

Managing Incidental Findings on Abdominal and Pelvic CT and MRI, Part 2: White Paper of the ACR Incidental Findings Committee II on Vascular Findings

Faisal Khosa, MD^a, Glenn Krinsky, MD^b, Michael Macari, MD^c,
E. Kent Yucel, MD^d, Lincoln L. Berland, MD^e

This white paper describes vascular incidental findings found on CT and MRI of the abdomen and pelvis. Recommendations for management are included. This represents the second of 4 such papers from the ACR Incidental Findings Committee II, which used a consensus method based on repeated reviews and revisions and a collective review and interpretation of relevant literature. Topics include definitions and recommended management for abdominal aortic, iliac, splenic, renal, and visceral artery aneurysms. Other incidentally discovered aortic conditions, systemic venous anomalies, compression syndromes, abdominal venous thrombosis, and gonadal and pelvic venous conditions are also discussed. A table is provided for reference.

Key Words: Incidental findings, incidentaloma, ACR, consensus, CT, MRI, aneurysm

J Am Coll Radiol 2013;10:789-794. Copyright © 2013 American College of Radiology

FOREWORD

This white paper does not comprehensively review the interpretation and management of vascular abnormalities but provides general guidance for managing common, incidental vascular findings on CT and MRI, appreciating that individual care will vary depending on each patient's specific circumstances, the clinical environment, available resources, and the judgment of practitioners. Also, the term *guidelines* is not used in this or prior white papers to avoid the implication that these represent components of the ACR Practice Guidelines

and Technical Standards (which represent official ACR policy, having undergone a rigorous drafting and review process culminating in approval by the ACR Council) or the ACR Appropriateness Criteria[®] (which use a formal consensus-building approach using a modified Delphi technique). This white paper, which represents the collective experience of the members of the ACR Incidental Findings Committee II, was developed through a less formal process of repeated reviews and revisions of the draft document and does not represent official ACR policy. For these reasons, this white paper should not be used to establish the legal standard of care in any particular situation.

Credits awarded for this enduring activity are designated "SA-CME" by the American Board of Radiology (ABR) and qualify toward fulfilling requirements for Maintenance of Certification (MOC) Part II: Lifelong Learning and Self-assessment.

^aDepartment of Radiology and Imaging Sciences, Emory University Hospital, Atlanta, Georgia. Dr. Khosa is an ARRS scholar.

^bDepartment of Radiology, Valley Hospital/Valley Health Care System, Ridgewood, New Jersey.

^cDepartment of Radiology, New York University, Langone Medical Center, New York, New York (deceased).

^dDepartment of Radiology, Tufts Medical Center, Boston, Massachusetts.

^eDepartment of Radiology, University of Alabama at Birmingham, Birmingham, Alabama.

Corresponding author and reprints: Lincoln L. Berland, MD, University of Alabama at Birmingham, Department of Radiology, 619 S 19th Street, N454, Birmingham, AL 35249; e-mail: lberland@uabmc.edu.

INTRODUCTION

Please refer to the overview of the work of the Incidental Findings Committee II [1] for a description of the purposes, structure and process, and conventions used in these 4 white papers, of which this is the second. The authors of this white paper represent the Vascular Subcommittee membership, as listed in the [appendix](#). The roster of the entire Incidental Findings Committee II is listed in the [appendix](#) of the overview of this project [1].

INCIDENTAL VASCULAR FINDINGS

This section describes the committee's consensus on nomenclature and recommendations for managing and fol-

lowing up the most common and important types of incidental vascular findings seen on CT and MRI of the abdomen and pelvis.

Abdominal Aortic Aneurysm (AAA)

AAA represents a progressive increase in the aortic luminal diameter and is the 10th most common cause of death in the Western world [2]. AAA is usually described by its relationship to renal arteries (ie, suprarenal or infrarenal). The normal diameter of the suprarenal abdominal aorta is up to 3.0 cm, and that of the infrarenal abdominal aorta is 2.0 cm. Aneurysmal dilation of the infrarenal aorta is defined as a diameter ≥ 3.0 cm or dilation of the aorta ≥ 1.5 times the normal diameter [3]; on the basis of these criteria, 9% of people aged >65 years have an AAA [4].

