The APDVS Medical Student Curriculum

The APDVS and contributors

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Preface

APDVS Association of Program Directors in Vascular Surgery

This content was developed as part of the Association of Program Directors in Vascular Surgery's (APDVS) medical student curriculum. Each chapter covers a key domain of vascular surgery pathology and treatment and is associated with an Audible Bleeding episode which you can access from the link embedded in the text.

This eBook would not have been possible without the Association of Program Directors in Vascular Surgery (APDVS), the guidance and resources of [Dr. Sharif Ellozy] (https://twitter.com/SharifEllozy) and Dr. Adam Johnson, the Audible Bleeding Vascular Surgery Exam Prep project, and the Audible Bleeding Team. This project is the direct result of Dr. Chelsea Dorsey et al.'s medical education research (see below) and the work of the Society of Vascular Surgery's Resident Student Outreach Committee (SVS RSOC).

Below, please see the two papers to understand the medical education need addressed by this eBook.

- Vascular Surgery Curriculum for Medical Students: A National Targeted Needs Assessment
- The Value of a Vascular Surgery Curriculum for Clinical Medical Students: Results of a National Survey of Nonvascular Educators

Editors: The Association of Program Directors in Vascular Surgery and Ezra Schwartz.

Version 01.02

Disclosures

This is an publication of the APDVS and therefore we share the same conflicts of interest. That noted, this eBook is the product of voluntarily donated time. This work has no financial backing and is not-for-profit. We are not receiving any funding or benefits from outside resources referenced. We include resources we believe will benefit you, the learner.

Usage

This eBook is intended to be a medical student level, easily accessible review for vascular surgery rotations and general medical education. This eBook is paired with slide decks, pre- and post-reading questions and a teaching case produced by the APDVS in addition to podcast content produced by Audible Bleeding, a publication of the Society of Vascular Surgery.

We are also excited to include an open source annotations software called hypothes.is. By creating a Hypothes.is account, you will be able to creates notes in the eBook as you read. You can choose to make your notes private or public. As this eBook is a community-led initiative, please consider making your notes public if you feel they would benefit your peers or the editors of the book. Given the public nature of annotations, if you choose to post public notes, these annotations are regarded as contributions to the eBook and we expect readers and listeners to follow a Contributor Code of Conduct. **We expect all participants, ourselves included, to maintain a safe space and behave professionally.**

Thanks to the flexibility and accessibility afforded as an eBook, we can (and will) update the book with extreme ease. Updates may include but are not limited to: slide decks, new pre- and post-reading questions, and relevant Audible Bleeding podcast episodes. **Please see the announcements page to stay up to date on developments.**

Additional Resources

We hope you use this eBook as a review for your rotations, undergraduate medical licencing examinations, and entry to independent practice. This resource is a summary and is by no means comprehensive. That noted, in every chapter we have included links to additional resources if you wish to gain a more comprehensive understanding of the topics covered. As our mission is to make vascular surgery education accessible, we prioritize open access publications and free materials. A few highly recommended additional resources include:

- Visit the ChooseVascular website toearn why we chose vascular surgery and determine if you wish to choose vascular too! This site is complete with a student resources page, and information to pair you with a vascular surgery mentor or a vascular surgery interest group, this website is a wonderful introduction the work and community of vascular surgery.
- The Gore Medical Vascular and Endovascular Surgery Combat Manual is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.
- TeachMe Surgery is a student friendly online resource complete with short quizzes to help cement learning and recommended readings from the literature. Clear and concise, this is a great just-in-time learning resource.
- The Audible Bleeding Medical Student Archive contains podcast episodes tailored for medical students.We suggest paying special attention to the *Holding Pressure* series. Whereas this eBook is organized by disease, *Holding Pressure* episodes are organized by surgical procedure. These episodes contain a wealth of high-yield information and we will include links to *Holding Pressure* episodes throughout the eBook when applicable.
  - You can listen to Audible Bleeding episodes on Apple Podcasts, Spotify, Google Podcasts, Stitcher, or Soundcloud
- The Audible Bleeding Exam Prep curriculum and the associated podcast series. While this resource is intended for postgraduate trainees and fellows and therefore beyond the scope of this curriculum, it is a well authored, no-nonsense resource for those who wish to deepen their knowledge base. The accompanying podcast series also allows you to learn or study on the go!

  - You can listen to Audible Bleeding episodes on Apple Podcasts, Spotify, Google Podcasts, Stitcher, or Soundcloud

- VascularTraining.org is an online repository of vascular surgery trainee resources compiled during the stages of the COVID-19 pandemic when providing traditional education was challenging and learning content was increasingly available online. Find videos, landmark and other informative papers, and device information here. Like the Audible Bleeding Exam Prep curriculum, the primary audience for this content is postgraduate trainees but is very accessible for undergraduate students who wish to deepen their knowledge base.

Comments, Questions or Contributions

Please visit our github page or send Audible Bleeding an email.

This book is built on Quarto.

Introduction

Announcements & Case of the Week

Please return to this page for updates on website, podcast, and acitivity developments.

eBook Chapters

The next chapter in development is peripheral artery disease including acute limb ischemia (ALI), chronic limb threatening ischemia (CLTI) and claudication.

Podcast Episodes

The next podcast in development is an introduction to the APDVS curriculum.

Case of the Week

To harness the learning principles of spaced practice, retrieval practice, interleaving and concrete examples, we invite students and residents to write a case of the week and submit it to APDVS for review.

Please try to build the case using material covered in the eBook. We ask you to write for the medical student level. Please include discussions to questions including why the correct is answer is correct and why the incorrect answers are incorrect.

Once reviewed, we will post it here for the community! We hope you will take part in this mini-publication exercise!

Please submit your case to ezraschwartz@hms.harvard.edu

Authors and Contributors

This eBook is the result of a partnership between the APDVS and the Audible Bleeding Podcast, each of which is comprised of hard working individuals dedicated to medical education and the promotion of vascular surgery. The authors and contributors of this eBook have freely donated their time and experience to create this resource. Please follow them on twitter or other social media platforms to return this debt of gratitude and to, as Audible Bleeding says, "keep a finger on the pulse."

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Approach to Vascular Patient

Patient History and Physical Examination of the Arterial System

Loay S Kabbani, MD, MHSA, FACS and Cassius Iyad Ochoa Chaar, MD, MS, RPVI

> Note

Introduction

If you pay close attention to the patient, *"the patient will tell you what is wrong."* Specialized testing should always be guided by the initial clinical impressions.

Though the electronic medical record helps eliminate repetitive questions and tests, it tends to divert attention from the importance of talking to and examining the patient. Interacting with the patient and family during the history and physical is extremely important to **build rapport with the patients and their family.**

For vascular patients passing the **"eyeball test"** is important before discussing complex procedures. This includes knowing the level of independent living.

Patient History of the Arterial System

As a vascular specialist, a focused arterial history should include the arteries of the neck, torso, and extremities. Many diseases are systemic. For example, atherosclerotic disease affects the carotids, heart, upper extremities, abdominal blood vessels, and lower extremities. Patients with an abdominal aortic aneurysm may have synchronous peripheral aneurysms.

Head and Neck History:

- The history should focus on the carotid artery disease—see **?@sec-carotidarterystenosis** for more detail—specifically, any history of stroke, transient ischemic attacks (TIA), or amaurosis fugax.
- Amaurosis fugax is described as a curtain or shade causing transient monocular blindness. This is usually caused by emboli from the carotid disease on the ipsilateral side but may be caused by other embolic sources, migraine, and giant cell arteritis.
- Syncopal episodes are usually not vascular in nature. However, in patients with subclavian stenosis may be a manifestation of subclavian steal syndrome.
- History of carotid interventions and studies should be elucidated and obtained.

Upper Extremity History:

- The evaluation depends on the timing (acute vs. chronic) and then the degree of ischemia (claudication vs. chronic limb-threatening ischemia) .
- **Acute limb ischemia (ALI)** is a sudden occlusion of the blood supply with no time for collateral vessels to develop. It is characterized by the **6 Ps.** *Pulselessness, pain, pallor, paresthesia, poikilothermia (cold), and paralysis.* The most common etiology of acute limb ischemia is embolic. However, uncommon causes may be thrombosis of a subclavian–axillary aneurysm.
- Intermittent hand coldness associated with pain, numbness may reflect Raynaud's syndrome (associated with cold exposure) or small vessel vasospasm like frostbite or scleroderma.
- Raynaud phenomena present as episodes of vasospasm in response to cold or stress. The patient describes the affected areas turning white, blue, and red during an attack. It is associated with feeling cold and numb. As the circulation improves and the affected areas turn red, there is throbbing, tingling, and swelling.
- Episodes of ischemia to the digits may be persistent and severe and associated with underlying obliterative microangiopathy and manifest with pain, tissue loss (ulceration or gangrene).
- Chronic ischemia of the upper extremity is uncommon can manifest with arm claudication (exertional fatigue). The etiology is most commonly proximal obstruction due to atherosclerotic disease in the subclavian artery. More uncommon causes are Takayasu arteritis and thoracic outlet syndrome.

- Exertion of the arm causing posterior cerebral circulation symptoms (diplopia, dysarthria, dizziness, drop attacks, vertigo, syncope, and ataxia) may reflect a subclavian steal syndrome caused by subclavian artery stenosis proximal to the vertebral artery.

Abdomen History:

- Aneurysm disease in the abdomen is usually silent.
- With weight loss and sitophobia (food fear), postprandial pain may reflect mesenteric artery stenosis.
- Severe hypertension, especially in young adults, may reflect renal artery stenosis.

- Aortoiliac occlusive disease may present with Leriche's syndrome. **Leriche's syndrome** is defined as bilateral hip and buttock claudication, absent femoral pulses, and impotence.

- Acute abdominal pain has multiple nonvascular etiologies. A ruptured aneurysm presents severe pain radiating to the back with a pulsatile abdominal mass. Acute mesenteric ischemia manifests as pain out of proportion to the physical exam.

