Sonographic Evaluation of Complications of Extracranial Carotid Artery Interventions

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Carotid endarterectomy and carotid artery stenting are among the most common peripheral vascular procedures performed worldwide. Sonography is the initial and often only imaging modality used in the evaluation of iatrogenic carotid arterial injuries. This pictorial essay provides an overview of the clinical and sonographic findings of complications after interventions in the extracranial carotid arteries, including dissection, fluid collections, pseudoaneurysm, thrombosis, thromboembolism, restenosis, and stent deformation. Grayscale, color, and pulsed Doppler imaging findings are reviewed, and correlations with computed tomography, magnetic resonance imaging, and angiography are provided.

Key Words—carotid artery; carotids/extracranial; complications; endarterectomy; sonography; stenting

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Abbreviations

CAS, carotid artery stenting; CCA, common carotid artery; CEA, carotid endarterectomy; CT, computed tomography; ICA, internal carotid artery; PSV, peak systolic velocity; MRI, magnetic resonance imaging

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Carotid endarterectomy (CEA) and carotid artery stenting (CAS) are among the most common peripheral vascular procedures performed worldwide.[1,2] In the United States, it is estimated that 1,350,000 CEAs were performed and 90,000 CASs were placed between 1998 and 2008.[3] During this same time, CAS use in the United States increased from 2.8% to 12.6% of all carotid artery revascularization procedures,[3] due in part to the publication of several large, multicenter studies demonstrating its safe use as a less-invasive alternative to CEA for certain patient populations.[4,5]

Sonography is considered the first-line imaging modality of choice for the evaluation of iatrogenic vascular injuries. As surgical and endovascular carotid artery interventions have become increasingly common, sonologists may expect to encounter iatrogenic complications from these procedures. Additionally, intraoperative sonography is performed after CEA at some institutions to assess for the presence of residual intimal flaps or residual stenosis and to exclude carotid arterial wall thrombi.[6,7] The purpose of this article is to provide a pictorial review of complications after surgical and percutaneous endovascular interventions in the extracranial carotid arteries (Table 1). The sonographic appearances of these complications are reviewed, and illustrative computed tomography (CT), magnetic resonance imaging (MRI), and angiographic correlations are provided.

Dissection

Arterial dissections result from tears between any of the 3 arterial wall layers (intima, media, and adventitia). Depending on whether

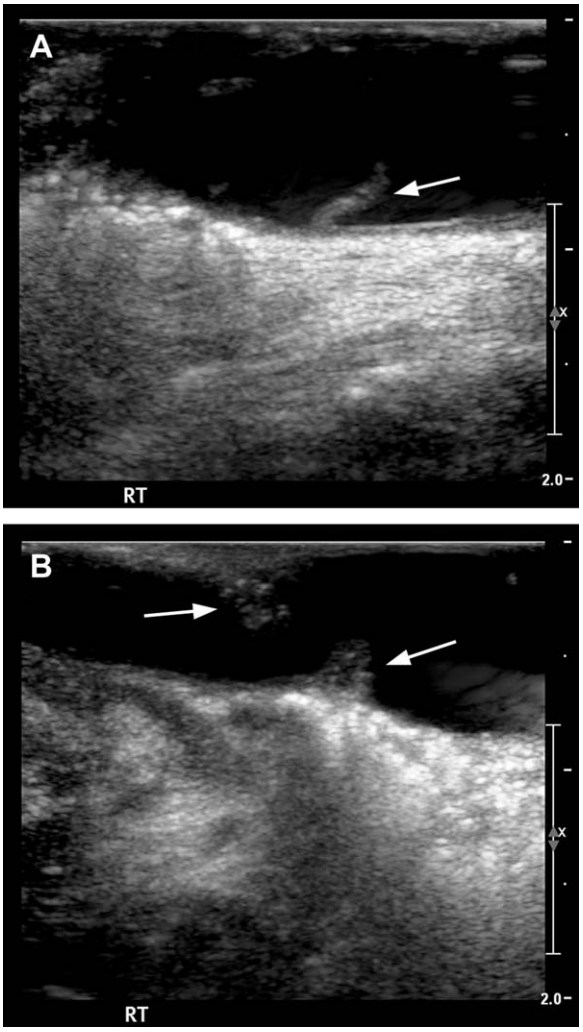
there is an exit as well as entrance tear, a dissection may result in an intramural hematoma or creation of a false lumen from blood flowing into the defect. Dissections may also result from spontaneous rupture of a vasa vasorum, with the direct introduction of blood between vessel layers.[8] The incidence of spontaneous carotid dissections has been estimated at 2.6 to 2.9 per 100,000 patients in all age groups.[9,10] In a retrospective analysis of 6981 diagnostic and therapeutic interventions in the carotid and vertebral arteries, iatrogenic arterial dissections occurred in 0.25% of cases, with 10 of 18 reported dissections occurring in the internal carotid arteries (ICAs).[11] In that series, no dissections were observed in the pediatric population, and the incidence increased to 0.35% in patients older than 35 years.[11] Iatrogenic causes of carotid dissections include vessel layer disruption during CEA and other open procedures of the neck or skull base,[12,13] intimal trauma from guide wire manipulation during percutaneous endovascular procedures,[11] trauma to the vessel wall from a contrast injection jet,[11] and inadvertent direct puncture of the carotid artery during an attempt at central venous catheterization.[12] Although carotid dissections are more common with increasing age,[11] other associated vascular risk factors include hypertension, diabetes, smoking, hyperlipidemia, and use of oral contraceptives.[14] Genetic connective tissue diseases that cause arteriopathy or weakness of the vessel wall, including Ehlers-Danlos syndrome, Marfan syndrome, and autosomal dominant polycystic kidney disease, also increase the dissection risk.[13] A history of recent infection, especially respiratory infection, has been shown to be more prevalent in patients with carotid artery dissections.[15]

Although intraprocedural dissections are often immediately identified, carotid dissections can cause a range of symptoms, including localized and unilateral neck, facial, or orbital pain, headaches (most commonly frontotemporal), partial Horner syndrome (unilateral miosis and ptosis), and cranial nerve palsies (most commonly hypoglossal).[16] Stroke is the most feared consequence of a carotid artery dissection. Dissection-related strokes are most commonly embolic in nature but can also be ischemic due to decreased blood flow and

