong)	B (Moderate)
2.2	We recommend emergent intervention for any jejunal, ileal, or colic artery aneurysm, any size, resulting in patient symptoms or rupture and all mesenteric branch vessel pseudoaneurysms.	1 (Strong)	A (High)

asymptomatic. Colic artery aneurysms, however, cause symptoms, primarily abdominal pain, in nearly 90% of patients.[166] Rupture may complicate up to 30% of jejunal and ileal artery aneurysms and up to 70% of colic artery aneurysms, resulting in gastrointestinal bleeding and mortality rates that approach 20% to 50%.[140,166] Treatment is recommended for all jejunal and ileal artery aneurysms >2 cm and for all colic branch aneurysms, regardless of size.[166,184]

We recommend emergent intervention for any jejunal, ileal, or colic artery aneurysm, any size, resulting in patient symptoms or rupture and all mesenteric branch vessel pseudoaneurysms (Grade 1A).

Interpretation of the existing data on mesenteric branch aneurysms is limited by small numbers and anecdotal reports. In addition, most of the existing studies do not delineate true aneurysms from false aneurysms, and the visceral bed is often excluded from analysis.

Surgical management has historically relied on simple arterial ligation or aneurysm excision without reconstruction.[56,87,140,169,184,185] This remains a conservative option for rupture, in which case exploratory laparotomy facilitates the evacuation of hematoma, definitive aneurysm treatment, and bowel assessment for viability. Enterectomy or colectomy may be required as intramural aneurysms and those associated with bowel necrosis require resection of the involved bowel at the time of aneurysm exclusion. Robust collateralization often permits simple aneurysm ligation or resection without reconstruction. However, special consideration is required in patients with a previous colectomy as the collateral network might be incomplete.

3. Treatment options

Recommendations for treatment of jejunal, ileal, and colic artery aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	We suggest open surgical ligation or aneurysm excision for cases of jejunal, ileal, and colic artery aneurysms when laparotomy is being considered for hematoma evacuation or bowel assessment for viability.	2 (Weak)	B (Moderate)
3.2	We suggest endovascular embolization for cases of jejunal, ileal, and colic artery aneurysm.	2 (Weak)	B (Moderate)
3.3	We suggest medical treatment of nonruptured, asymptomatic ileal, jejunal, and colic artery aneurysms associated with polyarteritis nodosa.	2 (Weak)	B (Moderate)

Transcatheter embolization with coils, Onyx, and glue (*n*-butyl cyanoacrylate) has been increasingly used, especially for cases of acute rupture and gastrointestinal bleeding.[1,7,111,186-188] Endovascular interventions offer precise localization of the aneurysm, assessment of collateral flow, lower risk for patients who are not good operative candidates, easier approach to aneurysms for which surgical exposure would be difficult, and decreased length of stay.[1,111] These benefits are balanced with a risk of intestinal necrosis, perforation, and late stricture requiring reoperation in addition to primary technical failures and failed hemostasis in the setting of rupture.[189-193]

Regression of aneurysms resulting from polyarteritis nodosa after cytotoxic or immunosuppressive treatment is well documented.[107] As such, medical therapy should be considered first line for these patients with nonruptured, asymptomatic aneurysms, with repeated arteriography staged at 3 to 4 months to ascertain regression.

4. Screening for concomitant aneurysms

Recommendations for screening of patients with jejunal, ileal, and colic artery aneurysms			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	We suggest abdominal axial imaging to screen for concomitant abdominal aneurysms.	2 (Weak)	B (Moderate)
4.2	We suggest one-time screening CTA (or MRA) of the head, neck, and chest for those patients with segmental arterial mediolysis.	2 (Weak)	B (Moderate)

Axial imaging as recommended will screen for concomitant nonaortic intra-abdominal arterial aneurysms, which are common.

We recommend screening for cerebrovascular and coronary artery aneurysm when the diagnosis of segmental arterial mediolysis is suspected, given the systemic nature of this arterial dysplasia, with one-time screening CTA or MRA of the head, neck, and chest.[146]

We recommend routine interval surveillance (ie, every 12 months) with axial imaging (ie, CTA or MRA) in cases of segmental medial arteriolysis in light of reported cases of rapid arterial transformation.[147]

We recommend postembolization surveillance every 1 to 2 years with axial imaging to assess for vascular remodeling.[1]

PANCREATICODUODENAL ARTERY ANEURYSM (PDAA) AND GASTRODUODENAL ARTERY ANEURYSM (GDAA)

CTA has become the most common diagnostic tool for PDAA and GDAA, given its accuracy, wide availability, and promptness.[194,195] Enhancement patterns vary according to the amount of thrombus within the aneurysm. Multiplanar reformations and three-dimensional reconstructions can aid in determining the relationship of the surrounding vasculature and parent vessels. In cases of ruptured GDAA and PDAA, CTA is the diagnostic

5. Follow-up and surveillance

Recommendations for follow-up and surveillance of jejunal, ileal, and colic artery aneurysm patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	We suggest interval surveillance (ie, every 12-24 months) with axial imaging (ie, CTA or MRA) for cases of segmental medial arteriolysis in light of reported cases of rapid arterial transformation and to monitor regression in cases of polyarteritis nodosa.	2 (Weak)	B (Moderate)
5.2	We suggest postembolization surveillance every 1 to 2 years with axial imaging to assess for vascular remodeling and evidence of aneurysm reperfusion.	2 (Weak)	B (Moderate)

1. Diagnosis and evaluation

Recommendations for diagnosis and evaluation of PDAA and GDAA			
	Recommendation	Strength of recommendation	Quality of evidence
1.1	In patients who are thought to have GDAA and PDAA, we recommend CTA as the diagnostic tool of choice.	1 (Strong)	B (Moderate)
1.2	In patients in whom celiac stenosis is suspected, we suggest further workup with duplex ultrasound to elucidate whether the stenosis is hemodynamically significant.	2 (Weak)	C (Low)
1.3	In patients with high radiation exposure risks or renal insufficiency, we suggest non-contrast-enhanced MRA for diagnosis.	2 (Weak)	C (Low)
	Technical remark: Non-contrast-enhanced MRA is best suited to children and women of childbearing potential or those who have contraindications to CTA or MRA contrast materials (ie, pregnancy, renal insufficiency, or gadolinium contrast material allergy).		

tool of choice because it can provide arterial-phase, venous-phase, and non-contrast-enhanced images in a rapid manner.[137] Furthermore, findings of diaphragmatic crura hypertrophy, a focal narrowing of the proximal celiac axis with a hooked appearance, and retrograde filling of the dorsal pancreatic and pancreaticoduodenal arteries on three-dimensional CTA are suggestive of median arcuate ligament syndrome and may direct the surgeon to consider a median arcuate ligament release in addition to treatment of the GDAA or PDAA.[196]

Although non-contrast-enhanced MRA sequences can be more time-consuming than contrast-enhanced MRA, non-contrast-enhanced MRA has almost the same sensitivity for detecting vascular abnormalities and a high negative predictive value. Non-contrast-enhanced MRA (like contrast-enhanced MRA) can be superior and complementary to ultrasound in evaluating most parts of the body because it is not limited by acoustic windows, particularly in the thoracoabdominal vasculature.[202-204]

2. Size criteria for invasive intervention

Recommended intervention criteria for PDAA and GDAA			
	Recommendation	Strength of recommendation	Quality of evidence
2.1	In patients with noncomplicated GDAA and PDAA of acceptable operative risk, we recommend treatment no matter the size of the aneurysm because of the risk of rupture.	1 (Strong)	B (Moderate)

Non-contrast-enhanced CT scans may show only a soft tissue mass in the aneurysm bed that can be confused with adenopathy or pancreatic or duodenal neoplasm.[160,194]

Contrast-enhanced MRA has been shown to correlate well with CTA,[86] but it should not be used in emergent cases as acquisition times are longer than for CTA.[197] Postprocessing maximum intensity projection images are similar to those of conventional angiography, providing a roadmap for therapeutic interventions.[179] Finally, duplex ultrasound may delineate larger aneurysms, but information on parent artery anatomy is insufficient.[59] Accuracy can be decreased because of the patient's body habitus, calcified vessel walls, and limited spatial resolution.[198,199]

Duplex ultrasound is one of the most common diagnostic techniques used to evaluate the celiac axis for median arcuate ligament syndrome.[200] It can provide real-time inspiratory and expiratory data, helping elucidate the cause of the GDAA or PDAA and its treatment.[201]

Previous studies have made the distinction in size threshold for repair of visceral artery aneurysms on the basis of whether the aneurysm is a true aneurysm or a pseudoaneurysm.[205] This is based on the fact that pseudoaneurysms are at higher risk of rupture and should therefore be repaired regardless of size. However, it is known that many of the PDAAs and GDAAs that have ruptured can be small and <10 mm.[206] In fact, a single-center review of all visceral aneurysms found that PDAAs were strongly associated with rupture ($P < .0002$).[207] Thus, GDAAs and PDAAs should be repaired regardless of size and regardless of true vs false aneurysm in patients who have acceptable operative or interventional risk.[117,208-213]

A Markov model decision analysis was performed to assess the effectiveness of preventive treatment of patients with PDAA based on risk.[214] The authors argued that whereas 60% of PDAAs present ruptured regardless of size, the natural history of unruptured aneurysms cannot be determined by that of ruptured aneurysms.