Lower Extremities History:

- The evaluation depends on the timing (acute vs. chronic) and then the degree of ischemia (claudication vs. chronic limb-threatening ischemia).
- **Acute limb ischemia (ALI)** is a sudden occlusion of the blood supply with no time for collateral vessels to develop. It is characterized by the **6 Ps.** *Pulselessness, pain, pallor, paresthesia, poikilothermia (cold), and paralysis.*
- Chronic limb ischemia is frequently asymptomatic and diagnosed on physical exam or vascular lab testing. In asymptomatic disease related to atherosclerotic disease, medical therapy is initiated to prevent disease progression.
- **Claudication** is defined by cramping pain in the leg induced by exercise and relieved by rest. It occurs at a fixed and reproducible distance and resolves with rest. The pain is described as discomfort, cramp, numbness, or tiredness in the legs. Claudication most commonly occurs in the calf muscles, but it can also affect the feet, thighs, hips, buttocks. It is crucial to determine how the patient's lifestyle is affected when taking a history. With proper medical therapy, including medication and exercise, claudication improves$>$ 50% of the time. Fewer than 5% progress to amputation. Amputation is more frequent in patients who continue to smoke, have uncontrolled diabetes, and have renal failure. Differential diagnosis of claudication is in **Table 1** below. Table 1 is also helpful in differentiating between other types of leg pain, pseudo claudication, and true claudication.

- **Chronic limb-threatening ischemia (CLTI).** These patients have multiple levels of arterial disease and are at risk of amputation. CLTI manifests as rest pain or tissue loss. Rest pain is characterized as pain in the dorsum of the foot and toes. It is increased with leg elevation and may wake the patient up at night and is relieved by dangling the limb, which increases perfusion by enlisting gravity to help perfuse the leg. Tissue loss can be in the form of an ulcer or gangrene.

Physical Exam of the Arterial System

The arterial system is diffuse. Therefore, the evaluation of vascular patients should include the entire arterial system.

The exam starts with the nurse checking the vital signs and bilateral upper extremity blood pressures. A difference of > 10 mmHg between the upper extremities indicates significant hemodynamic stenosis. Most of these stenoses are asymptomatic because of the extensive collateral network.

Key Notes

Arterial Bruit: Normal arterial flow is silent. However, turbulence can be heard (bruit) or palpated (thrill). Although murmur and bruit are synonymous, audible turbulence originating from the heart is called a murmur. Outside of the heart, audible turbulence is referred to as a bruit. The presence of a bruit does not necessarily indicate significant arterial stenosis.
Pulses are described as:

- 0 or absent
- 1+ or diminished
- 2+ or normal
- 3+ or prominent or aneurysmal.

In patients with weak pulses, the examiner must take care not confuse the patient's pulse with involuntary muscle twitches or their own pulse.
When a pulse is not palpated, a (portable) Doppler ultrasound probe is used to assess the blood flow. The Doppler signal can be triphasic, or biphasic, or monophasic.
Doppler Signals:

- Triphasic and biphasic doppler signals indicate good blood flow.
- Monophasic signal correlates with moderate to severe decrease in arterial blood flow.

In severe ischemia, a soft continuous venous signal may be all that is heard.
Listen to examples here.
On Exam:

- Temperature changes may help demarcate the level of disease.

- Sensory loss may be present in acute ischemia and chronic neuropathy.

Head and Neck Exam:

Inspection:

- Pulsatile masses in the neck are usually tortuous carotid arteries mistaken for carotid aneurysms. Carotid aneurysms are usually near the carotid bifurcation, while tortuous carotid arteries are usually at the base of the neck. Carotid body tumors are also at the carotid bifurcation. Both carotid body tumors and aneurysms are not visible until they are large.
- If the patient complains of amaurosis fugax, fundoscopy may reveal cholesterol plaques called Hollenhorst plaques. These are thought to originate from the carotid plaque.

Palpation:

- The carotid pulse is palpated on the medial border of the sternocleidomastoid muscle. Carotid palpation is generally not performed routinely as it may cause a syncopal episode in elderly patients with sensitive carotid bulbs.
- A robust temporal pulse anterior to the ear is a sign of a patent common and external carotid artery sign.
- A large supraclavicular pulse may indicate an enlarged subclavian artery. Otherwise, the subclavian artery is usually not palpable.

Auscultation:

- Include auscultation of the carotids for any bruits.
- Using the stethoscope bell, you can hear the S1, and S2 heart sounds in the carotid artery in the mid-neck. A bruit heard in the neck is not normal. This could be transmitted from the heart or could be from a kink or narrowing in the carotid artery. The carotid bruit is loudest in the mid neck over the carotid bifurcation. The heart bruit (called murmur) is the loudest in the upper chest.
- The intensity of the bruit and pitch do not correlate with the severity of stenosis. A tight stenosis may have low flow and thus a faint bruit.
- When a carotid bruit is heard, only 25% will have significant stenosis (75% or greater), and 50% will not have any stenosis.

Upper Extremity Exam:

Inspection

- Pink fingertips with capillary refill times < 2 seconds are a reliable sign of adequate perfusion. Ischemia manifests with pale extremity with poor to no capillary refill. Chronic ischemia manifests with muscle atrophy.
- Raynaud phenomenon is characterized by sharply demarcated triphasic color change after exposure to cold or emotional stress. First, the capillaries contract after the stressor, causing a characteristic white appearance. Then, as the capillaries open a little, the blood re-perfuses sluggishly and is deoxygenated; this causes a hypoxic blue color. Finally, when the capillaries recover and hyper-dilate, the affected limb becomes red and hyperemic. Raynaud's phenomena may occur idiopathically (Raynaud's Disease) or secondarily in autoimmune disease and is often provoked by exposure to cold or emotional distress.

Palpation

- Palpate the axillary artery in the upper arm in the groove between the biceps and triceps muscle.
- Palpate the brachial artery in the antecubital fossa just medial to the biceps tendon
- Palpate the radial artery on the wrist's flexor surface just medial to the radial styloid.
- Palpate the ulnar artery on the wrist's flexor surface just medial to the distal ulna; it lies deeper than the radial artery and may not be palpable.
- Absent pulses should initiate a search for a cause such as proximal atherosclerotic stenosis in older adults or autoimmune disease such as Takayasu's in young females.
- Aneurysm of the Subclavian artery and axillary artery (assessed above and below the clavicle) are difficult to palpate if small. Brachial artery aneurysms are usually pseudoaneurysms from trauma or arterial access. Ulnare artery aneurysm occurs from repetitive trauma in proximity to the hamate bone and manifests as hypothenar hammer syndrome.

Auscultation

- Listen for a bruit in the supraclavicular fossa over the subclavian artery.
- When pulses are not palpable, a Doppler is used to assess blood flow in the arteries.
- A blood pressure difference > 10 mmHg reflects hemodynamically significant stenosis in the innominate, subclavian, or axillary arteries. Therefore, the higher blood pressure is reflective of the actual blood pressure.

Chest and Abdominal Exam:

Inspection

- The aorta usually is not visible on the exam. However, a large aneurysm may be seen pulsating between the xiphoid and umbilicus, especially in thin patients.

Palpation

- The aorta bifurcates at the level of the umbilicus. To palpate the aorta, press our fingers on both sides of the midline between the umbilicus and the xiphoid. The patient should bend, his knees flex his hips, and relax his abdominal muscle helps. The goal is not only to feel the aortic pulse but also to estimate the size of the aorta. In most normal thin people, the aorta is palpable and is the size of the patient's thumb. A tender pulsatile aortic mass may represent a symptomatic aortic aneurysm or inflammatory aneurysm.
- The sensitivity of palpation to detect an abdominal aortic aneurysm is low (29%) for small (3.0- 4cm) aneurysms. Moreover, even larger aneurysms may not be detected on physical exam (76%) for aneurysms > 5 cm. false positives can be found in elderly patients who have tortuous anterior placed aorta. It is important to mention that palpation of an abdominal aortic aneurysm is safe and has never been reported to precipitate aortic rupture. When an aortic aneurysm is identified, A complete peripheral arterial examination should be performed looking for evidence of distal embolization, ischemia, or associated peripheral artery aneurysms (femoral, popliteal).
- The iliac arteries lie deep in the pelvis and are usually not palpable, even if they were an aneurysm.

Auscultation

- Cardiac auscultation is performed for rate and rhythm. And attention to any arrhythmias, gallops, and murmurs.
- Bruits in the abdomen is associated with arterial stenosis. The origin of the bruit could be renal, mesenteric, or aortic iliac. In young females, the bruit may reflect fibromuscular dysplasia.

Lower Extremity Exam:

Inspection

- Pallor, cyanosis, poor capillary refill are signs of chronic limb ischemia. Muscle atrophy, hair loss, and thick toenails may also be present.

- **Dependent rubor** and pallor with elevation indicate advanced peripheral occlusive disease. Dependent rubor is hyperemic erythematous discoloration of the limb in a dependent position (sitting or standing). Dependent rubor is due to the maximal dilation of the capillaries and the effect of gravity. However, the limb becomes pale once the foot is elevated (the patient lies down). It is usually associated with rest pain and edema. It is frequently misdiagnosed as cellulitis.
- Ulcers need to be identified as neuropathic ulcers or ischemic ulcers. Neuropathic ulcers are at pressure points over the plantar aspect of the metatarsal head. Ischemic ulcers are more on the tip of the toes.

- *Livedo Reticularis*: Violaceous mottling of the skin with a reticular pattern of the skin of the arms and legs. The term" livedo racemosa" is used for cutaneous findings in inflammatory or thrombotic vascular disease patients.
- *Acrocyanosis*: is defined as bluish discoloration of the extremities due to high deoxygenated blood in the capillaries. It is a persistent disorder without episodic triphasic color response.

- Microembolic disease can manifest as blue toe syndrome/trash foot.
- Dry skin is present in chronic limb ischemia because the sebaceous glands are not working well.
- Edema is called "pitting" when the indentation persists after applying pressure to a small area. Pitting edema is associated with systemic diseases like heart failure, chronic kidney disease, hypoproteinemia, or local disease of the veins or lymphatic. Non-pitting edema is observed when the indentation does not persist. It is associated with Myxedema, lipedema, and advanced lymphedema.