Table 1. Classification of Complications After Carotid Artery Interventions

Intraprocedural	Postprocedural
Dissection	Fluid collection
	Hematoma
	Abscess
	Suture dehiscence
	Pseudoaneurysm
	Thrombosis
	Thromboembolism
	Restenosis
	Stent deformation or fracture
	Arteriovenous fistula

Figure 1. Images from an 84-year-old woman after right CEA complicated by a difficult surgical reconstruction. **A**, Intraoperative sagittal grayscale sonogram shows a small intimal flap in the right carotid bulb (arrow). **B**, Smaller intimal flaps, adherent fibrin strands, or thrombi are also seen along the anterior and posterior walls of the superior aspect of the endarterectomy site (arrows). Given the small size of the intimal flap, the patient was treated medically with aspirin at 325 mg/d. She had an uneventful postoperative course and remained asymptomatic at a 3-month follow-up.



thrombus formation within the true lumen, which can result in complete thrombosis or occlusion of the carotid artery.[17] One meta-analysis that included 180 patients with acute strokes in the setting of carotid artery dissections showed similar safety and outcomes with the use of thrombolytics as in other stroke patients.[18] Antithrombotic therapy for 3 to 6 months with aspirin or a combination of heparin and warfarin has been advocated to reduce the risk of thromboembolism, and both treatments have shown similar efficacy in preventing recurrent stroke and disability.[19,20] Patients with evidence of expanding dissections, symptoms despite anticoagulation, or contraindications to anticoagulation may benefit from endovascular or surgical repair.[8]

On grayscale imaging, a carotid artery dissection is characterized by the presence of a mobile echogenic flap within the artery or false lumen. When thrombosed, the false lumen can appear hypoechoic or echogenic and may or may not encroach on the true lumen. A thrombosed false lumen can be difficult to differentiate from an intramural hematoma or plaque, both of which may also narrow the true lumen. Color Doppler imaging can show flow within a patent false lumen parallel to the true lumen, separated by a thin echogenic linear structure representing the intima. The spectral Doppler waveforms observed in carotid arterial dissections are variable and may be normal, damped (lower amplitude with a biphasic pattern), high resistance, or absent flow.[14] Although MR angiography and CT angiography may also be used to diagnose and evaluate carotid arterial dissections,[21] sonography is better suited for serial imaging to follow the resolution or progression.

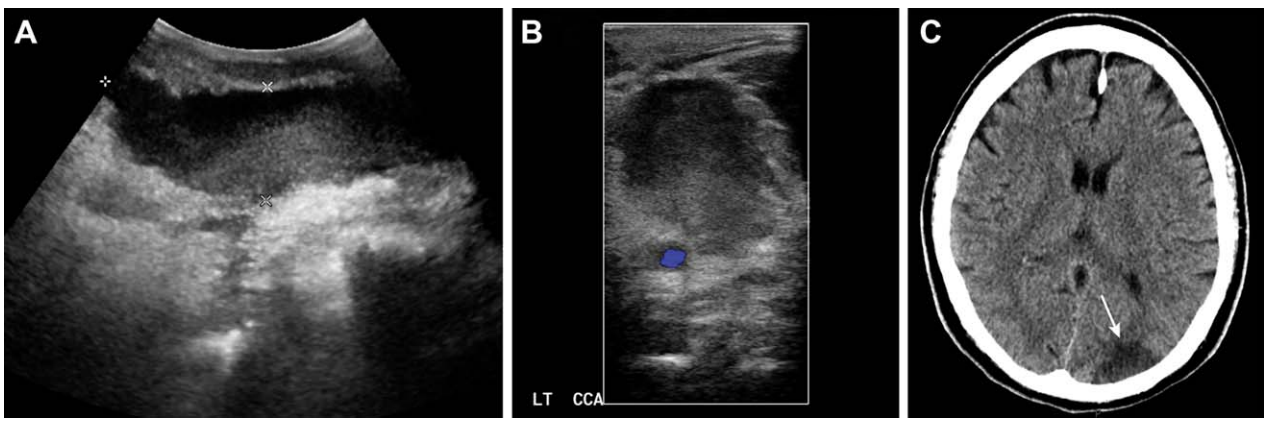
When intraoperative sonography is done, small mobile flaps may be seen within the artery on a grayscale sonogram (Figure 1). Typically, an arteriotomy followed by exploration is done if the flap is mobile and 2 mm or greater in length in the ICA, 3 mm or greater in the common carotid artery (CCA), or if there is a technical defect with greater than 30% luminal diameter reduction.[6,7] Intimal flaps and residual disease are usually left undisturbed in the external carotid artery.

Fluid Collections

Fluid collections, including hematomas, seromas, and abscesses, are well-known complications of CEA. Cervical hematomas can vary in size and severity and are dangerous because of their close vicinity to structures vital to respiration and cerebral blood flow and their ability to quickly expand.

In a randomized clinical trial, wound hematomas were found to occur in 7.1% (101 of 1415) patients who underwent CEA.[22] Although most wound complications were minor, 3.3% of patients (46 of 1415) required surgical reexploration, including 4 patients (0.3%) who died as a direct consequence of the wound hematomas.[22] Cervical hematomas after CEA are associated with an increased risk of mortality, intraoperative stroke, intraoperative myocardial infarction, and blood transfusion and an increased length of hospital stay.[23] Although a