Assuming a mortality rate of 21% after rupture, they found that preventive treatment was dominated by no treatment if mortality rates of preventive treatment were >1.4%, >2.6%, >3.8%, and >4.8% at annual rupture rates of 1%, 2%, 3%, and 4%, respectively, for an 80-year-old patient. Preventive treatment was dominated by no treatment if mortality rates of preventive treatment were >3.3%, >5.9%, >8.0%, and >9.7% at annual rupture rates of 1%, 2%, 3%, and 4%, respectively, for a 50-year-old patient.

Predictive factors of rupture, whether size, demographics, or comorbid disease, are few. A review of the English literature found that 32% of ruptured patients were female and 61% were male.[215] In one single-center study, rupture was associated with male sex ($P = .02$) and a trend toward rupture in patients with celiac stenosis ($P = .10$).[216] Thus, male patients should be considered for elective repair even in the presence of moderate operative risk.

Long-term success may be related to angiographic findings seen on completion imaging. The Raymond-Roy Occlusion Classification is a system for evaluating aneurysm occlusion after endovascular coiling with three classes based on completion imaging at the end of the procedure.[218] Class I is defined as complete obliteration; class II, a residual neck; and class III, a residual aneurysm. Class III can be further subdivided into IIIa, residual contrast within coil interstices, and IIIb, residual aneurysm with contrast material along the sidewall of the aneurysm. Class IIIa aneurysms tend to have a higher rate of subsequent thrombosis, whereas IIIb aneurysms tend to have a higher rate of re-treatment.[219]

Whereas survival is unrelated to operative technique in repair of intact aneurysms, endovascular repair of ruptured aneurysms is associated with both improved overall survival and aneurysm-related survival compared with open surgery.[4,209]

3. Treatment options

Recommendations for treatment of PDAA and GDAA			
	Recommendation	Strength of recommendation	Quality of evidence
3.1	In patients with intact and ruptured aneurysms, we recommend coil embolization as the treatment of choice.	1 (Strong)	B (Moderate)
3.2	In patients in whom coil embolization is not feasible, we suggest covered stenting or stent-assisted coil embolization as a treatment option in select cases of GDAA and PDAA.	2 (Weak)	C (Low)
3.3	In patients with appropriate anatomy, we suggest transcatheter embolization with liquid embolic agents as a treatment option for both GDAA and PDAA.	2 (Weak)	C (Low)
3.4	In patients with suitable anatomy, we suggest flow-diverting, multilayered stents as a treatment option for GDAA and PDAA, although these have not been adequately studied to be recommended as a primary treatment modality.	2 (Weak)	C (Low)
3.5	In patients with nonruptured aneurysms, we suggest open surgical reconstruction if needed to preserve flow.	2 (Weak)	B (Moderate)
3.6	In patients with concomitant stenosis or occlusion, we suggest celiac artery reconstruction.	2 (Weak)	B (Moderate)

Coil embolization of GDAA and PDAA has recently become the treatment of choice whether the aneurysm is ruptured or not.[4,190,195,209,211-213,216] Different catheter configurations can be employed, but a triaxial system consisting of a sheath-guiding catheter, a 4F or 5F catheter, and a microcatheter provides stable support through tortuous vessels or in treating distal arterial beds as well as rapid exchange of the microcatheter if necessary.[1] Although it is not imperative, exclusion of all inflow and outflow vessels is necessary to reduce the risk of recurrent sac pressurization from antegrade or retrograde perfusion.[6] End-organ perfusion is typically maintained by the dense collateral network of these vessels, but isolated coil packing of the aneurysm sac alone can be sufficient if the inflow vessel is vital for organ perfusion.[217] Immediate technical success rates are >90%.

Covered stenting may be performed when the artery proximal and distal to the aneurysm is of suitable diameter with a low degree of tortuosity.[212] Successful covered stenting with a Viabahn stent (W. L. Gore & Associates, Flagstaff, Ariz)[220] as well as with an Advanta V12 stent graft (Atrium Medical, Hudson, NH)[221] has been reported. Advantages of this technique include preservation of flow through the artery; however, it may be limited by the discrepancy in arterial seal zone diameters as well as by the ability to pass the stent through tortuous anatomy.[216]

The liquid embolic agent *n*-butyl 2-cyanoacrylate (NBCA) has been used to treat cases of both ruptured and unruptured GDAA and PDAA.[222] A theoretical advantage of embolization with liquid embolic agents is the ability to embolize small, tortuous vessels that

may be too small to selectively catheterize for coil embolization.[6,132,213,223] The NBCA must be premixed with iodized oil to control its polymerization and to make it radiopaque.[224] A 1:3 ratio, or 25% NBCA, increases the polymerization time to 4 seconds, although distal embolization may still occur.[225]

Ethylene vinyl alcohol (Onyx) has also been used successfully to treat PDAA.[65] Onyx is dissolved in dimethyl sulfoxide and is suspended in radiopaque micronized tantalum powder; it does not need to be mixed. When concomitant balloon occlusion of the parent vessel is used, injection of the Onyx directly into the aneurysm through a microcatheter preserves the inflow and outflow vessels. When balloon occlusion is not used, the inflow and outflow vessels can be obliterated as well.

Flow-diverting stents are a burgeoning technology that was first introduced for the treatment of intracranial aneurysms. Flow-diverting stents are placed in the parent artery in an effort to improve laminar flow within the parent artery and to reduce blood flow within the aneurysm sac to the point of thrombosis.[226] Furthermore, these stents preserve flow through collateral side branches. The stents require an overall porosity of 50% to 70%, or 30% to 50% metallic coverage. Stents such as the Pipeline Embolization Device (ev3, Plymouth, Minn), the SILK Arterial Reconstruction Device (Balt Extrusion, Montmorency, France), and the Cardiatis multilayer stent are available; however, only the Cardiatis multilayer stent has been used to treat a GDAA. At 6 months, the Cardiatis stent was patent with complete thrombosis of the aneurysm sac.[227] Patients are treated with dual antiplatelet regimens postoperatively. In patients with suitable anatomy, flow-diverting, multilayered stents may become a treatment option for GDAA and PDAA, although these have not been adequately studied to be recommended as a primary treatment modality at this point.

Open surgical treatment of GDAA and PDAA is technically feasible with a perioperative morbidity and mortality for nonruptured aneurysms of approximately 9.4% and 1.3%, respectively.[59] Techniques include ligation and excision with end-to-end anastomosis.[59,211,212,223] However, mortality for ruptured GDAA and PDAA undergoing open repair approaches 30%.[4,209]

Celiac axis stenosis or occlusion is frequently associated with aneurysms of the pancreaticoduodenal arcade.[228] This is thought to result in retrograde flow through the pancreaticoduodenal arteries, leading to turbulent flow, which ultimately causes aneurysmal dilation. Interestingly, celiac stenosis is more common in PDAA than in GDAA.[229] Whereas coil embolization can lead to end-organ hepatic ischemia and liver failure, the overall risk is likely to be low.[208] However, a low threshold for celiac revascularization should be considered in patients with symptoms of mesenteric insufficiency at baseline or when there is a risk of compromising end-organ perfusion with aneurysm treatment, and postoperative liver function test results should be monitored closely.[4,212] In cases of median arcuate ligament syndrome, celiac revascularization can be achieved with division of the median arcuate ligament and celiac plexus, aortoceliac bypass, or renohepatic bypass.[230] In cases not associated with median arcuate ligament compression, primary stenting of a celiac stenosis is an option.[216,231]

4. De novo screening and screening for concomitant aneurysms

Recommendations for screening of patients with PDAA and GDAA			
	Recommendation	Strength of recommendation	Quality of evidence
4.1	In patients with median arcuate ligament syndrome, we suggest screening for GDAA or PDAA with CTA or duplex ultrasound.	2 (Weak)	C (Low)

Few data exist regarding screening for GDAA and PDAA. However, based on the fact that celiac stenosis is associated with 50% to 60% of these aneurysms, it would seem prudent to examine the gastroduodenal artery and pancreaticoduodenal arcade by either duplex ultrasound or CTA once a celiac stenosis is diagnosed.[232] Pulsed Doppler ultrasound can distinguish between aneurysms and other masses of the pancreas, although it may be limited by patient factors such as bowel gas.[233]

There is no association of GDAA or PDAA with aneurysms outside the visceral circulation. Therefore, screening for concomitant aneurysms outside the abdomen is of little value.