- *Claudication*: claudicates may have no significant finding on inspection. They may have muscle atrophy or hair loss:

Auscultation

- Auscultation over the femoral region for any bruits. Auscultation may also find continuous bruits characterized by an arterio-venous fistula.

Palpation

- Femoral pulse: palpated under the inguinal ligament, two-finger breaths from the pubic tubercle.

- Popliteal pulse: with the patient's knee flexed, both hands are wrapped around the knee tendons, and the tips of the fingertips are pressed into the popliteal space. The

pulse is lightly lateral. A normal popliteal artery may not be palpable.

- Dorsalis Pedis: palpated in the dorsum of the foot between the first and second extensor tendons.
- Postier tibial: pulse is found behind the medial malleolus. It is easier to palpable with the foot passively dorsiflexed.

- Peroneal artery: not palpable.

- It is common not to palpate either the Dorsalis Pedis or posterior tibial pulse, but not in the same foot.
- When a pulse is not palpated, a Doppler is used to assess the blood flow. The Doppler signal can be triphasic, or biphasic, or monophasic.

- Triphasic and biphasic doppler signals indicate good blood flow.

- Monophasic signal correlates with moderate to severe decrease in arterial blood flow.

- In severe ischemia, a soft continuous venous signal may be all that is heard.

- Temperature changes may help demarcate the level of disease.

- Sensory loss may be present in acute ischemia and chronic neuropathy.

Carotid Disorders

Carotid Artery Stenosis

Angela Kokkosis, MD, FACS and Michael Malinowski, MD, MEHP, FACS

Note

By the end of this chapter, students will:

- Review the foundational knowledge to appraise asymptomatic and symptomatic carotid artery stenosis.
- Identify and describe risk factors, symptoms, and signs of carotid artery disease.
- Review and describe indications and contraindications for medical and surgical management of carotid artery stenosis.
- Propose next best steps in patient work-up and treatment of carotid artery disease.
- Describe the evidence-based outcomes of surgical management of carotid artery disease.

Key Facts

1. Stroke is the leading cause of disability nationally and 3rd leading cause of death with a breakdown of 80% occlusive (ischemic) and 20% hemorrhagic.
2. Symptomatic carotid stenosis presents with only three symptoms including: stroke, transient ischemic attack (TIA) and amaurosis fugax (transient retinal ischemia).
3. Risk factors include: age, smoking, CAD, diabetes, hypertension, hyperlipidemia and genetic/family history.
4. Seminal studies include ACAS and NASCET. They define an 11% five-year risk of stroke for asymptomatic carotid artery stenosis (>60%) and 24% two-year risk of stroke for symptomatic disease (>70%), respectively.
5. The CREST landmark study associates carotid artery stenting with higher perioperative risk of stroke and carotid endarterectomy (CEA) with higher risk of myocardial infarction (MI) in symptomatic patients.
6. Carotid artery stenting (CAS) is indicated for recurrent stenosis after CEA, neck immobility, high carotid bifurcation, contralateral occlusion, high risk open surgical candidate due to cardiopulmonary comorbidity and neck radiation.

7. Transcarotid artery revascularization (TCAR) is available for direct common carotid delivery of stent while avoiding aortic arch manipulation and providing embolic protection through flow reversal.
8. There is a marginal benefit for intervention for asymptomatic carotid artery stenosis patients with significant cardiopulmonary disease due to risks of associated perioperative events.

Carotid Disease Slide Deck

Please find the slide deck corresponding to this eBook chapter **here**.
Please find a video recording of Dr. Kokkosis and Dr. Malinowski's lecture using the above slide deck **here**.

How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

Pre/Post Questions

Case Based Questions

1. A 75-year-old male smoke presents with recent visual changes to his right eye that occurred yesterday. The patient reports a shading of his visual field that resulted in momentary monocular blindness followed by return to normal vision. He has a carotid duplex showing >50% diameter reduction to his right internal carotid artery and >80% stenosis to his left internal carotid artery. *What treatment should be offered to his patient?*

A. Emergent DC cardioversion to treat any underlying arrythmia.

B. Left carotid endarterectomy with shunt placement.

C. Left carotid TCAR.

D. Right carotid endarterectomy.

E. Placement on Apixaban and measurement of PF4 with medical management.

Answer

D. Right carotid endarterectomy
Discussion: The patient has evidence of amaurosis fugax with temporary monocular blindness as a sign of symptomatic right internal carotid artery disease. His left carotid artery is in a high-grade range and therefore is lower risk of stroke than the contralateral symptomatic lesion. There should be no immediate surgical treatment of the asymptomatic left carotid lesion. There is no evidence that the patient has an underlying arrythmia that needs cardioversion and medical management with Apixaban and PF4 levels are unrelated to this patient's current pathology.

2. A 50-year-old female patient with >80% right internal carotid artery stenosis presents to clinic for her first postoperative visit after carotid endarterectomy (CEA). She has no interval neurologic events since her discharge, has a soft neck with a clean incision. During your neurologic exam you notice an unintentional, subtle tongue deviation to the side of surgery. *What is the most likely facial nerve involved in this finding?*

A. Vagus Nerve.

B. Hypoglossal Nerve.

C. Glossopharyngeal Nerve.

D. Long Thoracic Nerve.

E. Hering's Nerve.

Answer

B. Hypoglossal Nerve
Discussion: This patient has evidence of hypoglossal nerve neuropraxia which can occur as the result of injury or traction on the nerve during distal dissection of the internal carotid artery. Although both glossopharyngeal nerve injury and vagal injuries can also occur during CEA, the former causes oropharyngeal dysfunction with swallowing and the latter causes vocal cord paralysis leading to a hoarse voice. The long thoracic nerve is more related to thoracic outlet surgery and innervation of the serratus anterior. The Nerve of Hering is related to carotid sinus innervation and is unrelated to this clinical finding.

3. During the initial evaluation of a patient with high grade symptomatic carotid disease, you notice that the patient has internal carotid plaque on that side this is above the angle of the mandible at the 1st cervical vertebral body (C1). This appears to be too high to access through open surgery. The patient has no know history of coronary artery

disease, has a preserved ejection fraction and good functional status. He has a low-density lipoprotein level (LDL) of 200 mg/dL. *What is the best treatment option for this individual?*

A. Transfemoral carotid artery angioplasty with placement on ASA only.

B. Carotid artery enterectomy with shunt placement.

C. Daily ASA therapy without any type of statin or antihypertensive therapy.

D. Trancarotid artery revascularization (TCAR) surgery with dual antiplatelet therapy.

E. No treatment is indicated.

Answer

D. Transcarotid artery revascularization surgery with dual antiplatelet therapy.
Discussion: This patient has symptomatic carotid stenosis with an overall optimal coronary health. Because his lesion is high at C1 and not surgically accessible, they would not qualify for a traditional carotid endarterectomy. Transfemoral carotid artery angioplasty is not indicated without stent placement, and daily ASA therapy without statin treatment for an LDL of 200mg/dL does not qualify as optimal medical management. Surgical treatment is indicated in this symptomatic patient since he has no significant cardiopulmonary disease and TCAR is the only option listed that could accomplish this outcome successfully.

4. A 60-year-old otherwise healthy woman, with no underlying comorbidities has a right carotid artery bruit on physical examination. She is concerned that she is at risk for stroke. She denies any episodes of vision changes, upper or lower extremity deficits, or speech impairments. She currently takes 81mg of aspirin daily, along with a multivitamin. *What is the next step in management for this patient?*

A. No further treatment necessary.

B. Carotid duplex.

C. CT angiogram head and neck.

D. Neurology evaluation.

E. Addition of statin therapy.

Answer

A. No further treatment necessary.
Discussion: This patient has an incidental finding of a carotid bruit on physical exam,

however she has no risk factors for carotid disease (such as smoking, hyperlipidemia, smoking, family history, diabetes or hypertension). Additionally, she is neurologically asymptomatic. Therefore, carotid duplex, CTA, neurology evaluation, and the addition of statin therapy are not indicated. The prevalence of >75% carotid stenosis for those with a carotid bruit has been found to be very low at 1.2%.

5. A 65-year-old man who is right-handed is undergoing evaluation for a coronary artery bypass graft surgery (CABG). As part of his workup, a carotid duplex is performed which demonstrates a chronic right carotid occlusion and a >80% stenosis of his left carotid artery. He has no history of ocular or cerebrovascular events. He has hypercholesterolemia and well-controlled hypertension. *What is the next best step for this patient?*

A. Proceed with the CABG as planned and continue medical therapy with aspirin and statin.

B. Left carotid endarterectomy before the CABG.

C. Place the patient on dual antiplatelet therapy, in addition to statin therapy.

D. Left TCAR (transcarotid stent) after the CABG.

E. Place the patient on anticoagulation.

Answer

B. Left carotid endarterectomy before CABG.
Discussion: It is standard of care to assess the carotid arteries prior to performing a CABG, with the goal of minimizing the risk of perioperative stroke. This patient has asymptomatic bilateral severe carotid disease (right occlusion and left >80% stenosis). Therefore, to reduce the risk of perioperative stroke, current guidelines recommend carotid revascularization prior or concomitant with the CABG. The patient does carry one high risk criterion for TCAR (coronary disease requiring revascularization), however this should not be performed after the CABG. Dual antiplatelet therapy is indicated in the event this patient undergoes carotid stenting, however medical management should not be the only management of his carotid disease. Lastly, anticoagulation has no role in atherosclerotic disease, such as carotid disease.