Multiple causes may contribute to the development and progression of aortic aneurysms, including smoking, male gender, age at detection, diabetes, hypertension, and hypercholesterolemia [5,6]. There is also a strong association between initial size and the subsequent rate of growth of an AAA. Therefore, we recommend tailoring follow-up intervals according to the size of the AAA at the time of detection, especially when previous growth patterns are unknown [7,8]. Emergency surgery for aortic aneurysm rupture is associated with 46% mortality (as opposed to 4%-6% for elective repair), and rupture occurs with increasing frequency as the aneurysm size exceeds 5 cm [9]. It is therefore valuable to detect AAAs and follow up until elective repair is indicated [10].

Detection and Characterization. An AAA may be encountered as an incidental finding on ultrasound, CT, or MRI. Ultrasound can detect and size AAAs, with the advantages of being relatively inexpensive and noninvasive and not requiring the use of intravenous contrast material. The limitations are that overlying bowel gas can obscure findings, and ultrasound is operator dependent. CT and MRI can define the extent and size of an aneurysm, depict intraluminal thrombus, and show the involvement and relationship of branch vessels, allowing the correct choice of therapy. Optimizing follow-up imaging intervals can help balance safety, cost, and patient anxiety. For abdominal aortic diameters <2.5 cm, follow-up is generally thought to be unnecessary. Because the rupture of smaller AAAs is less likely [10], and a recent meta-analysis [11] suggested that less frequent follow-up on smaller aneurysms in male patients may be equally safe, we recommend longer intervals between follow-up examinations. The follow-up intervals for imaging may also vary depending on comorbidities and the growth rate of the aneurysm [12]. Our recommendations, listed in Table 1, are based on published literature [7,13,14] and the consensus opinion of the committee.

Table 1. Recommended intervals for initial follow-up imaging of ectatic aortas and abdominal aortic aneurysms

Aortic Diameter (mm)	Imaging Interval
2.5-2.9	5 y
3.0-3.4	3 y
3.5-3.9	2 y
4.0-4.4	1 y
4.5-4.9	6 mo*
5.0-5.5	3-6 mo*

Note: For abdominal aortic diameters <2.5 cm, follow-up is generally thought to be unnecessary. Because the rupture of smaller abdominal aortic aneurysms is less likely, we recommend longer intervals between follow-up examinations. Follow-up intervals may vary depending on comorbidities and the growth rate of the aneurysm.

*In addition to planning follow-up imaging, one should also consider surgical or endovascular referral.

Penetrating Aortic Ulcer (PAU)

Penetrating aortic ulcers (PAUs) represent disruption of atherosclerotic plaque with penetration of luminal blood for variable distances into or through the aortic wall. They may present with acute symptoms and findings, but they may also be recognized as chronic, asymptomatic, incidental findings. A PAU may progress to an intramural hematoma, a focal dissection, or a pseudoaneurysm and rupture, or it may completely resolve [15]. It is typically seen in elderly patients with atherosclerosis, and because the symptoms of a rupturing PAU may be insidious, it may be difficult to determine by imaging alone if it is causing a patient's symptoms or is incidental. It also may be difficult to differentiate PAU from asymptomatic aortic conditions such as saccular pseudoaneurysms and true aneurysms on the basis of imaging alone. Studies have shown that the natural history of PAU is variable, unpredictable, and may be one of progressive enlargement resulting in rupture. Because the lack of symptoms does not necessarily imply stability, we recommend annual follow-up when asymptomatic and more frequent follow-up if symptoms arise [16], with consideration of surgical or endovascular intervention.

Dissections and intramural hematomas are almost always symptomatic and, therefore, are not usually incidental findings and are not further discussed in this white paper.