It is well known that endovascular treatment of GDAA and PDAA may be associated with the long-term complication of aneurysm reperfusion.[6,209] Although no studies have evaluated the recurrence rates of GDAA or PDAA after endovascular coiling, recanalization after endovascular treatment of visceral artery aneurysms occurs in 9% to 15% of patients.[111,113,190,234] This may be due to insufficient packing, long-term coil compaction, or delayed coil migration.[235,236] Thus, follow-up imaging is crucial.[59,235]

A radiopaque agent is necessary for embolization; however, this creates a significant radioartifact on follow-up imaging.[1,56] Whereas CTA is the most commonly used follow-up study modality, certain reports have found

5. Follow-up and surveillance

Recommendations for follow-up and surveillance of PDAA and GDAA patients			
	Recommendation	Strength of recommendation	Quality of evidence
5.1	In patients status post treatment of GDAA and PDAA, we recommend follow-up imaging after endovascular treatment of GDAA and PDAA to rule out persistent flow through the aneurysm sac.	1 (Strong)	B (Moderate)

that it cannot accurately determine reperfusion of an aneurysm sac because of the degree of radioartifact.[237]

More recently, three-dimensional contrast-enhanced MRA has been shown to be a safe and effective way to provide postoperative follow-up.[238] This technique has a 91% accuracy in defining hemodynamic status and complications with little metallic artifact.[239]

Concerns about repeated radiation exposure have prompted some to recommend duplex ultrasound as an alternative surveillance technique.[1,6,56,209] Contrast-enhanced ultrasound is a less expensive and noninvasive technique that has been shown to clearly identify flow in and around a metallic coil pack.[240] Thus, contrast-enhanced ultrasound may be optimal for long-term surveillance.

Finally, digital subtraction angiography is the "gold standard" for defining reperfusion of the aneurysm sac. However, this is typically reserved for cases in which reintervention of an enlarging aneurysm sac is necessary.[1,56,72]

The frequency of surveillance imaging is not well established, but it is unlikely that aneurysm reperfusion will occur if it is completely obliterated and thrombosed on the first postprocedural imaging study. As such, the value of long-term surveillance after embolization is not justified. In addition, given the lack of clear association of GDAA and PDAA with metachronous visceral aneurysms, surveillance for the development of new visceral aneurysms is also not well established.

REFERENCES

1. Tulsyan N, Kashyap VS, Greenberg RK, Sarac TP, Clair DG, Pierce G, et al. The endovascular management of visceral artery aneurysms and pseudoaneurysms. J Vasc Surg 2007;45:276-83; discussion: 283.
2. Cochennec F, Riga CV, Allaire E, Cheshire NJ, Hamady M, Jenkins MP, et al. Contemporary management of splanchnic and renal artery aneurysms: results of endovascular compared with open surgery from two European vascular centers. Eur J Vasc Endovasc Surg 2011;42:340-6.
3. Wagner WH, Allins AD, Treiman RL, Cohen JL, Foran RF, Levin PM, et al. Ruptured visceral artery aneurysms. Ann Vasc Surg 1997;11:342-7.
4. Suzuki K, Kashimura H, Sato M, Hassan M, Yokota H, Nakahara A, et al. Pancreaticoduodenal artery aneurysms associated with celiac axis stenosis due to compression by median arcuate ligament and celiac plexus. J Gastroenterol 1998;33:434-8.
5. Murad MH, Montori VM, Sidawy AN, Ascher E, Meissner MH, Chaikof EL, et al. Guideline methodology of the Society for Vascular Surgery including the experience with the GRADE framework. J Vasc Surg 2011;53:1375-80.
6. Lagana D, Carrafiello G, Mangini M, Dionigi G, Caronno R, Castelli P, et al. Multimodal approach to endovascular treatment of visceral artery aneurysms and pseudoaneurysms. Eur J Radiol 2006;59:104-11.
7. Sachdev U, Baril DT, Ellozy SH, Lookstein RA, Silverberg D, Jacobs TS, et al. Management of aneurysms involving branches of the celiac and superior mesenteric arteries: a comparison of surgical and endovascular therapy. J Vasc Surg 2006;44:718-24.
8. Barrionuevo P, Malas MB, Nejim B, Haddad A, Morrow A, Ponce O, et al. A systematic review and meta-analysis of the management of visceral artery aneurysms. J Vasc Surg 2019;70:1694-9.
9. Stanley JC, Rhodes EL, Gewertz BL, Chang CY, Walter JF, Fry WJ. Renal artery aneurysms. Significance of macroaneurysms exclusive of dissections and fibrodysplastic mural dilations. Arch Surg 1975;110:1327-33.
10. Leadbetter WF, Burkland CF. Hypertension in unilateral renal disease. J Urol 1938;39:611-26.
11. Plouin PF, Perdu J, La Batide-Alanore A, Boutouyrie P, Gimenez-Roqueplo AP, Jeunemaitre X. Fibromuscular dysplasia. Orphanet J Rare Dis 2007;2:28.
12. Cragg AH, Smith TP, Thompson BH, Maroney TP, Stanson AW, Shaw GT, et al. Incidental fibromuscular dysplasia in potential renal donors—long-term clinical follow-up. Radiology 1989;172:145-7.
13. Andreoni KA, Weeks SM, Gerber DA, Fair JH, Mauro MA, McCoy L, et al. Incidence of donor renal fibromuscular dysplasia: does it justify routine angiography? Transplantation 2002;73:1112-6.
14. Klausner JQ, Lawrence PF, Harlander-Locke MP, Coleman DM, Stanley JC, Fujimura N, et al. The contemporary management of renal artery aneurysms. J Vasc Surg 2015;61:978-84.
15. Zhang LJ, Yang GF, Qi J, Shen W. Renal artery aneurysm: diagnosis and surveillance with multidetector-row computed tomography. Acta Radiol 2007;48:274-9.
16. Smith PA, Fishman EK. Three-dimensional CT angiography: renal applications. Semin Ultrasound CT MR 1998;19:413-24.
17. Johnson PT, Fishman EK. Computed tomography angiography of the renal and mesenteric vasculature: concepts and applications. Semin Roentgenol 2011;46:115-24.
18. Angeretti MG, Lumia D, Cani A, Barresi M, Cardim LN, Piacentino F, et al. Non-enhanced MR angiography of renal arteries: comparison with contrast-enhanced MR angiography. Acta Radiol 2013;54:749-56.
19. Mohrs OK, Petersen SE, Schulze T, Zieschang M, Kux H, Schmitt P, et al. High-resolution 3D unenhanced ECG-gated respiratory-navigated MR angiography of the renal arteries: comparison with contrast-enhanced MR angiography. AJR Am J Roentgenol 2010;195:1423-8.