6. A 55-year-old woman has a past medical history of coronary artery disease status post coronary stenting in 2019, hypertension, hyperlipidemia, and previous smoking history of 60 pack-years. Her cardiologist sends her for a carotid duplex which demonstrates a 50-69% carotid stenosis on the left side, and mild atherosclerosis on the right side. She denies any prior signs or symptoms of stroke or transient ischemic attack. *How should this patient's carotid disease be managed?*

A. Left carotid endarterectomy, along with aspirin/statin therapy.

B. Left transfemoral carotid stent, along with dual antiplatelet/statin therapy.

C. Aspirin and statin therapy **only**, and routine surveillance carotid duplexes.

D. Aspirin, statin therapy, blood pressure management, and routine surveillance carotid duplexes.

E. No further management is indicated.

Answer

D. Aspirin, statin therapy, blood pressure management, and routine surveillance carotid duplexes.
Discussion: This patient has asymptomatic 50-69% left carotid stenosis with the associated risk factors of vascular disease which include hypertension, hyperlipidemia, and smoking history. Medical therapy needs to be employed to reduce these factors, thus a single antiplatelet, statin, and blood pressure medication are indicated. Fortunately, the patient is not a current smoker, however if she was, then smoking cessation counseling would be added to her treatment plan. Carotid revascularization is indicated only for asymptomatic carotid stenosis that is >70% based on current guidelines.

Operative Footage Questions

These questions are associated with the carotid endarterectomy (CEA) footage (**short version**) found at the bottom of the chapter.

1. What is the first muscle layer encountered in a CEA (i.e. the first muscle deep to skin)?

A. Sternocleidomastoid

B. Digastric

C. Platysma

D. Scalene

Answer

C. Platysma
Discussion: The platysma is the most superficial muscle in the neck. It covers most of the anterior and lateral aspect of the neck. It is the first muscle layer encountered in the neck during a CEA. It will be bisected and repaired upon neck closure. While the sternocleidomastoid is superficial, it is deep to the platysma. The sternocleidomastoid is an important landmark as it forms the anterolateral boundary of the carotid triangle.

You will dissect along its medial border and retract it laterally in order to access the carotid sheath.The digastric muscle is a small, "two-bellied" muscle located under the mandible. The posterior belly forms the superior border of the carotid triangle. It is often visualized in patients with high carotid bifurcations.The scalene muscles are deep to the sternocleidomastoid muscles and lateral to the cervical spine. They are not manipulated in a CEA.

2. What structure is **not** found in the carotid sheath?

A. Internal jugular vein

B. External jugular vein

C. Common carotid artery

D. Vasgus nerve

Answer

B. External jugular vein
Discussion: The external jugular vein is not in the carotid sheath. It runs superficial to and obliquely across the sternocleidomastoid before passing to the posterior border of the sternocleidomastoid as it descends deep into the neck. The carotid sheath contains the common carotid artery anteromedially, the internal jugular vein anterolaterally, and the vagus nerve posteriorly.

3. In what order do you **unclamp** the carotid vessels at the end of a carotid endarterectomy?

A. External carotid artery → common carotid artery → internal carotid artery

B. Common carotid artery → internal carotid artery → external carotid artery

C. Internal carotid artery → external carotid artery → common carotid artery

D. Internal carotid artery → common carotid artery → external carotid artery

Answer

D. External carotid artery → common carotid artery → internal carotid artery
Discussion: In conventional CEA, the internal carotid artery (ICA), the common carotid artery (CCA), and the external carotid artery (ECA) are clamped in that order such that any atherosclerotic debris loosened during vessel manipulation and/or clamping embolizes into the ECA where it could cause extracranial ischemia rather than the ICA where it could cause intracranial ischemia (i.e. ischemic stroke). A useful mnemonic to remember

the clamping order is **ICE** for **I**CA, **C**CA, and **E**CA. The order when unclamping is the opposite, **ECI**. The rational is similar. By unclamping the ECA first, remaining debris or debris loosened during removal of the clamps will flow up the ECA rather than the ICA. By unclamping the CCA second, and dislodged debris will flow down the open ECA rather than the ICA. The ICA is unclamped last. Options A and B are dangerous as they do not minimize the risk of embolus flowing into the ICA. Option C is simply incorrect.

Introduction

Hemispheric stroke related to carotid artery stenosis is a leading cause of both disability and death in the United States. Underlying etiologies for stroke include occlusive or hemorrhagic events with roughly 80% being related to occlusive pathology through embolus or in-situ thrombosis, the remaining 20% attributable to hemorrhage. Roughly 15% of stroke victims have a transient ischemic attack (TIA) that fully resolved prior to a later stroke event. Risk factors for carotid plaque formation are related to age, smoking, coronary artery disease, diabetes, hyperlipidemia, hypertension and family history of stroke. Due to carotid bulb anatomy, the most common area of plaque formation is within the proximal internal carotid artery. As plaque stenosis increases over time, the systolic velocity increases to maintain flow volumes which intensifies shear stress. This shear stress increases likelihood of plaque rupture, platelet aggregation and thromboembolization. There are multiple seminal studies that describe cohort comparisons of asymptomatic and symptomatic carotid artery stenosis with outcomes related to optimal medical management alone or as adjunct to surgical repair. Symptomatic carotid stenosis is described as carotid stenosis >50% with unilateral stroke, TIA or amaurosis fugax on the side of carotid disease. Amaurosis fugax is historically described as shade coming down across one eye on the side of stenosis to produce complete monocular visual loss related to transient retinal ischemia.

Etiology

Atherosclerosis is the most common cause for the development of carotid artery disease. This process is defined by deposition of lipid-laden plaque at the carotid bifurcation, and potentially across a larger territory of the common carotid, external carotid, and internal carotid arteries. This plaque may contain varying degrees of calcification and/or thrombus. The mechanisms by which atherosclerosis at the carotid bifurcation may lead to stroke or TIA are: occlusion

(cessation of blood flow to the internal carotid artery) or embolization (plaque debris break off and travel through the internal carotid artery to the brain). There are various risk factors which may contribute to the degree of atherosclerosis and its progression. These include history of cigarette smoking, hyperlipidemia, coronary artery disease, diabetes, hypertension, advanced age, and family history of carotid disease or stroke.

Diagnostics and Imaging

Three primary imaging modalities are used to evaluate carotid artery stenosis with the lowest cost option being color flow duplex ultrasound (DSA) that allows a physician to determine peak systolic and end diastolic velocities throughout the carotid bifurcation. Based on the internally validated vascular laboratory criteria of the institution, these velocities can be correlated to ranges of degree of stenosis, with high grade stenosis defined as >70-80%. Since the modality is based primarily on velocity range, it cannot give exact stenosis such as 66%. The modality can also provide adjunct information about the blood flow waveforms in each arterial segment, as well as whether that flow is laminar or turbulent with utilization of color flow imaging. Limitations of this imaging include technician skill, inability to obtain optimal angle of Doppler interrogation for velocity determination, shadowing from heavily calcified lesions, poor visualization due to patient habitus and tortuosity.

Axial imaging options include both computed tomography angiogram (CTA) and magnetic resonance angiogram (MRA). Both of these options require some form of intra-arterial contrast, either iodinated contrast or gadolinium, respectively. However, they offer a fuller perspective of relevant anatomy and a more precise determination of stenosis within the limitations of the modality, with MRA often overestimating the degree of stenosis due to intrinsic properties of MRA imaging acquisition. Both CTA and MRA, although superior to DSA in determination of exact degree of plaque stenosis and arterial anatomy, sacrifice the physiologic information offered through DSA that speak to flow patterns, flow direction and turbulence. Axial imaging of CTA and MRA can define patency but do not speak to the dynamic nature of blood flow or directionality of flow.

Definitive determination of flow and directionality can be augmented to a carotid artery stenosis workup by diagnostic angiography. This requires femoral artery access and includes contrast administration, as well as a small risk of periprocedural embolization. However, it offers additional physiologic evaluation that might not be present in DSA and that is inherently lacking in CTA and MRA studies.

Asymptomatic Carotid Atery Stenosis Screening

The 2021 SVS clinical practice guidelines outline the following recommendations. In asymptomatic patients who qualify for carotid artery stenosis screening, duplex ultrasound is the recommended choice over CTA, MRA, or other imaging modalities.

Routine screening is **not recommended** for clinically asymptomatic carotid artery stenosis for individuals **without significant risk factors** for carotid disease.

Screening is **recommended** for clinically **asymptomatic carotid artery stenosis in individuals** with significant risk factors** for carotid disease. High-risk groups include:

- Patients with lower extremity peripheral artery disease (PAD)
- Patients undergoing coronary artery bypass surgery (CABG)
- Patients aged >= 55 years with at least two traditional atherosclerotic risk factors (hyperlipidemia, hypertension, etc.)
- Patients aged >= 55 years and active cigarette smoking
- Patients with diabetes, hypertension, or coronary artery disease (CAD)
- Patients with clinically occult cerebral infarction noted on brain imaging studies.

The presence of a carotid bruit increases the likelihood of detecting significant stenosis. Asymptomatic patients with an abdominal aortic aneurysm (AAA) or previous radiotherapy to the neck *who do not meet the criteria of any of the high-risk groups* above do not require screening. It has been shown that the prevalence of carotid stenosis increases proportionally with the number of risk factors present.

Treatment

Carotid Artery Endarterectomy (CEA)

This procedure has been performed since the 1950s, either by plaque endarterectomy and patch angioplasty or primary arterial repair. To prevent arterial restenosis, patch angioplasty has become the standard of arterial closure after plaque removal. The procedure can involve cerebral monitoring including electroencephalography (EEG), transcranial Doppler (TCD) and stump pressure monitoring or be performed awake to directly monitor patient motor response. Endarterectomy and patch repair can be performed under a "clamp and sew" mentality or with an arterial shunt to maintain cerebral perfusion. Risks include cardiopulmonary risk of acute myocardial ischemia, <3% perioperative risk of neurologic event, neck hematoma or cranial nerve injury of roughly 5-10% affecting the vagus, marginal mandibular, recurrent laryngeal or hypoglossal nerves.

Carotid Artery Stenting (CAS)

Transfemoral Carotid Artery Stenting (TFCAS) with Embolic Protection

Transfemoral stenting requires some type of protection from embolization including a distal internal carotid artery retrievable filter or flow arrest procedure to prevent cerebral embolization during stent placement with or without angioplasty.