Iliac Artery Aneurysm

Aneurysms involve common and internal iliac arteries more commonly than external iliac arteries. Iliac artery aneurysm is defined as a vessel diameter ≥ 1.5 times the normal iliac artery diameter or ≥ 2.5 cm in diameter. Iliac artery aneurysms are rare in isolation; Lawrence et al [17] reported a prevalence of 6.58 per 100,000 hospitalized men and 0.26 per 100,000 hospitalized women in the United States. Aneurysms that are <3.0 cm in diameter tend to be asymptomatic, rarely rupture, and expand slowly; those that are 3.0 to 3.5 cm should be followed up with cross-sectional imaging initially at about 6 months. If stable, repeat scan-

ning can be performed annually. Iliac artery aneurysms >3.5 cm have a greater tendency to rupture and should be followed more closely or treated expeditiously [18].

Splenic Artery Aneurysm

Splenic artery aneurysms are the most common visceral aneurysms and the third most common intra-abdominal aneurysm, after those occurring in the aorta and iliac arteries [19-21]. In a series of >300 visceral artery aneurysms, 70.9% were of the splenic artery [20]. The vast majority are true aneurysms, although pseudoaneurysms related to prior inflammation, especially pancreatitis, or infection may occur [21]. The estimates of prevalence of splenic artery aneurysms vary, but a retrospective review of nonselective angiograms suggests that an incidence estimate of 0.8% may be the most accurate [21].

Risk factors for developing these aneurysms are similar to those for other aneurysms. In a review of the clinical features of 217 patients with splenic artery aneurysms, hypertension was present in 50.2%, obesity in 27.6%, coronary artery disease in 23.5%, and hypercholesterolemia in 21.7% [20]. Splenic artery aneurysms occur more frequently in women [21]. As mentioned previously, pseudoaneurysms of the splenic artery are rare [19]. In a series of 128 patients from the Cleveland Clinic with splenic artery aneurysms, only 6 (4.7%) were believed to be pseudoaneurysms, with 5 of these occurring in the setting of acute or chronic pancreatitis [21]. Most splenic artery aneurysms are incidentally detected during cross-sectional imaging [20,21] and it is difficult to confidently attribute abdominal symptoms to such aneurysms.

Spontaneous rupture of a splenic artery aneurysm is rare, especially for smaller (<2 cm) aneurysms [20], but may occur, usually with larger aneurysms. Additional risk factors associated with rupture include rapidly increasing size, occurrence in women of childbearing years, cirrhosis (especially associated with α_1 antitrypsin deficiency), and symptoms that can be attributable to the aneurysm [19-21].

The surgical literature suggests a consensus that such an aneurysm should be considered for endovascular therapy when ≥ 2 cm [20,21]. Smaller aneurysms probably can be safely followed, although the clinical risk factors for rupture should be carefully assessed. In one review of patients who were followed with small splenic artery aneurysms, the mean aneurysm growth rate was 0.06 cm/y, with the most rapid growth rate noted to be 1 cm over 63 months [20]. In this group of patients, none of the aneurysms ruptured [20]. Given these data, yearly surveillance for small splenic artery aneurysms is recommended, although for the smaller aneurysms among those ≥ 2 cm, surveillance intervals of >1 year may be reasonable, depending on comorbidities and life expectancy.

Renal Artery Aneurysm (RAA)

RAA is uncommon, occurring in about 0.09% of the population [22,23]. Etiologies include fibromuscular dysplasia (FMD), atherosclerosis, and pseudoaneurysms that may oc-

cur after trauma [23]. In a review of 168 patients with 252 RAAs, 34% had FMD, 25% had atherosclerosis, 6.5% had concurrent aneurysms of other vessels, and 73% had hypertension [22]. RAAs are usually detected incidentally at cross-sectional imaging [23], are small, are asymptomatic, and have uncertain clinical relevance [22]. However, they may rupture, especially if they enlarge, and may be associated with renal arterial hypertension [22,23].

RAAs related to FMD should be considered when there is a classic "string of beads" appearance to the renal artery or when aneurysms occur in younger women, especially when associated with hypertension [22]. Pseudoaneurysms typically occur after trauma and are usually located within the parenchyma of the kidney [22,23]. Other aneurysms, not related to FMD or trauma, typically occur at branch points in the renal artery.