20. Albert TS, Akahane M, Parienty I, Yellin N, Catala V, Alomar X, et al. An international multicenter comparison of time-SLIP unenhanced MR angiography and contrast-enhanced CT angiography for assessing renal artery stenosis: the renal artery contrast-free trial. AJR Am J Roentgenol 2015;204:182-8.
21. Granata A, Floccari F, Clementi A, Di Lullo L, Fiorini F. Contrast-enhanced ultrasound reveals renal artery aneurysm after detection of 'parapelvic lithiasis. Clin Exp Nephrol 2014;18:174-5.
22. Seo JM, Park KB, Kim KH, Jeon P, Shin SW, Park HS, et al. Clinical and multidetector CT follow-up results of renal artery aneurysms treated by detachable coil embolization using 3D rotational angiography. Acta Radiol 2011;52:854-9.
23. Endo H, Shimizu T, Kodama Y, Miyasaka K. Usefulness of 3-dimensional reconstructed images of renal arteries using rotational digital subtraction angiography. J Urol 2002;167:2046-8.
24. Kaufman JAK. Renal arteries. In: Kaufman JA, Lee MJ, editors. Vascular and interventional radiology. The requisites. Philadelphia: Mosby; 2004. p. 323-49.
25. Liu PS, Platt JF. CT angiography of the renal circulation. Radiol Clin North Am 2010;48:347-65. viii-ix.
26. Hirsch AT, Haskal ZJ, Hertzer NR, Bakal CW, Creager MA, Halperin JL, et al. ACC/AHA 2005 Practice Guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): Executive summary—a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease). Circulation 2006;113:1474-547.
27. Coleman DM, Stanley JC. Renal artery aneurysms. J Vasc Surg 2015;62:779-85.
28. Cerny JC, Chang CY, Fry WJ. Renal artery aneurysms. Arch Surg 1968;96:653-63.
29. Ippolito JJ, Leveen HH. Treatment of renal artery aneurysms. J Urol 1960;83:10-6.
30. Henriksson C, Lukes P, Nilson AE, Pettersson S. Angiographically discovered, non-operated renal artery aneurysms. Scand J Urol Nephrol 1984;18:59-62.
31. Robinson WP 3rd, Bafford R, Belkin M, Menard MT. Favorable outcomes with in situ techniques for surgical repair of complex renal artery aneurysms. J Vasc Surg 2011;53:684-91.
32. Pfeiffer H, Weiss FU, Karger B, Aghdassi A, Lerch MM, Brinkmann B. Fatal cerebro-renal oxalosis after appendectomy. Int J Legal Med 2004;118:98-100.
33. Hislop SJ, Patel SA, Abt PL, Singh MJ, Illig KA. Therapy of renal artery aneurysms in New York State: outcomes of patients undergoing open and endovascular repair. Ann Vasc Surg 2009;23:194-200.
34. Tsilimparis N, Reeves JG, Dayama A, Perez SD, Debus ES, Ricotta JJ 2nd. Endovascular vs open repair of renal artery aneurysms: outcomes of repair and long-term renal function. J Am Coll Surg 2013;217:263-9.
35. Tham G, Ekelund L, Herrlin K, Lindstedt EL, Olin T, Bergentz SE. Renal artery aneurysms. Natural history and prognosis. Ann Surg 1983;197:348-52.
36. Martin RS 3rd, Meacham PW, Ditesheim JA, Mulherin JL Jr, Edwards WH. Renal artery aneurysm: selective treatment for hypertension and prevention of rupture. J Vasc Surg 1989;9:26-34.
37. Henke PK, Cardneau JD, Welling TH 3rd, Upchurch GR Jr, Wakefield TW, Jacobs LA, et al. Renal artery aneurysms: a 35-year clinical experience with 252 aneurysms in 168 patients. Ann Surg 2001;234:454-62; discussion: 462-3.
38. Chandra A, O'Connell JB, Quinones-Baldrich WJ, Lawrence PF, Moore WS, Gelabert HA, et al. Aneurysmectomy with arterial reconstruction of renal artery aneurysms in the endovascular era: a safe, effective treatment for both aneurysm and associated hypertension. Ann Vasc Surg 2010;24:503-10.
39. Klausner JQ, Harlander-Locke MP, Plotnik AN, Lehrman E, DeRubertis BG, Lawrence PF. Current treatment of renal artery aneurysms may be too aggressive. J Vasc Surg 2014;59:1356-61.
40. Wayne EJ, Edwards MS, Stafford JM, Hansen KJ, Corriere MA. Anatomic characteristics and natural history of renal artery aneurysms during longitudinal imaging surveillance. J Vasc Surg 2014;60:448-52.
41. Cohen JR, Shamash FS. Ruptured renal artery aneurysms during pregnancy. J Vasc Surg 1987;6:51-9.
42. Wexler BC. Spontaneous arteriosclerosis of the mesenteric, renal, and peripheral arteries of repeatedly bred rats. Circ Res 1964;15:485-96.
43. Wexler BC. Spontaneous arteriosclerosis in repeatedly bred male and female rats. J Atheroscler Res 1964;4:57-80.
44. Hellmund A, Meyer C, Fingerhut D, Muller SC, Merz WM, Gembruch U. Rupture of renal artery aneurysm during late pregnancy: clinical features and diagnosis. Arch Gynecol Obstet 2016;293:505-8.
45. Hwang PF, Rice DC, Patel SV, Mukherjee D. Successful management of renal artery aneurysm rupture after cesarean section. J Vasc Surg 2011;54:519-21.
46. McCarron JP Jr, Marshall VF, Whitsell JC 2nd. Indications for surgery on renal artery aneurysms. J Urol 1975;114:177-80.
47. Soliman KB, Shawky Y, Abbas MM, Ammary M, Shaaban A. Ruptured renal artery aneurysm during pregnancy, a clinical dilemma. BMC Urol 2006;6:22.
48. Graff J, Schalte G, Jovanovic V. [Rupture of a renal artery aneurysm during delivery]. Aktuelle Urol 2003;34:350-3.
49. English WP, Pearce JD, Craven TE, Wilson DB, Edwards MS, Ayerdi J, et al. Surgical management of renal artery aneurysms. J Vasc Surg 2004;40:53-60.
50. Pfeiffer T, Reiher L, Grabitz K, Grunhage B, Hafele S, Voiculescu A, et al. Reconstruction for renal artery aneurysm: operative techniques and long-term results. J Vasc Surg 2003;37:293-300.
51. Hupp T, Allenberg JR, Post K, Roeren T, Meier M, Clorius JH. Renal artery aneurysm: surgical indications and results. Eur J Vasc Surg 1992;6:477-86.
52. Dzsinich C, Gloviczki P, McKusick MA, Pairolero PC, Bower TC, Hallett JW Jr, et al. Surgical management of renal artery aneurysm. Cardiovasc Surg 1993;1:243-7.
53. Heflin LA, Street CB, Papavassiliou DV, Kem DC, Wu DH, O'Rear EA. Transient stenotic-like occlusions as a possible mechanism for renovascular hypertension due to aneurysm. J Am Soc Hypertens 2009;3:192-200.
54. Down LA, Papavassiliou DV, O'Rear EA. Arterial deformation with renal artery aneurysm as a basis for secondary hypertension. Biorheology 2013;50:17-31.
55. Bastounis E, Pikoulis E, Georgopoulos S, Alexiou D, Leppaniemi A, Boulafendis D. Surgery for renal artery aneurysms: a combined series of two large centers. Eur Urol 1998;33:22-7.
56. Pulli R, Dorigo W, Troisi N, Pratesi G, Innocenti AA, Pratesi C. Surgical treatment of visceral artery aneurysms: a 25-year experience. J Vasc Surg 2008;48:334-42.