Transcarotid Artery Revascularization (TCAR)

Treatment of a carotid stenosis that avoids aortic arch manipulation involving direct common carotid artery exposure and sheath placement to allow for transcarotid stent delivery to the internal carotid artery. The common carotid artery sheath is connected to a femoral vein sheath so that the natural arterial pressure gradient reverses flow across the distal internal carotid artery driving blood and possible embolus into the arterial tubing circuit and across a filter before it reenters the venous circulation. As FDA approval for the device was delivered in 2016, the technology is less than 10 years old without robust long-term follow up data. This technique offers a lower perioperative stroke risk than transfemoral stenting, for multiple reasons including lack of transaortic arch manipulation and great vessel cannulation which can result in embolus prior to placement of an internal carotid artery embolic protection device.

Optimal Medical Management

Understandably, optimal medical management requires full risk evaluation of the individual patient in question including other comorbidities, drug allergies, compliance, etc. We have listed a few broad recommendations to follow that offer general guidance surrounding the dynamic target of optimal medical management for arterial disease.

Antiplatelet Therapy

- ASA offers a 22% risk reduction in major vascular events with no difference in protection based on dosage (81 versus 325 mg).
- Clopidogrel can be used as an adjunct or alternative to ASA, but the added benefit from dual antiplatelet combination in asymptomatic carotid artery stenosis is unproven.

Anticoagulants

- Only useful for prevention of cardioembolic strokes due to arrhythmia or prosthetic valve.

Hypertension Treatment

- Recommended blood pressure range of <130/80 with individual antihypertensive regimen based on other comorbidities and patient risk factors.

Diabetic Control

- In accordance with best practice for diabetes management, the patient's hemoglobin A1c should be <7.0

Smoking Cessation

- Treatments offered include nicotine replacement therapy (NRT), varenicline or bupropion as first line agents.

Hyperlipidemia Management

- Regimen goals of LDL <100mg/dl, or <70mg/dl depending on risk profile.

Outcomes and Surveillance

Asymptomatic carotid stenosis

- The historically touted Asymptomatic Carotid Atherosclerosis Study (ACAS) demonstrated that patients with >60% carotid stenosis who underwent CEA benefited significantly from stroke risk reduction at 5 years (5.1% for CEA vs. 11% for optimal medical therapy consisting of aspirin alone).
- More recent studies suggest that with the current optimal medical management, which consists of antiplatelet medication and statin therapy, 5-year stroke risk is highest in patients with >70% carotid stenosis, and therefore this patient population would benefit from carotid endarterectomy.
- Patients who are deemed high risk, either due to an anatomic (such as surgically inaccessible bifurcation or restenosis after previous CEA) or physiologic findings (congestive heart failure, severe coronary artery disease, or chronic obstructive pulmonary disease), may be considered for TCAR given the equivocal results of perioperative stroke or death at 1.3%, as compared to CEA.

- Asymptomatic patients **with significant risk factors** found to have moderate stenoses (50%-79%) should be followed every 6-months to detect disease progression. High risk patients with <50% stenosis can be followed-up annually.

Symptomatic carotid stenosis

- Patients who have >50% carotid stenosis and have developed symptoms of TIA or stroke were found to benefit from CEA in the pivotal North American Symptomatic Carotid Endarterectomy Trial (NASCET) because of the significant 2-year stroke risk reduction as compared to optimal medical management (15.7% vs. 22.2%.). An even greater stroke risk reduction was seen in patients with >70% carotid stenosis (9% CEA vs. 26% medical management).
- Current management of patients with symptomatic >50% carotid stenosis who are low/standard risk is carotid endarterectomy over transfemoral carotid stenting (TFCAS), as there are no studies to date which have shown benefit of TFCAS.
- Patients who are deemed high risk, as defined above, may be considered for TCAR over TFCAS due to the significantly lower incidence of in-hospital stroke and death (1.6% vs. 3.1%).
- Post-operative surveillance (by duplex ultrasound) after open (CEA) or endovascular (TF-CAS) repair of the carotid artery is strongly recommended by the SVS to monitor for signs of restenosis in the repaired artery or atherosclerotic disease progression in the unoperated, contralateral artery. Duplex ultrasound testing is recommended within 30 days of the procedure, then every 6 months for 2 years, then annually.
- Restenosis <50% warrants the regular surveillance protocol; 50-99% warrants closer follow-up, confirmation with a CTA, and possible angiographic evaluation; 100% restenosis warrants surveillance and medical treatment of the contralateral carotid artery.
- It should be noted, there is some debate as to the economic and medical value of continuing post-operative duplex ultrasound surveillance after successful CEA with patch closure when the immediate post-operative duplex was normal or showed minimal disease.

Teaching Case

Scenario

An 81 year old male with a significant smoking history and prior three vessel CABG five years ago, presents with monocular right eye blindness that occurred two days ago. He has no prior ophthalmologic conditions and states that he describes the process of a veil coming down over

his right eye with resolution about a minute later with complete return of normal vision at that point. He denies any other symptoms during the event or since, such as motor or sensory deficits, speech, etc. He did not think much of the event but presented after his wife told him to see someone about the event.

Exam

HEENT: No prior neck incisions, good cervical extension.

Cardiac: Regular rate and rhythm. Healed sternotomy scar.

Pulmonary: Clear to auscultation throughout.

Abdominal: Soft and nontender.

Neurologic: All cranial nerves 2-12 intact, no lateralizing deficits, 5/5 strength to all extremities.

Optho: No visual deficits at 20 feet from eye chart.

Imaging

Duplex Ultrasound (Peak Systolic Velocity/End Diastolic Velocity)

Location	Right	Left
Proximal ICA	540/240 cm/s	120/45 cm/s
Mid ICA	230/145 cm/s	119/37 cm/s
Distal ICA	240/110 cm/s	110/23 cm/s

Duplex Report: Based on color flow duplex imaging there is evidence of 80-99% stenosis of the right internal carotid artery segment and <50% stenosis to the contralateral side.

Discussion Points

> **i** N.B. There is no prepared answers for the questions below.
>
> However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. Please explain the pathophysiology of the visual event for this patient? Describe why it can be termed amaurosis fugax. Ensure understandinf that amaurosis fugax is the result of carotid plaque embolization to the retina.

2. Please list the patient's risk factors for carotid disease? What is best medical management to optimize these risk factors?
3. Is this patient asymptomatic or symptomatic based on the clinical scenario presented?
4. What next steps should be pursued to offer effective and timely treatment to this patient? Please discuss adjunct imaging such as CTA or MRA to determine anatomic characteristics of the lesions such as ulceration, vessel patency, level (accessible or high lesions), etc.
5. What surgical managements could be suggested to this patient? Please include a discussion of carotid endarterectomy, transfemoral stenting or TCAR.
6. What medications should be started in this scenario? Please consider ASA, Plavix, statin medications, etc.
7. What are some possible relevant complications of surgical intervention, including periprocedural stroke risk?

Key Articles

1. Ricotta JJ, Aburahma A, Ascher E, Eskandari M, Faries P, Lal BK; Society for Vascular Surgery. Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease. J Vasc Surg. 2011 Sep;54(3):e1-31.(Ricotta et al. 2011)

2. AbuRahma AF, Avgerinos EM, Chang RW, Darling RC 3rd, Duncan AA, Forbes TL, Malas MB, Murad MH, Perler BA, Powell RJ, Rockman CB, Zhou W. SOCIETY FOR VASCULAR SURGERY CLINICAL PRACTICE GUIDELINES FOR MANAGEMENT OF EXTRACRANIAL CEREBROVASCULAR DISEASE. J Vasc Surg. 2021 Jun 18. (AbuRahma et al. 2022)

3. Endarterectomy for asymptomatic carotid stenosis.Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. JAMA 1995;273(18):1421-8. (Walker 1995)

4. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade stenosis. North American Symptomatic Carotid Endarterectomy (NASCET) Trial Collaborators. N Engl J Med 1991;325(7):445-53.("Beneficial Effect of Carotid Endarterectomy in Symptomatic Patients with High-Grade Carotid Stenosis" 1991)

5. Howard D.P.J., Gaziano L., Rothwell P.M.: Risk of stroke in relation to degree of asymptomatic carotid stenosis: a population-based cohort study, systematic review, and meta-analysis. Lancet Neurol 2021; 20: pp. 193-202.(Howard, Gaziano, and Rothwell 2021)

Additional Resources

Audible Bleeding Content

- Audible Bleeding Exam Prep: Cerebrovascular Chapter
- Audible Bleeding has an episode covering the NASCET trial. Listen to it below and find additional information here, or find the episode wherever you listen to podcasts.

Websites

- TeachMe Surgery: Carotid Artery Disease

Serious Games

Touch Surgery Simulations.

- Must download the Medtronic Touch Surgery mobile application to access the modules. Available for Apple and Android mobile devices.
- Carotid Endarterectomy
- Carotid Artery Stenting

Operative Footage

Developed by the Debakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.*

Carotid Endarterectomy (Short Version)

Carotid Endarterectomy (Long Version) Part 1

Carotid Endarterectomy (Long Version) Part 2

Carotid Endarterectomy (Long Version) Part 2

Transcarotid Artery Revascularization (TCAR)

Aortic Disorders

Abdominal Aortic Aneurysm (AAA)

Erin K. Greenleaf, MD, MS, Jonathan Bath, MD, Dawn M. Coleman, MD, FACS and Ezra Schwartz, MD,CM, MS

Note

By the end of this chapter, students will:

- Review abdominal aortic anatomy and aneurysm pathophysiology.
- Recognize history, physical exam, and radiologic findings typical of patients presenting with an abdominal aortic aneurysm.
- Identify indications for intervention on abdominal aortic aneurysms and the evidence-based foundation for these recommendations.
- Describe the basic procedural steps of both an endovascular and an open surgical approach for abdominal aortic aneurysm repair.
- Delineate appropriate surveillance intervals for imaging an abdominal aortic aneurysm, both before and after intervention.