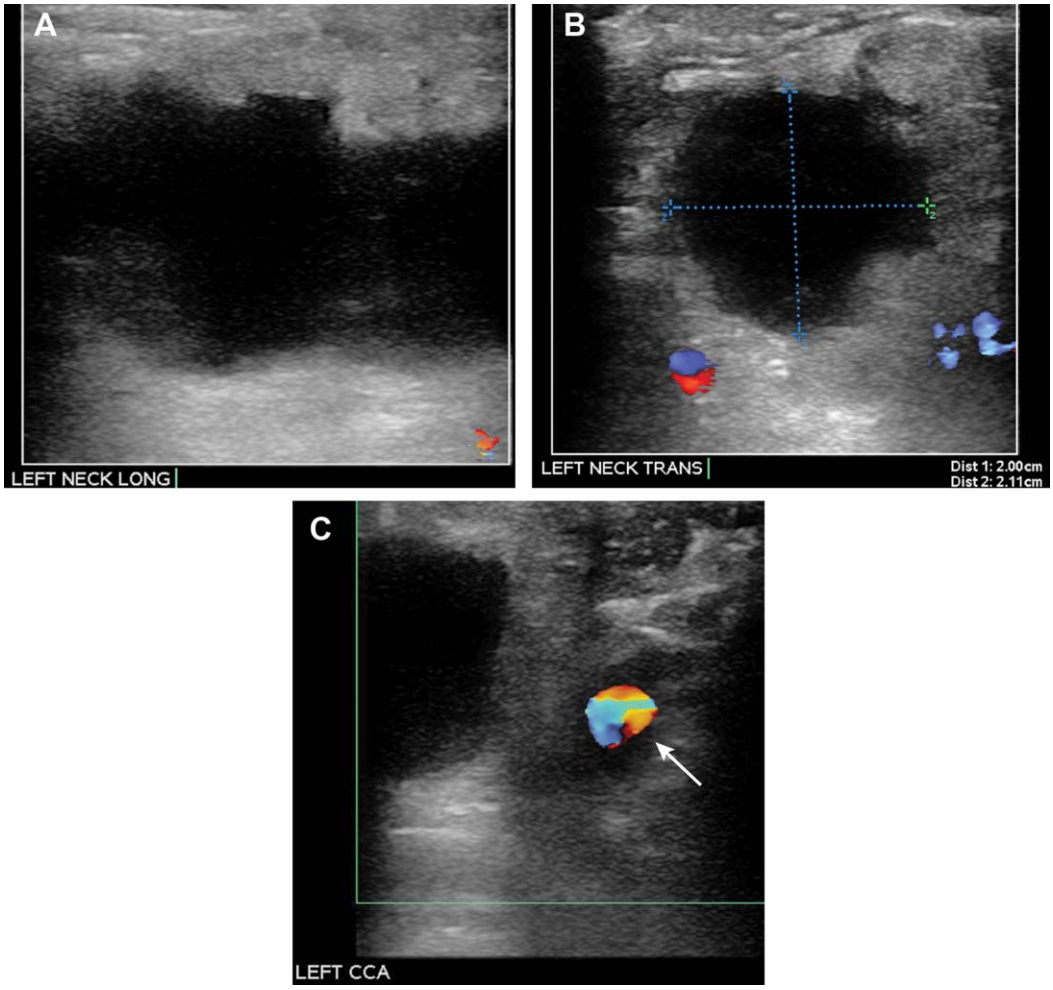
Figure 2. Images from a 65-year-old man who underwent left CEA 1 day previously and developed bleeding from the incision site. **A** and **B**, Sagittal grayscale and transverse color Doppler sonograms of the neck show a large collection with a fluid-fluid level, consistent with an acute hematoma, presumably secondary to suture dehiscence. He developed a stroke soon afterwards. **C**, Computed tomogram of the head without contrast shows a left posterior circulation infarct (arrow).



rare occurrence, the wires used during CAS can perforate branches of the external carotid artery, leading to hematoma formation.[24] In a systematic review and meta-analysis of randomized clinical trials comparing CAS to CEA, the 30-day risk for hematoma formation was lower with CAS, with a reported odds ratio of 0.59.[25] Anticoagulation is a well-known risk factor for hematoma formation during carotid artery procedures. In an analysis of 4587 CEAs, reversal of heparin anticoagulation with protamine during CEA significantly reduced the incidence of post-CEA bleeding requiring reoperation from 1.66% to 0.64% without increasing the risk of myocardial infarction, stroke, or death.[26] Other risk factors for hematoma formation after CEA include intraoperative hypotension and carotid shunt placement.[17] Acute and large hematomas are potentially hazardous entities and when not addressed in a timely manner can lead to stroke (Figure 2). Some hematomas may resolve without intervention; however, many surgeons will elect for immediate surgical revision when a hematoma is diagnosed.[27]

Abscesses can result from superinfection of a post-CEA hematoma or may occur as a result of an infected carotid patch. The incidence of carotid patch infections is rare, at 0.25% to 0.5%,[28] and may be greater when prosthetic patches are used.[29] Neck swelling and formation of a local draining sinus or a pseudoaneurysm are the most common presentations of an infected carotid

Figure 3. Images from a 69-year-old man who presented with a nonpulsatile left neck mass after left CEA. **A** and **B**, Sagittal and transverse color Doppler sonograms show an anechoic fluid collection adjacent to the left CCA and ICA, measuring 2.0 × 2.1 cm. **C**, The left CCA is patent (arrow). Findings were consistent with a postoperative hematoma versus seroma.



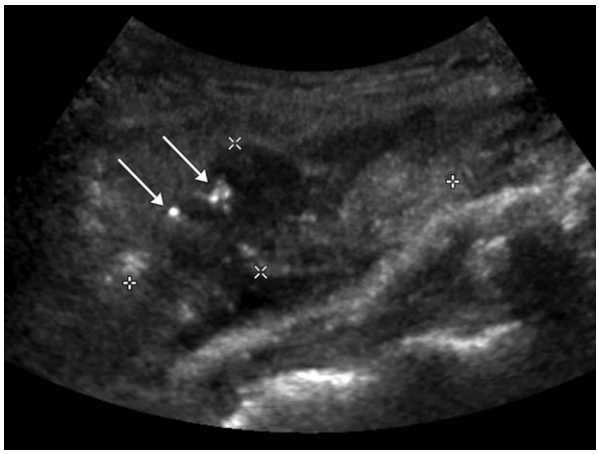
patch, with half of patients presenting 3 months postoperatively.[30]

On grayscale sonography, hematomas are usually visualized as soft tissue masses with heterogeneous echogenicity, often adjacent to nearby vessels (Figure 3). Echogenicity varies depending on the age of the hematoma. Fluid-fluid levels may be observed (Figure 2). Absence of flow should be confirmed on color and pulsed Doppler imaging. Abscesses can present as poorly defined heterogeneously echogenic masses. In some cases, punctate echogenic foci representing gas bubbles may be seen (Figure 4).