57. Morita K, Seki T, Iwami D, Sasaki H, Fukuzawa N, Nonomura K. Long-term outcome of single institutional experience with conservative and surgical management for renal artery aneurysm. Transplant Proc 2012;44:1795-9.
58. Lumsden AB, Salam TA, Walton KG. Renal artery aneurysm: a report of 28 cases. Cardiovasc Surg 1996;4:185-9.
59. Marone EM, Mascia D, Kahlberg A, Brioschi C, Tshomba Y, Chiesa R. Is open repair still the gold standard in visceral artery aneurysm management? Ann Vasc Surg 2011;25:936-46.
60. Jibiki M, Inoue Y, Kudo T, Toyofuku T. Surgical procedures for renal artery aneurysms. Ann Vasc Dis 2012;5:157-60.
61. Murray SP, Kent C, Salvatierra O, Stoney RJ. Complex branch renovascular disease: management options and late results. J Vasc Surg 1994;20:338-45; discussion: 346.
62. Gallagher KA, Phelan MW, Stern T, Bartlett ST. Repair of complex renal artery aneurysms by laparoscopic nephrectomy with ex vivo repair and autotransplantation. J Vasc Surg 2008;48:1408-13.
63. Abath C, Andrade G, Cavalcanti D, Brito N, Marques R. Complex renal artery aneurysms: liquids or coils? Tech Vasc Interv Radiol 2007;10:299-307.
64. Dib M, Sedat J, Raffaelli C, Petit I, Robertson WG, Jaeger P. Endovascular treatment of a wide-neck renal artery bifurcation aneurysm. J Vasc Interv Radiol 2003;14:1461-4.
65. Bratby MJ, Lehmann ED, Bottomley J, Kessel DO, Nicholson AA, McPherson SJ, et al. Endovascular embolization of visceral artery aneurysms with ethylene-vinyl alcohol (Onyx): a case series. Cardiovasc Intervent Radiol 2006;29:1125-8.
66. Meyer C, Verrel F, Weyer G, Wilhelm K. Endovascular management of complex renal artery aneurysms using the multilayer stent. Cardiovasc Intervent Radiol 2011;34:637-41.
67. Paci E, Mincarelli C, Fichetti M, Alborino S, Antico E, Candelari R. Stent-assisted coil embolization of renal artery bifurcation aneurysm using the kissing stent technique. J Vasc Interv Radiol 2011;22:1485-7.
68. Somarouthu B, Rabinov J, Waichi W, Kalva SP. Stent-assisted coil embolization of an intraparenchymal renal artery aneurysm in a patient with neurofibromatosis. Vasc Endovascular Surg 2011;45:368-71.
69. Kitzing B, Vedelago J, Bajic N, Lai G, Waugh R. Stent-assisted coil embolization of a wide-necked renal artery aneurysm. J Radiol Case Rep 2010;4:20-4.
70. Manninen HI, Berg M, Vanninen RL. Stent-assisted coil embolization of wide-necked renal artery bifurcation aneurysms. J Vasc Interv Radiol 2008;19:487-92.
71. Ikeda O, Tamura Y, Nakasone Y, Iryou Y, Yamashita Y. Nonoperative management of unruptured visceral artery aneurysms: treatment by transcatheter coil embolization. J Vasc Surg 2008;47:1212-9.
72. Etezadi V, Gandhi RT, Benenati JF, Rochon P, Gordon M, Benenati MJ, et al. Endovascular treatment of visceral and renal artery aneurysms. J Vasc Interv Radiol 2011;22:1246-53.
73. Abdel-Kerim A, Cassagnes L, Alfidja A, Gageanu C, Favrolt G, Dumousset E, et al. Endovascular treatment of eight renal artery aneurysms. Acta Radiol 2012;53:430-4.
74. Sedat J, Chau Y, Baque J. Endovascular treatment of renal aneurysms: a series of 18 cases. Eur J Radiol 2012;81:3973-8.
75. Zhang Z, Yang M, Song L, Tong X, Zou Y. Endovascular treatment of renal artery aneurysms and renal arteriovenous fistulas. J Vasc Surg 2013;57:765-70.
76. Gandini R, Morosetti D, Chiocchi M, Chiaravalloti A, Citraro D, Loreni G, et al. Long-term follow-up of endovascular treatment of renal artery aneurysms with covered stent deployment. J Cardiovasc Surg (Torino) 2016;57:625-33.
77. Luke P, Knudsen BE, Nguan CY, Pautler SE, Swinnimer S, Kiaii R, et al. Robot-assisted laparoscopic renal artery aneurysm reconstruction. J Vasc Surg 2006;44:651-3.
78. Giulianotti PC, Bianco FM, Addeo P, Lombardi A, Coratti A, Sbrana F. Robot-assisted laparoscopic repair of renal artery aneurysms. J Vasc Surg 2010;51:842-9.
79. Gheza F, Coratti F, Masrur M, Calatayud D, Annecchiarico M, Coratti A, et al. Robot-assisted renal artery aneurysm repair with a saphenous vein Y-graft interposition. Surg Endosc 2013;27:1404-5.
80. Samarasekera D, Autorino R, Khalifeh A, Kaouk JH. Robot-assisted laparoscopic renal artery aneurysm repair with selective arterial clamping. Int J Urol 2014;21:114-6.
81. Olin JW, Gornik HL, Bacharach JM, Biller J, Fine LJ, Gray BH, et al. Fibromuscular dysplasia: state of the science and critical unanswered questions: a scientific statement from the American Heart Association. Circulation 2014;129:1048-78.
82. Agrawal GA, Johnson PT, Fishman EK. Splenic artery aneurysms and pseudoaneurysms: clinical distinctions and CT appearances. AJR Am J Roentgenol 2007;188:992-9.
83. Saba L, Anzidei M, Lucatelli P, Mallarini G. The multidetector computed tomography angiography (MDCTA) in the diagnosis of splenic artery aneurysm and pseudoaneurysm. Acta Radiol 2011;52:488-98.
84. Al-Habbal Y, Christophi C, Muralidharan V. Aneurysms of the splenic artery—a review. Surgeon 2010;8:223-31.
85. Russo A, Francia C, Zaottini A, Pagliei M. Giant splenic artery aneurysm, incidentally diagnosed. Ann Ital Chir 2008;79:371-5.
86. Pilleul F, Forest J, Beuf O. [Magnetic resonance angiography of splanchnic artery aneurysms and pseudoaneurysms]. J Radiol 2006;87(Pt 1):127-31.
87. Stanley JC, Wakefield TW, Graham LM, Whitehouse WM Jr, Zelenock GB, Lindenauer SM. Clinical importance and management of splanchnic artery aneurysms. J Vasc Surg 1986;3:836-40.
88. Dave SP, Reis ED, Hossain A, Taub PJ, Kerstein MD, Hollier LH. Splenic artery aneurysm in the 1990s. Ann Vasc Surg 2000;14:223-9.
89. Stanley JC, Fry WJ. Pathogenesis and clinical significance of splenic artery aneurysms. Surgery 1974;76:898-909.
90. Barrett JM, Van Hooydonk JE, Boehm FH. Pregnancy-related rupture of arterial aneurysms. Obstet Gynecol Surv 1982;37:557-66.
91. Holdsworth RJ, Gunn A. Ruptured splenic artery aneurysm in pregnancy. A review. Br J Obstet Gynaecol 1992;99:595-7.
92. Pitton MB, Dappa E, Jungmann F, Kloeckner R, Schotten S, Wirth GM, et al. Visceral artery aneurysms: incidence, management, and outcome analysis in a tertiary care center over one decade. Eur Radiol 2015;25:2004-14.
93. Berceli SA. Hepatic and splenic artery aneurysms. Semin Vasc Surg 2005;18:196-201.
94. Lakin RO, Bena JF, Sarac TP, Shah S, Krajewski LP, Srivastava SD, et al. The contemporary management of splenic artery aneurysms. J Vasc Surg 2011;53:958-64; discussion: 965.
95. Tessier DJ, Stone WM, Fowl RJ, Abbas MA, Andrews JC, Bower TC, et al. Clinical features and management of splenic artery pseudoaneurysm: case series and cumulative review of literature. J Vasc Surg 2003;38:969-74.
96. Parangi S, Levine D, Henry A, Isakovich N, Pories S. Surgical gastrointestinal disorders during pregnancy. Am J Surg 2007;193:223-32.
97. Hemp JH, Sabri SS. Endovascular management of visceral arterial aneurysms. Tech Vasc Interv Radiol 2015;18:14-23.

98. Messina LM, Shanley CJ. Visceral artery aneurysms. Surg Clin North Am 1997;77:425-42.
99. Nosher JL, Chung J, Brevetti LS, Graham AM, Siegel RL. Visceral and renal artery aneurysms: a pictorial essay on endovascular therapy. Radiographics 2006;26:1687-704.
100. Arca MJ, Gagner M, Heniford BT, Sullivan TM, Beven EG. Splenic artery aneurysms: methods of laparoscopic repair. J Vasc Surg 1999;30:184-8.
101. Mastracci TM, Cadeddu M, Colopinto RF, Cina C. A minimally invasive approach to the treatment of aberrant splenic artery aneurysms: a report of two cases. J Vasc Surg 2005;41:1053-7.
102. Mattar SG, Lumsden AB. The management of splenic artery aneurysms: experience with 23 cases. Am J Surg 1995;169:580-4.
103. Huang IH, Zuckerman DA, Matthews JB. Occlusion of a giant splenic artery pseudoaneurysm with percutaneous thrombin-collagen injection. J Vasc Surg 2004;40:574-7.
104. Carroccio A, Jacobs TS, Faries P, Ellozy SH, Teodorescu VJ, Ting W, et al. Endovascular treatment of visceral artery aneurysms. Vasc Endovascular Surg 2007;41:373-82.
105. Saltzberg SS, Maldonado TS, Lamparello PJ, Cayne NS, Nalbandian MM, Rosen RJ, et al. Is endovascular therapy the preferred treatment for all visceral artery aneurysms? Ann Vasc Surg 2005;19:507-15.
106. Hogendoorn W, Lavida A, Hunink MG, Moll FL, Geroulakos G, Muhs BE, et al. Cost-effectiveness of endovascular repair, open repair, and conservative management of splenic artery aneurysms. J Vasc Surg 2015;61:1432-40.
107. Chadha M, Ahuja C. Visceral artery aneurysms: diagnosis and percutaneous management. Semin Intervent Radiol 2009;26:196-206.
108. Abbas MA, Stone WM, Fowl RJ, Gloviczki P, Oldenburg WA, Pairolero PC, et al. Splenic artery aneurysms: two decades experience at Mayo Clinic. Ann Vasc Surg 2002;16:442-9.
109. Schanzer H, Papa MC, Miller CM. Rupture of surgically thrombosed abdominal aortic aneurysm. J Vasc Surg 1985;2:278-80.
110. Schwartz RA, Nichols WK, Silver D. Is thrombosis of the infrarenal abdominal aortic aneurysm an acceptable alternative? J Vasc Surg 1986;3:448-55.
111. Carr SC, Pearce WH, Vogelzang RL, McCarthy WJ, Nemcek AA Jr, Yao JS. Current management of visceral artery aneurysms. Surgery 1996;120:627-33; discussion: 633-4.
112. Onohara T, Okadome K, Mii S, Yasumori K, Muto Y, Sugimachi K. Rupture of embolised coeliac artery pseudoaneurysm into the stomach: is coil embolisation an effective treatment for coeliac anastomotic pseudoaneurysm? Eur J Vasc Surg 1992;6:330-2.
113. Sessa C, Tinelli G, Porcu P, Aubert A, Thony F, Magne JL. Treatment of visceral artery aneurysms: description of a retrospective series of 42 aneurysms in 34 patients. Ann Vasc Surg 2004;18:695-703.
114. Grosso M, Zanon E, Mancini A, Gomes Pavanello I, Bocchio A, Zanlungo D, et al. [Percutaneous transcatheter therapy of visceral pseudoaneurysms]. Minerva Chir 1998;53:363-8.
115. Miller DW Jr, Royster TS. Celiac artery aneurysm: rationale for celiac axis ligation with excisional treatment. Vasc Surg 1971;5:42-7.
116. Graham LM, Stanley JC, Whitehouse WM Jr, Zelenock GB, Wakefield TW, Cronenwett JL, et al. Celiac artery aneurysms: historic (1745-1949) versus contemporary (1950-1984) differences in etiology and clinical importance. J Vasc Surg 1985;2:757-64.
117. Stone WM, Abbas MA, Gloviczki P, Fowl RJ, Cherry KJ. Celiac arterial aneurysms: a critical reappraisal of a rare entity. Arch Surg 2002;137:670-4.
118. McMullan DM, McBride M, Livesay JJ, Dougherty KG, Krajcer Z. Celiac artery aneurysm: a case report. Tex Heart Inst J 2006;33:235-40.
119. Rokke O, Sondenaa K, Amundsen S, Bjerke-Larssen T, Jensen D. The diagnosis and management of splanchnic artery aneurysms. Scand J Gastroenterol 1996;31:737-43.
120. Hosaka A, Nemoto M, Miyata T. Outcomes of conservative management of spontaneous celiac artery dissection. J Vasc Surg 2017;65:760-5.e1.
121. Otsuka H, Sato T, Aoki H, Nakagawa Y, Inokuchi S. Optimal management strategy for spontaneous isolated dissection of a visceral artery. Vascular 2018;26:169-74.
122. Guo B, Guo D, Xu X, Chen B, Shi Z, Luo J, et al. Early and intermediate results of endovascular treatment of symptomatic and asymptomatic visceral artery aneurysms. J Vasc Surg 2016;64:140-8.
123. Tetreau R, Beji H, Henry L, Valette PJ, Pilleul F. Arterial splanchnic aneurysms: presentation, treatment and outcome in 112 patients. Diagn Interv Imaging 2016;97:81-90.
124. Atkins BZ, Ryan JM, Gray JL. Treatment of a celiac artery aneurysm with endovascular stent grafting—a case report. Vasc Endovascular Surg 2003;37:367-73.
125. D'Ayala M, Deitch JS, deGraft-Johnson J, Nguyen E, McGagh D, Gwertzman GA, et al. Giant celiac artery aneurysm with associated visceral occlusive disease. Vascular 2004;12:390-3.
126. Glickerman DJ, Hathaway PB, Hatsukami T, Daly CP, Althaus S, Kohler TR. Transluminal treatment of a celiac artery pseudoaneurysm with a stent graft occlusion device. Cardiovasc Intervent Radiol 1997;20:466-9.
127. Matsukura I, Iwai T, Inoue Y. Celiac artery aneurysm: report of two surgical cases. Surg Today 1999;29:948-52.
128. McIntyre TP, Simone ST, Stahlfeld KR. Intraoperative thrombin occlusion of a visceral artery aneurysm. J Vasc Surg 2002;36:393-5.
129. Rodrigus IE, Berry MC, Gdeedo A, Van Schil PE, Vanmaele RG. Surgical management of concurrent, coeliac and bilateral iliac artery aneurysms. Cardiovasc Surg 1995;3:501-3.
130. Waldenberger P, Bendix N, Petersen J, Tauscher T, Glodny B. Clinical outcome of endovascular therapeutic occlusion of the celiac artery. J Vasc Surg 2007;46:655-61.
131. Dinter DJ, Rexin M, Kaehler G, Neff W. Fatal coil migration into the stomach 10 years after endovascular celiac aneurysm repair. J Vasc Interv Radiol 2007;18(Pt 1):117-20.
132. Roberts KJ, McCulloch N, Forde C, Mahon B, Mangat K, Olliff SP, et al. Emergency treatment of haemorrhaging coeliac or mesenteric artery aneurysms and pseudoaneurysms in the era of endovascular management. Eur J Vasc Endovasc Surg 2015;49:382-9.
133. Hossain A, Reis ED, Dave SP, Kerstein MD, Hollier LH. Visceral artery aneurysms: experience in a tertiary-care center. Am Surg 2001;67:432-7.
134. Carmeci C, McClenathan J. Visceral artery aneurysms as seen in a community hospital. Am J Surg 2000;179:486-9.
135. Vasconcelos L, Garcia AC, Silva e Castro J, Albuquerque e Castro J, Mota Capitao L. Celiac artery aneurysms. Ann Vasc Surg 2010;24:554.e17-22.
136. Inada K, Maeda M, Ikeda T. Segmental arterial mediolysis: unrecognized cases culled from cases of ruptured aneurysm of abdominal visceral arteries reported in the Japanese literature. Pathol Res Pract 2007;203:771-8.