Key Facts

1. Abdominal aortic aneurysms occur in 2-8% of the population in Western countries, with a slightly higher prevalence among males relative to females.
2. The average size of an abdominal aorta is approximately 2cm; hence, an aneurysm is diagnosed when the diameter of the aorta in this segment reaches 3cm (i.e. 1.5x normal diameter).
3. Risk factors for an abdominal aortic aneurysm growth include age, male sex, white race, smoking, atherosclerosis, family history of arterial aneurysms, and personal history of arterial aneurysms.
4. According to vascular surgery guidelines, elective repair of a fusiform abdominal aortic aneurysm is recommended once the aneurysm has reached 5.5cm diameter in males and 5 cm diameter in females, measured from outer wall to outer wall. Circumstances in which repair is indicated for smaller aneurysms include those associated with symptoms and/or rupture, saccular morphology, rapid ex-

pansion, or those in females (as mentioned above), immunosuppressed patients (i.e. chemotherapy, radiation therapy, or solid organ transplantation) and patients with aortopathies (e.g. Marfan Syndrome, Ehlers-Danlos Syndrome, etc.).
5. Nearly 80% of abdominal aortic aneurysms are currently repaired via an endovascular approach, with an open surgical approach generally reserved for patients in which a stent graft would perform poorly.
6. Outcomes following endovascular abdominal aortic aneurysm repair have improved over time, as experience has accrued with endovascular technique. This pattern of progressive improvement has not similarly been seen for open surgical repair.
7. Surveillance regimens for patients with unrepaired abdominal aortic aneurysms suggest imaging via ultrasound or computerized tomography (CT) scan every three years if the aneurysm is 3.0-3.9cm diameter, every year if the aneurysm is 4.0-4.9cm, and every six months if the aneurysm is 5.0-5.4cm.

How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

Pre/Post Questions

Case Based Questions

1. A 75-year-old male with history of hypertension, hyperlipidemia, diabetes and degenerative disk disease presents to the emergency department with abdominal pain and a blood pressure of 90/60 mm Hg. The emergency department physician already obtained a CT scan showing what appears to be a contained rupture of an infrarenal abdominal aortic aneurysm. *What is the next step in management?*

A. Resuscitate the patient to ensure a systolic blood pressure of at least 120 mm Hg.

B. Two large bore IVs and admission to the ICU for closer monitoring.

C. Transfer to the operating room for emergent repair of the aneurysm.

D. Corroboration of the CT scan with an abdominal ultrasound.

E. Contact the next of kin to request their presence at bedside for a family meeting.

Answer

D. Transfer to the operating room for emergent repair of the aneurysm.
Discussion: **Explanations to come shortly**

2. An 83-year-old female presents to your clinic with a 5.1cm abdominal aortic aneurysm. She has a history of chronic tobacco abuse, hyperlipidemia, osteoarthritis, and renal cell carcinoma status post left nephrectomy. She denies abdominal pain and is without significant physical exam findings. *Based on her presentation, what is your recommendation to her?*

A. Continue surveillance with imaging every 6 months until her aneurysm reaches 5.5cm in diameter.

B. Discuss repair of the aneurysm at this size.

C. Prescribe an antiplatelet, statin, and beta blocker prior to discussing operative repair of the aortic aneurysm.

D. Continue surveillance with imaging every 12 months until her aneurysm reaches 5.5cm in diameter.

E. Tobacco cessation should be confirmed prior to committing to aneurysm repair.

Answer

B. Discuss repair of the aneurysm at this size.
Discussion: **Explanations to come shortly**

3. A 68-year-old male with history of hypertension, hyperlipidemia, tobacco abuse, obesity, and gout is found to have a 5.9cm infrarenal abdominal aortic aneurysm with a 21mm neck, patent common femoral arteries, but highly calcified/nearly occluded bilateral iliac arteries. *Upon discussing repair with him in your surgery clinic, what would you advise the patient about his repair?*

A. He should undergo endovascular repair because there is at least 20mm of neck below the lowest renal artery before the aneurysm begins.

B. He should undergo endovascular repair with percutaneous access because his common femoral arteries are widely patent.

C. He should undergo endovascular repair because this will minimize his inpatient stay while maximizing the primary patency of the stent graft.

D. He should undergo open repair because his diseased iliac arteries will preclude safe passage of sheaths and a device to the level of the aorta.

E. He should undergo open repair because this will minimize his short-term morbidity.

Answer

D. He should undergo open repair because his diseased iliac arteries will preclude safe passage of sheaths and a device to the level of the aorta.
Discussion: **Explanations to come shortly**

4. A 78-year-old female is referred by her primary care physician after she was incidentally found to have a saccular aneurysm immediately distal to her right renal artery, which is the lower of the bilateral renal arteries. *When documenting the location of this aneurysm, what terminology should be used to label it?*

A. Infrarenal

B. Juxtarenal

C. Suprarenal

D. Infrainguinal

E. No specific term exists

Answer

A. Juxtarenal
Discussion: **Explanations to come shortly**

5. Two days following an open transperitoneal repair of an infrarenal abdominal aortic aneurysm, an 81-year-old male with a history of a right colectomy for colon cancer has bright red blood in his stool. He remains in the ICU with a leukocytosis and appropriately tender abdomen without peritonitis. *What is the next best step in management?*

A. Broad spectrum antibiotics and flexible sigmoidoscopy.

B. Broad spectrum antibiotics and CTA.

C. Observation as this is likely related to his previous colon resection.

D. NPO order and immediate return to the operating room.

E. Stool softeners to mitigate the risk of hemorrhoids and anal fissures.

Answer

B. Left carotid endarterectomy before CABG.
Discussion: **Explanations to come shortly**

6. A 70-year-old female is undergoing an endovascular repair of an abdominal aortic aneurysm in the operating room under general anesthesia and a completion aortogram was just taken. A late blush is noted in the sac, seemingly arising from a lumbar artery. *What kind of endoleak would this suggest is present?*

A. Type Ia endoleak; blood is filling the sac from the proximal extent of the stent graft.

B. Type III endoleak; blood is filling the sac from the middle and can appear to be coming from a lumbar artery.

C. Type Ib endoleak; blood is filling the sac retrograde from the iliac arteries and so is only seen with a late aortogram view.

D. Type II endoleak; flow from a lumbar artery would cause a late blush and can be followed conservatively provided that the sac does not continue to expand.

E. No endoleak.

Answer

D. Type 2 endoleak; flow from a lumbar artery would cause a late blush and can be followed conservatively provided that the sac does not continue to expand.
Discussion: **Explanations to come shortly**

Operative Footage Questions

To come shortly. Stay tuned!

Introduction

The aorta is invariably the largest arterial conduit in the body, serving as the channel through which blood from the heart travels to every other body part. The wall of the aorta is comprised of three layers, which include the intima, media and adventitia. Its typical course begins at the distal side of the aortic valve and terminates at the aortic bifurcation, where it splits into

the two common iliac arteries. The thoracic aortic segment provides blood flow to the great vessels of the aortic arch, ensuring perfusion to the head and bilateral upper extremities via the innominate artery, the left carotid artery, and the left subclavian artery (in that order). The abdominal segment is a retroperitoneal structure that enters the abdominal cavity through the aortic hiatus of the diaphragm, at the level of T12. The branches of the abdominal aorta include, in descending order, the two inferior phrenic arteries, the celiac axis or trunk, the superior mesenteric artery (SMA), the renal arteries, the inferior mesenteric artery (IMA), and ultimately, the bilateral common iliac arteries. The branches of both the thoracic and abdominal aorta provide blood flow to areas of critically vital viscera.

While the aorta serves a critical role for the rest of the body, it can be the site of significant pathology. Aneurysms, which are common pathology in both the thoracic and abdominal aorta, are defined by an increase in arterial diameter to 1.5 times the normal diameter of the vessel. As the diameter of the aortic aneurysm increases, the risk of rupture increases in direct proportion. For example, a patient with a 4cm abdominal aortic aneurysm (AAA) has an approximate 1-year rupture risk of 1%; a patient with a 5.5cm aneurysm has up to a 10% 1-year rupture risk. With the widespread use of cross-sectional imaging, many aortic aneurysms are discovered incidentally. Others are found on screening exams. The US Preventative Services Task Force recommends that men aged 65 to 75 years of age who have ever smoked tobacco undergo an abdominal ultrasound to screen for aneurysmal disease. Aortic aneurysms may also present symptomatically, with chest, abdominal and/or back pain and occasionally a palpable pulsatile abdominal mass, which is widely believed to represent an increasingly unstable aneurysm.

Etiology

Certain risk factors, some modifiable and others non-modifiable (a.k.a. innate), predispose individuals to the development of aortic aneurysms. These include age, male gender, family history of aneurysms or aortopathies, and comorbidities including hypertension, hyperlipidemia, and peripheral vascular disease. Interestingly, female sex, African American race, and a history of diabetes have been found to be protective against the development and rupture of aortic aneurysms. A personal history of Marfan's syndrome, Loeys Dietz syndrome, or Ehlers Danlos Type IV (a.k.a. vascular Ehlers-Danlos or vEDS) is associated with a particularly elevated risk of aneurysmal degeneration. Medical providers should counsel and assist their patients to mitigate all potentially modifiable risk factors.

Diagnostics and Imaging

When evaluating a patient with a suspected aortic aneurysm, a thorough history, physical examination, and imaging will help to narrow the differential diagnosis. Most often, patients present without symptoms; however, if symptomatic, typical complaints include chest, abdominal or back pain. Physical exam findings may not be overt but vital signs may provide a first clue in a symptomatic patient as heart rate and blood pressure might deviate from normal ranges. Patients with thoracic aortic aneurysms (TAA) often have no other easily identifiable physical exam findings aside from vital sign derangements in those with symptomatic or ruptured aneurysms. In thin patients with suspected AAA, the abdominal exam may demonstrate a pulsatile mass at or adjacent to the midline.

Although the history and physical exam are indispensable to patient evaluation, the diagnosis of an aneurysm is usually secured with imaging. The mainstay of imaging is computed tomography (CT), while ultrasound, magnetic resonance imaging (MRI), and angiography are useful modalities in non-urgent or non-emergent settings. The benefits of CT, particularly the richness of anatomic data obtained, must be weighed against the potential risks related to contrast administration and exposure to radiation. Since the 2019 statement from the U.S. Preventive Services Task Force regarding one-time screening with ultrasonography in men aged 65 to 75 years who have ever smoked, the use of ultrasound has a more prominent role in AAA screening. Equipped with history, exam, and imaging data, the diagnosis of an aortic aneurysm can usually be made.