Pseudoaneurysm

A pseudoaneurysm typically results from disruption of all 3 layers of the arterial wall, resulting in a jet or extravasation of blood through the neck of the pseudoaneurysm into the adjacent sac, which is contained by a compacted layer of the surrounding soft tissues. Partial thrombosis of the pseudoaneurysm sac is common. However, some pseudoaneurysms arise secondary to weakening of the arterial wall from damage to only 1 or 2 layers (eg, after a dissection), resulting in an outpouching or focal bulge of the remainder of the arterial wall, usually the adventitial layer and sometimes the media into the surrounding tissues.[31] Iatrogenic pseudoaneurysms of the carotid arteries are rare. The incidence of pseudoaneurysms after CEA has been reported to be 0.37%, with underlying causes including suture failure, arterial wall degeneration, injury to the vessel wall from surgical clamping, dissection, and infection.[32] Additional cases have been reported after carotid stenting[33] and placement of central lines.[34] Risk factors for iatrogenic pseudoaneurysm formation include advanced age, female sex, calcified vessels, obesity, and anticoagulation.[31]

Figure 4. Image from an 81-year-old man after left CEA with bovine patch angioplasty 1 week previously, who presented with left neck pain. Transverse grayscale sonogram of the left neck shows a poorly defined area of heterogeneous echogenicity measuring 5.1 × 2.0 × 4.3 cm, outlined with calipers. Note the punctate echogenic foci (arrows) within the collection, which likely represent gas bubbles. Although this appearance may be seen with both an abscess and a hematoma (secondary to aseptic breakdown of blood products), in this case, the collection resolved without intervention.



A carotid pseudoaneurysm may present immediately after an inciting event or years later.[35] In one reported case, a man presented with a pseudoaneurysm 29 years after CEA.[36] Presenting symptoms may include pulsatile neck swelling, a palpable cervical mass, pain, persistent bleeding, or infection at the surgical site.[32] Pseudoaneurysms may also present with neurologic symptoms due to compression of nearby nerves and can be found in asymptomatic patients undergoing follow-up imaging.[35] Traditionally, treatment for carotid pseudoaneurysms has involved open surgery.[35] However, endovascular repair is becoming more common. A recent systematic review of endovascular stenting for true and false aneurysm repair found it comparable in efficacy to open repair.[37]

On grayscale imaging, pseudoaneurysms appear as soft tissue or fluid-filled masses that are contiguous with or adjacent to the vessel with varying echogenicity depending on the presence or lack of a thrombus. Persistent swirling blood flow on color Doppler imaging within the pseudoaneurysm sac, with a "yin-yang" pattern, can help distinguish a pseudoaneurysm from a hematoma. The neck connecting the pseudoaneurysm to the carotid artery may be wide or thin, depending on etiology. If thin, a typical "to-and-fro" waveform pattern characterized by flow heading toward the pseudoaneurysm during systole and away from the pseudoaneurysm during diastole will be observed. Angiography, CT, or MRI can be useful to confirm the diagnosis of a carotid pseudoaneurysm, size, presence or absence of an intraluminal thrombus, and anatomy (width and length) of the neck, all of which are important for determining optimal patient treatment (Figure 5).

Thrombosis and Thromboembolism

Carotid thrombosis and thromboembolism leading to stroke are among the most feared complications of carotid artery interventions and may occur independently or in conjunction with one of the other

complications described above. The long-term (>2 years) incidence of major ipsilateral stroke after revascularization ranges from 0.5% to 3.0% for CEA and 1.2% to 4.6% for CAS, with no significant difference yet observed between the approaches.[38] However, studies have found the long-term risk of all strokes (major and minor) to be significantly higher for CAS than CEA,[39,40] with incidence rates reported to range from 7.9% to 10.1% for CEA and 9.0% to 14.2% for CAS.[38] A few trials have also reported a significantly higher rate of stroke in the periprocedure period for CAS,[39–41] although this risk may be mitigated by the use of embolic protective devices during stenting.[38]

Acute thrombosis is a catastrophic event that requires immediate recognition and treatment with thrombolysis, endovascular thrombosuction, or open surgery.[42] The most frequently cited causes of acute thrombosis after CAS are failure to receive antiplatelet medication and technical failure, such as stent malposition and carotid dissection.[43] Risk factors for stroke after CEA include female sex,[44] nonwhite race, prior stroke or transient ischemic attack, contralateral stenosis of greater than 50%, severe disability, and coronary artery disease,[45] whereas risk factors for stroke after CAS include advanced age (≥80 years), chronic renal failure, diabetes mellitus, symptomatic status, operator inexperience, and use of multiple stents.[46] High-grade stenosis and heavily calcified plaque are also believed to increase the risk of stroke.

On sonography, an acute carotid thrombus appears as a hypoechoic area within the vessel or stent lumen. A

Figure 5. Images from an 83-year-old woman after left CEA 3 years previously who presented with a hard, enlarging left neck mass. **A**, Sagittal grayscale sonogram of the left CCA shows an outpouching, consistent with a wide-neck pseudoaneurysm. Note internal echoes (arrow) that fill in approximately half of the pseudoaneurysm lumen, consistent with partial thrombosis. **B**, A yin-yang pattern of swirling blood flow was observed within the residual lumen on color Doppler interrogation. **C**, Angiography confirmed the presence of a CCA pseudoaneurysm (arrow).