The decision to treat or repair an RAA should be based on factors including patient age and gender and anatomic features of the aneurysm, including size [22]. One approach that has been suggested is to repair all aneurysms ≥ 1 cm in patients with uncontrolled hypertension [22]. An incidentally discovered RAA measuring 1.0 to 1.5 cm can be safely followed [22,23]. In a series of 86 RAAs with a mean size of 1.3 cm, none ruptured after an average follow-up of 72 months [22]. We recommend that a reasonable imaging follow-up interval in these asymptomatic individuals is every 1 to 2 years. Larger aneurysms, measuring >1.5 to 2.0 cm, should be considered for surgical or endovascular repair [22].

Other Visceral Artery Findings

Outside of the splenic and renal circulations, visceral aneurysms can affect the celiac, hepatic, gastroduodenal, pancreaticoduodenal, gastric, or mesenteric arteries. After splenic and renal arterial aneurysms, the hepatic artery is the next most common location [24]. When discovered incidentally, these aneurysms are typically caused by atherosclerosis and may be associated with aneurysmal disease elsewhere [25]. They can also be mycotic, traumatic (including iatrogenic trauma for hepatic aneurysms after liver biopsy), or, less commonly, related to polyarteritis nodosa, FMD, or visceral inflammatory disease, such as pancreatitis [25,26]. As with all aneurysms, rupture is the feared risk and is the reason for considering surgical or endovascular treatment.

Treatment is generally recommended for aneurysms >2 cm in diameter, possibly with a smaller threshold for nonatherosclerotic aneurysms [27,28]. For hepatic aneurysms, Abbas et al [29] established that multiplicity and nonatherosclerotic origin were linked to increased rupture rate. Criteria for which it is safe to observe visceral arterial aneurysms have not been clearly established. In the study of Abbas et al, of 21 patients with a mean follow-up interval of 68.4 months and mean diameter of 2.3 cm, none required intervention during the follow-up period.

However, pancreaticoduodenal aneurysms are felt to be at higher risk for rupture, and some authors recom-

ment that all of these aneurysms undergo surgical or endovascular treatment, regardless of size [30,31]. If a decision is made to observe rather than treat, repeat scanning at annual intervals is recommended to assess for interval growth. Initial scanning could be at a shorter interval, especially if pseudoaneurysm is a consideration.

Isolated visceral arterial dissection, typically of the superior mesenteric artery, can occasionally be seen as an incidental finding. Cho et al [32] published a study of 30 patients, 13 of whom were asymptomatic and 25 of whom were treated conservatively without surgical or percutaneous intervention. In the 15 patients followed with CT for a mean of 15.6 months, none progressed, 7 showed no change, 4 improved, and 4 completely resolved. The researchers concluded that follow-up imaging, rather than prompt treatment, is appropriate for asymptomatic dissection.

The arcuate ligament can occasionally compress the celiac axis origin; this is demonstrable on cross-sectional imaging performed at end-expiration [33]. When noted incidentally in a patient without relevant symptoms, no further action is necessary. The celiac, superior, and inferior mesenteric artery origins are frequently affected by atherosclerotic stenosis. As long as this remains well compensated by collateral vessels and is not symptomatic with postprandial abdominal pain or weight loss, no further evaluation or follow-up is recommended.

Systemic Venous Anomalies and Compression Syndromes

A variety of congenital anomalies and pathologic conditions can affect the inferior vena cava (IVC) and branch vessels, including caval duplication, left-sided IVC, azygous continuation of the IVC and retroaortic or circum-aortic renal veins [34]. Most congenital anomalies are asymptomatic, but familiarity with their appearance helps avoid misinterpreting them as abnormal findings. Awareness of vascular anatomic variants becomes important when an asymptomatic patient volunteers to donate a kidney or a liver segment or if an interventional or vascular procedure is planned.