When looking at an imaging study, abdominal aortic aneurysms are labeled in regard to their location relative to the renal arteries: - Aortic aneurysms are termed **"suprarenal"** when their most proximal extent is above the level of the renal arteries. Suprarenal abdominal aortic aneurysms typically involve visceral branches of the aorta as well (i.e. the SMA or celiac trunk). - Aneurysms are labeled **"pararenal"** when the proximal extent of the aneurysm is at the level of the renal arteries and includes the origins of the renal arteries. - Abdominal aortic aneurysms are termed **"juxtarenal"** when their most proximal extent is immediately below the take-off of the renal arteries. - Lastly, aneurysms are labeled **"infrarenal"** when they begin below the renal arteries, often with a neck of non-dilated aorta between the lowest renal artery and the top of the aneurysm. As one might expect, the location of the aneurysm has implications for treatment options.

> Tip
>
> Please click here to open an UpToDate image depicting the anatomic classifications of AAAs.

Treatment

Once a diagnosis has been obtained, considerations for management follow. Similar to a thoracic aortic aneurysm (TAA), indications for repair are reflective of risk for rupture. The aim is to intervene when the rupture risk exceeds the risks posed by surgery. Intervention is considered when an aneurysm reaches the size threshold of 5.5cm in males and 5cm in females or if the growth rate exceeds 5 mm in 6 months. Again, this threshold is modified in patients with a strong family history of aneurysmal disease or collagen vascular disorder, or when patients present symptomatically or with a ruptured aorta. The topography of the aneurysm must also be considered as a saccular aneurysm, an isolated pouch off the sidewall of the aorta, presents a much greater risk of rupture and should be fixed when identified.

On first evaluation, patients with AAAs should be medically optimized to minimize their perioperative risk of morbidity and mortality. Multiple factors must be considered when deciding between an open surgical approach and an endovascular one. While the prevalence of endovascular aortic repair (EVAR) has exponentially increased in the past two decades relative to open surgical repair (OSR), each aneurysm poses a unique set of potential constraints that suggest a preference for one type of repair over another.

Please see the Section section and watch the included videos.

Endovascular Aortic Repair (EVAR)

Endovascular aortic repair (EVAR) is a novel approach to AAA treatment that has gained widespread popularity since its first successful use by Dr. Juan Parodi and Dr. Julio Palmaz in 1991. EVAR necessitates cross-sectional imaging to measure the AAA dimensions and determine the appropriate size of an aortic stent graft. Stent grafts are straight or bifurcated tubes with a wire framework covered by non-porous fabric. Simply, it is a tube that can be deployed from inside a vessel.

EVAR Procedural Steps

A step-by-step guide of a typical, uncomplicated EVAR for an infrarenal AAA is listed below. Please see the Section section to a video guide of a typical EVAR.

1. Bilateral femoral access via percutaneous or open surgical exposure (a.k.a. “femoral cut-downs”)
2. Heparinization to achieve an activated clotting time (ACT) greater than 250 seconds.
3. Placement of stiff wires into the abdominal aorta and upsizing of sheaths into the access vessels.

4. Aortogram to visualize renal arteries and deployment of the endograft main body under the origins of the renal arteries.
5. Cannulation of contralateral limb gate using a wire and catheter with subsequent confirmation that both the wire and catheter are within the main body.
6. Aortoiliac angiogram to measure the distance from the contralateral gate to the contralateral hypogastric (i.e. internal iliac) artery.
7. Deployment of contralateral limb with the distal landing site just proximal to the hypogastric artery.
8. Partial deployment of the ipsilateral limb.
9. Aortoiliac angiogram to measure the distance from the ipsilateral gate of the main body to the ipsilateral hypogastric artery.
10. Deployment of the remainder of the ipsilateral limb.
11. Ballooning of the proximal and distal landing zones, and areas of stent overlap.
12. Remove wires and sheaths and close arteriotomies (often with closure devices).
13. Reverse heparin with protamine.
14. Apply pressure to surgical sites and monitor for hematoma formation.

EVAR Considerations and Planning

The need for imaging can be a limiting factor such as when patients with a ruptured aneurysm are too hemodynamically unstable to stop at the CT scanner prior to repair or when a patient's kidney function is too impaired to tolerate a large contrast bolus.

If the situation is amenable to getting a CT angiogram to measure anatomic dimensions, several components of the aneurysm anatomy should be assessed to determine anatomic suitability for EVAR. These include the length, width, angulation of the aneurysm, and presence of disease at the aneurysm neck (i.e. the aortic segment between the lowest renal artery and the proximal extent of the aortic dilatation).

> Tip
>
> - See page 71 of the Gore Vascular/Endovascular Surgery Combat Manual in the additional resources (**?@sec-additionalresources**) for a **high-yield** EVAR planning and sizing considerations chart.
> - Video Tutorial: Endovascular Aortic Repair Preoperative Sizing with Dr. Sharif Ellozy. *This is above the level of medical students*, but for those who are interested, watch it here.

The access vessels, including the bilateral iliac and femoral arteries, must be healthy enough to allow sheaths, wires and catheters to pass through them. Calcification due to atherosclerosis can make arterial access difficult, and if vessels are so diseased as to be completely occluded, wires and devices may not be able to travel past the occlusion. Additionally, the iliac arteries must be within the appropriate diameter and length dimensions to accommodate the chosen stent graft. Lastly, certain anatomic anomalies such as a horseshoe kidney must be evaluated to ensure the appropriateness of EVAR for repair. With recent innovations in endovascular devices and intra-operative imaging, many factors that were once contraindications to EVAR have now become considerations when planning for EVAR. This is to say, as technology advances, EVAR is increasingly possible and safe for patients with difficult anatomy and co-morbidities.

EVAR Complications

Although a more recent development relative to open surgical repair (OSR), EVAR is not without risk of complications. EVAR may be complicated by injury to access vessels such as femoral occlusion or iliac avulsion (aka, "iliac-on-a-stick"), wire trauma from the relatively stiff wires that are used to deliver the stent graft, rupture of the aneurysm, and endoleak. Endoleak is the term used to describe ongoing blood flow in the aneurysm sac after stent graft placement (e.g. from lumbar arteries or the inferior mesenteric artery, inadequate proximal or distal endograft seal, a defect in the endograft, etc.). There are various types of endoleak and each has its own management approach.

Endoleaks are classified as follows:

- Type Ia: caused by an improper **proximal** endograft seal (i.e. the top of the graft is not well opposed to the abdominal aorta and blood can flow around it into the aneurysm sac).
- Type Ib: caused by an improper **distal** endograft seal (i.e. the distal iliac limb is not well opposed to the iliac artery and blood can flow retrograde into the aneurysm sac).
- Type II: caused by retrograde blood flow into the aneurysm sac from aortic side branches such as lumbar arteries or the IMA. This is the most common type and does not require intervention provided a stable aneurysm sac size is observed on follow-up. If the sac continues to expand, the feeding vessels may need to be occluded via embolization (e.g. coil embolization).
- Type III: caused by a defect in the endograft such as a fabric tear, or disconnection of the contralateral iliac limb from the main body of the graft.
- Type IV: caused by graft wall porosity. To explain graft wall porosity, imagine filling up a pillowcase with water. Even if the pillowcase has the tightest of weaves, eventually, water will leak *through* the pillowcase.

- Type V: increase in aneurysm size with no identifiable cause.

> **Tip**
>
> Please click here to open an article by Dr. Tamer W. Kassem with a simple image depicting the endoleak classifications.

Additionally, one should monitor for abdominal compartment syndrome after repair of AAA rupture, as much of the large volume resuscitation can leak from the intravascular compartment into the interstitium of the abdominal tissues causing distention. In the event of rupture, the retroperitoneal hematoma can cause a mass effect and increase of abdominal pressures. Lastly, a well-known complication of EVAR is ischemic colitis, although it is only encountered in less than 3% of endovascular aortic repairs. This occurs when the colon becomes ischemic secondary to vascular hypoperfusion caused by a variety of quoted sources including atheroembolization and systemic hypotension. Albeit rare, ischemic colitis has a significant associated mortality risk and should be addressed promptly with bowel rest, antibiotic coverage, a flexible sigmoidoscopy, and potentially further intervention, if warranted. This complication is not limited to EVAR and has been identified postoperatively with the same incidence in OSR.

Open Surgical Repair (OSR)

Open surgical repair (OSR) remains the traditional means of repairing AAA and is still the most appropriate modality when EVAR is not feasible for anatomic reasons. In the elective setting, the ideal patient is physiologically fit enough for OSR as it is a procedure with the potential for significant morbidity and a lengthy recovery.

OSR Considerations and Planning

OSR can be conducted via a transperitoneal or a retroperitoneal exposure. Other decision points for open repair include the following:

- Location of the proximal clamp: A clamp must be placed on the aorta in a location free of significant disease, with enough clearance below it to allow space for suturing the proximal anastomosis.
- Configuration of the proximal anastomosis: Anastomoses performed for aneurysmal disease are sewn in an end-to-end fashion, whereas aortas being reconstructed for occlusive disease can be performed either end-to-end or end-to-side.
- Location of the distal anastomosis: the end of the graft can be sewn to either the distal aorta, iliac arteries, or femoral arteries depending on the extent of the native vessel that needs to be excluded and the distal location where the vessel is healthy enough to clamp and receive sutures (e.g. is not heavily calcified).

- Management of the inferior mesenteric artery when an end-to-end anastomosis is created: The IMA can be ligated If collateral flow is adequate or reimplanted into the graft if collateral flow is inadequate.
- Management of aberrant intraperitoneal anatomy and pathology: Examples include adhesions or scarred tissue due to previous abdominal surgery, accessory renal arteries, horseshoe kidney, history of kidney transplant, and previous aortic replacement, among many other possibilities.

Depending on the urgency of repair, many of these considerations might only be entertained intraoperatively.