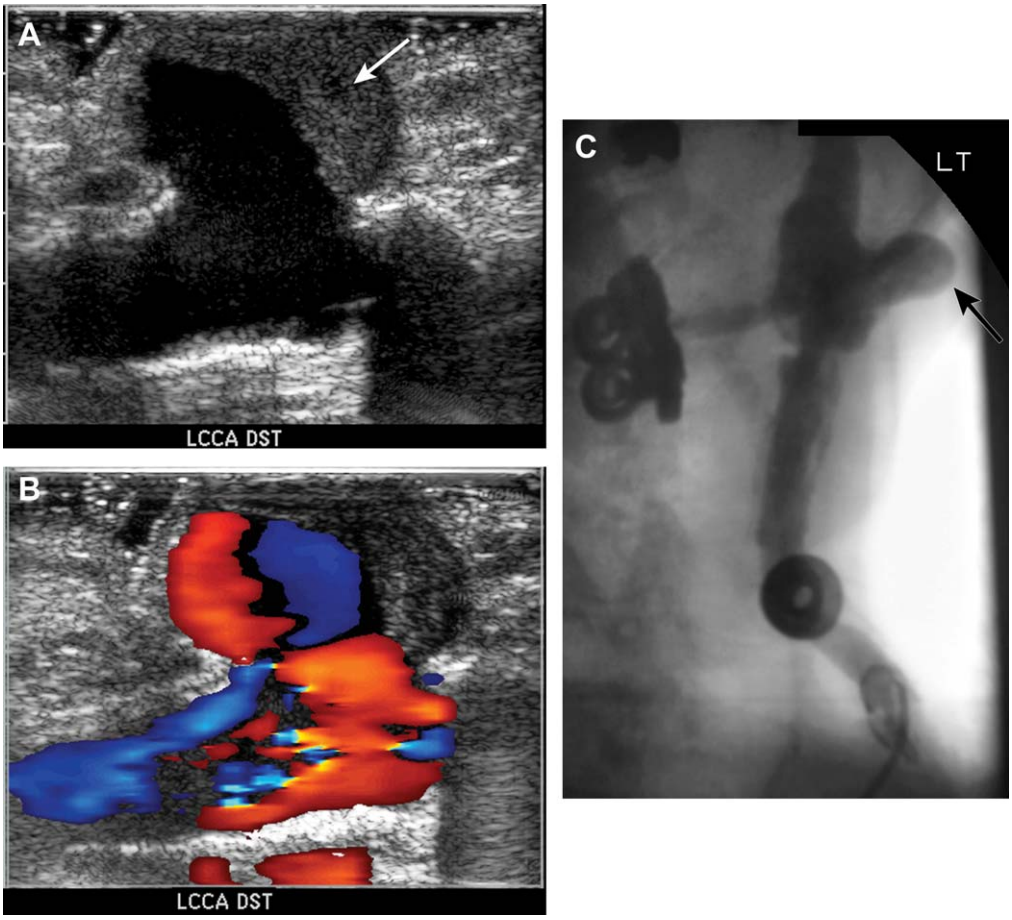
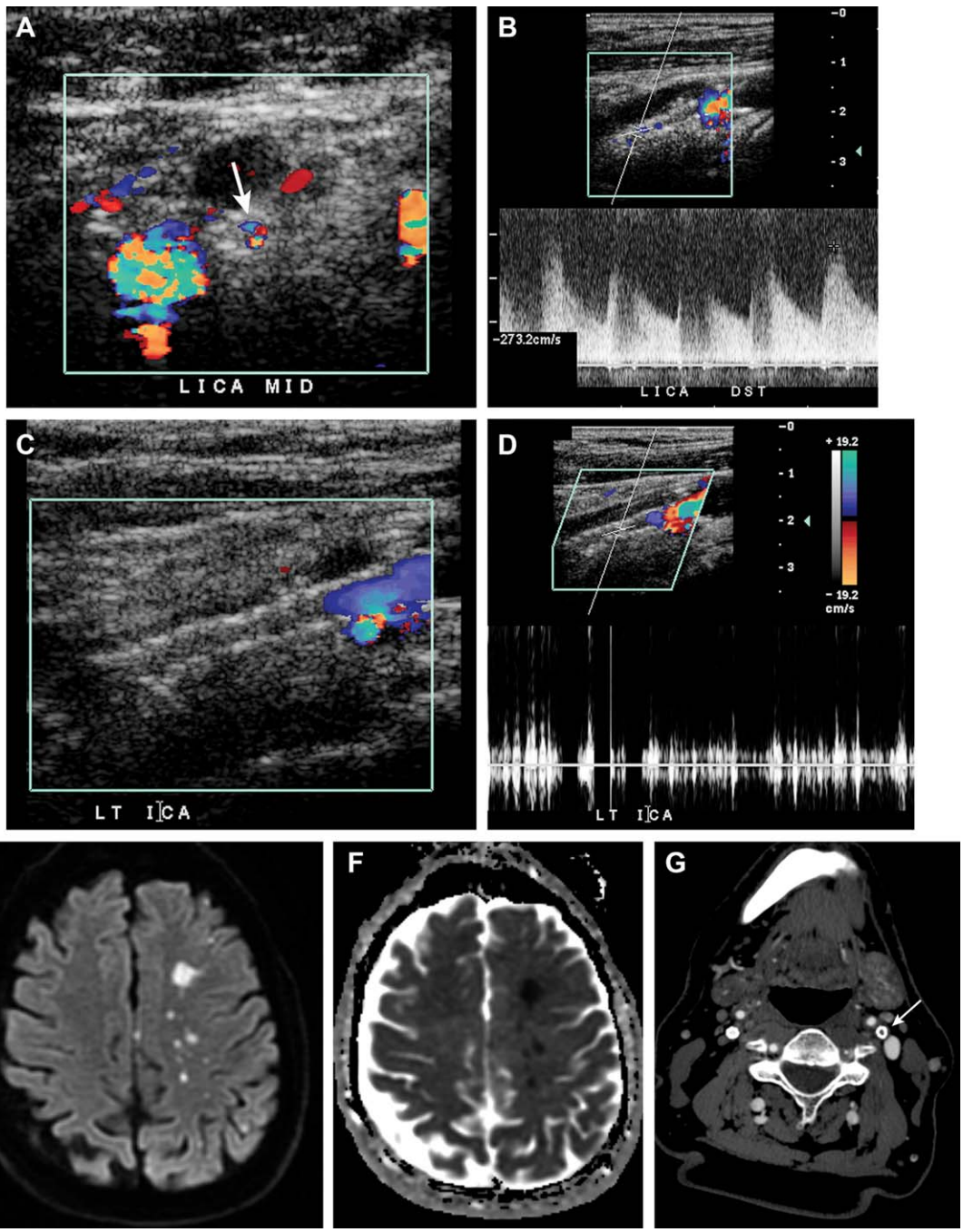