The size and shape of the IVC can vary with respiration, volume status, and cardiac output. A "flat" IVC, when associated with other CT abnormalities, may be caused by volume depletion or shock but can also be seen as a normal variant [35]. Dilation or reflux opacification of the intrahepatic IVC or hepatic veins has been associated with cardiac disease but the latter can also be seen in normal patients undergoing CT with high injection rates (>3 mL/s) [36]. The most common IVC filling defect seen on CT is pseudothrombosis caused by laminar flow of enhanced blood from the renal veins streaming parallel to the column of unopacified blood returning from the lower body [37]. Similar artifactual filling defects in other vessels may also result from mixing of enhanced and unenhanced blood or from laminar reflux of opacified blood from the heart into the IVC. Delayed CT,

ultrasound, or MR venography can be performed for indeterminate cases [38].

Compression of the left common iliac vein by the anteriorly crossing right common iliac artery, known as May-Thurner or iliocaval compression syndrome, may be associated with left lower-extremity venous hypertension, edema, and venous thrombosis (VT). However, both cadaveric and retrospective CT studies from asymptomatic patients [39] suggest that this anatomic variant is present in approximately 25% of the population, indicating that most patients with compression are not symptomatic [40], and follow-up is not necessary unless the patient develops unilateral symptoms of leg swelling or VT. Similarly, compression of the left renal vein between the aorta and superior mesenteric artery with localized varices, known as the nutcracker syndrome, is an occasional asymptomatic incidental finding. If the patient is not hypertensive and does not have proteinuria or hematuria, this anatomic finding is likely clinically unimportant.

Incidental Abdominal VT

VT has many causes, including hypercoagulable states, portal hypertension, vessel wall damage, and cancer. Imaging signs of VT may be direct or indirect, with imaging often helping differentiate between acute and chronic thrombi and between bland and tumor thrombi [38,41]. In a recent retrospective study of 2,619 consecutive abdominal CT examinations performed in a nonselected patient population, the prevalence of VT was 1.74% [42]. In the same study, when patients with malignancy and cirrhosis were excluded, the prevalence decreased to 0.36%. However, these data are from a single retrospective study in which pelvic veins were not evaluated, so these results may not be generalizable.

How to further evaluate VT depends on location and the local availability and expertise for particular techniques. Peripheral and superficial VT is usually best studied with ultrasound; for intracranial, intrathoracic, or abdominopelvic VT, CT or MRI may be the preferred modality. Nuclear medicine flow studies have essentially been replaced by cross-sectional imaging. Once VT is diagnosed as chronic (small vein, wall thickening, extensive collateral vessels, absence of intraluminal filling defects other than webs), usually no follow-up imaging is recommended.

Gonadal and Pelvic Veins

Ovarian veins originate from the plexus in the broad ligament near the ovary and fallopian tubes and communicate with the uterine plexus, then course anterior to the psoas muscle and the ureter [43]. The right ovarian vein typically drains into the IVC and the left ovarian vein into the left renal vein. Autopsy studies have shown that valves are absent in the cranial portion of the ovarian vein in 15% of women on the left and 6% on the right [44]. The valves are incompetent on either side in 35% to 43%, with a higher frequency in multiparous women, resulting in dilation >8 mm and incompetence in many asymptomatic patients

who undergo CT [45]. Anatomic differences of the ovarian veins cannot solely explain different rates of reflux between the right and left systems, but the decreased diameter of the left renal vein at the aortomesenteric portion likely contributes to this finding [46], similar to the nutcracker syndrome described above.

Incompetence of the ovarian and draining pelvic veins and venous reflux are considered the main cause of pelvic congestion syndrome in women, symptoms of which include persistent dull pelvic pain lasting >6 months, dysmenorrhea, dyspareunia, postcoital ache, and urinary symptoms. However, dilated pelvic veins are often seen incidentally in asymptomatic multiparous women [46]. If dilated pelvic veins are noted in a woman and are asymptomatic, no further imaging or intervention is recommended. In a CT angiographic study of potential renal donors, dilated ovarian veins were found in 16 (47%) of 34 asymptomatic women [45]. In another CT study of patients with severe ovarian vein reflux, but without PCS, both right and left parauterine veins were tortuous and dilated in all cases, with a mean vein diameter of 5.9 ± 1.6 mm (range, 4.3–8.0 mm) [46]. Pelvic varices, and early opacification and dilation of the gonadal veins, may occur without venous reflux, particularly if uterine fibroids [47] or other pelvic abnormalities are present [48].