137. Chiaradia M, Novelli L, Deux JF, Tacher V, Mayer J, You K, et al. Ruptured visceral artery aneurysms. Diagn Interv Imaging 2015;96:797-806.
138. Vargas HA, Cousins C, Higgins JN, See TC. Left gastric artery aneurysm: successful embolization with ethylene vinyl alcohol copolymer (Onyx). Cardiovasc Intervent Radiol 2008;31:418-21.
139. Sandstrom A, Jha P. Ruptured left gastric artery aneurysms: three cases managed successfully with open surgical repair. Ann Vasc Surg 2016;36:296.e9-12.
140. Carr SC, Mahvi DM, Hoch JR, Archer CW, Turnipseed WD. Visceral artery aneurysm rupture. J Vasc Surg 2001;33:806-11.
141. Lagoudianakis EE, Filis KA, Tsekouras DK, Genetzakis M, Archontovassilis F, Pattas M, et al. Endovascular obliteration of a ruptured right gastric artery aneurysm. Minerva Gastroenterol Dietol 2006;52:333-7.
142. Schellhammer F, Steinhaus D, Cohnen M, Hoppe J, Modder U, Furst G. Minimally invasive therapy of pseudoaneurysms of the trunk: application of thrombin. Cardiovasc Intervent Radiol 2008;31:535-41.
143. Ikeda O, Nakasone Y, Tamura Y, Yamashita Y. Endovascular management of visceral artery pseudoaneurysms: transcatheter coil embolization using the isolation technique. Cardiovasc Intervent Radiol 2010;33:1128-34.
144. Lagana D, Carrafiello G, Mangini M, Dizonno M, Fugazzola C. Emergency endovascular treatment with stent graft of a gastric artery aneurysms (GAA). Emerg Radiol 2008;15:141-4.
145. Tseng KC, Hsieh YH, Lin CW, Chang SM, Wei CK. Aneurysms of the left gastric and splenic arteries presenting with massive upper gastrointestinal bleeding. Endoscopy 2009;41(Suppl 2):E131-2.
146. Shinoda N, Hirai O, Mikami K, Bando T, Shimo D, Kuroyama T, et al. Segmental arterial mediolysis involving both vertebral and middle colic arteries leading to subarachnoid and intraperitoneal hemorrhage. World Neurosurg 2016;88:694.e5-10.
147. Shimohira M, Ogino H, Sasaki S, Ishikawa K, Koyama M, Watanabe K, et al. Transcatheter arterial embolization for segmental arterial mediolysis. J Endovasc Ther 2008;15:493-7.
148. Fankhauser GT, Stone WM, Naidu SG, Oderich GS, Ricotta JJ, Bjarnason H, et al. The minimally invasive management of visceral artery aneurysms and pseudoaneurysms. J Vasc Surg 2011;53:966-70.
149. Batagini NC, El-Arousy H, Clair DG, Kirksey L. Open versus endovascular treatment of visceral artery aneurysms and pseudoaneurysms. Ann Vasc Surg 2016;35:1-8.
150. Abbas MA, Fowl RJ, Stone WM, Panneton JM, Oldenburg WA, Bower TC, et al. Hepatic artery aneurysm: factors that predict complications. J Vasc Surg 2003;38:41-5.
151. Lumsden AB, Mattar SG, Allen RC, Bacha EA. Hepatic artery aneurysms: the management of 22 patients. J Surg Res 1996;60:345-50.
152. Erben Y, De Martino RR, Bjarnason H, Duncan AA, Kalra M, Oderich GS, et al. Operative management of hepatic artery aneurysms. J Vasc Surg 2015;62:610-5.
153. Lal RB, Strohl JA, Piazza S, Aslam M, Ball D, Patel K. Hepatic artery aneurysm. J Cardiovasc Surg (Torino) 1989;30:509-13.
154. Chan RJ, Goodman TA, Aretz TH, Lie JT. Segmental mediolytic arteriopathy of the splenic and hepatic arteries mimicking systemic necrotizing vasculitis. Arthritis Rheum 1998;41:935-8.
155. den Bakker MA, Tangkau PL, Steffens TW, Tjiam SL, van der Loo EM. Rupture of a hepatic artery aneurysm caused by Wegener's granulomatosis. Pathol Res Pract 1997;193:61-6.
156. Caputo AE, Roberts WN, Yee YS, Posner MP. Hepatic artery aneurysm in corticosteroid-treated, adult Kawasaki's disease. Ann Vasc Surg 1991;5:533-7.
157. Hassen-Khodja R, Declemy S, Batt M, Castanet J, Perrin C, Ortonne JP, et al. Visceral artery aneurysms in von Recklinghausen's neurofibromatosis. J Vasc Surg 1997;25:572-5.
158. Koyama M, Tanaka M, Shimizu M, Nomura S, Kako N, Suzuki S, et al. Surgical treatment of mesenteric infarction, thoracoabdominal aortic aneurysm, and proper hepatic aneurysm in a middle-aged woman with Takayasu's arteritis. J Cardiovasc Surg (Torino) 1995;36:337-41.
159. Erskine JM. Hepatic artery aneurysm. Vasc Surg 1973;7:106-25.
160. Cordova AC, Sumpio BE. Visceral artery aneurysms and pseduoaneurysms—should they all be managed by endovascular techniques? Ann Vasc Dis 2013;6:687-93.
161. Hidalgo F, Narvaez JA, Rene M, Dominguez J, Sancho C, Montanya X. Treatment of hemobilia with selective hepatic artery embolization. J Vasc Interv Radiol 1995;6:793-8.
162. Tarazov PG, Ryzhkov VK, Polysalov VN, Prozorovskii KV, Polikarpov AA. Extraorganic hepatic artery aneurysm: failure of transcatheter embolization. HPB Surg 1998;11:55-9; discussion: 59-60.
163. McNamara MF, Griska LB. Superior mesenteric artery branch aneurysms. Surgery 1980;88:625-30.
164. Busuttil RW, Brin BJ. The diagnosis and management of visceral artery aneurysms. Surgery 1980;88:619-24.
165. Honarbakhsh A, Madjlessi HM, Davaii M, Saldjooghi H. Aneurysm of superior mesenteric artery: identification with ultrasonography. J Clin Ultrasound 1993;21:207-8.
166. Shanley CJ, Shah NL, Messina LM. Uncommon splanchnic artery aneurysms: pancreaticoduodenal, gastroduodenal, superior mesenteric, inferior mesenteric, and colic. Ann Vasc Surg 1996;10:506-15.
167. Dong Z, Fu W, Chen B, Guo D, Xu X, Wang Y. Treatment of symptomatic isolated dissection of superior mesenteric artery. J Vasc Surg 2013;57(Suppl):69S-76S.
168. Yun WS, Kim YW, Park KB, Cho SK, Do YS, Lee KB, et al. Clinical and angiographic follow-up of spontaneous isolated superior mesenteric artery dissection. Eur J Vasc Endovasc Surg 2009;37:572-7.
169. Lorelli DR, Cambria RA, Seabrook GR, Towne JB. Diagnosis and management of aneurysms involving the superior mesenteric artery and its branches—a report of four cases. Vasc Endovascular Surg 2003;37:59-66.
170. Kanazawa S, Inada H, Murakami T, Masaki H, Morita I, Tabuchi A, et al. The diagnosis and management of splanchnic artery aneurysms. Report of 8 cases. J Cardiovasc Surg (Torino) 1997;38:479-85.
171. Zelenock GB, Stanley JC. Splanchnic artery aneurysms. In: Rutherford RB, editor. Vascular surgery. Philadelphia: WB Saunders; 2000. p. 1369-82.
172. Tan C, Reul R. Inferior mesenteric artery aneurysm in the setting of celiac and superior mesenteric artery occlusion. J Vasc Surg Cases Innov Tech 2019;5:197-9.
173. Rahman Q, Naidu SG, Chong BW, Stone WM. Percutaneous embolization of an inferior mesenteric artery aneurysm in a patient with type IV Ehlers-Danlos syndrome. Vasc Endovascular Surg 2019;53:343-7.
174. Hansraj N, Hamdi A, Wise ES, DiChiacchio L, Sarkar R, Toursavadkohi S. Open and endovascular management of inferior mesenteric artery aneurysms: a report of two cases. Ann Vasc Surg 2017;43:316.e9-14.
175. Farber A, Wagner WH, Cossman DV, Cohen JL, Walsh DB, Fillinger MF, et al. Isolated dissection of the abdominal aorta: clinical presentation and therapeutic options. J Vasc Surg 2002;36:205-10.