Figure 6. Images from a 60-year-old man who had a transient ischemic attack a few hours after an uneventful stenting of the left ICA. **A** and **B**, Transverse color and sagittal spectral Doppler sonograms of the left ICA show a string sign (arrow in **A**) and an increased velocity of 273 cm/s within the stent. He was subsequently discharged to home and received dual antiplatelet therapy. On postprocedure day 5, he presented to the emergency department with aphasia. **C** and **D**, Subsequent sagittal color and spectral Doppler sonograms of the left ICA show no detectable flow within the stent, which was concerning for complete thrombosis. **E**, Axial diffusion-weighted MRI of the brain shows multiple foci with an increased signal in the left cerebral hemisphere. **F**, These foci are hypointense on the corresponding apparent diffusion coefficient map, consistent with a thromboembolic stroke. **G**, Computed tomographic angiography of the neck performed a few months later for evaluation of the right side reshows the known left ICA stent occlusion (arrow).



"string sign," showing a thin stringlike line of residual blood flow on color Doppler imaging, may be seen adjacent to the area of the thrombus (Figure 6). High-resistance waveforms may be observed in the CCA proximal to the site of the thrombus. Flow velocities may be elevated on spectral Doppler sonography at the site of the thrombus. When the thrombus completely occludes the vessel or stent lumen, no blood flow will be detected. Downstream cerebral ischemia resulting from thromboembolism may be seen with other imaging modalities, such as diffusion-weighted brain MRI (Figure 7).

Restenosis

The incidence of restenosis after CEA and CAS has been reported to range from 5% to 20%, with variation in rates between studies influenced in part by the duration of follow-up and the exact definition of restenosis that is used.[47] Although some studies have reported significantly higher rates of restenosis with CAS than with CEA,[25,48] the Carotid Revascularization Endarterectomy Versus Stenting Trial found no significant difference in rates between the procedures, with restenosis rates after 2 years reported as 6.0% and 6.3% for CAS and CEA, respectively.[47] Patients who develop restenosis after CEA are also likely to develop in-stent restenosis after CAS.[49]

Risk factors for the development of restenosis after CEA or CAS include female sex, diabetes, and dyslipidemia.[47,50–52] Coronary artery disease and smoking have been identified as additional risk factors for restenosis after CEA,[47,53] whereas advanced age, the use of balloon-expandable stents, and implantation of multiple stents may contribute to in-stent restenosis after CAS.[54] Other risk factors include fibrous or inflammatory plaque, history of neck radiation therapy, history of restenosis after CEA, residual waist or narrowing immediately after treatment, and increased age. There is currently no clear consensus on how to manage restenosis after carotid revascularization, and follow-up imaging should therefore be handled on an individualized basis.[55–57] Generally, baseline sonography is performed, followed by sonography every 6 months for the first 2 years and yearly thereafter.[47] If sonography shows moderate stenosis, continued follow-up every 6 months is done.

On grayscale sonography, stenosis appears as a focal area of luminal narrowing within the carotid vessel or stent lumen, possibly with wall thickening and plaque. Blood flow velocities will be elevated at the point of stenosis on spectral Doppler imaging, and color Doppler imaging may show narrowing of the lumen and color aliasing (Figure 8). The peak systolic velocity (PSV) at the site of the stenosis is useful in determining the degree of stenosis.[58] There are no widely accepted Doppler criteria for grading restenosis after CEA and CAS. To compound matters further, no clear consensus exists as to what percentage or degree of recurrent stenosis after CEA or CAS should be treated.

Figure 7. Images from an 82-year-old woman with a surgical history of left CEA who developed left-sided weakness, left facial droop, and dysarthria 4 hours after right CEA. **A**, There is no demonstrable flow within the right distal CCA on sagittal power Doppler sonography, suggestive of complete thrombosis. **B**, Axial diffusion-weighted MRI of the brain shows an increased signal in portions of the right parietal and frontal lobes, consistent with an acute infarction in the territory of the right middle cerebral artery.

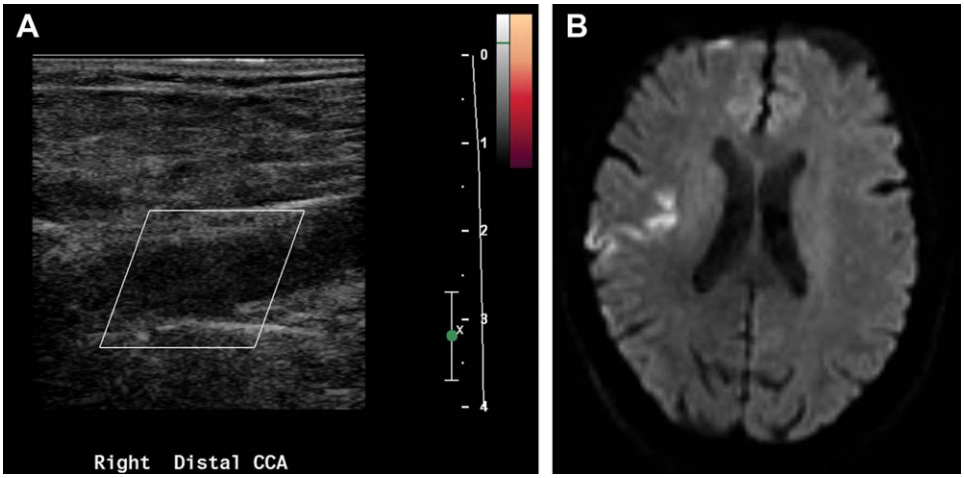


Figure 8. Images from a 65-year-old man after left CEA 9 years previously. **A** and **B**, Sagittal color and spectral Doppler sonograms show color aliasing with an increased flow velocity of 234 cm/s within the left proximal ICA, which was concerning for high-grade stenosis. **C**, Digital subtraction angiogram confirms a high-grade focal left proximal ICA stenosis (arrow). The patient subsequently underwent a technically successful CAS (not shown). **D** and **E**, Sagittal color and spectral Doppler sonograms 1 month later show a widely patent stent with a normal PSV of 106 cm/s. **F**, Angiogram 2 years later shows high-grade focal in-stent restenosis (arrow). The patient was treated with angioplasty (not shown).