Gonadal vein thrombosis can be seen in up to 80% of asymptomatic women who undergo routine CT after hysterectomy and lymphadenectomy for neoplasm [49]. When acute, the central thrombus typically demonstrates low attenuation and is associated with mural enhancement. The vessel chronically becomes fibrotic and contracted, and phleboliths may develop [49].

TAKE-HOME POINTS

- The committee recommends follow-up imaging at decreasing intervals with increasing diameter for AAA (3 years for 3.0–3.4 cm, 2 years for 3.5–3.9 cm, 1 year for 4.0–4.4 cm, 6 months for 4.5–4.9 cm, and 3–6 months for 5.0–5.5 cm) and consideration of vascular or endoscopic referral for the larger AAAs in these ranges.
- Iliac artery aneurysms 3.0 to 3.5 cm in size should be followed up with cross-sectional imaging initially at about 6 months.
- The surgical literature suggests a consensus that RAAs should be considered for endovascular therapy when ≥ 2 cm, while smaller aneurysms probably can be safely followed.
- For visceral artery aneurysms, treatment is generally recommended for aneurysms > 2 cm in diameter, possibly with a smaller threshold for nonatherosclerotic aneurysms.
- Pancreaticoduodenal aneurysms are felt to be at higher risk for rupture, and all of these aneurysms should be considered for surgical or endovascular treatment.
- Incompetence of the ovarian and draining pelvic veins and venous reflux are considered the main cause of

pelvic congestion syndrome, symptoms of which include persistent dull pelvic pain lasting >6 months, dysmenorrhea, dyspareunia, postcoital ache, and urinary symptoms.

ACKNOWLEDGMENTS

The authors thank David Kurth of the ACR staff, who helped support and coordinate the activities that led to this report, and Dr Joel Platt for review and comments.

APPENDIX

Committee Members

Incidental Findings Committee II: Lincoln L. Berland, MD (chair).

Vascular Subcommittee: Faisal Khosa, MD (chair), Glenn Krinsky, MD, Michael Macari, MD, E. Kent Yucel, MD.

Ex Officio: James A. Brink, MD (chair), ACR Body Imaging Commission.