176. Kim SK, Lee J, Duncan JR, Picus DD, Darcy MD, Sauk S. Endovascular treatment of superior mesenteric artery pseudoaneurysms using covered stents in six patients. AJR Am J Roentgenol 2014;203:432-8.
177. Shanley CJ, Shah NL, Messina LM. Common splanchnic artery aneurysms: splenic, hepatic, and celiac. Ann Vasc Surg 1996;10:315-22.
178. Morra A, Rimondini A, Adovasio R. Jejunal artery aneurysm: diagnostic efficacy of spiral CT angiography. A case report. Radiol Med 2002;104:95-8.
179. Pilleul F, Beuf O. Diagnosis of splanchnic artery aneurysms and pseudoaneurysms, with special reference to contrast enhanced 3D magnetic resonance angiography: a review. Acta Radiol 2004;45:702-8.
180. Sachdev-Ost U. Visceral artery aneurysms: review of current management options. Mt Sinai J Med 2010;77:296-303.
181. Goffi L, Chan R, Boccoli G, Ghiselli R, Saba V. Aneurysm of a jejunal branch of the superior mesenteric artery in a patient with Marfan's syndrome. J Cardiovasc Surg (Torino) 2000;41:321-3.
182. Hong YK, Yoo WH. Massive gastrointestinal bleeding due to the rupture of arterial aneurysm in Behçet's disease: case report and literature review. Rheumatol Int 2008;28:1151-4.
183. Sellke FW, Williams GB, Donovan DL, Clarke RE. Management of intra-abdominal aneurysms associated with periarteritis nodosa. J Vasc Surg 1986;4:294-8.
184. Tessier DJ, Abbas MA, Fowl RJ, Stone WM, Bower TC, McKusick MA, et al. Management of rare mesenteric arterial branch aneurysms. Ann Vasc Surg 2002;16:586-90.
185. Costa S, Costa A, Pereira T, Maciel J. Ruptured jejunal artery aneurysm. BMJ Case Rep 2013;2013. bcr2013008772.
186. Balderi A, Antonietti A, Ferro L, Peano E, Pedrazzini F, Fonio P, et al. Endovascular treatment of visceral artery aneurysms and pseudoaneurysms: our experience. Radiol Med 2012;117:815-30.
187. Rossi UG, Seitun S, Ferro C. Endovascular embolization of a third jejunal artery aneurysm: isolation technique using the Amplatzer vascular plug 4. Catheter Cardiovasc Interv 2013;81:1049-52.
188. Turkbey B, Peynircioglu B, Akpinar E, Cil BE, Karcaaltincaba M. Isolated aneurysm of the distal branch of the jejunal artery: MDCT angiographic diagnosis and endovascular management. Cardiovasc Intervent Radiol 2008;31(Suppl 2):S34-7.
189. Ham EM, Cho BS, Ye JB, Mun YS, Choi YJ, Kwon OS. The endovascular treatment of a ruptured aneurysm of the middle colic artery combined with an isolated dissection of superior mesenteric artery: report of a case. Vasc Endovascular Surg 2014;48:352-5.
190. Huang YK, Hsieh HC, Tsai FC, Chang SH, Lu MS, Ko PJ. Visceral artery aneurysm: risk factor analysis and therapeutic opinion. Eur J Vasc Endovasc Surg 2007;33:293-301.
191. Huo CW. Middle colic artery aneurysm: a case report and review of the literature. Ann Vasc Surg 2012;26:571.e1-6.
192. Moskowitz R, Rundback J. Middle colic artery branch aneurysm presenting as spontaneous hemoperitoneum. Ann Vasc Surg 2014;28:1797.e15-7.
193. Nishimura K, Hamasaki T, Ota R, Ohno T, Kodama W, Uchida N, et al. Elective treatment of middle colic artery aneurysm. Ann Vasc Dis 2014;7:328-30.
194. Lu M, Weiss C, Fishman EK, Johnson PT, Verde F. Review of visceral aneurysms and pseudoaneurysms. J Comput Assist Tomogr 2015;39:1-6.
195. Chivot C, Rebibo L, Robert B, Regimbeau JM, Yzet T. Ruptured pancreaticoduodenal artery aneurysms associated with celiac stenosis caused by the median arcuate ligament: a poorly known etiology of acute abdominal pain. Eur J Vasc Endovasc Surg 2016;51:295-301.
196. Ilica AT, Kocaoglu M, Bilici A, Ors F, Bukte Y, Senol A, et al. Median arcuate ligament syndrome: multidetector computed tomography findings. J Comput Assist Tomogr 2007;31:728-31.
197. Kallamadi R, Demoya MA, Kalva SP. Inferior pancreaticoduodenal artery aneurysms in association with celiac stenosis/occlusion. Semin Intervent Radiol 2009;26:215-23.
198. Perez C, Llauger J, Pallardo Y, Sanchis E, Sabate JM. Radiologic diagnosis of pseudoaneurysms complicating pancreatitis. Eur J Radiol 1993;16:102-6.
199. Uno A, Ishida H, Naganuma H, Niizawa M, Sawabe J, Masamune O. Color Doppler findings in small abdominal aneurysms. Abdom Imaging 1994;19:410-2.
200. Columbo JA, Trus T, Nolan B, Goodney P, Rzucidlo E, Powell R, et al. Contemporary management of median arcuate ligament syndrome provides early symptom improvement. J Vasc Surg 2015;62:151-6.
201. Gruber H, Loizides A, Peer S, Gruber I. Ultrasound of the median arcuate ligament syndrome: a new approach to diagnosis. Med Ultrason 2012;14:5-9.
202. Glockner JF, Takahashi N, Kawashima A, Woodrum DA, Stanley DW, Takei N, et al. Non-contrast renal artery MRA using an inflow inversion recovery steady state free precession technique (Inhance): comparison with 3D contrast-enhanced MRA. J Magn Reson Imaging 2010;31:1411-8.
203. Hartung MP, Grist TM, Francois CJ. Magnetic resonance angiography: current status and future directions. J Cardiovasc Magn Reson 2011;13:19.
204. Khoo MM, Deeab D, Gedroyc WM, Duncan N, Taube D, Dick EA. Renal artery stenosis: comparative assessment by unenhanced renal artery MRA versus contrast-enhanced MRA. Eur Radiol 2011;21:1470-6.
205. Loffroy R, Favelier S, Pottecher P, Genson PY, Estivalet L, Gehin S, et al. Endovascular management of visceral artery aneurysms: when to watch, when to intervene? World J Radiol 2015;7:143-8.
206. de Perrot M, Berney T, Deleaval J, Buhler L, Mentha G, Morel P. Management of true aneurysms of the pancreaticoduodenal arteries. Ann Surg 1999;229:416-20.
207. Corey MJ, Ergul EA, Cambria RP, English SJ, Patel VI, Lancaster RT, et al. SS24. The natural history of splanchnic artery aneurysms (SAAs) and outcomes following operative intervention. J Vasc Surg 2015;61(Suppl):110s-1s.
208. Katsura M, Gushimiyagi M, Takara H, Mototake H. True aneurysm of the pancreaticoduodenal arteries: a single institution experience. J Gastrointest Surg 2010;14:1409-13.
209. Shukla AJ, Eid R, Fish L, Avgerinos E, Marone L, Makaroun M, et al. Contemporary outcomes of intact and ruptured visceral artery aneurysms. J Vasc Surg 2015;61:1442-7.
210. De Santis F, Bruni A, Da Ros V, Chaves Brait CM, Scevola G, Di Cintio V. Multiple pancreatoduodenal artery arcade aneurysms associated with celiac axis root segmental stenosis presenting as aneurysm rupture. Ann Vasc Surg 2015;29:1657.e1-7.
211. Boll JM, Sharp KW, Garrard CL, Naslund TC, Curci JA, Valentine RJ. Does management of true aneurysms of peripancreatic arteries require repair of associated celiac artery stenosis? J Am Coll Surg 2017;224:199-203.
212. Corey MR, Ergul EA, Cambria RP, English SJ, Patel VI, Lancaster RT, et al. The natural history of splanchnic artery aneurysms and outcomes after operative intervention. J Vasc Surg 2016;63:949-57.
213. Ferrero E, Ferri M, Viazzo A, Robaldo A, Carbonatto P, Pecchio A, et al. Visceral artery aneurysms, an experience