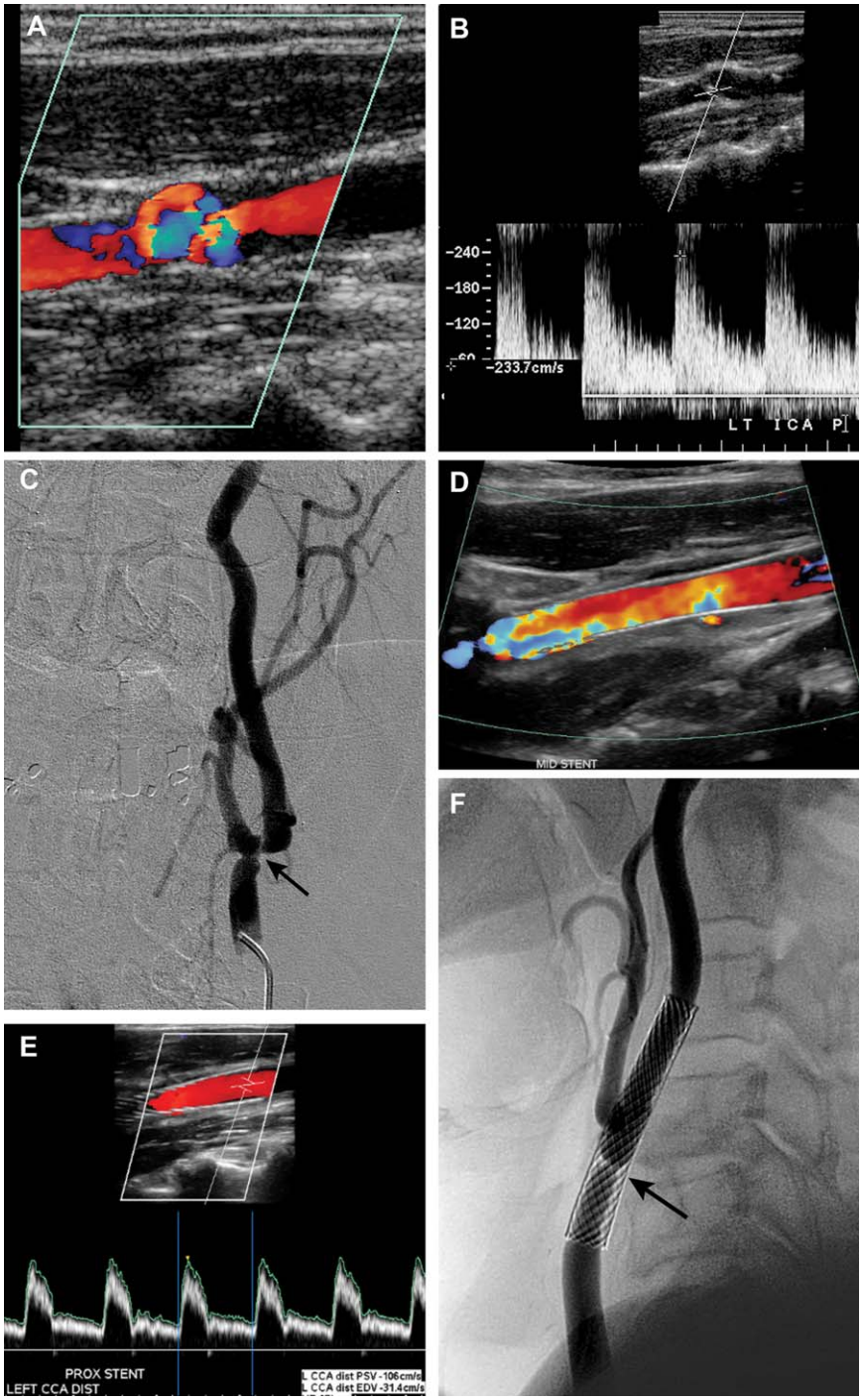
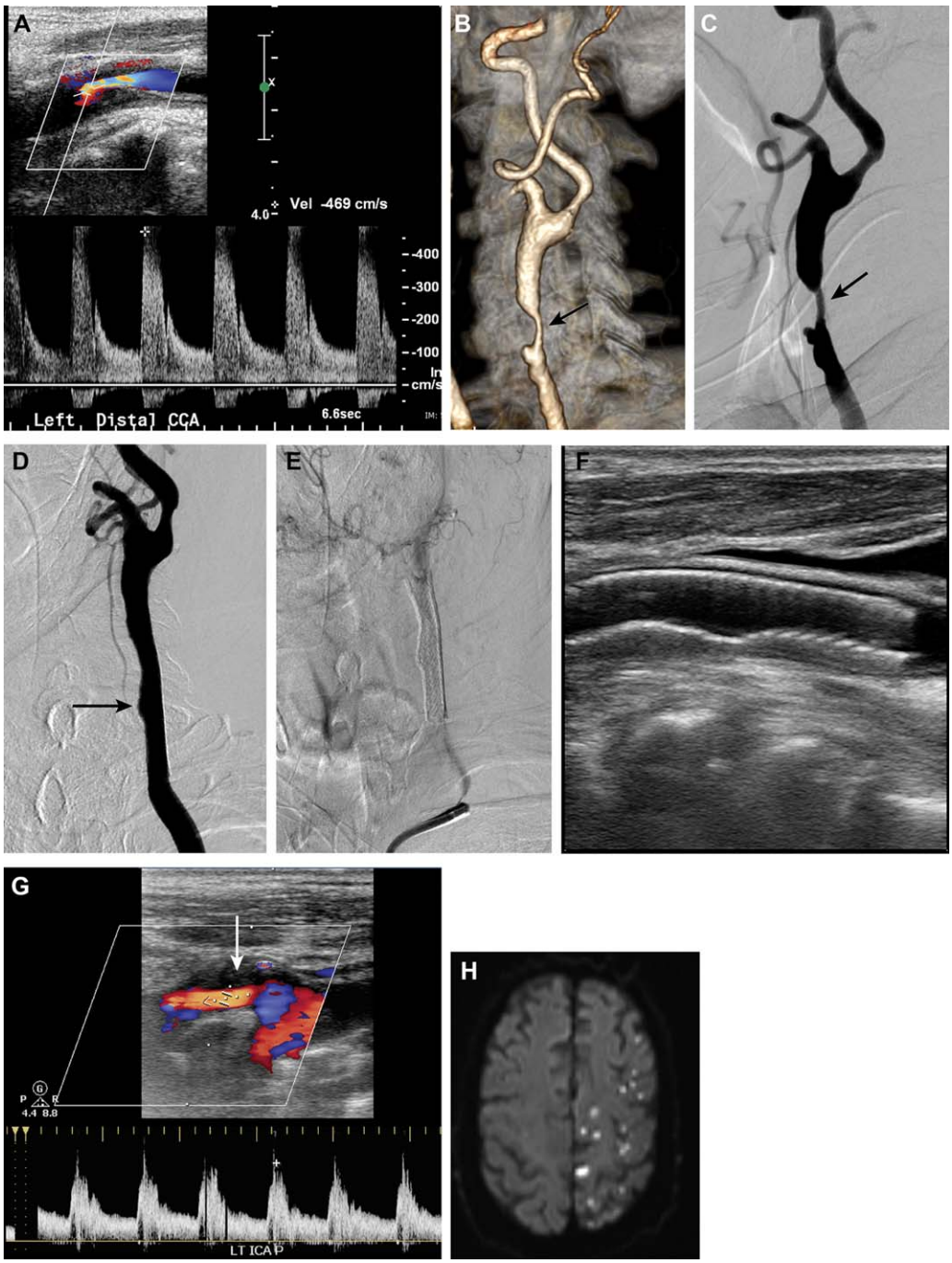