REFERENCES

1. Berland LL. Overview of white papers of the ACR Incidental Findings Committee II on ovarian and paraovarian, vascular, splenic, nodal, gallbladder, and biliary findings. *J Am Coll Radiol* 2013;10:672-4.
2. Bengtsson H, Sonesson B, Bergqvist D. Incidence and prevalence of abdominal aortic aneurysms, estimated by necropsy studies and population screening by ultrasound. *Ann N Y Acad Sci* 1996;800:1-24.
3. Golledge J, Muller J, Daugherty A, Norman P. Abdominal aortic aneurysm: pathogenesis and implications for management. *Arterioscler Thromb Vasc Biol* 2006;26:2605-13.
4. Alcorn HG, Wolfson SK Jr, Sutton-Tyrrell K, Kuller LH, O'Leary D. Risk factors for abdominal aortic aneurysms in older adults enrolled in the Cardiovascular Health Study. *Arterioscler Thromb Vasc Biol* 1996;16:963-70.
5. Chang JB, Stein TA, Liu JP, Dunn ME. Risk factors associated with rapid growth of small abdominal aortic aneurysms. *Surgery* 1997;121:117-22.
6. Iribarren C, Darbinian JA, Go AS, Fireman BH, Lee CD, Grey DP. Traditional and novel risk factors for clinically diagnosed abdominal aortic aneurysm: the Kaiser multiphasic health checkup cohort study. *Ann Epidemiol* 2007;17:669-78.
7. Brady AR, Thompson SG, Fowkes FG, Greenhalgh RM, Powell JT. Abdominal aortic aneurysm expansion: risk factors and time intervals for surveillance. *Circulation* 2004;110:16-21.
8. Golledge J, Muller J, Coomans D, Walker PJ, Norman PE. The small abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 2006;31:237-8.
9. Brown PM, Pattenden R, Vernooy C, Zelt DT, Gutelius JR. Selective management of abdominal aortic aneurysms in a prospective measurement program. *J Vasc Surg* 1996;23:213-20.
10. Brown PM, Sobolev B, Zelt DT. Selective management of abdominal aortic aneurysms smaller than 5.0 cm in a prospective sizing program with gender-specific analysis. *J Vasc Surg* 2003;38:762-5.
11. The RESCAN Collaborators. Surveillance intervals for small abdominal aortic aneurysms. *JAMA* 2013;309:806-13.
12. Hirsch AT, Haskal ZJ, Hertzner NR, et al. ACC/AHA 2005 practice guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): 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): endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. *Circulation* 2006;113:e463-654.
13. McCarthy RJ, Shaw E, Whyman MR, Earnshaw JJ, Poskitt KR, Heather BP. Recommendations for screening intervals for small aortic aneurysms. *Br J Surg*. Jul 2003;90:821-6.
 14. Powell JT, Brady AR. Detection, management, and prospects for the medical treatment of small abdominal aortic aneurysms. *Arterioscler Thromb Vasc Biol* 2004;24:241-5.
 15. Braverman AC. Penetrating atherosclerotic ulcers of the aorta. *Curr Opin Cardiol* 1994;9:591-7.
 16. Quint LE, Williams DM, Francis IR, et al. Ulcerlike lesions of the aorta: imaging features and natural history. *Radiology* 2001;218:719-23.
 17. Lawrence PF, Lorenzo-Rivero S, Lyon JL. The incidence of iliac, femoral, and popliteal artery aneurysms in hospitalized patients. *J Vasc Surg* 1995;22:409-15.
 18. Santilli SM, Wernsing SE, Lee ES. Expansion rates and outcomes for iliac artery aneurysms. *J Vasc Surg* 2000;31:114-21.
 19. Agrawal GA, Johnson PT, Fishman EK. Splenic artery aneurysms and pseudoaneurysms: clinical distinctions and CT appearances. *AJR Am J Roentgenol* 2007;188:992-9.
 20. Abbas MA, Stone WM, Fowl RJ, et al. Splenic artery aneurysms: two decades experience at Mayo clinic. *Ann Vasc Surg* 2002;16:442-9.
 21. Lakin RO, Bena JF, Sarac TP, et al. The contemporary management of splenic artery aneurysms. *J Vasc Surg* 2011;53:958-64.
 22. Henke PK, Cardneau JD, Welling TH III, et al. Renal artery aneurysms: a 35-year clinical experience with 252 aneurysms in 168 patients. *Ann Surg* 2001;234:454-62.
 23. Cura M, Elmerhi F, Bugnogne A, Palacios R, Suri R, Dalsaso T. Renal aneurysms and pseudoaneurysms. *Clin Imaging* 2011;35:29-41.
 24. 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.
 25. Carr SC, Mahvi DM, Hoch JR, Archer CW, Turnipseed WD. Visceral artery aneurysm rupture. *J Vasc Surg* 2001;33:806-11.