on 32 cases in a single center: treatment from surgery to multilayer stent. Ann Vasc Surg 2011;25:923-35.
214. Takao H, Nojo T, Ohtomo K. True pancreaticoduodenal artery aneurysms: a decision analysis. Eur J Radiol 2010;75:110-3.
215. Brocker JA, Maher JL, Smith RW. True pancreaticoduodenal aneurysms with celiac stenosis or occlusion. Am J Surg 2012;204:762-8.
216. Orion KC, Najafian A, Ehlert BA, Malas MB, Black JH 3rd, Abularrage CJ. Gender predicts rupture of pancreaticoduodenal artery aneurysms. Ann Vasc Surg 2016;36:1-6.
217. Guillon R, Garcier JM, Abergel A, Mofid R, Garcia V, Chahid T, et al. Management of splenic artery aneurysms and false aneurysms with endovascular treatment in 12 patients. Cardiovasc Intervent Radiol 2003;26:256-60.
218. Roy D, Milot G, Raymond J. Endovascular treatment of unruptured aneurysms. Stroke 2001;32:1998-2004.
219. Mascitelli JR, Moyle H, Oermann EK, Polykarpou MF, Patel AA, Doshi AH, et al. An update to the Raymond-Roy Occlusion Classification of intracranial aneurysms treated with coil embolization. J Neurointerv Surg 2015;7:496-502.
220. Marjanovic IR, Jevtic M, Misovic S, Rusovic S, Zoranovic U, Sarac M. Endovascular reconstruction of giant gastroduodenal artery aneurysm with stent graft: case report. Vasc Endovascular Surg 2010;44:392-4.
221. Lykoudis PM, Stafyla VK, Koutoulidis V, Xatziioannou A, Arkadopoulos N, Mourikis I, et al. Stenting of a gastroduodenal artery aneurysm: report of a case. Surg Today 2012;42:72-4.
222. Won Y, Lee SL, Kim Y, Ku YM. Clinical efficacy of transcatheter embolization of visceral artery pseudoaneurysms using N-butyl cyanoacrylate (NBCA). Diagn Interv Imaging 2015;96:563-9.
223. Mazzaccaro D, Carmo M, Nano G, Barbetta I, Settembrini AM, Occhiuto MT, et al. Treatment options for visceral artery aneurysms: ten year experience. J Cardiovasc Surg (Torino) 2015;56:423-32.
224. Song HH, Won YD, Kim YJ. Transcatheter N-butyl cyanoacrylate embolization of pseudoaneurysms. J Vasc Interv Radiol 2010;21:1508-11.
225. Pollak JS, White RI Jr. The use of cyanoacrylate adhesives in peripheral embolization. J Vasc Interv Radiol 2001;12:907-13.
226. Shankar JJ, Tampieri D, Iancu D, Cortes M, Agid R, Krings T, et al. SILK flow diverter for complex intracranial aneurysms: a Canadian registry. J Neurointerv Surg 2016;8:273-8.
227. Ruffino M, Rabbia C. Endovascular treatment of visceral artery aneurysms with Cardiatis multilayer flow modulator: preliminary results at six-month follow-up. J Cardiovasc Surg (Torino) 2011;52:311-21.
228. Sutton D, Lawton G. Coeliac stenosis or occlusion with aneurysm of the collateral supply. Clin Radiol 1973;24:49-53.
229. Moore E, Matthews MR, Minion DJ, Quick R, Schwarcz TH, Loh FK, et al. Surgical management of peripancreatic arterial aneurysms. J Vasc Surg 2004;40:247-53.
230. Bowens NM, Woo EY, Fairman RM. Reno-hepatic artery bypass for an inferior pancreaticoduodenal artery aneurysm with associated celiac occlusion. J Vasc Surg 2011;53:1696-8.
231. Tien YW, Kao HL, Wang HP. Celiac artery stenting: a new strategy for patients with pancreaticoduodenal artery aneurysm associated with stenosis of the celiac artery. J Gastroenterol 2004;39:81-5.
232. Sugiyama K, Takehara Y. Analysis of five cases of splanchnic artery aneurysm associated with coeliac artery stenosis due to compression by the median arcuate ligament. Clin Radiol 2007;62:688-93.
233. Grech P, Rowlands P, Crofton M. Aneurysm of the inferior pancreaticoduodenal artery diagnosed by real-time ultrasound and pulsed Doppler. Br J Radiol 1989;62:753-5.
234. Salam TA, Lumsden AB, Martin LG, Smith RB 3rd. Nonoperative management of visceral aneurysms and pseudoaneurysms. Am J Surg 1992;164:215-9.
235. Yasumoto T, Osuga K, Yamamoto H, Ono Y, Masada M, Mikami K, et al. Long-term outcomes of coil packing for visceral aneurysms: correlation between packing density and incidence of coil compaction or recanalization. J Vasc Interv Radiol 2013;24:1798-807.
236. Skipworth JR, Morkane C, Raptis DA, Kennedy L, Johal K, Pendse D, et al. Coil migration—a rare complication of endovascular exclusion of visceral artery pseudoaneurysms and aneurysms. Ann R Coll Surg Engl 2011;93:e19-23.
237. Yamamoto S, Hirota S, Maeda H, Achiwa S, Arai K, Kobayashi K, et al. Transcatheter coil embolization of splenic artery aneurysm. Cardiovasc Intervent Radiol 2008;31:527-34.
238. Koganemaru M, Abe T, Uchiyama D, Iwamoto R, Yoshida S, Hayabuchi N, et al. Detection of neck recanalization with follow-up contrast-enhanced MR angiography after renal artery aneurysm coil embolization. J Vasc Interv Radiol 2010;21:298-300.
239. Koganemaru M, Abe T, Nonoshita M, Iwamoto R, Kusumoto M, Kuhara A, et al. Follow-up of true visceral artery aneurysm after coil embolization by three-dimensional contrast-enhanced MR angiography. Diagn Interv Radiol 2014;20:129-35.
240. Piscaglia F, Gualandi S, Galassi M, Giampalma E, Golfieri R, Bolondi L. Contrast enhanced ultrasonography for the evaluation of coil embolization of splenic artery aneurysm. Circulation 2010;122:e451-4.

Submitted Jan 21, 2020; accepted Jan 22, 2020.