Figure 9. Images from a 73-year-old man with history of strokes who had also received radiation therapy for throat cancer. **A**, Sagittal spectral Doppler sonogram shows high-grade stenosis within the left distal CCA with a PSV of 469 cm/s. **B**, Severe CCA stenosis is confirmed on 3-dimensional reconstructed CT angiography (arrow). **C**, The stenosis is also shown on digital subtraction angiography (arrow). The patient underwent successful CAS with angioplasty. **D** and **E**, Poststent angiogram shows a focal bulge in the location of the prior stenosis (arrow), consistent with stent deformation without evidence of a fracture. A few days after the procedure, the patient had a transient ischemic attack. **F**, Grayscale sonogram shows the mildly deformed but intact stent struts. **G**, Sagittal color and spectral Doppler sonogram of the proximal left ICA shows an interval appearance of a hypoechoic area at the origin of the left ICA (arrow) with an elevated PSV of 191 cm/s (scale not shown), consistent with an acute thrombus. **H**, Diffusion-weighted MRI of the brain shows multiple hyperintense foci confined to the left cerebral hemisphere, consistent with multiple thromboemboli from a carotid source.



After CAS, when evaluating for in-stent restenosis, the rigid stent–arterial wall complex can cause higher overall PSVs.[51,59] Armstrong et al[60] used a stent PSV of greater than 300 cm/s, an end-diastolic velocity of greater than than 125 cm/s, and ratio of the in-stent PSV divided by the proximal CCA of greater than 4 to diagnose greater than 75% stenosis. Criteria used for greater than 50% stent stenosis was a PSV of greater than 150 cm/s and a PSV stent ratio of greater than 2. Other published criteria include stent PSVs of greater than 300 and greater than 450 cm/s for diagnosing 70% stenosis within the stent.[61,62] It is often helpful to obtain baseline measurements to later assess the change in the PSV over time. In case of doubt, one must have a lower threshold for obtaining a confirmatory CT angiogram, MR angiogram, or angiogram. After CEA, Doppler velocity criteria are likely to be lower, as placing an end-arterectomy patch in most cases increases the diameter of the carotid bulb, thereby lowering the PSV, and vessel wall compliance likely increases.

Stent Deformation and Fracture

Carotid stents are exposed to a wide range of forces that over time can result in stent deformation or fracture.[63] Deformation is defined as stent shape and caliber distortions without disruption of stent struts, whereas fracture results from strut failure and breakage.[21,64] In a case series that followed 116 carotid stents yearly for up to 9 years, the prevalence of deformation (23%) was greater than that of fracture (4%), and the incidence of both increased over time.[64] The risk of stent deformation and fracture may increase with local vessel calcification,[65] vessel tortuosity,[66] and a history of local radiation therapy.[63] The use of open-cell–design stents may help mitigate the fracture risk in patients with heavily calcified or tortuous vessels because of their increased flexibility.[66] Although stent fractures are usually treated conservatively, yearly imaging follow-up has been recommended because of the increased risk of restenosis.[63] Restenosis rates have been reported to range from 12.5% to 21% in patients with confirmed carotid stent fracture.[63,65]

On grayscale sonography, properly deployed, well-functioning stents appear as thin parallel echogenic lines adjacent to the intima of the vessel wall. Malapposition between the stent and the vessel wall with intervening flow on Doppler imaging is indicative of mechanical stent failure. Bends appear as wavy irregularities throughout the stent wall (Figure 9), whereas fractures may be seen as stent wall disruptions with or without protrusion of fragmented struts into the vessel lumen. These findings can be confirmed with plain radiography, digital subtraction angiography, or CT. In some cases, the intimal flap of a dissection may be confused with stent fracture, especially in the setting of a highly calcified dissecting flap.

Arteriovenous Fistula

An arteriovenous fistula has been described as an extremely rare complication after CEA.[67] In a case report, an arteriovenous fistula was noted to have developed between the external carotid artery to the internal jugular vein approximately 6 years after CEA.[67] It was treated by using coil embolization of the fistula and adjoining portion of the internal jugular vein.

Conclusions

Sonography is often the initial imaging modality used in the evaluation of many iatrogenic carotid arterial injuries, whether clinically suspected or found incidentally at routine follow-up imaging. Knowledge of the sonographic appearance of complications after carotid arterial interventions is essential to their early detection, may enable a reduction in patient morbidity and mortality, and may help determine the optimal management method.

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