 26. Messina LM, Shanley CJ. Visceral artery aneurysms. *Surg Clin North Am* 1997;77:425-42.
 27. 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.
 28. Berceli SA. Hepatic and splenic artery aneurysms. *Semin Vasc Surg* 2005;18:196-201.
 29. Abbas MA, Fowl RJ, Stone WM, et al. Hepatic artery aneurysm: factors that predict complications. *J Vasc Surg* 2003;38:41-5.
 30. 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.
 31. Bageacu S, Cuilleron M, Kaczmarek D, Porcheron J. True aneurysms of the pancreaticoduodenal artery: successful non-operative management. *Surgery* 2006;139:608-16.
 32. Cho BS, Lee MS, Lee MK, et al. Treatment guidelines for isolated dissection of the superior mesenteric artery based on follow-up CT findings. *Eur J Vasc Endovasc Surg* 2011;41:780-5.
 33. Lee VS, Morgan JN, Tan AG. Celiac artery compression by median arcuate ligament: a pit fall of end expiratory MR imaging. *Radiology* 2003;228:437-42.
 34. Bass JE, Redwine MD, Kramer LA, Huynh PT, Harris JH Jr. Spectrum of congenital anomalies of the inferior vena cava: cross-sectional imaging findings. *Radiographics* 2000;20:639-52.
 35. Eisenstat RS, Whitford AC, Lane MJ, Katz DS. The "flat cava" sign revisited: what is its significance in patients without trauma? *AJR Am J Roentgenol* 2002;178:21-5.
 36. Yeh BM, Kurzman P, Foster E, Qayyum A, Joe B, Coakley F. Clinical relevance of retrograde inferior vena cava or hepatic vein opacification during contrast-enhanced CT. *AJR Am J Roentgenol* 2004;183:1227-32.
 37. Kaufman LB, Yeh BM, Breiman RS, Joe BN, Qayyum A, Coakley FV. Inferior vena cava filling defects on CT and MRI. *AJR Am J Roentgenol* 2005;185:717-26.
 38. Khosa F, Otero HJ, Prevedello LM, Rybicki FJ, Di Salvo DN. Imaging presentation of venous thrombosis in patients with cancer. *AJR Am J Roentgenol* 2010;194:1099-108.
 39. Kibbe MR, Ujiki M, Goodwin AL, Eskandari M, Yao J, Matsumura J. Iliac vein compression in an asymptomatic patient population. *J Vasc Surg* 2004;39:937-43.
 40. Suwanabol PA, Tefera G, Schwarze ML. Syndromes associated with the deep veins: phlegmasia cerulea dolens, May-Thurner syndrome, and nutcracker syndrome. *Perspect Vasc Surg Endovasc Ther* 2010;22:223-30.
 41. Khosa F, Magoon P, Bedi H, Khan AN, Otero H, Yucel K. Primary and metastatic vascular neoplasms: imaging findings. *AJR Am J Roentgenol* 2012;198:700-4.
 42. Ageno W, Squizzato A, Togna A, et al. Incidental diagnosis of deep vein thrombosis in consecutive patients undergoing a CT Scan of the abdomen: a retrospective cohort study. *J Thromb Haemost*. In press.
 43. Karaosmanoglu D, Karcaaltincaba M, Karcaaltincaba D, Akata D, Ozmen M. MDCT of the ovarian vein: normal anatomy and pathology. *AJR Am J Roentgenol* 2009;192:295-9.
 44. Ahlberg NE, Bartley O, Chidekel N. Right and left gonadal veins. An anatomical and statistical study. *Acta Radiol Diagn (Stockh)* 1966;4:593-601.
 45. Rozenblit AM, Ricci ZJ, Tuvia J, Amis ES Jr. Incompetent and dilated ovarian veins: a common CT finding in asymptomatic parous women. *AJR Am J Roentgenol* 2001;176:119-22.
 46. Hiromura T, Nishioka T, Nishioka S, Ikeda H, Tomita K. Reflux in the left ovarian vein: analysis of MDCT findings in asymptomatic women. *AJR Am J Roentgenol* 2004;183:1411-5.
 47. Kim CY, Miller MJ Jr, Merkle EM. Time-resolved MR angiography as a useful sequence for assessment of ovarian vein reflux. *AJR Am J Roentgenol* 2009;193:W458-63.
 48. Coakley FV, Varghese SL, Hricak H. CT and MRI of pelvic varices in women. *J Comput Assist Tomogr* 1999;23:429-34.
 49. Yassa NA, Ryst E. Ovarian vein thrombosis: a common incidental finding in patients who have undergone total abdominal hysterectomy and bilateral salpingo-oophorectomy with retroperitoneal lymph node dissection. *AJR Am J Roentgenol* 1999;172:45-7.



Credits awarded for this enduring activity are designated "SA-CME" by the American Board of Radiology (ABR) and qualify toward fulfilling requirements for Maintenance of Certification (MOC) Part II: Lifelong Learning and Self-assessment.