OSR Complications

Complications related to OSR differ from those of EVAR, owing greatly to the open abdominal exposure and the lack of intraluminal wire manipulation. Just as with most other laparotomies or open retroperitoneal dissections, incisional pain can be significant and cause atelectasis or other respiratory compromise. Additionally, ileus and wound infections can impair the return of proper bowel function. Similar to EVAR, ischemic colitis can manifest after OSR, particularly in the setting of rupture.

Outcomes

Much experience has been gained since the first EVAR in 1990 and ample research has been dedicated to the comparison of EVAR to OSR. Multiple trials have demonstrated an early survival advantage for EVAR over OSR that tends to diminish over time. Certainly, the perioperative period for EVAR is notable for reduced morbidity and mortality relative to OSR with length of hospital stay significantly longer in those undergoing OSR. However, this early benefit is often balanced by a necessity for life-long follow-up including imaging at regular intervals and a tendency for re-interventions during the surveillance period (e.g. to manage endoleaks). Hence, the risk-benefit ratio must be weighed individually for each patient.

Contemporary management of abdominal aortic aneurysms is most often undertaken with EVAR. Endovascular repair has a third of the peri-operative all-cause mortality attributed to open repair (1.6% vs 4.8%, respectively). This survival benefit tends to dissipate by three to four years post-operatively. Moreover, the overall complication rate of EVAR in the perioperative period is up to 10%.

Among patients who undergo open abdominal aortic aneurysm repair, mortality in the immediate post-operative period can be as high as 5%. Of patients who survive, later death is

typically related to cardiovascular disease burden. Complications arise in 9% of patients and most often include myocardial infarction, respiratory insufficiency, pneumonia, acute kidney injury, and ischemic colitis. Late Infection of the graft is possible, is challenging to manage, and occurs in less than 1% of patients.

Surveillance

The Society for Vascular Surgery guidelines suggest that for patients found to have an abdominal aortic aneurysm, surveillance imaging should occur at intervals specific to the maximum diameter of their aneurysm.

- For patients with abdominal aortic aneurysms between 3.0 and 3.9cm in diameter, CT or ultrasound should be undertaken every 3 years.
- Patients with abdominal aortic aneurysms between 4.0 and 4.9cm in diameter, imaging is indicated every 12 months.
- When the aneurysm reaches a maximum diameter of 5.0 cm, imaging with CT is recommended at intervals of every 6 months.

After the abdominal aortic aneurysm has been repaired, ongoing surveillance is warranted to monitor for post-repair complications and the need for reintervention. As mentioned, endovascular repairs can develop endoleaks over time, which may necessitate another intervention if the aneurysm sac is expanding. Therefore, patients who have undergone EVAR should undergo CTA at one month and 12 months post-operatively. A six-month scan is indicated in the presence of a persistent endoleak observed on the one-month scan. If the endograft is in a good position without complications, yearly interval follow-up with concomitant imaging is warranted. Open repairs can similarly develop problems that require repair, albeit with less frequency, and hence need surveillance imaging every five years.

Teaching Case

Scenario

A 73-year-old male with a history of atrial fibrillation, diabetes, hypertension, ongoing tobacco use, hyperlipidemia, chronic kidney disease, and peripheral arterial disease undergoes a screening ultrasound of the abdominal aorta. The ultrasound shows a 5.6cm infrarenal fusiform

aortic aneurysm extending into the proximal common iliac arteries. His primary care physician refers him to the vascular surgery clinic, where he presents without subjective complaints. CTA is recommended to assess the patient's anatomy.

Exam

HEENT: No carotid bruits.

Cardiac: Irregularly irregular, no murmurs.

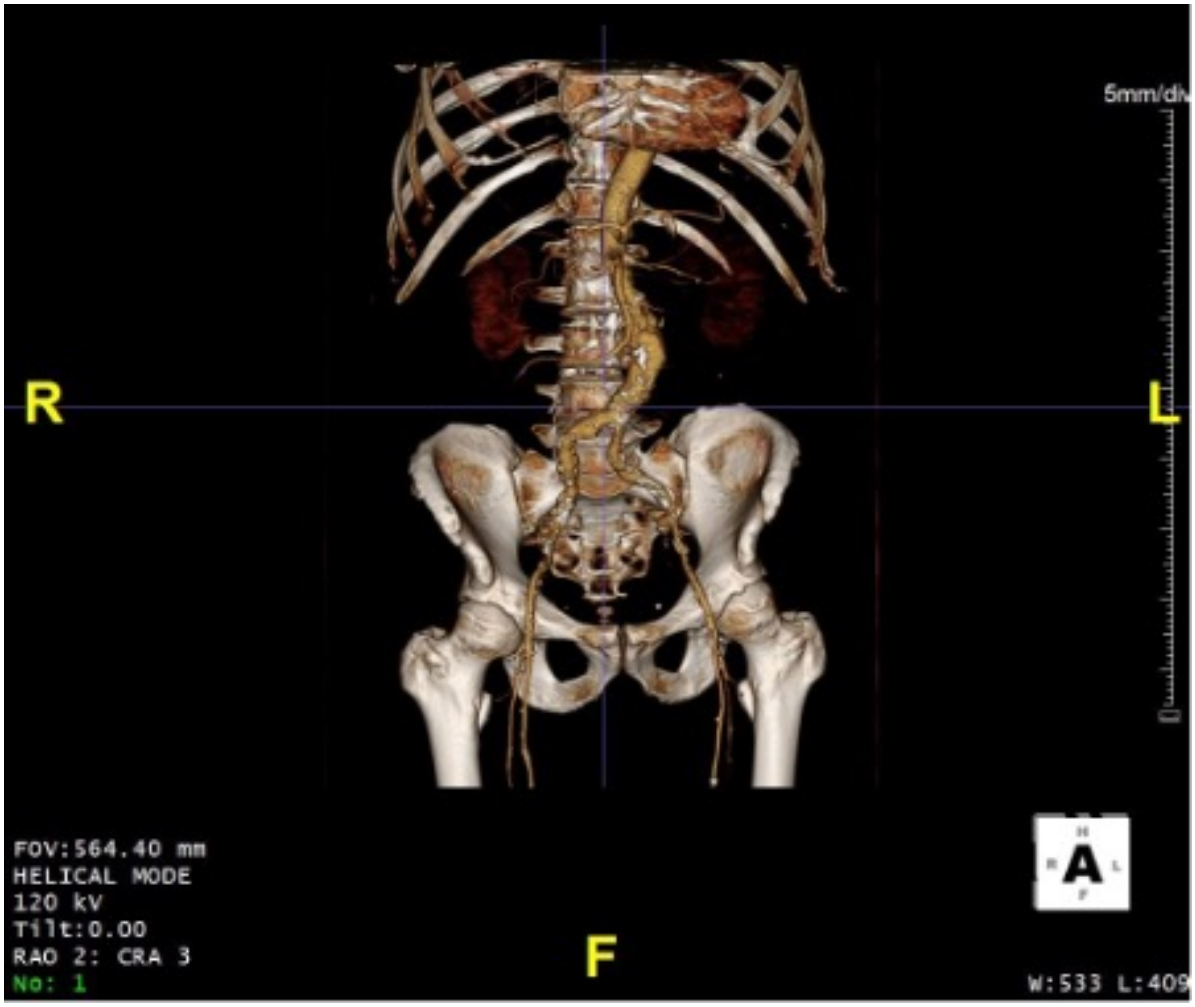
Pulmonary: Clear to auscultation in all lung fields.

Abdominal: Obese, soft, non-tender, no pulsatile masses felt.

Vascular: Palpable femoral pulses, multiphasic pedal signals bilaterally.

Imaging

CTA Abdo/Pelvis



CTA Report: 5.6cm infrarenal bilobed aortic aneurysm with bilateral accessory renal arteries inferior to main renal arteries. 14mm infrarenal neck cranial to aneurysm. Widely patent iliac systems with non-diseased femoral arteries.

Discussion Points

> **i** N.B. There is no prepared answers for the questions below.
>
> However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. What recommendations for medical management should be made to optimize this patient prior to surgical intervention?

2. What is the important information to ascertain from history, physical exam and imaging that can help you determine the best modality of treatment.
3. With what urgency should the patient be scheduled for operative repair?
4. What kind of repair would you suggest for this patient and why? Open or endovascular?
5. What complications are possible after an endovascular repair of the abdominal aorta? After an open repair?
6. Following repair, how should the patient be surveilled? With what frequency?
7. If the patient's aneurysm was 4.6cm in greatest transverse diameter, how often should he undergo imaging and follow-up?
8. What aortopathies and collagen vascular diseases would convince you to repair the aneurysm at a size less than 5.5cm?

Key Articles

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Additional Resources

Audible Bleeding Content

- Audible Bleeding Exam Prep: AAA Chapter
- Audible Bleeding has an episode covering the IMPROVE trial. Listen to it below and find additional information here, or find the episode wherever you listen to podcasts.
- Holding Pressure Case Prep: EVAR. Listen to it here, or find the episode wherever you listen to podcasts. **Be sure to take a quick look at the shownotes.**
- VSITE Review - AAA. Listen to it below and find additional information here, or find the episode wherever you listen to podcasts.
- Video Tutorial: Dr. Kristina Giles - "Decision-Making and Treatment: Elective Infrarenal and Juxtarenal AAA." Watch it here.
- Video Tutorial: Open AAA repair: How I Do It with Dr. Ashlee Vinyard and Dr. John Eidt. Watch it here.
- Vascular Origin Stories: Bridging the Gap - The Fabric of Aortic Repair. Listen to it below and find additional information here, or find the episode wherever you listen to podcasts.

Websites

- TeachMe Surgery: Abdominal Aortic Aneurysm

Operative Footage

Developed by the Debakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.*

Endovascular Aortic Repair (EVAR)

Open Surgical Repair (OSR) of a AAA, Transperitoneal Access

EVAR Explantation with Aortic Reconstruction

Anonymous Feedback

We welcome suggestions for updates. We want to hear from you! Please help us help you!

To this end, please complete this anonymous Google Form to send us any feedback you may have.

Thank you for your continued support!

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